Echocardiography in the Management of Pulmonary Embolism

Samuel Z. Goldhaber, MD

Echocardiography is not recommended as a routine imaging test to diagnose suspected pulmonary embolism. However, it is useful for identifying patients with pulmonary embolism who may have a poor prognosis. It can be used for rapid and accurate risk assessment. Moderate or severe right ventricular hypokinesis, persistent pulmonary hypertension, a patent foramen ovale, and free-floating right-heart thrombus are echocardiographic markers that identify patients at risk for death or recurrent thromboembolism. Such patients warrant consideration for thrombolysis or embolectomy. Serial imaging of right ventricular function can help physicians monitor the effect of treatment and judge whether the selected management strategy is successful. Further research will clarify and define more precisely the utility and limitations of echocardiography in the management of pulmonary embolism.

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For author affiliation and current address, see end of text.

Patients with acute pulmonary embolism are undergoing echocardiography with increasing frequency (1). Because the use of echocardiography in these patients is burgeoning, I provide my perspective on the utility of this test in bedside management.

I do not recommend echocardiography as a routine imaging test to diagnose suspected pulmonary embolism because most patients with pulmonary embolism have normal echocardiograms. Nevertheless, among patients who undergo echocardiography for suspected left-heart failure, the imaging of a normal left ventricle and dilated, hypokinetic right ventricle may serendipitously suggest the diagnosis of pulmonary embolism. In addition, echocardiography is useful for diagnosis in hemodynamically unstable patients with unexplained dyspnea, syncope, or right-heart failure. Transesophageal echocardiography is best reserved for critically ill patients in whom the cause of cardiovascular instability remains uncertain.

Qualitative Abnormalities on Transthoracic Echocardiography

Among patients with large pulmonary embolism, abnormalities are often apparent on the transthoracic echocardiogram (**Table**). These include right ventricular dilatation and hypokinesis, abnormal motion of the interventricular septum, tricuspid regurgitation, and lack of collapse of the inferior vena cava during inspiration (2). These abnormalities on echocardiography have clinical correlates, such as jugular venous distention, tricuspid regurgitation, and an accentuated pulmonic heart sound. Electrocardiographic features of right ventricular strain include a new right bundle-branch block, a finding of an S wave in lead I, a Q wave in lead III or an inverted T wave in lead III, and new T-wave inversion in leads V1 through V4 (3).

Among patients with hemodynamic impairment, there is a small difference in left ventricular area during diastole and during systole; this difference indicates low cardiac output (**Figure 1**) (4). Typically, this represents cardiogenic shock due to right ventricular failure (the usual cause of death from pulmonary embolism) despite the presence of an intrinsically normal left ventricle. Rarely, pulmonary embolism may be visualized directly on transthoracic echocardiography, but only if it is large and located in the central vasculature (**Figure 2**).

Pulmonary Hypertension

An abrupt increase in pulmonary artery pressure causes the right ventricular dysfunction observed in some patients with acute pulmonary embolism. The hemodynamic response to pulmonary embolism depends on the size of the embolus, coexistent cardiopulmonary disease, and neurohumoral effects. Pulmonary artery obstruction and circulating neurohumoral substances increase right ventricular afterload. As right ventricular and pulmonary artery pressures increase, the right ventricle dilates, becomes hypokinetic, and ultimately fails. Acute increases in right ventricular pressure can cause left ventricular dysfunction because of the anatomic juxtaposition of the two ventricles and "ventricular interdependency." With underfilling of the left ventricle, systemic cardiac output and pressure both decrease,

Table. **Abnormal Echocardiographic Findings in Patients with Pulmonary Embolism**

Abnormal Finding	Description
Right ventricular dilatation and hypokinesis	Associated with leftward septal shift; the ratio of the right ventricular end-diastolic area to left ventricular end-diastolic area exceeds the upper limit of normal (0.6). Associated with right atrial enlargement and tricuspid regurgitation.
Septal flattening and paradoxical septal motion	Right ventricular contraction continues even after the left ventricle starts relaxing at end-systole; therefore, the interventricular septum bulges toward the left ventricle.
Diastolic left ventricular impairment with a small difference between left ventricular area during diastole and systole, indicating low cardiac output	Due to septal displacement and reduced left ventricular distensibility during diastole; consequently, Doppler mitral flow exhibits a prominent A wave, much higher than the E wave, with an increased contribution of atrial contraction to left ventricular filling.
Direct visualization of pulmonary embolism	Only if pulmonary embolism is large and centrally located; much more easily visualized on transesophageal than transthoracic echocardiography.
Pulmonary arterial hypertension detected by Doppler flow velocity in the right ventricular outflow tract	Shortened acceleration time, with peak velocity occurring close to the onset of ejection. Biphasic ejection curve, with midsystolic reduction in velocity.
Right ventricular hypertrophy	With mildly increased right ventricular thickness (often about 6 mm, with 4 mm as upper limit of normal); clear visualization of right ventricular muscle trabeculations.
Patent foramen ovale	When right atrial pressure exceeds left atrial pressure, the foramen ovale may open and cause worsening hypoxemia or stroke.

potentially compromising coronary perfusion and producing myocardial ischemia.

The pulmonary artery systolic pressure can be estimated by measuring the peak velocity of the tricuspid regurgitant jet obtained with Doppler echocardiography. The gradient across the tricuspid valve can be estimated by using the modified Bernoulli equation, $P = 4V^2$; P represents the peak pressure difference between the right atrium and right ventricle, and V is the peak velocity of the regurgitant jet (5). Estimated right atrial pressure is added to the gradient to estimate pulmonary artery systolic pressure. Note, however, that acute and massive pulmonary embolism can produce right ventricular failure without causing substantial elevations in pulmonary artery pressure (6).

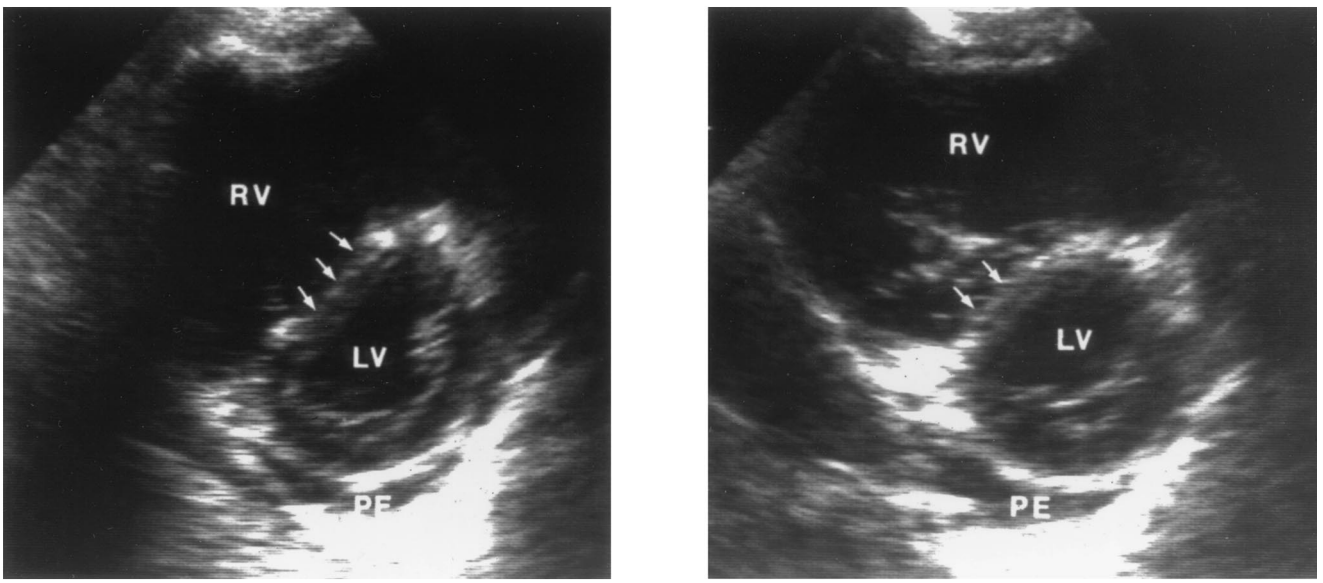
This estimated pressure usually correlates well with invasive measurements obtained at pulmonary artery catheterization, although it is subject to error in estimating right atrial pressure. In addition, considerable technical skill is required to ensure that the complete tricuspid regurgitant envelope is visualized. If necessary, agitated saline contrast can be used to enhance the tricuspid regurgitant Doppler tracing. In the absence of tricuspid regurgitation, other techniques can be considered for estimating pulmonary artery pressure. For example, the time to peak velocity in the right ventricular outflow tract is reduced with elevated pulmonary artery pressures. However, this measurement is not nearly as precise as the modified Bernoulli equation (5).

Relationship between Right Ventricular Dysfunction and Size of Pulmonary Embolism

At what point does a pulmonary embolus become large enough to cause right ventricular dysfunction visible on echocardiography? This depends on the patient population. In a clinical trial of patients with moderately large pulmonary emboli but without hemodynamic instability at baseline (7), lung scans and echocardiograms were obtained on the day of enrollment. Whereas echocardiograms were assessed qualitatively as either showing right ventricular dysfunction (defined as impaired contraction of the right ventricular systolic wall or increased right ventricular end-diastolic area detected by observation alone, without planimetry) or not, the lung scans were graded according to a precise, quantitative segmental perfusion score (8). Each lung segment was graded in terms of perfusion reduction (0 = normal and 3 = absent) and size (0 = no defect and 3 = whole segment). The defect score for each segment was defined as the volume of the abnormal perfusion times the perfusion reduction in the abnormality. The defect score for each patient was the average of 18 segmental scores, 10 from the right lung and 8 from the left lung.

A receiver-operating characteristic curve was then generated by plotting the quantitative segmental perfusion score in relation to the presence of right ventricular

Figure 1. **Typical findings on transthoracic echocardiography in patients with massive pulmonary embolism.**



In patients with pulmonary embolism, the transthoracic echocardiogram rarely shows thrombus; instead, findings suggestive of pulmonary embolism may be observed. Shown here are parasternal short-axis views of the right ventricle (*RV*) and left ventricle (*LV*) in diastole (*left*) and systole (*right*) in a patient with angiographically proved pulmonary embolism. There is diastolic and systolic bowing of the interventricular septum (*arrows*) into the left ventricle, a finding compatible with the presence of right ventricular volume overload and pressure overload, respectively. The left ventricle has assumed a classic D-shaped configuration, indicating impaired left ventricular relaxation. The right ventricle is appreciably dilated and markedly hypokinetic, with little change in the apparent right ventricular area from diastole to systole. There is a small pericardial effusion (*PE*). Reprinted with permission from Come (4).

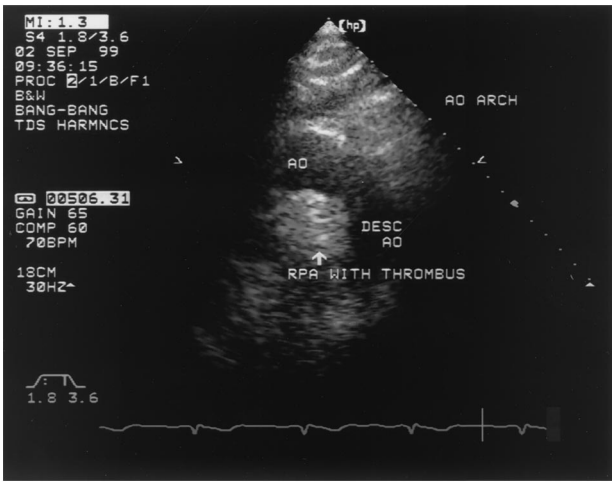
hypokinesis (9). Patients in whom less than 30% of the pulmonary vascular cross-sectional area was impaired by pulmonary embolism on lung scanning were 6.8 times more likely to have normal right ventricular function on echocardiography. In contrast, among patients with 30% or more of the lung not perfused, 92% of those with right ventricular hypokinesis were identified. Similar results were obtained in another cohort of patients with pulmonary embolism who underwent both baseline lung scanning and echocardiography (10). On the basis of these findings, I suspect right ventricular dysfunction and obtain an echocardiogram in patients with pulmonary embolism who have approximately one third or more of the lung nonperfused on lung scanning.

Regional Right Ventricular Dysfunction

In reviewing serial echocardiograms in patients with known, large pulmonary emboli, my colleagues and I detected a qualitative finding that typified these patients (11). Despite moderate or severe right ventricular free-wall hypokinesis, we noted relatively normal contraction and "sparing" of the right ventricular apex. We quantified this abnormality (11), which has become widely known as the McConnell sign. First, we designed a computer program for quantitative analysis of right ventricular free-wall motion. Then, we traced the right ventricular free-wall endocardium in the apical four-chamber view from base to apex at end-systole and end-diastole for three separate cardiac cycles (**Figure 3**). For pulmonary embolism, the McConnell sign had a sensitivity of 77%, specificity of 94%, positive predictive value of 71%, and negative predictive value of 96%. The McConnell sign appears to be a useful screening test to help distinguish between right ventricular dysfunction due to pulmonary embolism and dysfunction due to other conditions, such as primary pulmonary hypertension.

For patients with right ventricular hypokinesis due to acute pulmonary embolism, the excursion diminished markedly when measured in the middle of the right

Figure 2. **Right main pulmonary artery embolism visualized on transthoracic echocardiography.**



This aortic arch view shows the relationship between the aorta and the right main pulmonary artery. Thrombus is seen in the right pulmonary artery (*RPA*). AO = aorta; AO arch = aortic arch; DESC AO = descending aorta; hp = Hewlett Packard. Courtesy of José Rivero, with permission.

ventricular free wall. However, the excursion improved progressively when segments closer to the apex were measured. This pattern of regional right ventricular dysfunction appeared highly specific for acute pulmonary embolism; in patients with right ventricular dysfunction due to primary pulmonary hypertension, right ventricular hypokinesis was not improved upon assessment of apical segments (11) (**Figure 4**). However, no completely satisfying explanation accounts for this observation. Potential mechanisms include tethering of the right ventricular apex to a hyperdynamic left ventricle, lower wall stress in the right ventricular apex, and localized ischemia of the right ventricular free wall (11).

TRANSESOPHAGEAL ECHOCARDIOGRAPHY

Transesophageal echocardiography holds the promise of diagnosing pulmonary embolism by direct visualization of a thrombus rather than by relying on indirect signs, such as right ventricular enlargement and hypokinesis. The examination assesses the extent of thromboembolism as well as its surgical accessibility.

This technique requires far more resources and operator skill than does transthoracic echocardiography. Technically, transesophageal echocardiography is easiest to perform in the unconscious patient. Conscious patients almost always require topical anesthesia of the pharynx and often benefit from small, sedating doses of midazolam or fentanyl. In general, the main pulmonary artery and then the right pulmonary artery are first visualized. The right pulmonary artery can be followed until it branches to the right lobar pulmonary arteries. The transducer is then rotated to examine the left pulmonary artery. However, interposition of the left main bronchus interferes with the ultrasound beam in the middle portion of the left pulmonary artery (12, 13). Therefore, thromboembolism is more difficult to detect in the left pulmonary artery.

Transesophageal echocardiography may have a uniquely valuable role in patients who present with unexplained sudden cardiac arrest and pulseless electrical activity (14). Two echocardiographers performed transesophageal echocardiography in 25 patients who presented with pulseless electrical activity. Eleven patients did not have isolated right ventricular enlargement. In 14, however, the ultrasonographers found right ventricular enlargement without left ventricular enlargement. Of these 14, 5 had no pulmonary embolism. The explanations for isolated right ventricular enlargement were cardiac contusion; right ventricular infarction; cor pulmonale; and, in 2 of the 5 patients, ventricular hypertrophy. The remaining 9 patients had pulmonary embolism. One case of pulmonary embolism was missed during transesophageal echocardiography but was subsequently diagnosed at autopsy. For the remaining 8 patients, pulmonary embolism was diagnosed during cardiac arrest (**Figure 5**). Two survived to hospital discharge: One received thrombolysis, and the other underwent emergent surgical embolectomy. This approach to cardiac arrest with pulseless electrical activity is preliminary, and more research is needed to determine the role of echocardiography in this situation.

Although this approach may appear to have limited utility, 5% of 1246 patients in Vienna, Austria, who presented with cardiac arrest had pulmonary embolism (15). Of those with pulmonary embolism, 63% presented with pulseless electrical activity. Therefore, occult pulmonary embolism should be considered a possible cause of cardiac arrest in patients with pulseless electrical activity. If feasible, transesophageal echocardiography should be done as quickly as possible in appropriate patients during resuscitative efforts.

MANAGEMENT OF PULMONARY EMBOLISM

Echocardiography is excellent for selecting patients with pulmonary embolism who may have a poor prognosis. Echocardiography can facilitate a change in management of patients with pulmonary embolism by identifying those at high risk who might otherwise escape early detection. It can also help assess whether thrombus is sufficiently central to warrant potential surgical embolectomy. Finally, the echocardiogram can be used to assess whether a selected intervention such as thrombolysis is improving right ventricular function and diminishing elevated pulmonary artery pressures.

Risk Stratification and Prognostication

The clinical spectrum of patients with pulmonary embolism is wide. Patients range from those with minimal symptoms and small clot burden to those with cardiogenic shock due to acute right ventricular failure. Most patients with pulmonary embolism fall between these two extremes. Rapid risk stratification of patients with pulmonary embolism may ensure optimal management that is tailored to prognosis, with use of thrombolysis or embolectomy when appropriate.

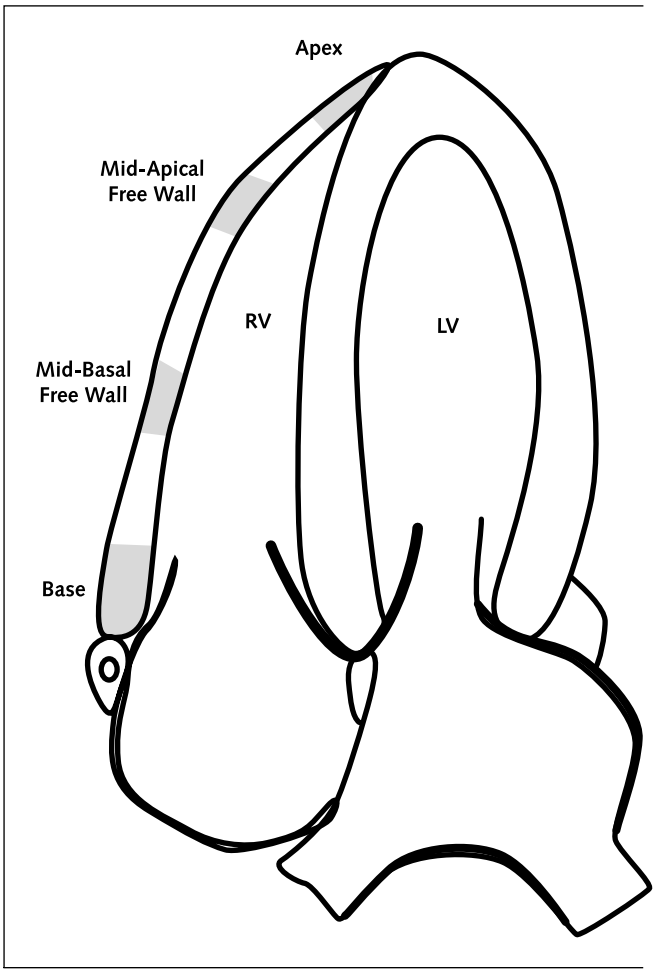
Right Ventricular Dysfunction

Right ventricular dysfunction has been reliably established as a predictor of increased likelihood of death from pulmonary embolism. Moderate or severe right ventricular dysfunction is usually identified qualitatively, and it is ordinarily readily apparent to observers with only modest experience. The most commonly used quantitative standards are 1) a right ventricular–to–left ventricular end-diastolic diameter ratio greater than 1 in the apical four-chamber view, 2) a right ventricular end-diastolic diameter greater than 30 mm, or 3) paradoxical right ventricular septal systolic motion.

At the Karolinska Institute in Stockholm, Sweden, 126 consecutive patients with pulmonary embolism were examined with echocardiography on the day of diagnosis (16). Overall, 56 patients had normal or near-normal right ventricular function, and 70 had moderately or severely impaired right ventricular function. After multivariate analysis, right ventricular dysfunction emerged as the most powerful predictor of in-hospital death, with a 6-fold increase in relative risk compared with normal right ventricular function. The presence of cancer doubled the risk for in-hospital death. After 1 year of follow-up, the relative risk for death was attenuated to 2.4-fold among patients with pulmonary embolism and moderate or severe right ventricular dysfunction. Neither embolism size nor the amount of vascular bed occluded was studied.

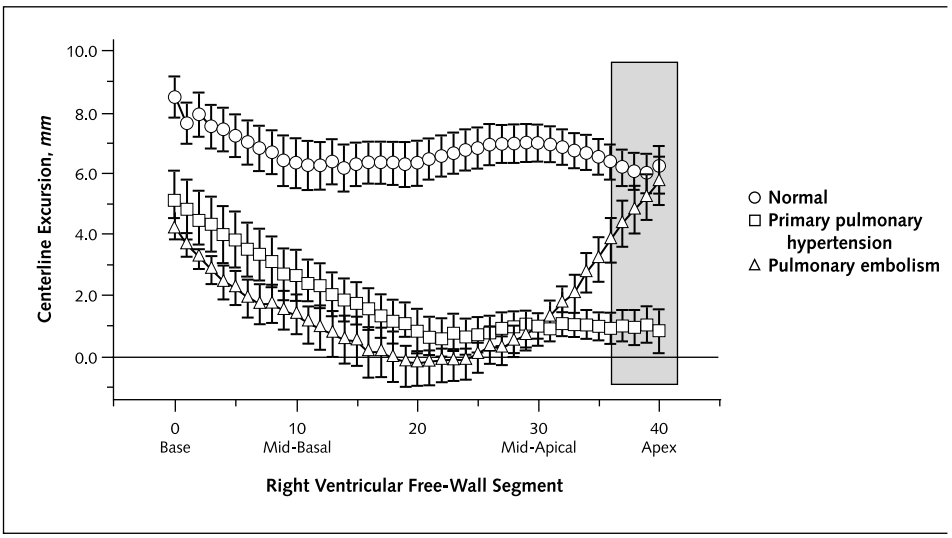
In a cohort of 209 consecutive patients with pulmonary embolism, 65 (31%) presented with the combination of normal systemic arterial pressure and echocardio-

Figure 3. **Apical four-chamber view from a transthoracic two-dimensional echocardiogram.**



Qualitative wall-motion scores were assigned at four locations of the right ventricular free wall (shaded areas). The excursion of the right ventricular free wall was measured from end-diastole to end-systole, and a centerline was defined midway between the diastolic and systolic curves. Chord lengths were then defined perpendicular to the centerline extending from the diastolic to the systolic curve. Forty measurements were obtained from the right ventricular base to the right ventricular apex. LV = left ventricle; RV = right ventricle. Reprinted from McConnell et al. (11); Am J Cardiol, pp 469-473, copyright 1996, with permission of Excerpta Medica.

Figure 4. **Segmental right ventricular free-wall excursion (mean ±SE) by centerline analysis as a function of right ventricular free-wall segment.**



The 40 right ventricular free-wall segments were arbitrarily numbered from 1 at the base to 40 at the apex. The centerline excursion was then plotted at each of these 40 intervals. This process generated a curve for normal right ventricular function in which the centerline excursion remained fairly constant at 7 to 8 mm. Centerline excursion in patients with acute pulmonary embolism was near normal at the apex (shaded area) but abnormal at the mid-free wall and base ($P < 0.02$ compared with normal). Centerline excursion in patients with primary pulmonary hypertension was reduced compared with that in normal persons in all segments ($P < 0.03$). Reprinted from McConnell et al. (11); Am J Cardiol, pp 469-473, copyright 1996, with permission of Excerpta Medica.

graphic evidence of right ventricular dysfunction (17). Of this group, 6 (10%) developed cardiogenic shock within 24 hours of diagnosis and 3 (5%) died during the initial hospitalization. Conversely, none of the 97 normotensive patients with normal right ventricular function on echocardiography died of pulmonary embolism (17).

In the International Cooperative Pulmonary Embolism Registry, the 90-day mortality rate was increased in patients who had right ventricular dysfunction on baseline echocardiography. After multiple regression modeling, right ventricular hypokinesis emerged as a powerful independent predictor of death at 90 days. It doubled the hazard ratio of death at 90 days and was as important a prognosticator as age older than 70 years, cancer, congestive heart failure, chronic obstructive pulmonary disease, and renal insufficiency (1).

Persistent Pulmonary Hypertension

Doppler echocardiography also has an important role in assessing 5-year survival. At the Karolinska Institute, initial echocardiography in patients with pulmonary embolism was followed by repeated echocardiography in the year after hospitalization for pulmonary embolism (18). During a "dynamic phase" that extended for the first 6 weeks after hospitalization, the estimated pulmonary artery systolic pressure decreased exponentially and the right ventricular function recovered markedly. Then, a stable "plateau phase" occurred in most patients.

However, patients with a pulmonary artery systolic pressure estimated as 50 mm Hg or greater at admission were three times more likely and patients 70 years of age or older were four times more likely to have persistent pulmonary hypertension and right ventricular dysfunction at 6 weeks compared with younger patients who presented with normal or only mildly elevated pulmonary artery pressures. Patients with pulmonary embolism and persistent pulmonary hypertension and right ventricular dysfunction at 6 weeks were less likely to survive during the ensuing 5 years than patients whose pulmonary pressures and right ventricular function had normalized.

Patent Foramen Ovale

A patent foramen ovale with a diameter greater than 4 mm increases the risk for embolic cerebrovascular

events (19). In patients with major pulmonary embolism, detection of a patent foramen ovale signifies a particularly high risk for death and arterial thromboembolic complications (20). In Konstantinides and colleagues' case series of 139 consecutive patients with pulmonary embolism, the presence of a patent foramen ovale was associated with a mortality rate more than twice as high as in patients without evidence of right-to-left shunt (20). Multivariate analysis indicated a greater than 10-fold increase in the risk for death and a 5-fold increase in the risk for major adverse events among patients with pulmonary embolism and a patent foramen ovale. Therefore, in patients with pulmonary embolism and elevated right atrial pressure, a patent foramen ovale should be sought on echocardiography with color-flow Doppler imaging or agitated saline contrast.

Free-Floating Right-Heart Thrombi

In a series of 38 consecutive patients with free-floating right heart thrombi detected on echocardiography, 37 patients had pulmonary embolism (21). The thrombi were usually wormlike and occasionally extended through a patent foramen ovale. Almost all of these patients had right ventricular dysfunction and pulmonary hypertension. Their prognosis was dismal, and 45% died.

Role of Thrombolysis

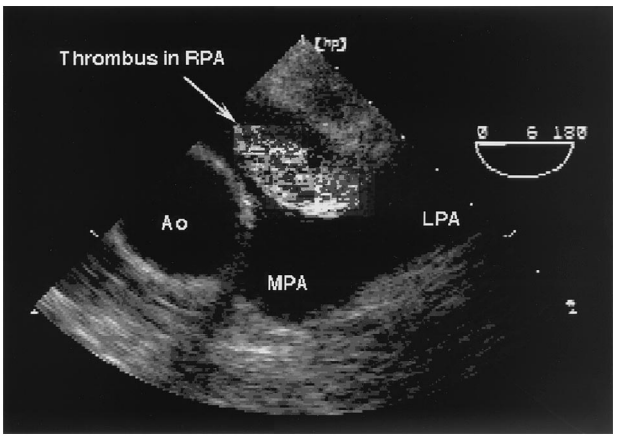
In the Management Strategy and Prognosis of Pulmonary Embolism (MAPPET) registry of 1001 patients with acute major pulmonary embolism (22), thrombolysis appeared to be beneficial in the subgroup of 719 patients with moderate or severe right ventricular dysfunction and initially normal systemic arterial pressure (23). The 30-day mortality rate was lower in the 169 patients who immediately received thrombolytic therapy than in the 550 patients who initially received anticoagulation alone (4.7% vs. 11.1%). The major bleeding rate was 22% in the thrombolysis group compared with 7.8% in the anticoagulation-alone group. Multivariate analysis indicated that thrombolysis halved the death rate, at the expense of an increase in major bleeding complications. In this registry, the decision to use thrombolysis was not randomized and was left to the discretion of the treating physician. However, MAPPET provides an important link between thrombolysis and clinically relevant end points.

In a randomized trial of thrombolysis plus anticoagulation versus anticoagulation alone, the investigators enrolled 101 patients with pulmonary embolism who had normal systemic arterial pressure (7). The 55 patients who were randomly assigned to anticoagulation alone had a good outcome in the absence of right ventricular dysfunction. However, of the 23 patients with right ventricular hypokinesis and dilatation, 5 had recurrent pulmonary embolism, even though they received intravenous heparin and had therapeutic partial thromboplastin times. Pulmonary embolism did not recur among the 46 patients randomly assigned to receive thrombolysis plus anticoagulation, even though 23 had right ventricular dysfunction.

After thrombolysis for acute pulmonary embolism, both regional and global right ventricular function usually recover markedly (24). This finding can be demonstrated quantitatively by assessing right ventricular wall-motion excursion and right ventricular end-diastolic and end-systolic areas (**Figure 6**).

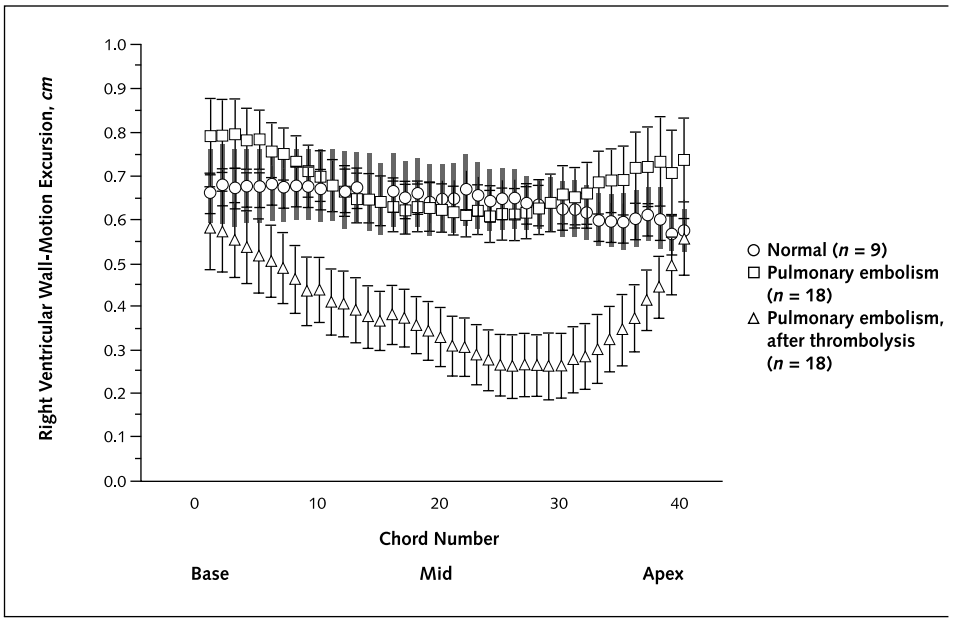
Echocardiography has been used successfully to monitor patients undergoing thrombolysis for right-

Figure 5. **Transesophageal short-axis view at the level of the pulmonary artery bifurcation, demonstrating thrombus in the right pulmonary artery (*RPA*).**



Also shown are the left pulmonary artery (*LPA*) and aorta (*Ao*). hp = Hewlett Packard; MPA = main pulmonary artery. Reprinted with permission from Comess et al. (14); Am J Med, pp 351-356, copyright 2000, with permission of Excerpta Medica. The electronic original of the figure was kindly provided by Keith A. Comess, MD.

Figure 6. **Right ventricular wall-motion excursion and range for normal persons, patients with acute pulmonary embolism, and patients with pulmonary embolism on follow-up after thrombolysis.**



Wall-motion excursion was significantly different from normal in chord numbers 9 to 37 in patients with acute pulmonary embolism. Wall-motion excursion did not significantly differ from normal at any location on follow-up echocardiography. The bars represent SEs. Reprinted with permission from Nass et al. (24); Am J Cardiol, pp 804-806, copyright 1999, with permission of Excerpta Medica.

heart thrombi. In a series of seven patients who received tissue plasminogen activator, 100 mg over 2 hours, continuous echocardiographic monitoring demonstrated clot dissolution within 1 hour in all patients (25).

I believe that thrombolysis should be considered in patients with pulmonary embolism who have moderate or severe right ventricular dysfunction. I obtain an echocardiogram at baseline in almost all patients being considered for lytic therapy, unless computed tomography of the chest shows saddle pulmonary embolism or the patient is hemodynamically unstable.

Bedside Echocardiography for Critically Ill Patients

Bedside echocardiography can help physicians decide whether critically ill patients with pulmonary embolism should have thrombolysis, catheter embolectomy, or surgical embolectomy in addition to anticoagulation. Inspecting the echocardiogram can quickly identify moderate or severe right ventricular dysfunction, and transesophageal echocardiography can visualize centrally located thromboembolism in the main pulmonary arteries that is surgically accessible. Occasionally, transthoracic echocardiography can visualize saddle embolism, but using this strategy to identify potential surgical candidates is not consistently reliable. Among 49 consecutive patients suspected of having massive pulmonary embolism who had abnormal transthoracic echocardiograms, Pruszczyk and colleagues (13) compared the gold standard (computed tomography of the chest) with transesophageal echocardiography. The sensitivity of transesophageal echocardiography for detecting pulmonary embolism of any size was 80%; its specificity was 100% (13).

In my institution, critically ill patients with contraindications to thrombolysis or with large atrial thrombi at risk for embolization are directly transferred from referring hospitals to the cardiac surgical operating room, without evaluation in the emergency department or intensive care unit (26). In the operating room, before sternotomy, surgeons perform emergent transesophageal echocardiography to ensure that surgery is appropriate. At times, patients transferred with a large right atrial thrombus on transthoracic echocardiography have sustained embolization to the central pulmonary arteries before surgery. Catheter embolectomy is a less invasive strategy that requires skill and experience (27).

An Integrated Approach to Risk Stratification

To predict which patients will have adverse outcomes, echocardiography is ideally used in conjunction with pertinent clinical and laboratory measures. Those with massive pulmonary embolism most often present clinically with unexplained breathlessness. Syncope and presyncope due to pulmonary embolism are uncommon but ominous findings. Tachypnea is often present with acute, major pulmonary embolism, but cyanosis is rare. Patients with difficulty maintaining an adequate systemic arterial pressure or elderly patients are at high risk for death or recurrent pulmonary embolism. In the International Cooperative Pulmonary Embolism Registry, right ventricular dysfunction on echocardiography was an independent predictor of death (1). Other adverse prognostic markers included age older than 70 years, cancer, congestive heart failure, chronic obstructive pulmonary disease, and systemic arterial hypotension.

Recently, elevation of the troponin level has emerged as a correlate of right ventricular dysfunction (28) and as an independent prognostic indicator of a poor outcome after pulmonary embolism (29). Acute pressure overload due to pulmonary embolism may cause regional myocardial ischemia and release of troponin without coronary atherosclerosis.

Ultrasound Thrombolysis

Echocardiography has the potential to lyse pulmonary embolism. In a rabbit femoral artery thrombosis model, ultrasound at 40 kHz accelerated fibrinolysis, improved tissue perfusion, and reversed acidosis (30). In a canine model of acute myocardial infarction, transthoracic application of low-frequency ultrasound augmented the efficacy of thrombolysis with tissue plasminogen activator (31). An in vitro study using bovine arterial segments showed that external high-intensity focused ultrasound thrombolysis appears safe and effective. An attractive futuristic approach is using echocardiography to monitor the results of attempted ultrasound thrombolysis (32).

CONCLUSIONS

Echocardiography is a convenient and safe imaging technique that may provide critical information on the physiologic effect of pulmonary embolism on right ventricular function. In the management of pulmonary embolism, echocardiography can provide rapid and accurate risk assessment and can help direct therapy toward thrombolysis or embolectomy. The success or failure of an individual patient's therapy can be monitored by serial assessment of right ventricular function. Further research will clarify and define more precisely the utility and limitations of echocardiography in the management of pulmonary embolism.

From Brigham and Women's Hospital, Boston, Massachusetts.

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Requests for Single Reprints: Samuel Z. Goldhaber, MD, Cardiovascular Division, Brigham and Women's Hospital, 75 Francis Street, Boston, MA 02115; e-mail, sgoldhaber@partners.org.

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