As fewer pulmonary angiograms are required to solve diagnostic dilemmas, more are undertaken as the necessary prelude to mechanical intervention. Contemporary noninvasive diagnostic evaluation can be integrated by judicious use of the plasma D-dimer ELISA blood test, transthoracic or transesophageal echocardiography, and spiral chest computed tomography (CT) scanning with contrast, in addition to traditional ventilation-perfusion lung scanning. Therefore, the diagnosis of pulmonary embolism can usually be established or excluded without resorting to pulmonary angiography. In the new millennium, we have devised algorithms in which diagnostic pulmonary angiography is undertaken only after a series of noninvasive tests are deemed unhelpful (Fig. 31.1). Since lung scanning only occasionally provides a definitive “high probability” or “normal” result, chest CT scanning with contrast is increasingly utilized as the initial imaging test.

Techniques for fragmentation, rheolytic, and aspiration thrombectomy (Table 31.1) are improving, and these procedures are being utilized with increasing frequency among patients with right ventricular dysfunction due to pulmonary embolism. We also carry out pulmonary angiography as part of the preoperative evaluation for patients with chronic pulmonary embolism and pulmonary hypertension who are being considered for pulmonary thromboendarterectomy (Fig. 31.2).

FIG. 31.1.

Proposed diagnostic strategy that integrates lung and chest CT scanning, plasma D-dimer ELISA, echocardiography, leg ultrasonography, and pulmonary angiography.

Our concept of hemodynamic impairment has undergone an important evolution. We used to define hemodynamic instability rather simplistically as persistent systemic arterial hypotension requiring fluid resuscitation or pressors. We currently judge patients with pulmonary embolism to be hemodynamically unstable if they present with right ventricular hypokinesis, usually documented on echocardiogram, even in the presence of a normal systemic arterial pressure (1),(2). Such patients may initially appear deceptively stable based on the clinical evaluation alone. However, despite adequate heparin anticoagulation, patients with right ventricular hypokinesis are at high risk of recurrent pulmonary embolism and clinical deterioration, even if they are normotensive initially (3–5). Such patients, therefore, are prime candidates for more aggressive treatment with thrombolytic therapy or mechanical intervention.

FIG. 31.2.

Pulmonary angiogram of chronic pulmonary embolism. A 31-year-old man with progressive dyspnea and clinical evidence of pulmonary hypertension had right heart pressures as follows: right atrium, 12; right ventricle, = 98/18; pulmonary artery, 90/40; mean, 65; and pulmonary capillary wedge, 8 mm Hg. The contrast pulmonary angiogram shows occlusion of the descending left pulmonary artery and 80% stenosis of the anterior segmental pulmonary artery of the left upper lobe.

**DIAGNOSIS**

Maintaining a high degree of clinical suspicion for possible pulmonary embolism is of paramount importance. The onset of symptoms may be sudden, gradual, or intermittent. The most common symptoms and signs are nonspecific:
dyspnea, chest pain, tachypnea, and tachycardia. Usually, pulmonary embolism patients with severe chest pain or hemoptysis have anatomically small emboli near the periphery of the lung, where nerve innervation is greatest and where pulmonary infarction is most likely to occur due to poor collateral circulation. Ironically, patients with life-threatening pulmonary embolism often have a painless presentation characterized by dyspnea, syncope, or cyanosis.

Pulmonary embolism should be suspected in hypotensive patients when (a) there is evidence of, or there are predisposing factors for, venous thrombosis and (b) there is clinical evidence of acute cor pulmonale (acute right ventricular failure) such as distended neck veins, an S3 gallop, a right ventricular heave, tachycardia, or tachypnea, especially if (c) there is electrocardiographic evidence of acute cor pulmonale manifested by a new S1–Q3–T3 pattern, new incomplete right bundle branch block, or right ventricular ischemia. Under such circumstances, a bedside echocardiogram is especially helpful.

**Laboratory and Imaging Tests**

Chest x-ray abnormalities include focal oligemia (Westermark's sign), indicating massive central embolic occlusion, or a peripheral wedge-shaped density above the diaphragm (Hampton's hump), indicating pulmonary infarction. An enlarged right descending (6) pulmonary artery (Palla's sign) is also a useful clue. Furthermore, the chest radiograph can help identify patients with other diseases, such as lobar pneumonia or pneumothorax, that can mimic pulmonary embolism. However, patients with these illnesses can also have concomitant pulmonary embolism.

The electrocardiogram helps to exclude acute myocardial infarction and to identify electrocardiographic manifestations of right heart strain. The finding of T-wave inversion in leads V1 to V4 is surprisingly common in pulmonary embolism (7). The differential diagnosis of new right heart strain includes acute pulmonary embolism, acute asthma, or exacerbation of chronic bronchitis in patients with chronic obstructive pulmonary disease. Unfortunately, the time-honored screening test of abnormal room-air arterial blood gases is not helpful in triaging the population of patients suspected of pulmonary embolism (8). Although arterial blood gases are inexpensive and readily available, extensive analyses of the large Prospective Investigation of Pulmonary Embolism Diagnosis (PIOPED) database indicate that even sophisticated calculations of the alveolar/arterial oxygen difference do not accurately differentiate patients with pulmonary embolism from those without pulmonary embolism (9). Therefore, arterial blood gases should not be obtained as a screening test in patients suspected of pulmonary embolism. An abnormally elevated level of ELISA-determined plasma D-dimer (>500 ng/mL) has a more than 90% sensitivity for identifying patients with pulmonary embolism proven by lung scan (10) or by angiogram (11). This test relies on the principle that most patients with pulmonary embolism have ongoing endogenous fibrinolysis that is not effective enough to prevent pulmonary embolism, but that does break down some of the fibrin clot to D-dimers (Fig. 31.3). These D-dimers can be assayed by monoclonal antibodies that are commercially available.

**FIG. 31.3.**

Plasma D-dimer is generated exclusively from plasmin breakdown of fibrin clot. The D-dimers can be measured by commercially available ELISA kits. Plasma D-dimer ELISA is an excellent screening test for pulmonary embolism. Elevated levels are sensitive and normal levels have a high negative predictive value for pulmonary embolism at angiography.

Although elevated plasma concentrations of D-dimers are sensitive for the presence of pulmonary embolism, they are not specific. Levels will be elevated in patients for at least 1 week postoperatively and will also be abnormally high in patients with myocardial infarction, sepsis or almost any other systemic illness. Therefore, the plasma D-dimer ELISA is best used in patients without coexisting acute systemic illness. A normal plasma D-dimer ELISA has a greater than 90% probability of excluding PE.

*Ventilation-perfusion (V-Q) lung scanning* has traditionally served as the principal diagnostic imaging test when the clinical suspicion for pulmonary embolism is high. The V-Q scan is most useful if it is clearly normal or if it demonstrates a pattern indicating a high probability for pulmonary embolism. Intermediate-probability scans or low-probability scans with high clinical suspicion do not exclude pulmonary embolism (12). Patients in these latter categories may require chest CT scanning with contrast or pulmonary angiography, particularly if the plasma D-dimer level is elevated in the presence of normal leg ultrasonography and echocardiography.
Spiral chest CT scanning with contrast is beginning to replace lung scanning as the initial imaging test and is often viewed as a noninvasive alternative to conventional diagnostic pulmonary angiography. This technique is superb for the identification of proximal lesions. Its success rate for detection of clinically important distal pulmonary embolism is controversial (13).

In the future, gadolinium-enhanced magnetic resonance pulmonary angiography will be especially useful (14). This technique can combine detailed anatomy with cine loops of right ventricular wall motion.

Ultrasonography of the leg veins is usually accurate in diagnosing proximal leg deep venous thrombosis in symptomatic outpatients (15) and may serve as a useful surrogate for pulmonary embolism. However, about one-third of pulmonary embolism patients have no venographic evidence of leg deep venous thrombosis (16). Therefore, if clinical suspicion of pulmonary embolism is high, patients without clinical or imaging evidence of deep venous thrombosis should still be worked up for pulmonary embolism.

Echocardiography is most useful among hemodynamically unstable patients who appear to be too ill to be transported for lung scanning or pulmonary angiography. If transthoracic echocardiographic images are technically inadequate, then transesophageal echocardiography should be considered. Rarely, echocardiography demonstrates a thrombus in the main pulmonary artery or at its proximal bifurcation. More often, bedside echocardiography will suggest pulmonary embolism if a constellation of findings indicates right heart failure, especially with sparing of the right ventricular apex (McConnell’s sign) (Table 31.2). Echocardiography in this setting can also help exclude other life-threatening conditions, such as ventricular septal rupture, aortic dissection, and pericardial tamponade. Nevertheless, patients can have a normal echocardiogram despite anatomically extensive pulmonary embolism. Therefore, except for clinically unstable patients, echocardiography should be considered an ancillary rather than a principal diagnostic test.

Before undertaking diagnostic pulmonary angiography, it is of paramount importance to obtain accurate and high-quality recordings of right heart pressures and waveforms. Before injection of the contrast agent, a carefully performed right heart catheterization may provide important clues to alternative diagnoses not suspected by the referring physicians. For example, the cause of unexplained shortness of breath might be cardiac tamponade or left ventricular failure rather than pulmonary embolism. Patients with dyspnea and pulmonary hypertension might have intracardiac shunting, which can be defined most precisely by an oxygen saturation run. Therefore, information gleaned from catheterization may be more valuable than angiography and may occasionally make angiography unnecessary.

If the pressure tracing “dampens” or “wedges” in the proximal pulmonary artery without balloon expansion, anatomically massive pulmonary embolism should be suspected before injection of the contrast agent. Even when the angiographic diagnosis is, in fact, pulmonary embolism, a carefully performed right heart catheterization can provide clues about the age of the thrombus, based on the degree of elevation of the pulmonary artery systolic pressure. In general, if the pulmonary artery systolic pressure exceeds approximately 50 mm Hg, the differential diagnosis should include chronic pulmonary embolism or acute superimposed on chronic pulmonary embolism.

Among patients undergoing pulmonary angiography, an intraluminal filling defect seen in more than one projection is the most reliable feature to diagnose pulmonary embolism. Secondary signs of pulmonary embolism reflect decreased perfusion and consist of abrupt occlusion (“cutoff”) of vessels, oligemia or avascularity of a segment, a prolonged arterial phase with slow filling and emptying of veins, and tortuous tapering peripheral vessels. Standard-contrast pulmonary angiography can detect emboli accurately in peripheral vessels as small as 1 or 2 mm. Neither spiral CT nor magnetic resonance (MR) have achieved comparable imaging resolution of small vessels.

Pulmonary angiography may also help diagnose chronic pulmonary embolism (Fig. 31.2). Arteries may appear “pouched,” and thrombus appears organized, with a concave edge. Bandlike defects called webs may be present, in addition to intimal irregularities and abrupt narrowing or occlusion of lobar vessels (17).

Pulmonary angiography can almost always be accomplished safely if (a) selective angiography is performed, with the perfusion lung scan or chest CT serving as a road map to the angiographer; (b) soft, flexible catheters with side-holes
are employed, rather than stiff catheters with end-holes; and (c) a low-osmolar contrast agent is utilized to minimize the transient hypotension, heat, and coughing sensation that often occurs with conventional radiocontrast agents. In general, pulmonary angiography is exceedingly safe and uncomplicated. Nevertheless, if the diagnosis of pulmonary embolism (PE) is reliably established noninvasively, pulmonary angiography is not necessary, even if thrombolysis is planned.

Case Presentation: Diagnostic Dilemma in a 21-Year-Old Woman Who Is 8 Weeks Pregnant

A 21-year-old woman, 8 weeks pregnant and nulliparous, was hospitalized with suspicion of pulmonary embolism. She had complained of pleuritic chest and back discomfort for 1 week. A ventilation-perfusion lung scan was interpreted as demonstrating intermediate probability for pulmonary embolism. Leg ultrasonography and echocardiography were both normal. A plasma D-dimer ELISA was 2,003 ng/mL (normal less than 500 ng/mL).

Her physician estimated the overall likelihood of pulmonary embolism to be 50%. With an intermediate clinical suspicion and intermediate-probability lung scan, the PIOPED estimate of her likelihood of pulmonary embolism was 29%. However, the presence of a markedly elevated plasma D-dimer level in the setting of pregnancy increased the overall likelihood estimate to 50%. The dilemma was whether to treat her empirically as having a pulmonary embolism or to undertake pulmonary angiography, despite her being pregnant.

If she did not have pulmonary embolism, she would be exposed needlessly to the immediate risk of heparin-associated osteopenia. She would also suffer the unnecessary burden of continued hospitalization and prolonged outpatient treatment with anticoagulation. She would subsequently be prohibited from taking oral contraceptives during child-bearing years and hormone replacement therapy after menopause. In contrast, if pulmonary angiography were normal, she could be discharged as “healthy” within several hours of completing the procedure.

FIG. 31.4.

Pulmonary angiography with digital subtraction (left anterior oblique projection) demonstrates a large, acute embolus in the right lower lobar pulmonary artery (arrowhead).

The fetal exposure to radiation during pulmonary angiography is well below the recommended maximum for pregnancy. After considerable discussion, the patient and her physician agreed to proceed with cardiac catheterization and pulmonary angiography. Appropriate lead-shielding of the abdomen was employed, and fluoroscopy time was kept to a minimum. Right heart pressures were entirely normal (RA = 5 mm Hg, PA = 25/9 mm Hg), but angiography demonstrated a large right lower lobar pulmonary embolism (Fig. 31.4). Therefore, she was maintained on therapeutic levels of heparin for her entire pregnancy and was anticoagulated postpartum with warfarin.

PATHOPHYSIOLOGY: IMPORTANCE OF RIGHT VENTRICULAR FUNCTION

The hemodynamic response to pulmonary embolism depends on the size of the embolus, coexistent cardiopulmonary disease, and neurohumoral activation. Pulmonary artery obstruction and circulating neurohumoral substances reduce the pulmonary vascular bed and cause an increase in right ventricular afterload. As right ventricular and pulmonary artery pressures rise, the right ventricle dilates, becomes hypokinetic, and ultimately fails. Progressive right heart failure leads to reduced forward cardiac output and is usually the cause of death from acute pulmonary embolism.

FIG. 31.5.

Parasternal short-axis views of the right ventricle (RV) and left ventricle (LV) in diastole (left) and systole (right). There is diastolic and systolic bowing of the interventricular septum (arrows) into the left ventricle compatible with
right ventricular volume and pressure overloads, respectively. The right ventricle is appreciably dilated and markedly hypokinetic, with little change in apparent right ventricular area from diastole to systole. PE, small pericardial effusion. (Reprinted with permission from Come PC. Echocardiographic evaluation of pulmonary embolism and its response to therapeutic interventions. Chest 1992;101:151S.)

Sudden increases in right ventricular pressure adversely affect left ventricular function because of the anatomic juxtaposition of the two ventricles and “ventricular interdependence.” Moderate right ventricular hypertension can displace the interventricular septum toward the left ventricle, resulting in decreased left ventricular diastolic filling and end-diastolic volume (Fig. 31.5). The subsequent reduction in coronary artery perfusion pressure to the overloaded right ventricle may cause progressive right ventricular ischemia and failure. Ultimately, right ventricular infarction, circulatory arrest, and death may ensue.

Thrombolysis, mechanical interventions (e.g., suction embolectomy, pulverization, or fragmentation and distal embolization of clot), or open surgical embolectomy can relieve obstruction to major pulmonary artery blood flow, thereby rapidly lowering the abnormally elevated pulmonary artery pressure. As a result, right ventricular function usually improves quickly. Because of ventricular interdependence, improved right ventricular function leads to better left ventricular function, which helps reverse cardiogenic shock. The use of thrombolysis or mechanical intervention in patients at high risk for adverse outcomes with anticoagulation alone might reduce the mortality rate from pulmonary embolism by quickly restoring normotensive pulmonary artery and right ventricular pressures and by normalizing right ventricular wall motion.

THROMBOLYSIS, MECHANICAL INTERVENTION, AND SURGICAL EMBOLECTOMY

In the past, a normal blood pressure and heart rate too often engendered a sense of complacency among physicians caring for pulmonary embolism patients. Typically, clinically undetected right heart failure worsened, caused pressor dependence, and led to unremitting cardiogenic shock. As rapid overt deterioration ensued, desperate clinicians considered employing thrombolysis or surgical embolectomy as a last resort, often with poor results. After high-risk PE patients are identified with moderate or severe right ventricular dysfunction, adequate anticoagulation with heparin should be followed by screening to determine suitability for pharmacologic thrombolysis or other aggressive treatment modalities.

FIG. 31.6.

A: A large embolus is present in the right pulmonary artery (arrow). B: After a 2-hour infusion of rt-PA through a peripheral vein, there is pronounced resolution, with only a small amount of residual thrombus in segmental branches. (Reprinted with permission from Goldhaber SZ et al. Acute pulmonary embolism treated with tissue plasminogen activator. Lancet 1986;2:886.)

Thrombolysis (Fig. 31.6) (20), mechanical catheter interventions (21), and open surgical embolectomy debulk clot and provide primary treatment of pulmonary embolism, whereas intensive anticoagulation is critical for prevention of recurrent pulmonary embolism. These aggressive approaches to pulmonary embolism management are almost always successful if undertaken before the onset of cardiogenic shock. Echocardiography showing moderate or severe right ventricular dysfunction should serve as a trigger for consideration of aggressive intervention. However, patients with normal right ventricular function on echocardiography can be treated conservatively and have an excellent prognosis when managed with anticoagulation alone (3).

Over the past decade, the administration of thrombolysis to pulmonary embolism patients has been streamlined so that it is safer, less expensive, and less time-consuming than previously. There is a wide 14-day “window” for effective use of thrombolysis (22). Increasing age, catheterization for pulmonary angiography, and obesity are risk factors for major hemorrhage after thrombolysis (23). At least half of high-risk PE patients will be relatively unsuitable candidates for thrombolysis.
An inferior vena caval (IVC) filter does not treat an established pulmonary embolism directly, nor does it halt the thrombotic process. Accepted indications for filter insertion to prevent pulmonary embolism include (a) established venous thrombosis with active, clinically important bleeding that prohibits the use of heparin, or (b) recurrent pulmonary embolism despite adequate anticoagulation. An IVC filter may also be used adjutively to prevent recurrent pulmonary embolism among hemodynamically compromised patients in whom pulmonary embolism cannot be treated with thrombolytic therapy. Whenever possible, anticoagulation should be utilized in combination with a filter to prevent further thrombosis (24).

There has been a resurgence of interest in catheter-based embolectomy, including fragmentation (25), rheolytic (26), and aspiration (27) thrombectomy (Table 31.1). Other interventional techniques are under development (28–30). At times, thrombolysis and mechanical intervention can be combined (31). If catheter-based strategies fail, emergent surgical embolectomy with cardiopulmonary bypass can be undertaken (32,33).

Case Presentation: Combined Approach of Suction Catheter Embolectomy and Thrombolysis in a 78-Year-Old Woman with Massive Pulmonary Embolism and Hemodynamic Instability

A 78-year-old woman presented with marked shortness of breath, persistent hypotension (systemic arterial pressure 78/51 mm Hg), and right ventricular dilatation and hypokinesis on echocardiogram. Pulmonary angiogram showed a massive right pulmonary artery embolism, as well as a small left lung volume because of a prior thoracoplasty to treat tuberculosis (Fig. 31.7A). She received heparin and placement of a Greenfield filter. Hypoxemia persisted despite ventilatory support. She developed melena on heparin. Cardiac surgeons felt she would not survive surgical embolectomy because of the prior left lung thoracoplasty.

Because of her hemodynamic compromise, with melena on heparin and surgical inoperability, aspiration thrombectomy was undertaken in the catheterization laboratory by Michael F. Meyerovitz using the Meyerovitz technique. The right common femoral vein was accessed with a single wall puncture needle. A guidewire was advanced across the Greenfield filter. A 7F pigtail catheter was used with a tip-deflecting guidewire to enter the pulmonary artery. The catheter was exchanged for a 9F multipurpose coronary guiding catheter. Pressures were 18 mm Hg (mean) in the right atrium, 90/18 mm Hg in the right ventricle, and 90/40 mm Hg in the pulmonary artery. Suction catheter embolectomy removed both fresh and old clot from the pulmonary artery branches of the upper and lower right lobar arteries.

Systemic arterial hypotension persisted and therefore 50 mg of rt-PA was administered over 15 minutes through the pulmonary artery catheter. Pulmonary angiography then showed an approximately 30% reduction in the overall clot burden (Fig. 31.7B).

FIG. 31.7.

A: Massive right main pulmonary artery embolism in the presence of markedly diminished left lung volume due to prior thoracoplasty. Cardiopulmonary diseases and cardiac tumors. Philadelphia: Current Medicine, 1995:3.1.) B: Digital subtraction pulmonary angiography immediately following combined suction catheter embolectomy and thrombolysis. There is an approximately 30% reduction in overall clot burden compared with the baseline angiogram (A). (Reprinted with permission from Goldhaber SZ. Treatment of acute pulmonary embolism. In: Goldhaber SZ, ed. Cardiopulmonary diseases and cardiac tumors. Philadelphia: Current Medicine, 1995:3.1.)

The procedure was complicated by a retroperitoneal bleed that was corrected with 12 units (U) of packed red blood cells. She also developed pneumonia and acute respiratory distress syndrome. Nonetheless, her clinical picture gradually improved. She was successfully weaned from the ventilator and was transferred to a rehabilitation facility. Two years later, she wrote to me and stated, “I am able to get around with a walker and portable cannister of oxygen. I celebrated my 80th birthday last May, so I guess I’m a tough old bird.”

Case Presentation: Failed Aspiration Thrombectomy Followed by Open Surgical Embolectomy

A 65-year-old dentist underwent right frontal craniotomy for resection of a malignant astrocytoma. He received venous thromboembolism prophylaxis with heparin 5,000 U subcutaneously twice daily and intermittent pneumatic...
Massive right main pulmonary artery embolism. Massive left main pulmonary artery embolism. Several centimeters of thrombus removed in the Interventional Laboratory. Placement below the renal veins of a bird's nest filter. Large amount of thrombus surgically extracted from the right and left pulmonary arteries, respectively.

Left pulmonary arteriogram of a 53-year-old man with chronic pulmonary embolism causing total occlusion of left lower lobar pulmonary arteries. This patient underwent pulmonary thromboendarterectomy at Brigham and Women's Hospital, where large and extensive thrombi were surgically removed. The specimen contains laminated thrombus that is adherent to the endothelial wall of the endarterectomy.

Nevertheless, on postoperative day 11, he developed pulmonary embolism with a systolic blood pressure of 100 mm Hg and severe right ventricular dysfunction on echocardiogram. A right (Fig. 31.8A) and left (Fig. 31.8B) pulmonary angiogram was done as a prelude to catheter aspiration embolectomy, which yielded only a small amount of thrombus (Fig. 31.8C) and did not improve his clinical condition. A Bird's nest filter (Cook, Bloomington, IN) was then placed (Fig. 31.8D) and he was taken to the operating room, where a large volume of thrombus was removed from the right (Fig. 31.8E) and left pulmonary artery (Fig. 31.8F). He subsequently recuperated uneventfully and is clinically stable more than 2 years postoperatively. In his case, cardiac surgical backup during interventional angiography was crucial to ensure a successful outcome.

**FIG. 31.8.**

A: Massive right main pulmonary artery embolism. B: Massive left main pulmonary artery embolism. C: Several centimeters of thrombus removed in the Interventional Laboratory. D: Placement below the renal veins of a bird's nest filter. E,F: Large amount of thrombus surgically extracted from the right and left pulmonary arteries, respectively.

**CHRONIC PULMONARY EMBOLISM**

Patients with chronic pulmonary hypertension due to prior pulmonary embolism may be virtually bedridden with breathlessness due to high pulmonary arterial pressures. They should be considered for pulmonary thromboendarterectomy, which, if successful, can reduce and at times even cure pulmonary hypertension (34).

The operation involves a median sternotomy, institution of cardiopulmonary bypass, and deep hypothermia with circulatory arrest periods. Incisions are made in both pulmonary arteries. The surgeon who performs thromboendarterectomy creates an endarterectomy plane and then dissects endothelialized thrombus from as many involved pulmonary vessels as possible.

At the University of California at San Diego Medical Center, the operative mortality rate is approximately 6%. The two major causes of postoperative mortality are (a) inability to remove sufficient thrombotic material at the time of operation, resulting in persistent postoperative pulmonary hypertension and right ventricular dysfunction, and (b) severe reperfusion lung injury (35). Thus, at selected centers, pulmonary thromboendarterectomy can be performed with good results and at an acceptable risk among patients debilitated from chronic pulmonary hypertension due to pulmonary embolism.

**Case Presentation: Pulmonary Thromboendarterectomy for Treatment of Chronic Pulmonary Embolism in a 53-Year-Old Man with Pulmonary Hypertension and Right Ventricular Dysfunction**

A 53-year-old man presented with gradually worsening dyspnea on exertion. He complained of fatigue and inability to work and pursue leisure activities without marked shortness of breath. Echocardiography showed a severely enlarged and somewhat hypertrophied right ventricle with moderately reduced systolic function. The left ventricle was relatively small with marked septal flattening and abnormal septal motion but preserved systolic function.

**FIG. 31.9.**

A: Left pulmonary arteriogram of a 53-year-old man with chronic pulmonary embolism causing total occlusion of left lower lobar pulmonary arteries. B: This patient underwent pulmonary thromboendarterectomy at Brigham and Women's Hospital, where large and extensive thrombi were surgically removed. The specimen contains laminated thrombus that is adherent to the endothelial wall of the endarterectomy.

At age 25, he had suffered bilateral deep venous thrombosis of the legs but did not receive a prolonged course of anticoagulation due to a duodenal ulcer 3 years previously. At age 36, he presented with syncope accompanied by tachycardia and diaphoresis. His electrocardiogram was notable for atrial fibrillation and inverted T waves in leads
V1 through V3. Five years later, he complained of exertional dyspnea. A lung scan showed perfusion defects that were of high probability for pulmonary embolism. At that time, his mean pulmonary artery pressure was 32 mm Hg, and a pulmonary angiogram was reportedly positive for pulmonary embolism. He was placed on warfarin.

Despite 12 years of anticoagulation, his dyspnea worsened to the point where he could not pursue the active lifestyle that he desired. Chronic pulmonary embolism was suspected, and he was referred for possible pulmonary thromboendarterectomy. Therefore, right heart catheterization and pulmonary angiography were repeated.

Catheterization demonstrated a right atrial pressure of 10 mm Hg, right ventricular pressure of 55/10 mm Hg, and pulmonary artery pressure of 55/28 mm Hg, with a mean pulmonary artery pressure of 35 mm Hg. Pulmonary angiography (Fig. 31.9A) revealed total occlusion of his left lower lobe pulmonary arteries. He underwent pulmonary thromboendarterectomy at Brigham and Women's Hospital. The surgeon endarterectomized multiple large thrombi that were chronic and laminated (Fig. 31.9B). The patient has subsequently done well and is no longer incapacitated in any way. He runs a factory and hunts and fishes in his leisure time.