

The RUSH Exam: Rapid Ultrasound in SHock in the Evaluation of the Critically Ill

Phillips Perera, MD, RDMS, FACEP^{a,*}, Thomas Mailhot, MD, RDMS^b,
David Riley, MD, MS, RDMS^a, Diku Mandavia, MD, FACEP, FRCPC^{b,c}

KEYWORDS

- Rapid ultrasound in shock examination • RUSH exam
- Shock • Ultrasound

Care of the patient with shock can be one of the most challenging issues in emergency medicine. Even the most seasoned clinician, standing at the bedside of the patient in extremis, can be unclear about the cause of shock and the optimal initial therapeutic approach. Traditional physical examination techniques can be misleading given the complex physiology of shock.¹ Patients in shock have high mortality rates, and these rates are correlated to the amount and duration of hypotension. Therefore, diagnosis and initial care must be accurate and prompt to optimize patient outcomes.² Failure to make the correct diagnosis and act appropriately can lead to potentially disastrous outcomes and high-risk situations.

Ultrasound technology has been rapidly integrated into Emergency Department care in the last decade. More practicing emergency physicians (EPs) are now trained in bedside point of care or goal-directed ultrasound, and this training is now included in all United States Accreditation Council for Graduate Medical Education Emergency Medicine residency programs.^{3,4} Furthermore, the American College of Emergency Physicians (ACEP) has formally endorsed and embraced bedside ultrasound by the EP for multiple applications.⁵ This technology is ideal in the care of the critical patient in shock, and the most recent ACEP guidelines further delineate a new category of

^a New York Presbyterian Hospital, Columbia University Medical Center, Division of Emergency Medicine, 622 West 168th Street, PH1-137, New York, NY 10032, USA

^b LA County+USC Medical Center, Department of Emergency Medicine, General Hospital, 1200 State Street, Room 1011, Los Angeles, California 90033, USA

^c Cedars-Sinai Medical Center, Department of Emergency Medicine, General Hospital, 1200 State Street, Room 1011, Los Angeles, California 90033, USA

* Corresponding author.

E-mail address: pperera1@mac.com (P. Perera).

“resuscitative” ultrasound. Studies have demonstrated that initial integration of bedside ultrasound into the evaluation of the patient with shock results in a more accurate initial diagnosis with an improved patient care plan.^{1,6,7} Instead of relying on older techniques, like listening for changes in sound coming from the patient’s body suggestive of specific pathology, bedside ultrasound now allows direct visualization of pathology or abnormal physiological states. Thus, bedside ultrasound has become an essential component in the evaluation of the hypotensive patient.

CLASSIFICATIONS OF SHOCK

Many authorities categorize shock into 4 classic subtypes.⁸ The first is hypovolemic shock. This condition is commonly encountered in the patient who is hemorrhaging from trauma, or from a nontraumatic source of brisk bleeding such as from the gastrointestinal (GI) tract or a rupturing aortic aneurysm. Hypovolemic shock may also result from nonhemorrhagic conditions with extensive loss of body fluids, such as GI fluid loss from vomiting and diarrhea. The second subtype of shock is distributive shock. The classic example of this class of shock is sepsis, in which the vascular system is vasodilated to the point that the core vascular blood volume is insufficient to maintain end organ perfusion. Other examples of distributive shock include neurogenic shock, caused by a spinal cord injury, and anaphylactic shock, a severe form of allergic response. The third major form of shock is cardiogenic shock, resulting from pump failure and the inability of the heart to propel the needed oxygenated blood forward to vital organs. Cardiogenic shock can be seen in patients with advanced cardiomyopathy, myocardial infarction, or acute valvular failure. The last type of shock is obstructive shock. This type is most commonly caused by cardiac tamponade, tension pneumothorax, or large pulmonary embolus. Many patients with obstructive shock will need an acute intervention such as pericardiocentesis, tube thoracostomy or anticoagulation, or thrombolysis.

At the bedside of a critical patient, it is often difficult to assess clinically which classification of shock best fits the patient’s current clinical status. Physical findings often overlap between the subtypes. For example, patients with tamponade, cardiogenic shock and sepsis (when myocardial depression compounds this form of distributive shock) may all present with distended neck veins and respiratory distress. Because of this diagnostic challenge, practitioners used to perform Swan-Ganz catheterization in hypotensive patients, providing immediate intravascular hemodynamic data. Although the data obtained from these catheters was detailed and often helpful at the bedside, large studies demonstrated no improvement in mortality in the patients who received such prolonged invasive monitoring.⁹ Swan-Ganz catheterization has thus declined in use, and the stage has now been set for development of a noninvasive hemodynamic assessment using point of care ultrasound.

SHOCK ULTRASOUND PROTOCOL: THE RUSH EXAM

Given the advantages of early integration of bedside ultrasound into the diagnostic workup of the patient in shock, this article outlines an easily learned and quickly performed 3-step shock ultrasound protocol. The authors term this new ultrasound protocol the RUSH exam (*R*apid *U*ltrasound in *S*Hock). This protocol involves a 3-part bedside physiologic assessment simplified as:

Step 1: The pump

Step 2: The tank

Step 3: The pipes

This examination is performed using standard ultrasound equipment present in many emergency departments today. The authors recommend a phased-array transducer (3.5–5 MHz) to allow adequate thoracoabdominal intercostal scanning, and a linear array transducer (7.5–10 MHz) for the required venous examinations and for the evaluation of pneumothorax.

The first, and most crucial, step in evaluation of the patient in shock is determination of cardiac status, termed for simplicity “the pump” (Table 1). Clinicians caring for the patient in shock begin with a limited echocardiogram. The echo examination is focused on looking for 3 main findings. First, the pericardial sac can be visualized to determine if the patient has a pericardial effusion that may be compressing the heart, leading to a mechanical cause of obstructive shock. Second, the left ventricle can be analyzed for global contractility. Determination of the size and contractility status of the left ventricle will allow for those patients with a cardiogenic cause of shock to be rapidly identified.^{10,11} The third goal-directed examination of the heart focuses on determining the relative size of the left ventricle to the right ventricle. A heart that has an increased size of the right ventricle relative to the left ventricle may be a sign of acute right ventricular strain from a massive pulmonary embolus in the hypotensive patient.^{12,13}

The second part of the RUSH shock ultrasound protocol focuses on the determination of effective intravascular volume status, which will be referred to as “the tank.” Placement of the probe in the subxiphoid position, along both the long and short axis of the inferior vena cava (IVC), will allow correct determination of the size of the vessel. Looking at the respiratory dynamics of the IVC will provide an assessment of the patient’s volume status to answer the clinical question, “how full is the

Table 1
Rapid Ultrasound in SHock (RUSH) protocol: ultrasonographic findings seen with classic shock states

| RUSH Evaluation | Hypovolemic Shock | Cardiogenic Shock | Obstructive Shock | Distributive Shock |
|-----------------|---|---|--|---|
| Pump | Hypercontractile heart Small chamber size | Hypocontractile heart Dilated heart | Hypercontractile heart Pericardial effusion Cardiac tamponade RV strain Cardiac thrombus | Hypercontractile heart (early sepsis) Hypocontractile heart (late sepsis) |
| Tank | Flat IVC Flat jugular veins Peritoneal fluid (fluid loss) Pleural fluid (fluid loss) | Distended IVC Distended jugular veins Lung rockets (pulmonary edema) Pleural fluid Peritoneal fluid (ascites) | Distended IVC Distended jugular veins Absent lung sliding (pneumothorax) | Normal or small IVC (early sepsis) Peritoneal fluid (sepsis source) Pleural fluid (sepsis source) |
| Pipes | Abdominal aneurysm Aortic dissection | Normal | DVT | Normal |

Abbreviations: DVT, deep venous thrombosis; IVC, inferior vena cava; RV, right ventricle.

tank?”^{14,15} The clinician can also place a transducer on the internal jugular veins to view their size and changes in diameter with breathing to further assess volume.¹⁶ Also included in evaluation of the tank is an assessment of the lung, pleural cavity, and abdominal cavities for pathology that could signal a compromised vascular volume. Integration of lung ultrasound techniques can quickly allow the clinician to identify a pneumothorax, which in the hypotensive patient may represent a tension pneumothorax requiring immediate decompression. Tension pneumothorax presumably limits venous return into the heart due to increased pressure within the chest cavity.^{17,18} The lung can also be examined for ultrasonic B lines, a potential sign of volume overload and pulmonary edema. The clinician can further examine the thoracic cavity for a pleural effusion. Last, the clinician can perform a FAST exam (Focused Assessment with Sonography in Trauma examination), to look for fluid in the abdomen, indicating a source for “loss of fluid from the tank.”

The third and final part of the shock ultrasound protocol is evaluation of the large arteries and veins of the body, referred to as “the pipes.” Clinicians should answer the clinical question “are the pipes ruptured or obstructed” by first evaluating the arterial side of the vascular system to specifically examine the abdominal and thoracic aorta for an aneurysm or dissection. Next the clinician should turn to evaluation of the venous side of the vascular system. The femoral and popliteal veins can be examined with a high frequency linear array transducer for compressibility. Lack of full venous compression with direct pressure is highly suggestive of a deep venous thrombosis (DVT).^{19,20} Presence of a venous thrombus in the hypotensive patient may signal a large pulmonary thromboembolus.

RUSH Protocol: Step 1—Evaluation of the Pump

Focused echocardiography is a skill that is readily learned by the EP. A smaller footprint phased-array transducer is ideal for this examination as it permits the intercostal scanning required of the heart. Imaging of the heart usually involves 4 views. The traditional views of the heart for bedside echocardiography are the parasternal long- and short-axis views, the subxiphoid view, and the apical 4-chamber view (Fig. 1). The parasternal views are taken with the probe positioned just left of the sternum at intercostal space 3 or 4. The subxiphoid 4-chamber view is obtained with the probe aimed up toward the left shoulder from a position just below the subxiphoid tip of the sternum (Fig. 2). The apical 4-chamber view of the heart is best evaluated by turning the patient into a left lateral decubitus position and placing the probe just below the nipple line at the point of maximal impulse of the heart. It is important for the EP to know all 4 views

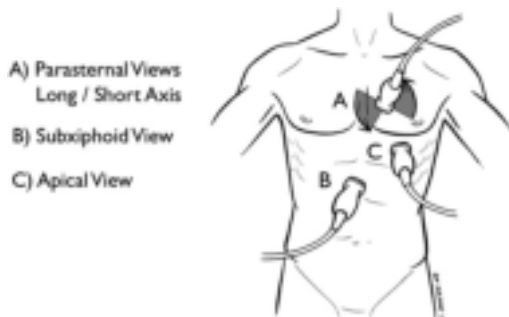


Fig. 1. Rapid Ultrasound in SHock (RUSH) step 1. Evaluation of the pump.

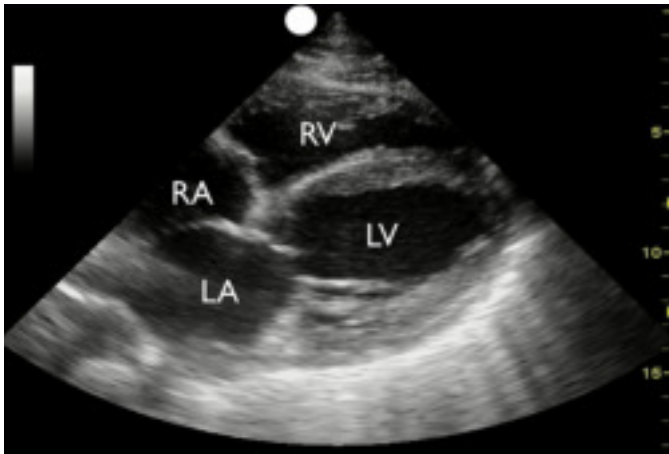


Fig. 2. Subxiphoid view: cardiomyopathy with enlarged heart. LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle.

of the heart, as some views may not be well seen in individual patients, and an alternative view may be needed to answer the clinical question at hand.

“Effusion around the pump”: evaluation of the pericardium

The first priority is to search for the presence of a pericardial effusion, which may be a cause of the patient’s hemodynamic instability. The heart should be imaged in the planes described here, with close attention to the presence of fluid, usually appearing as a dark or anechoic area, within the pericardial space (**Fig. 3**). Small effusions may be seen as a thin stripe inside the pericardial space, whereas larger effusions tend to wrap circumferentially around the heart.^{21,22} Isolated small anterior anechoic areas on the parasternal long-axis view often represent a pericardial fat pad, as free flowing pericardial effusions will tend to layer posteriorly and inferiorly with gravity. Fresh fluid

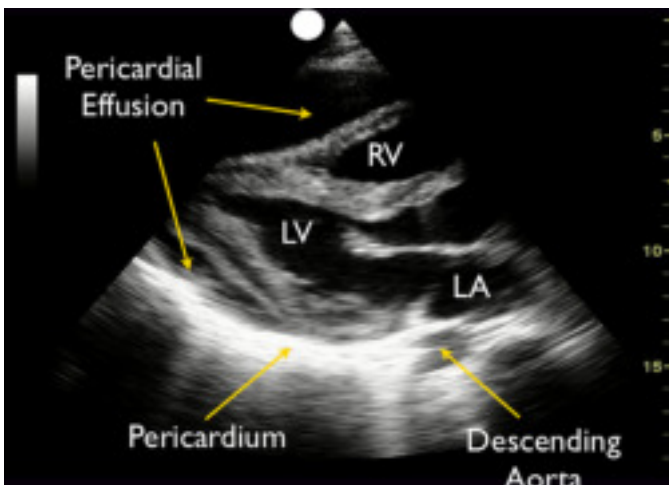


Fig. 3. Parasternal long-axis view: large pericardial effusion.

or blood tends to have a darker or anechoic appearance, whereas clotted blood or exudates may have a lighter or more echogenic look.

Pericardial effusions can result in hemodynamic instability, due to increased pressure within the sac leading to compression of the heart. Because the pericardium is a relatively thick and fibrous structure, acute pericardial effusions may result in cardiac tamponade despite only small amounts of fluid. In contrast, chronic effusions can grow to a large volume without hemodynamic instability.²³ Once a pericardial effusion is identified, the next step is to evaluate the heart for signs of tamponade. Thinking of the heart as a dual chamber in-line pump, the left side of the heart is under considerably more pressure, due to the high systemic pressures against which it must pump. The right side of the heart is under relatively less pressure, due to the lower pressure within the pulmonary vascular circuit. Thus, most echocardiographers define tamponade as compression of the right side of the heart (**Fig. 4**). High pressure within the pericardial sac keeps the chamber from fully expanding during the relaxation phase of the cardiac cycle and thus is best recognized during diastole. As either chamber may be affected by the effusion, both the right atrium and right ventricle should be closely inspected for diastolic collapse. Diastolic collapse of the right atrium or right ventricle appears as a spectrum from a subtle inward serpentine deflection of the outer wall to complete compression of a chamber.²⁴ Whereas most pericardial effusions are free flowing in the pericardial sac, occasionally effusions may be loculated. This phenomenon is more commonly seen in patients following heart surgery, in whom a clot can form in only one area of the sac.²⁵ In these cases, effusions can preferentially form posteriorly, and in tamponade, the left side of the heart may be compressed before the right side of the heart. The IVC can also be evaluated for additional confirmatory signs of tamponade.²⁶ IVC plethora will be recognized by distention of the IVC without normal respiratory changes. (see later discussion on IVC in the section “Evaluation of the tank”).

Previous published studies have demonstrated that EPs, with a limited amount of training, can correctly and accurately identify the presence of a pericardial effusion.²⁷ Studies examining the incidence of pericardial effusions in Emergency Department or

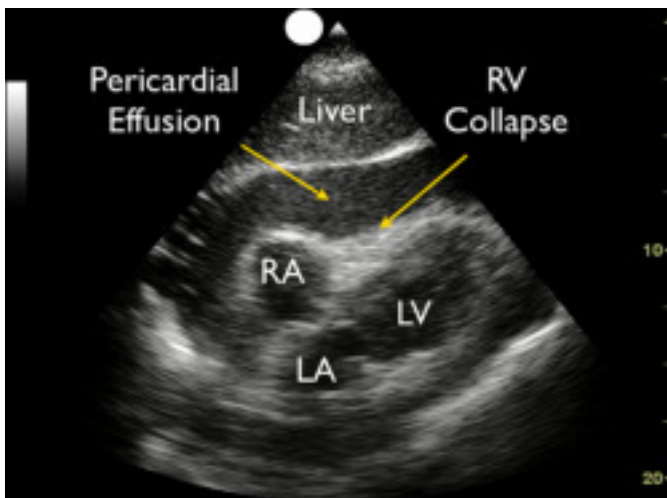


Fig. 4. Subxiphoid view: cardiac tamponade.

Intensive Care patients suffering acute shortness of breath, respiratory failure, or shock have found effusions in as many as 13% of these patients.²¹ Another study looked specifically at patients arriving at the Emergency Department in near-cardiac arrest states, and found a relatively large number of these cases had pericardial effusions.²⁸ Thus, symptomatic pericardial effusions may be a cause of hemodynamic instability in a significant number of acute patients, and EPs can quickly and accurately diagnose this condition using bedside ultrasound.

As a general principle, it is easier for an EP to diagnose a pericardial effusion than to evaluate for the specific signs of tamponade.²⁹ It is thus safer to assume tamponade physiology in the hypotensive patient if a significant pericardial effusion is identified. Under ideal circumstances, the EP can obtain a formal echocardiogram in conjunction with Cardiology to specifically examine for cardiac tamponade. In the rare cases where there is not enough time for consultation and the patient is unstable, a pericardiocentesis under echo guidance by the EP may be life-saving. In these cases, employing bedside echocardiography also allows the EP to determine the optimal needle insertion site for pericardiocentesis. Of note, most EPs have classically been taught the subxiphoid approach for pericardiocentesis. However, a large review from the Mayo Clinic looked at 1127 pericardiocentesis procedures, and found that the optimal placement of the needle was where the distance to the effusion was the least and the effusion size was maximal.³⁰ The apical position at the point of maximal impulse on the left lateral chest wall was chosen in 80% of these procedures, based on these variables. The subxiphoid approach was only chosen in 20% of these procedures, as the investigators recognized the large distance the needle had to travel through the liver to enter the pericardial sac. EPs should therefore anatomically map out the effusion before a pericardiocentesis procedure to plan the most direct and safest route. If the apical approach is selected, the patient should optimally be rolled into a left lateral decubitus position to bring the heart closer to the chest wall, and after local anesthesia, a pericardiocentesis drainage catheter should be introduced over the rib and into the pericardial sac. To maximize success and to avoid complications, the transducer should be placed in a sterile sleeve adjacent to the needle, and the procedure performed under real-time ultrasound guidance.

“Squeeze of the pump”: determination of global left ventricular function

The next step in the RUSH protocol is to evaluate the heart for contractility of the left ventricle. This assessment will give a determination of “how strong the pump is.” The examination focuses on evaluating motion of the left ventricular endocardial walls, as judged by a visual calculation of the percentage change from diastole to systole. Whereas in the past echocardiographers used radionuclide imaging to determine ejection fraction, published studies have demonstrated that visual determination of contractility is roughly equivalent.³¹ A ventricle that has good contractility will be observed to have a large percentage change from the 2 cycles, with the walls almost coming together and touching during systole. As an example, a vigorously contracting ventricle will almost completely obliterate the ventricular cavity during systole. In comparison, a poorly contracting heart will have a small percentage change in the movement of the walls between diastole and systole. In these hearts, the walls will be observed to move little during the cardiac cycle, and the heart may also be dilated in size, especially if a long-standing cardiomyopathy with severe systolic dysfunction is present. Motion of anterior leaflet of the mitral valve can also be used to assess contractility. In a normal contractile state, the anterior leaflet will vigorously touch the wall of the septum during ventricular filling when examined using the parasternal long-axis view.

The parasternal long-axis view of the heart is an excellent starting view to assess ventricular contractility. Moving the probe into the parasternal short-axis orientation will give confirmatory data on the strength of contractions. In this view, a left ventricle with good contraction will appear as a muscular ring that squeezes down concentrically during systole. Whereas cardiologists often use the parasternal short-axis view to evaluate for segmental wall motion abnormalities, this is a more subjective measurement, and determinations may differ among different clinicians. For that reason, it is better for the EP to initially concentrate on the overall contractility of the ventricle, rather than to evaluate for segmental wall motion deficits. An easy system of grading is to judge the strength of contractions as good, with the walls of the ventricle contracting well during systole; poor, with the endocardial walls changing little in position from diastole to systole; and intermediate, with the walls moving with a percentage change in between the previous 2 categories. If the parasternal views are inadequate for these determinations, moving the patient into the left lateral decubitus position and examining from the apical view often gives crucial data on left ventricular contractility. The subxiphoid view can be used for this determination, but the left ventricle is farther away from the probe in this view.

Published studies confirm that EPs can perform this examination and get an estimate of left ventricular contractility that compares well with that measured by a cardiologist.³² Because a large proportion of shock patients (up to 60% in one study) will have a cardiac cause for their hypotension, this part of the examination can be very high yield.¹⁰ Immediate identification of cardiogenic shock by the EP can lead to more rapid transfer of the patient to the cardiac catheterization suite for revascularization, especially in suspected cases of cardiac ischemia.^{33,34} Other types of shock can be evaluated by knowing the strength of the left ventricle during systole. Strong ventricular contractility (often termed hyperdynamic, because of the strength of contractions of the left ventricle in addition to a rapid heart rate) is often seen in early sepsis and in hypovolemic shock.³⁵ In severe hypovolemic conditions, the heart is often small in size with complete obliteration of the ventricular cavity during systole. Bedside echocardiography also allows for repeated evaluation of the patient's heart, looking for changes in contractility over time, especially when if a patient's status deteriorates. For example, later in the course of sepsis there may be a decrease in contractility of the left ventricle due to myocardial depression.³⁶

Knowing the strength of left ventricular contractility will give the EP a better idea of how much fluid "the pump" or heart of the patient can handle, before manifesting signs and symptoms of fluid overload. This knowledge will serve as a critical guide for the clinician to determine the amount of fluid that can be safely given to a patient. As an example, in a heart with poor contractility, the threshold for initiation of vasopressor agents for hemodynamic support should be lower. In contrast, sepsis patients have been shown to benefit with aggressive early goal-directed therapy, starting with large amounts of fluids before use of vasopressor medications.³⁷ Because many Emergency Departments do not currently use the invasive catheter needed to optimally monitor the hemodynamic goals outlined for treatment of sepsis patients, bedside ultrasound gives the clinician a noninvasive means to identify and follow a best management strategy.

In cardiac arrest, the clinician should specifically examine for the presence or absence of cardiac contractions. If contractions are seen, the clinician should look for the coordinated movements of the mitral and aortic valves. In this scenario, the absence of coordinated opening of mitral and aortic valves will require chest compressions to maintain cardiac output. Furthermore, if after prolonged advanced cardiac life

support resuscitation the bedside echocardiogram shows cardiac standstill, it is unlikely that the adult patient will have return of spontaneous circulation.^{38,39}

“Strain of the pump”: assessment of right ventricular strain

In the normal heart, the left ventricle is larger than the right ventricle. This aspect is predominantly a cause of the muscular hypertrophy that takes place in the myocardium of the left ventricle after birth, with the closure of the ductus arteriosus. The left ventricle is under considerably more stress than the right ventricle, to meet the demands of the higher systemic pressure, and hypertrophy is a normal compensatory mechanism. On bedside echocardiography, the normal ratio of the left to right ventricle is 1:0.6.⁴⁰ The optimal cardiac views for determining this ratio of size between the 2 ventricles are the parasternal long and short-axis views and the apical 4-chamber view. The subxiphoid view can be used, but care must be taken to fan through the entire right ventricle, as it is easy to underestimate the true right ventricular size in this view.

Any condition that causes pressure to suddenly increase within the pulmonary vascular circuit will result in acute dilation of the right heart in an effort to maintain forward flow into the pulmonary artery. The classic cause of acute right heart strain is a large central pulmonary embolus. Due to the sudden obstruction of the pulmonary outflow tract by a large pulmonary embolus, the right ventricle will attempt to compensate with acute dilation. This process can be seen on bedside echocardiography by a right ventricular chamber that is as large, or larger, than the left ventricle (Fig. 5).⁴¹ In addition, deflection of the interventricular septum from right to left toward the left ventricle may signal higher pressures within the pulmonary artery.⁴² In rare cases, intracardiac thrombus may be seen floating free within the heart (Fig. 6).⁴³ In comparison, a condition that causes a more gradual increase in pulmonary artery pressure over time, such as smaller and recurrent pulmonary emboli, cor pulmonale with predominant right heart strain, or primary pulmonary artery hypertension, will cause both dilation and thickening or hypertrophy of the right ventricular wall.⁴⁴ These mechanisms can allow the right ventricle to compensate over time and to adapt to pumping

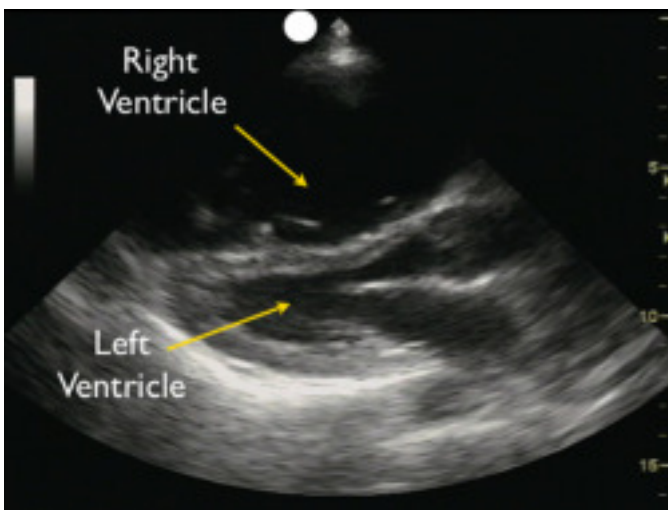


Fig. 5. Parasternal long-axis view: right ventricular strain.

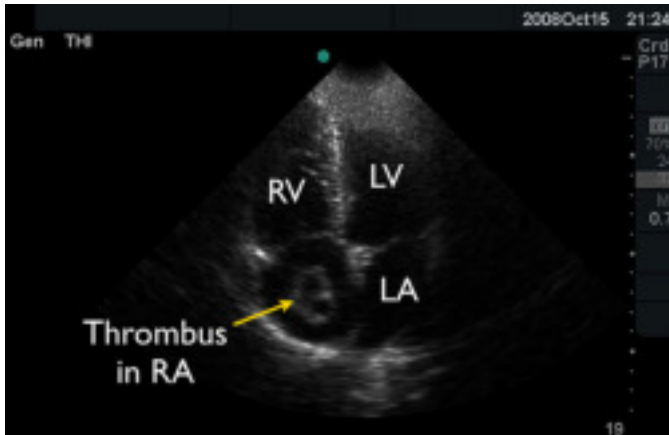


Fig. 6. Apical view: floating thrombus in right atrium.

blood against the higher pressures in the pulmonary vascular circuit. Acute right heart strain thus differs from chronic right heart strain in that although both conditions cause dilation of the chamber, the ventricle will not have the time to hypertrophy if the time course is sudden.

Previous published studies have looked at the sensitivity of the finding of right heart dilation in helping the clinician to diagnose a pulmonary embolus. The results show that the sensitivity is moderate, but the specificity and positive predictive value of this finding are high in the correct clinical scenario, especially if hypotension is present.^{12,13,45–47} The finding of acute right heart strain due to a pulmonary embolus correlates with a poorer prognosis.^{12,48,49} This finding, in the setting of suspected pulmonary embolus, suggests the need for immediate evaluation and treatment of thromboembolism.⁵⁰ The EP should also proceed directly to evaluation of the leg veins for a DVT (covered in detail later under “Evaluation of the pipes”).

The literature suggests that in general, patients with a pulmonary embolus should be immediately started on heparin. However, a hypotensive patient with a pulmonary embolus should be considered for thrombolysis.^{51,52} Besides, ultrasound gives the treating clinician the clinical confidence to proceed in this more aggressive fashion. Clinical status permitting, a chest computed tomography (CT) scan using a dedicated pulmonary embolus protocol should be obtained. If the patient is not stable enough for CT, an emergent echocardiogram in conjunction with Cardiology or bilateral duplex ultrasound of the legs should be considered.

RUSH Protocol Step 2: Evaluation of the Tank

“Fullness of the tank”: evaluation of the inferior cava and jugular veins for size and collapse with inspiration

The next step for the clinician using the RUSH protocol in the hypotensive patient is to evaluate the effective intravascular volume as well as to look for areas where the intravascular volume might be compromised (**Fig. 7**). An estimate of the intravascular volume can be determined noninvasively by looking initially at the IVC.^{14,15} The ultrasound transducer should be positioned in the epigastric area in a long-axis configuration along the IVC as it runs from the abdomen into the heart. A good way of obtaining this image is to first examine the heart in the subxiphoid 4-chamber plane and then to move the probe into the subxiphoid 2-chamber plane, with the probe marker oriented

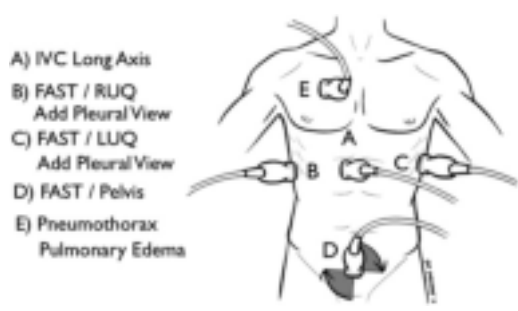


Fig. 7. RUSH step 2. Evaluation of the tank. IVC exam, inferior vena cava; FAST views (Focused Sonography in Trauma), right upper quadrant, left upper quadrant and suprapubic; lung exam, pneumothorax and pulmonary edema.

anteriorly. The aorta will often come quickly into view from this plane as a thicker walled and deeper structure. Moving the probe to the patient's right will bring the IVC into view, running longitudinally adjacent to the aorta. The IVC should be examined at the junction of the right atrium and the cava and followed 2 to 3 cm caudally along the vessel. Moving the probe into short axis to further evaluate the IVC is complementary as it will display the structure as an oval and allow confirmation of size by avoiding underestimation of the structure with the cylinder effect. As the patient breathes, the IVC will have a normal pattern of collapse during inspiration, due to the negative pressure generated in the chest, causing increased flow from the abdominal to the thoracic cavity (**Fig. 8**). This respiratory variation can be further augmented by having the patient sniff or inspire forcefully. M-mode Doppler, positioned on the IVC, can graphically document the dynamic changes in the vessel caliber during the patient's respiratory cycle (**Fig. 9**).

Previous studies have demonstrated a correlation between the size and percentage change of the IVC with respiratory variation to central venous pressure (CVP) using an indwelling catheter. A smaller caliber IVC (<2 cm diameter) with an inspiratory collapse

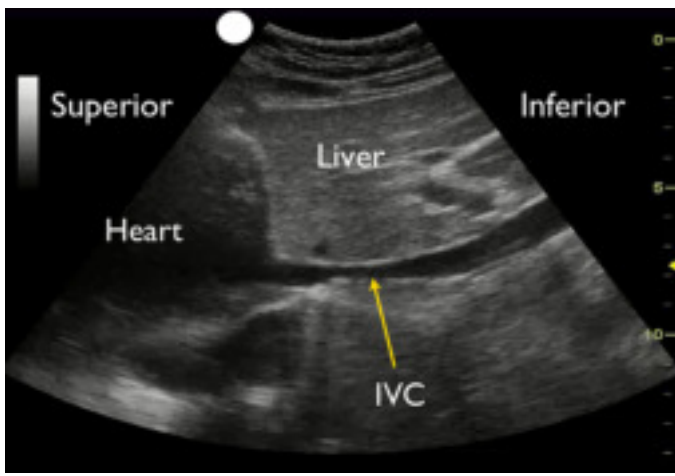


Fig. 8. Inferior vena cava sniff test: low cardiac filling pressures.

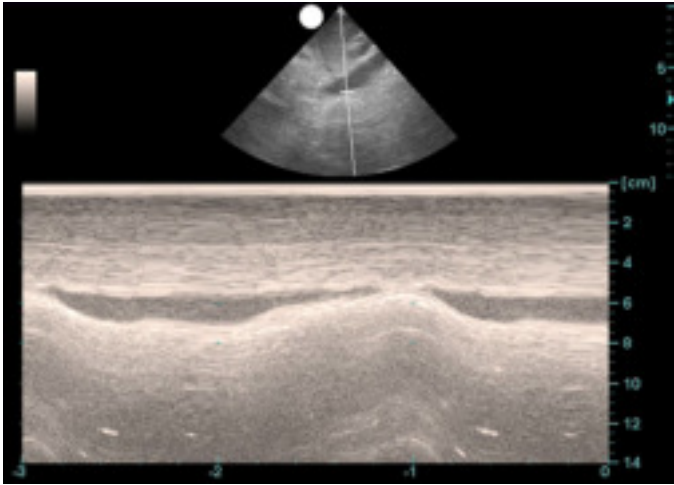


Fig. 9. Inferior vena cava sniff test: M-mode Doppler showing collapsible IVC.

greater than 50% roughly correlates to a CVP of less than 10 cm of water. This phenomenon may be observed in hypovolemic and distributive shock states. A larger sized IVC (>2 cm diameter) that collapses less than 50% with inspiration correlates to a CVP of more than 10 cm of water (Fig. 10).^{53,54} This phenomenon may be seen in cardiogenic and obstructive shock states. Two caveats to this rule exist. The first is in patients who have received treatment with vasodilators and/or diuretics prior to ultrasound evaluation in whom the IVC may be smaller than prior to treatment, altering the initial physiological state. The second caveat exists in intubated patients receiving positive pressure ventilation, in which the respiratory dynamics of the IVC are reversed. In these patients, the IVC is also less compliant and more distended throughout all respiratory cycles. However, crucial physiologic data can still be

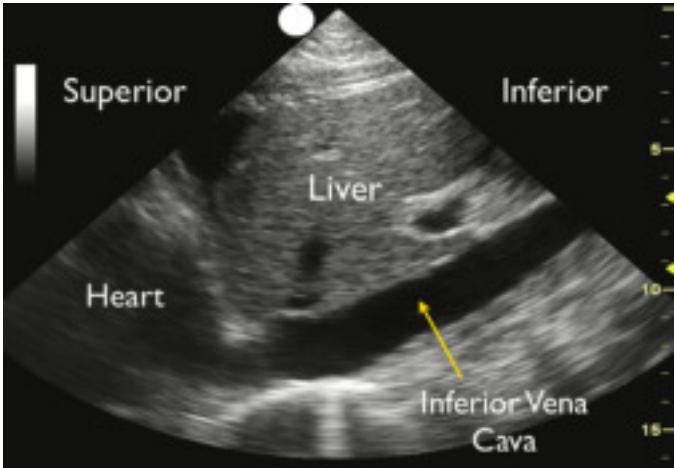


Fig. 10. Inferior vena cava sniff test: high cardiac filling pressures.

obtained in these ventilated patients, as fluid responsiveness has been correlated with an increase in IVC diameter over time.⁵⁵

Evidence suggests that the bedside ultrasound estimate of CVP is most accurate when the IVC is small and inspiratory collapse is high. However, rather than relying on a single measurement of the IVC, it is better to determine the effective vascular volume by following changes in size and respiratory dynamics over time with fluid challenges.⁵⁶ Observing a change in IVC size from small, with a high inspiratory collapse, to a larger IVC with little inspiratory collapse, suggests that the CVP is increasing and “the tank” is more full.⁵⁷

The internal jugular veins can also be examined with ultrasound to further evaluate the intravascular volume. As with visual evaluation of the jugular veins, the patient’s head is placed at a 30° angle. Using a high-frequency linear array transducer, the internal jugular veins can first be found in the short-axis plane, then evaluated more closely by moving the probe into a long-axis configuration. The location of the superior closing meniscus is determined by the point at which the walls of the vein touch each other. Similar to the IVC, the jugular veins can also be examined during respiratory phases to view inspiratory collapse. Veins that are distended, with a closing meniscus level that is high in the course of the neck, suggest a higher CVP.¹⁶ Coupling this data with the evaluation of the IVC may give a better overall assessment of the effective intravascular volume.

“Leakiness of the tank”: FAST exam and pleural fluid assessment

Once a patient’s intravascular volume status has been determined, the next step in assessing the tank is to look for “abnormal leakiness of the tank.” Leakiness of the tank refers to 1 of 3 things leading to hemodynamic compromise: internal blood loss, fluid extravasation, or other pathologic fluid collections. In traumatic conditions, the clinician must quickly determine whether hemoperitoneum or hemothorax is present, as a result of a “hole in the tank,” leading to hypovolemic shock. In nontraumatic conditions, accumulation of excess fluid into the abdominal and chest cavities often signifies “tank overload,” with resultant pleural effusions and ascites that may build-up with failure of the heart, kidneys, and/or liver. However, many patients with intrathoracic or intra-abdominal fluid collections are actually intravascularly volume depleted, confusing the clinical picture. Focusing on “tank fullness” by assessment of IVC and jugular veins in conjunction with the aforementioned findings can be very helpful in elucidating these conditions. In infectious states, pneumonia may be accompanied by a complicating parapneumonic pleural effusion, and ascites may lead to spontaneous bacterial peritonitis. Depending on the clinical scenario, small fluid collections within the peritoneal cavity may also represent intra-abdominal abscesses leading to a sepsis picture.

The peritoneal cavity can be readily evaluated with bedside ultrasound for the presence of an abnormal fluid collection in both trauma and nontrauma states. This assessment is accomplished with the FAST exam. This examination consists of an inspection of the potential spaces in the right and left upper abdominal quadrants and in the pelvis. Specific views include the space between the liver and kidney (hepatorenal space or Morison pouch), the area around the spleen (perisplenic space), and the area around and behind the bladder (rectovesicular/rectovaginal space or pouch of Douglas). A dark or anechoic area in any of these 3 potential spaces represents free intraperitoneal fluid (**Fig. 11**). These 3 areas represent the most common places for free fluid to collect, and correspond to the most dependent areas of the peritoneal cavity in the supine patient. Because the FAST exam relies on free fluid settling into these dependent areas, the patient’s position should be taken into account while

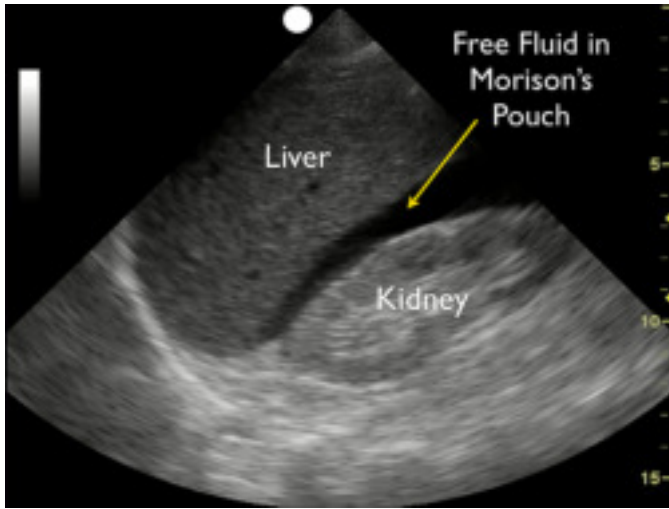


Fig. 11. Right upper quadrant/hepatorenal view: free fluid.

interpreting the examination. Trendelenburg positioning will cause fluid to shift to the upper abdominal regions, whereas an upright position will cause shift of fluid into the pelvis.

The FAST exam has been reported to detect intraperitoneal fluid collections as small as 100 mL, with a range of 250 to 620 mL commonly cited.^{58–60} How much fluid can be detected depends on the clinician's experience as well as the location of the free fluid, with the pelvic view best able to detect small quantities of fluid.⁶⁰ The overall sensitivity and specificity of the FAST exam have been reported to be approximately 79% and 99%, respectively.⁶¹

Ultrasound can also assist in evaluating the thoracic cavity for free fluid (pleural effusion or hemothorax) in an examination known as the extended FAST, or E-FAST. This evaluation is easily accomplished by including views of the thoracic cavity with the FAST examination. In both the hepatorenal and perisplenic views, the diaphragms appear as bright or hyperechoic lines immediately above, or cephalad to, the liver and spleen respectively. Aiming the probe above the diaphragm will allow for identification of a thoracic fluid collection. If fluid is found, movement of the probe 1 or 2 intercostal spaces cephalad provides a better view of the thoracic cavity, allowing quantification of the fluid present. In the normal supradiaphragmatic view, there are no dark areas of fluid in the thoracic cavity, and the lung can often be visualized as a moving structure. In the presence of an effusion or hemothorax, the normally visualized lung above the diaphragm is replaced with a dark, or anechoic, space. The lung may also be visualized floating within the pleural fluid (Fig. 12). Pleural effusions often exert compression on the lung, causing "hepatization," or an appearance of the lung in the effusion similar to a solid organ, like the liver. The literature supports the use of bedside ultrasound for the detection of pleural effusion and hemothorax. Several studies have found Emergency Department ultrasound to have a sensitivity in excess of 92% and a specificity approaching 100% in the detection of hemothorax.^{62–65} Assessing the patient with the head slightly elevated may improve the sensitivity of this examination, as this will cause intrathoracic fluid to accumulate just above the diaphragms.

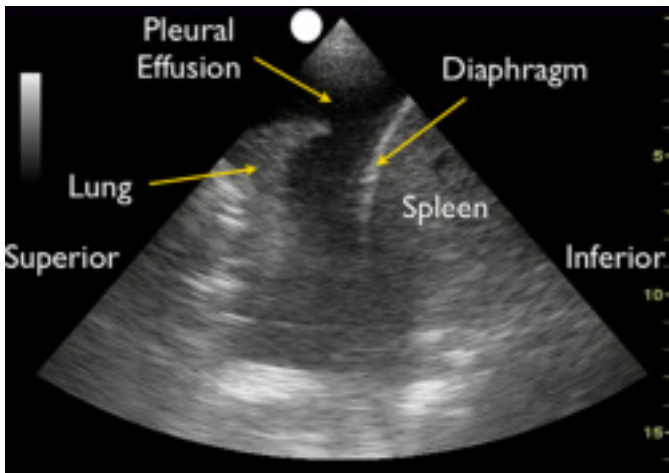


Fig. 12. Left upper quadrant: pleural effusion.

Free fluid in the peritoneal or thoracic cavities in a hypotensive patient in whom a history of trauma is present or suspected should initially be presumed to be blood, leading to a diagnosis of hemorrhagic shock. Although a history of trauma is commonly elicited in such cases, the trauma may be occult or minor, making diagnosis sometimes difficult. One circumstance of occult trauma is a delayed splenic rupture resulting from an enlarged and more fragile spleen, such as in a patient with infectious mononucleosis. Although rare, this entity may occur several days following a minor trauma, and may thus be easily overlooked by both patient and clinician.⁶⁶ Leakage of intestinal contents from rupture of a hollow viscus or urine extravasation from intraperitoneal bladder rupture may also demonstrate free intraperitoneal fluid.

Nontraumatic conditions may also lead to hemorrhagic shock, and must remain on the EP's differential diagnosis. Ruptured ectopic pregnancy and hemorrhagic corpus luteum cyst are 2 diagnoses that should not be overlooked in women of childbearing age. In an elderly patient, an abdominal aortic aneurysm may occasionally rupture into the peritoneal cavity and thoracic aneurysms may rupture into the chest cavity. Once the diagnosis of hemorrhagic shock is made, treatment should be directed toward transfusion of blood products and surgical or angiographic intervention.

In the nontrauma patient, ascites and pleural effusions will appear as dark, or anechoic, fluid collections, similar to blood. Parapneumonic inflammation may cause considerable pleural effusions and/or empyema. Differentiating blood from other fluids can be suggested from the history, clinical examination, and chest radiograph. There may occasionally be some signature sonographic findings that help make a diagnosis. In hemorrhagic conditions, blood often has a mixed appearance, with areas of both anechoic fresh blood and more echogenic blood clot present. In an infectious parapneumonic pleural effusion, gas bubbles may be seen within the fluid. In cases of uncertainty, a diagnostic thoracentesis or paracentesis (under ultrasound guidance) will most accurately evaluate the nature of the fluid.⁶⁷

"Tank compromise": pneumothorax

Although the exact mechanism by which tension pneumothorax causes shock is controversial, it has historically been thought to produce obstructive shock.^{17,18,68} According to this theory, severely increased intrathoracic pressure produces mediastinal

shift, which kinks and compresses the inferior and superior vena cava at their insertion into the right atrium, obstructing venous return to the heart. Regardless of the exact mechanism, detection is critical.

Although chest radiography reveals characteristic findings in tension pneumothorax, therapy should not be delayed while awaiting radiographic studies. With bedside ultrasound, the diagnosis of tension pneumothorax can be accomplished within seconds. Pneumothorax detection with ultrasound relies on the fact that free air (pneumothorax) is lighter than normal aerated lung tissue, and thus will accumulate in the nondependent areas of the thoracic cavity. Therefore, in a supine patient a pneumothorax will be found anteriorly, while in an upright patient a pneumothorax will be found superiorly at the lung apex.

Multiple studies have shown ultrasound to be more sensitive than supine chest radiography for the detection of pneumothorax.^{69–74} Sensitivities for these various studies ranged from 86% to 100%, with specificities ranging from 92% to 100%. A study by Zhang and colleagues⁷¹ that focused on trauma victims found the sensitivity of ultrasound for pneumothorax was 86% versus 27% for chest radiography; furthermore, this same study reported the average time to obtain ultrasound was 2.3 minutes versus 19.9 minutes for chest radiography.

To assess for pneumothorax with ultrasound, the patient should be supine. Position a high-frequency linear array or a phased-array transducer in the mid-clavicular line at approximately the third through fifth intercostal spaces to identify the pleural line. This line appears as an echogenic horizontal line located approximately half a centimeter deep to the ribs. The pleural line consists of both the visceral and parietal pleura closely apposed to one another. In the normal lung, the visceral and parietal pleura can be seen to slide against each other, with a glistening or shimmering appearance, as the patient breathes (**Fig. 13**). The presence of this lung sliding excludes a pneumothorax.⁷⁵ This lung sliding motion can be graphically depicted by using M-mode Doppler. A normal image will depict “waves on the beach,” with no motion of the chest wall anteriorly, represented as linear “waves,” and the motion of the lung posteriorly, representing “the beach” (**Fig. 14**). When a pneumothorax is present, air gathers between the parietal and visceral pleura, preventing the ultrasound beam from detecting lung sliding. In pneumothorax, the pleural line seen consists only of the parietal layer, seen as a stationary line. M-mode Doppler through the chest will show only repeating horizontal linear lines, demonstrating a lack of lung sliding or absence of the “beach” (see **Fig. 14**). Although the presence of lung sliding is sufficient to rule

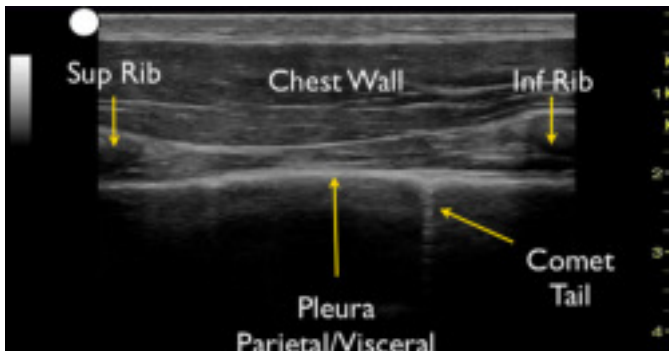


Fig. 13. Long-axis view: normal lung.

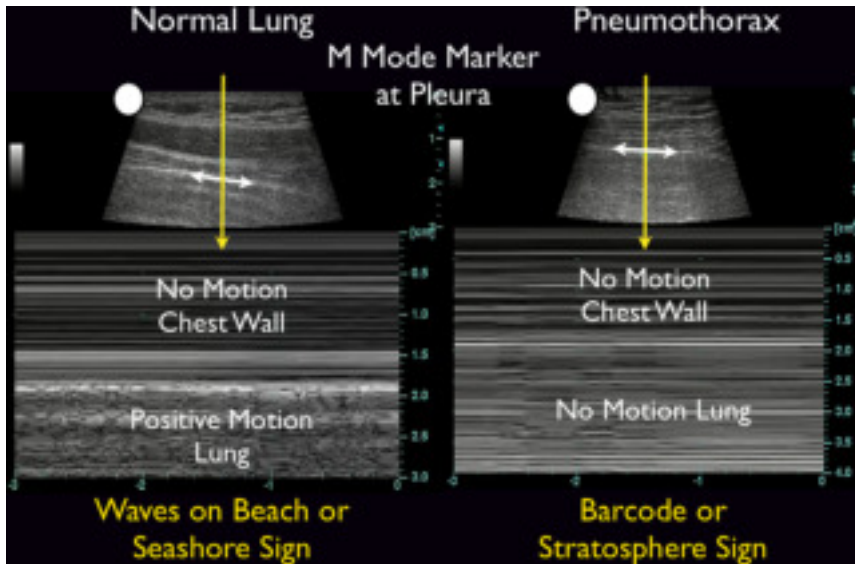


Fig. 14. M-mode: normal lung versus pneumothorax.

out pneumothorax, the absence of lung sliding may be seen in other conditions in addition to pneumothorax, such as a chronic obstructive pulmonary disease bleb, consolidated pneumonia, atelectasis, or mainstem intubation.⁷⁶⁻⁷⁸ Thus the absence of lung sliding, especially as defined in one intercostal space, is not by itself diagnostic of a pneumothorax. The clinician can examine through several more intercostal spaces, moving the transducer more inferiorly and lateral, to increase the utility of the test. This maneuver may also help identify the lung point, or the area where an incomplete pneumothorax interfaces with the chest wall, as visualized by the presence of lung sliding on one side and the lack of lung sliding on the other.⁷⁹

Another sonographic finding seen in normal lung, but absent in pneumothorax, is the comet tail artifact. Comet tail artifact is a form of reverberation echo that arises from irregularity of the lung surface. This phenomenon appears as a vertical echoic line originating from the pleural line and extending down into the lung tissue. The presence of comet tail artifact rules out a pneumothorax.⁸⁰ The combination of a lack of lung sliding and absent comet tail artifacts strongly suggests pneumothorax. In the setting of undifferentiated shock, the EP should strongly consider that a tension pneumothorax may be present, and immediate needle decompression followed by tube thoracostomy should be considered.

"Tank overload": pulmonary edema

Pulmonary edema often accompanies cardiogenic shock, in which weakened cardiac function causes a backup of blood into the pulmonary vasculature, leading to tank overload. Yet the clinical picture can be misleading, as patients in pulmonary edema may present with wheezing, rather than rales, or may have relatively clear lung sounds. The ability to quickly image the lung fields with ultrasound can rapidly lead the EP to the correct diagnosis. Although it is a relatively new concept, ultrasound has been shown to be helpful in the detection of pulmonary edema.⁸¹ The sonographic signs of pulmonary edema correlate well with chest radiography.⁸²

To assess for pulmonary edema with ultrasound, the lungs are scanned with the phased-array transducer in the anterolateral chest between the second and fifth rib interspaces. Detection of pulmonary edema with ultrasound relies on seeing a special subtype of comet tail artifact, called B lines (**Fig. 15**). These B lines appear as a series of diffuse, brightly echogenic lines originating from the pleural line and projecting in a fanlike pattern into the thorax (described as “lung rockets”). In contrast to the smaller comet tail artifacts seen in normal lung that fade out within a few centimeters of the pleural line, the B lines of pulmonary edema are better defined and extend to the far field of the ultrasound image. B lines result from thickening of the interlobular septa, as extravascular water accumulates within the pulmonary interstitium.^{81,83} The presence of B lines coupled with decreased cardiac contractility and a plethoric IVC on focused sonographic evaluation should prompt the clinician to consider the presence of pulmonary edema and initiate appropriate treatment.

RUSH Protocol: Step 3—Evaluation of the Pipes

“Rupture of the pipes”: aortic aneurysm and dissection

The next step in the RUSH exam is to examine the ‘Pipes’ looking first at arterial side of circulatory system and then at the venous side (**Fig. 16**). Vascular catastrophes, such as ruptured abdominal aortic aneurysms (AAA) and aortic dissections, are life-threatening causes of hypotension. The survival of such patients may often be measured in minutes, and the ability to quickly diagnose these diseases is crucial.

A ruptured AAA is classically depicted as presenting with back pain, hypotension, and a pulsatile abdominal mass. However, fewer than half of cases occur with this triad, and some cases will present with shock as the only finding.⁸⁴ A large or rupturing AAA can also mimic a kidney stone, with flank pain and hematuria. Fortunately for the EP, ultrasound can be used to rapidly diagnose both conditions.⁸⁵ Numerous studies have shown that EPs can make the diagnosis of AAA using bedside ultrasound, with a high sensitivity and specificity.^{86–89} The sensitivity of EP-performed ultrasound for the detection of AAA ranges from 93% to 100%, with specificities approaching 100%.^{86–88}

A complete ultrasound examination of the abdominal aorta involves imaging from the epigastrium down to the iliac bifurcation using a phased-array or curvilinear

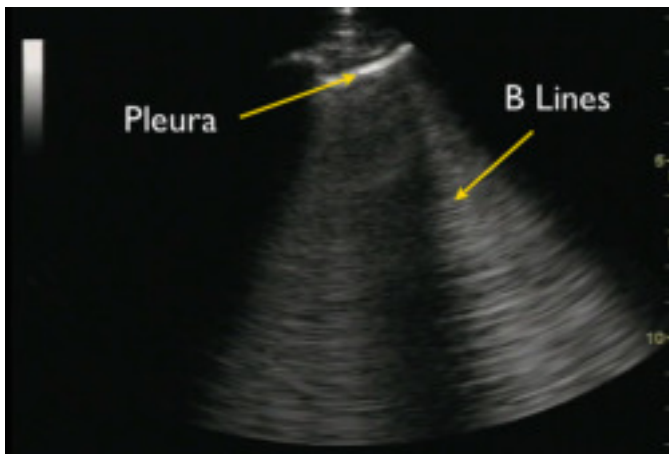


Fig. 15. Lung ultrasound: edema with B lines.

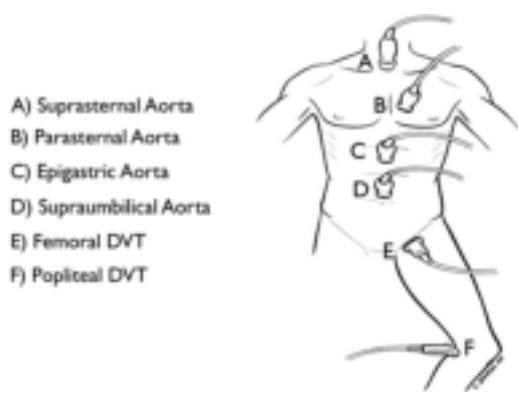


Fig. 16. RUSH step 3. Evaluation of the pipes.

transducer. Aiming the transducer posteriorly in a transverse orientation in the epigastric area, the abdominal aorta can be visualized as a circular vessel seen immediately anterior to the vertebral body and to the left of the paired IVC. Application of steady pressure to the transducer to displace bowel gas, while sliding the probe inferiorly from a position just below the xiphoid process down to the umbilicus, allows for visualization of the entire abdominal aorta. The aorta should also be imaged in the longitudinal orientation for completion. Measurements should be obtained in the short axis, measuring the maximal diameter of the aorta from outer wall to outer wall, and should include any thrombus present in the vessel. A measurement of greater than 3 cm is abnormal and defines an abdominal aortic aneurysm (**Fig. 17**). Smaller aneurysms may be symptomatic, although rupture is more common with aneurysms measuring larger than 5 cm.⁹⁰ Studies have also confirmed that the EP can reliably make a correct determination of the size of an AAA.^{87,91}

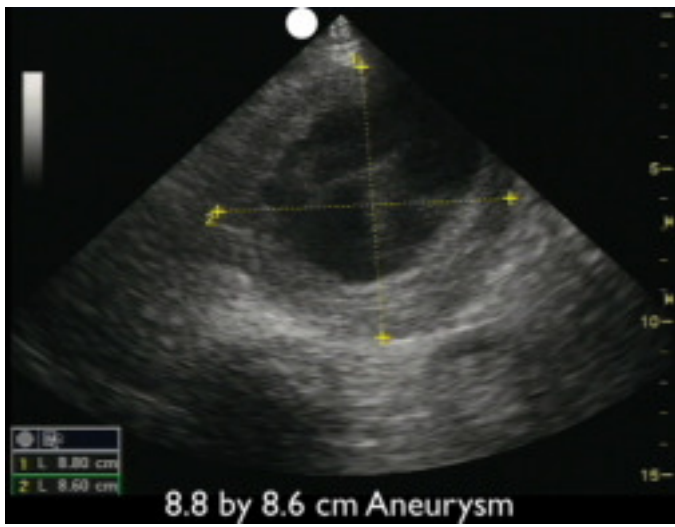


Fig. 17. Short-axis view: large abdominal aortic aneurysm.

Identifying the abdominal aorta along its entire course is essential to rule out an aneurysm, paying special attention below the renal arteries where most AAAs are located. Rupture of an abdominal aortic aneurysm typically occurs into the retroperitoneal space, which unfortunately is an area difficult to visualize with ultrasound. In a stable patient, a CT scan with intravenous contrast can be ordered to investigate leakage of an aneurysm. However, a hypotensive patient with sonographic evidence of an AAA should be considered to have acute rupture, and a surgeon should be consulted with plans for immediate transport to the operating room.

Another crucial part of “the pipes” protocol is evaluation for an aortic dissection. The sensitivity of transthoracic echocardiography to detect aortic dissection is poor (approximately 65% according to one study), and is limited compared with CT, MRI, or transesophageal echocardiography.⁹² Despite this, EP-performed bedside ultrasound has been used to detect aortic dissections and has helped many patients.^{93–95} Sonographic findings suggestive of the diagnosis include the presence of aortic root dilation and an aortic intimal flap. The parasternal long-axis view of the heart permits an evaluation of the proximal aortic root, and a measurement of more than 3.8 cm is considered abnormal. An echogenic intimal flap may be recognized within the dilated root or anywhere along the course of the thoracic or abdominal aorta (**Fig. 18**). The suprasternal view allows imaging of the aortic arch and should be performed in high-suspicion scenarios by placing the phased-array transducer within the suprasternal notch and aiming caudally and anteriorly (**Fig. 19**). Color flow imaging can further delineate 2 lumens with distinct blood flow, confirming the diagnosis. In patients with acute proximal dissection, aortic regurgitation or a pericardial effusion may also be recognized. Abdominal aortic ultrasound may reveal a distal thoracic aortic dissection that extends below the diaphragm, and in the hands of skilled sonographers has been shown to be 98% sensitive.⁹⁶

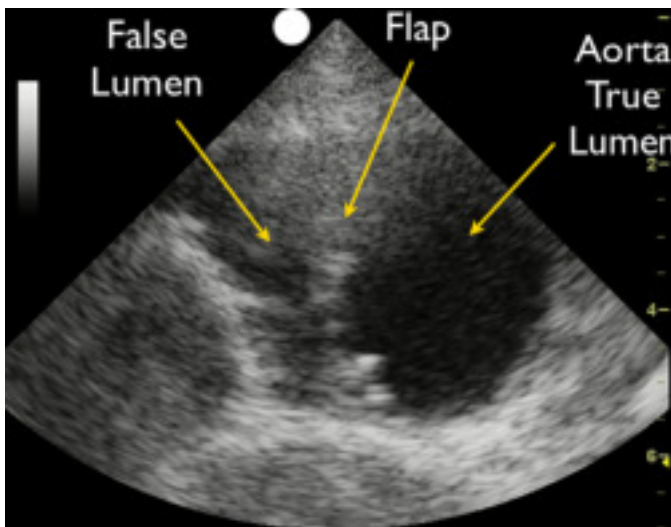


Fig. 18. Short-axis view: aortic dissection.

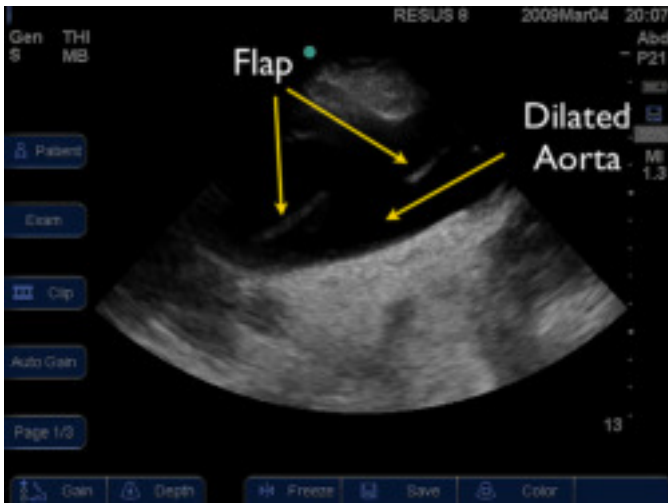


Fig. 19. Suprasternal view: aortic dissection.

“Clogging of the pipes”: venous thromboembolism

Bedside ultrasound for DVT In the patient in whom a thromboembolic event is suspected as a cause of shock, the EP should then move to an assessment of the venous side of “the pipes.” As the majority of pulmonary emboli originate from lower extremity DVT, the examination is concentrated on a limited compression evaluation of the leg veins. Simple compression ultrasonography, which uses a high frequency linear probe to apply direct pressure to the vein, has a good overall sensitivity for detection of DVT of the leg.⁹⁷ An acute blood clot forms a mass in the lumen of the vein, and the pathognomonic finding of DVT will be incomplete compression of the anterior and posterior walls of the vein (**Fig. 20**).^{98,99} In contrast, a normal vein will completely collapse with simple compression. Most distal deep venous thromboses can be detected through

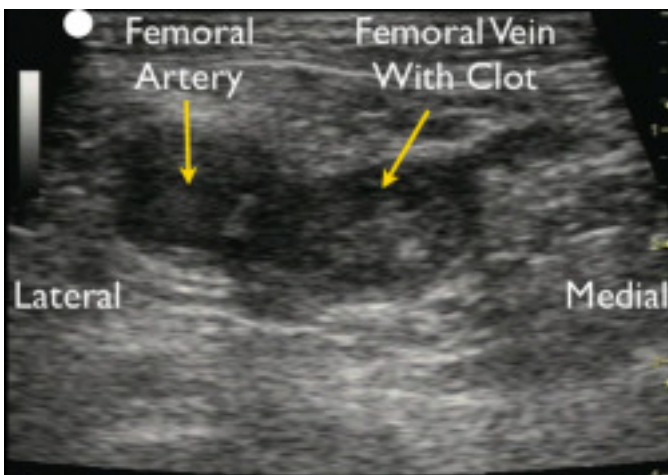


Fig. 20. Femoral vein deep venous thrombosis with fresh clot.

simple compression ultrasonography of the leg using standard B-mode imaging, and more complicated Doppler techniques add little utility to the examination.¹⁰⁰

Ultrasound may miss some clots that have formed in the calf veins, a difficult area to evaluate with sonography.¹⁰¹ However, most proximal DVTs can be detected by a limited compression examination of the leg that can be rapidly performed by focusing on 2 major areas.^{102,103} The proximal femoral vein just below the inguinal ligament is evaluated first, beginning at the common femoral vein, found below the inguinal ligament. Scanning should continue down the vein through the confluence with the saphenous vein to the bifurcation of the vessel into the deep and superficial femoral veins. The second area of evaluation is the popliteal fossa. The popliteal vein, the continuation of the superficial femoral vein, can be examined from high in the popliteal fossa down to trifurcation into the calf veins. If an upper extremity thrombus is clinically suspected, the same compression techniques can be employed, following the arm veins up to the axillary vein and into the subclavian vein. Although a good initial test, the sensitivity of ultrasound for proximal upper extremity clots is lower than for lower extremity clots, as the subclavian vein cannot be fully compressed behind the clavicle.¹⁰⁴ Although clinically less common, an internal jugular vein thrombosis that may form in a patient with a previous central line can also be well seen with ultrasound.

Previous studies have shown that EPs can perform limited ultrasound compression for lower extremity venous clots with good sensitivity in patients with a high pretest probability for the disease.^{105–108} The examination can also be performed rapidly, and can be integrated into the overall RUSH protocol with a minimum of added time.¹⁰⁹

SUMMARY

Bedside ultrasound technology has evolved to the point that it offers a powerful, yet easy to use tool for the clinician faced with a critical patient. The initial imaging focus of ultrasound as used by Radiologists was on anatomy and pathology. Now with clinicians actively using this technology at the bedside, attention has shifted to the crucial evaluation of *physiology*. The ability to recognize both abnormal pathology and physiology in a critical patient, recognize a distinctive shock state, and arrive at a more precise diagnosis represents a new paradigm in resuscitation care. Clinicians around the world are recognizing the power of point of care ultrasound and the impact it will have on critical care resuscitation in the Emergency Department, as well as in Intensive Care Units.

The individual components of the *Rapid Ultrasound in SHock* protocol have been studied and published prior, but this new protocol represents the first synthesis of these sonographic techniques into a unified 3 step algorithm. The protocol simplifies the ultrasound evaluation into the physiological paradigm of “pump, tank, and pipes,” allowing the clinician to easily remember the critical aspects of the exam components. Although described in a sequential 3-step approach, clinicians are expected to alter the components and sequence of sonographic techniques based on the clinical scenario presented. Unlike previous published studies that have examined ultrasound protocols in the hypotensive patient, the described RUSH exam presents the most detailed shock algorithm for use by EPs to date.¹¹⁰ By focusing on both the anatomy and the physiology of these complex patients, in shock, bedside ultrasound provides the opportunity for improved clinical treatments and patient outcomes.

For educational ultrasound videos covering all RUSH applications, please go to <http://www.sound-bytes.tv> where a downloadable pocket card on RUSH is also available.

ACKNOWLEDGMENTS

We would like to acknowledge the work of Scott Weingart, MD and Brett Nelson, MD on ultrasound evaluation of the hypotensive patient.

REFERENCES

1. Jones AE, Tayal VS, Sullivan DM, et al. Randomized, controlled trial of immediate versus delayed goal directed ultrasound to identify the cause of nontraumatic hypotension in emergency department patients. *Crit Care Med* 2004;32:1703–8.
2. Jones AE, Aborn LS, Kline JA. Severity of emergency department hypotension predicts adverse hospital outcome. *Shock* 2004;22:410–4.
3. Thomas HA, Beeson MS, Binder LS, et al. The 2005 model of the clinical practice of emergency medicine: the 2007 update. *Acad Emerg Med* 2008;15(8):776–9.
4. Heller MB, Mandavia D, Tayal VS. Residency training in emergency ultrasound: fulfilling the mandate. *Acad Emerg Med* 2002;9:835–9.
5. ACEP emergency ultrasound guidelines. *Ann Emerg Med* 2009;14:550–70.
6. Pershad J, Myers S, Plouman C, et al. Bedside limited echocardiography by the emergency physician is accurate during evaluation of the critically ill patient. *Pediatrics* 2004;114:e667–71.
7. Plummer D, Heegaard W, Dries D, et al. Ultrasound in HEMS: its role in differentiating shock states. *Air Med J* 2003;22:33–6.
8. Kline JA. Shock. In: Rosen P, Marx J, editors. *Emergency medicine: concepts and clinical practice*. 5th edition. St Louis (MO): Mosby; 2002. p. 33–47.
9. Shah MR, Hasselblad V, Stevenson LW, et al. Impact of the pulmonary artery catheter in critically ill patients: meta-analysis of randomized clinical trials. *JAMA* 2005;294:1664–70.
10. Joseph M, Disney P. Transthoracic echocardiography to identify or exclude cardiac cause of shock. *Chest* 2004;126:1592–7.
11. Bealieu Y. Specific skill set and goals of focused echocardiography for critical care physicians. *Crit Care Med* 2007;35:S144–9.
12. Viellard-Baron A, Page B, Augarde R, et al. Acute cor pulmonale in massive pulmonary embolism: incidence, echocardiography pattern, clinical implications and recovery rate. *Intensive Care Med* 2001;27:1481–6.
13. Grifoni S, Olivotto I, Cecchini P, et al. Utility of an integrated clinical, echocardiographic and venous ultrasound approach for triage of patients with suspected pulmonary embolism. *Am J Cardiol* 1998;82:1230–5.
14. Jardin F, Veillard-Baron A. Ultrasonographic examination of the vena cavae. *Intensive Care Med* 2006;32:203–6.
15. Randazzo MR, Snoey ER, Levi MA, et al. Accuracy of emergency physician assessment of left ventricular ejection fraction and central venous pressure using echocardiography. *Acad Emerg Med* 2003;10:973–7.
16. Jang T, Aubin C, Naunheim R, et al. Ultrasonography of the internal jugular vein in patients with dyspnea without jugular venous distention on physical examination. *Ann Emerg Med* 2004;44:160–8.
17. Connolly JP. Hemodynamic measurements during a tension pneumothorax. *Crit Care Med* 1993;21:294–6.
18. Carvalho P, Hilderbrandt J, Charan NB. Changes in bronchial and pulmonary arterial blood flow with progressive tension pneumothorax. *J Appl Physiol* 1996;81:1664–9.

19. Lensing AW, Prandoni P, Brandjes D, et al. Detection of deep vein thrombosis by real time B-mode ultrasonography. *N Engl J Med* 1989;320:342–5.
20. Birdwell BG, Raskob GE, Whitsett TL, et al. The clinical validity of normal compression ultrasonography in outpatients suspected of having deep venous thrombosis. *Ann Intern Med* 1998;128:1–7.
21. Blaivas M. Incidence of pericardial effusions in patients presenting to the emergency department with unexplained dyspnea. *Acad Emerg Med* 2001;8:1143–6.
22. Shabetai R. Pericardial effusions: haemodynamic spectrum. *Heart* 2004;90:255–6.
23. Spodick DH. Acute cardiac tamponade. *N Engl J Med* 2003;349:684–90.
24. Trojanos CA, Porembka DT. Assessment of left ventricular function and hemodynamics with transesophageal echocardiography. *Crit Care Clin* 1996;12:253–72.
25. Russo AM, O'Connor WH, Waxman HL. Atypical presentations and echocardiographic findings in patients with cardiac tamponade occurring early and late after cardiac surgery. *Chest* 1993;104:71–8.
26. Poelaert J, Schmidt C, Colardyn F. Transesophageal echocardiography in the critically ill patient. *Anaesthesia* 1998;53:55–68.
27. Mandavia DP, Hoffner RJ, Mahaney K, et al. Bedside echocardiography by emergency physicians. *Ann Emerg Med* 2001;38:377–82.
28. Tayal VS, Kline JA. Emergency echocardiography to determine pericardial effusions in patients with PEA and near PEA states. *Resuscitation* 2003;59:315–8.
29. Merce J, Sagrista SJ. Correlation between clinical and Doppler echocardiographic findings in patients with moderate and large pericardial effusions. *Am Heart J* 1999;138:759–64.
30. Tsang T, Enriquez-Sarano M, Freeman WK. Consecutive 1127 therapeutic echocardiographically guided pericardiocenteses: clinical profile, practice patterns and outcomes spanning 21 years. *Mayo Clin Proc* 2002;77:429–36.
31. Amico AF, Lichtenberg GS, Reisner SA, et al. Superiority of visual versus computerized echocardiographic estimation of radionuclide left ventricular ejection fraction. *Am Heart J* 1989;118:1259–65.
32. Moore CL, Rose GA, Tayal VS, et al. Determination of left ventricular function by emergency physician echocardiography of hypotensive patients. *Acad Emerg Med* 2002;9:186–93.
33. Reynolds HR, Hochman JS. Cardiogenic shock: current concepts and improving outcomes. *Circulation* 2008;117:686–97.
34. Picard MH, Davidoff R, Sleeper LA. Echocardiographic predictors of survival and response to early revascularization in cardiogenic shock. *Circulation* 2003;107:279–84.
35. Jones AE, Craddock PA, Tayal VS, et al. Diagnostic accuracy of identification of left ventricular function among emergency department patients with nontraumatic symptomatic undifferentiated hypotension. *Shock* 2005;24:513–7.
36. Parker M, Shelhamer J, Baruch S, et al. Profound but reversible myocardial depression in patients with septic shock. *Ann Intern Med* 1984;100:483–90.
37. Rivers E, Nguyen B, Haystad S, et al. Early goal directed therapy in the treatment of severe sepsis and septic shock. *N Engl J Med* 2001;345:1368–77.
38. Blaivas M, Fox JC. Outcome in cardiac arrest patients found to have cardiac standstill on bedside emergency department echocardiogram. *Acad Emerg Med* 2001;8:616–21.

39. Salen P, Melniker L, Choolijan C, et al. Does the presence or absence of sonographically identified cardiac activity predict resuscitation outcomes of cardiac arrest patients? *Am J Emerg Med* 2005;23:459–62.
40. Nazeyrollas D, Metz D, Jolly D, et al. Use of transthoracic Doppler echocardiography combined with clinical and electrographic data to predict acute pulmonary embolism. *Eur Heart J* 1996;17:779–86.
41. Jardin F, Duborg O, Bourdarias JP. Echocardiographic pattern of acute cor pulmonale. *Chest* 1997;111:209–17.
42. Jardin F, Dubourg O, Gueret P, et al. Quantitative two dimensional echocardiography in massive pulmonary embolism: emphasis on ventricular interdependence and leftward septal displacement. *J Am Coll Cardiol* 1987;10:1201–6.
43. Madan A, Schwartz C. Echocardiographic visualization of acute pulmonary embolus and thrombolysis in the ED. *Am J Emerg Med* 2004;22:294–300.
44. Stein J. Opinions regarding the diagnosis and management of venous thromboembolic disease. ACCP Consensus Committee on pulmonary embolism. *Chest* 1996;109:233–7.
45. Jackson RE, Rudoni RR, Hauser AM, et al. Prospective evaluation of two dimensional transthoracic echocardiography in emergency department patients with suspected pulmonary embolism. *Acad Emerg Med* 2000;7:994–8.
46. Rudoni R, Jackson R. Use of two-dimensional echocardiography for the diagnosis of pulmonary embolus. *J Emerg Med* 1998;16:5–8.
47. Miniati M, Monti S, Pratali L, et al. Value of transthoracic echocardiography in the diagnosis of pulmonary embolism: results of a prospective study in unselected patients. *Am J Med* 2001;110(7):528–35.
48. Gifroni S, Olivotto I, Cecchini P, et al. Short term clinical outcome of patients with acute pulmonary embolism, normal blood pressure and echocardiographic right ventricular dysfunction. *Circulation* 2000;101:2817–22.
49. Becattini C, Agnelli G. Acute pulmonary embolism: risk stratification in the emergency department. *Intern Emerg Med* 2007;2:119–29.
50. Frazee BW, Snoey ER. Diagnostic role of ED ultrasound in deep venous thrombosis and pulmonary embolism. *Am J Emerg Med* 1999;17:271–8.
51. Konstantinides S, Geibel A, Heusel G, et al. Heparin plus alteplase compared with heparin alone in patients with submassive pulmonary embolus. *N Engl J Med* 2002;347:1143–50.
52. Kucher N, Goldhaber SZ. Management of massive pulmonary embolism. *Circulation* 2005;112:e28–32.
53. Kircher B, Himelman R. Noninvasive estimation of right atrial pressure from the inspiratory collapse of the inferior vena cava. *Am J Cardiol* 1990;66:493–6.
54. Simonson JS, Schiller NB. Sonospirometry: a new method for noninvasive measurement of mean right atrial pressure based on two dimensional echocardiographic measurements of the inferior vena cava during measured inspiration. *J Am Coll Cardiol* 1988;11:557–64.
55. Barbier C, Loubieres Y, Schmit C, et al. Respiratory changes in inferior vena cava diameter are helpful in predicting fluid responsiveness in ventilated septic patients. *Intensive Care Med* 2004;30:1704–6.
56. Lyon M, Blaivas M, Brannam L. Sonographic measurement of the inferior vena cava as a marker of blood loss. *Am J Emerg Med* 2005;23:45–50.
57. Feissel M, Michard F. The respiratory variation in inferior vena cava diameter as a guide to fluid therapy. *Intensive Care Med* 2004;30:1834–7.

58. Gracias VH, Frankel HL, Gupta R, et al. Defining the learning curve for the Focused Abdominal Sonogram for Trauma (FAST) examination: implications for credentialing. *Am Surg* 2001;67:364–8.
59. Branney SW, Wolfe RE, Moore EE, et al. Quantitative sensitivity of ultrasound in detecting free intraperitoneal fluid. *J Trauma* 1995;39:375–80.
60. Von Kuenssberg Jehle D, Stiller G, Wagner D. Sensitivity in detecting free intraperitoneal fluid with the pelvic views of the FAST exam. *Am J Emerg Med* 2003; 21:476–8.
61. Stengel D, Bauwens K, Rademacher G, et al. Association between compliance with methodological standards of diagnostic research and reported test accuracy: meta-analysis of focused assessment of US for trauma. *Radiology* 2005;236: 102–11.
62. Sisley AC, Rozycki GS, Ballard RB, et al. Rapid detection of traumatic effusion using surgeon-performed ultrasonography. *J Trauma* 1998;44:291–6.
63. Ma OJ, Mateer JR. Trauma ultrasound examination versus chest radiography in the detection of hemothorax. *Ann Emerg Med* 1997;29:312–5.
64. Brooks A, Davies B, Smethhurst M, et al. Emergency ultrasound in the acute assessment of haemothorax. *Emerg Med J* 2004;21:44–6.
65. McEwan K, Thompson P. Ultrasound to detect haemothorax after chest injury. *Emerg Med J* 2007;24:581–2.
66. Gamblin TC, Wall CE Jr, Rover GM, et al. Delayed splenic rupture: case reports and review of the literature. *J Trauma* 2005;59:1231–4.
67. Blaivas M. Emergency department paracentesis to determine intraperitoneal fluid identity discovered on bedside ultrasound of unstable patients. *J Emerg Med* 2005;29:461–5.
68. Subotich D, Mandarich D. Accidentally created tension pneumothorax in patient with primary spontaneous pneumothorax—confirmation of the experimental studies, putting into question the classical explanation. *Med Hypotheses* 2005;64:170–3.
69. Soldati G, Testa A, Sher S, et al. Occult traumatic pneumothorax: diagnostic accuracy of lung ultrasonography in the emergency department. *Chest* 2008; 133:204–11.
70. Sartori S, Tombesi P, Trevisani L, et al. Accuracy of transthoracic sonography in detection of pneumothorax after sonographically guided lung biopsy: prospective comparison with chest radiography. *Am J Roentgenol* 2007;188:37–41.
71. Zhang M, Liu ZH, Yang JX, et al. Rapid detection of pneumothorax by ultrasonography in patients with multiple trauma. *Crit Care* 2006;10:R112.
72. Garofalo G, Busso M, Perotto F, et al. Ultrasound diagnosis of pneumothorax. *Radiol Med* 2006;11:516–25.
73. Blaivas M, Lyon M, Duggal S. A prospective comparison of supine chest radiography and bedside ultrasound for the diagnosis of traumatic pneumothorax. *Acad Emerg Med* 2005;12:844–9.
74. Knudtson JL, Dort JM, Helmer SD, et al. Surgeon-performed ultrasound for pneumothorax in the trauma suite. *J Trauma* 2004;56:527–30.
75. Lichtenstein DA, Menu Y. A bedside ultrasound sign ruling out pneumothorax in the critically ill. Lung sliding. *Chest* 1995;108:1345–8.
76. Slater A, Goodwin M, Anderson KE, et al. COPD can mimic the appearance of pneumothorax on thoracic ultrasound. *Chest* 2006;129:545–50.
77. Lichtenstein DA, Meziere GA. Relevance of lung ultrasound in the diagnosis of acute respiratory failure: the BLUE protocol. *Chest* 2008;134:117–25.

78. Blaivas M, Tsung JW. Point-of-care sonographic detection of left endobronchial main stem intubation and obstruction versus endotracheal intubation. *J Ultrasound Med* 2008;27:785–9.
79. Lichtenstein D, Meziere G, Biderman P, et al. The “lung point”: an ultrasound sign specific to pneumothorax. *Intensive Care Med* 2000;26:1434–40.
80. Lichtenstein D, Meziere G, Biderman P, et al. The comet-tail artifact: an ultrasound sign ruling out pneumothorax. *Intensive Care Med* 1999;25:383–8.
81. Lichtenstein D, Meziere G, Biderman P, et al. The comet-tail artifact. An ultrasound sign of alveolar-interstitial syndrome. *Am J Respir Crit Care Med* 1997;156:1640–6.
82. Agricola E, Bove T, Oppizzi M, et al. “Ultrasound comet-tail images”: a marker of pulmonary edema: a comparative study with wedge pressure and extravascular lung water. *Chest* 2005;127:1690–5.
83. Soldati G, Copetti R, Sher S. Sonographic interstitial syndrome: the sound of lung water. *J Ultrasound Med* 2009;28:163–74.
84. Rohrer MJ, Cutler BS, Wheeler HB. Long-term survival and quality of life following ruptured abdominal aneurysm. *Arch Surg* 1988;123:1213–7.
85. Hendrickson RG, Dean AJ, Costantino TG. A novel use of ultrasound in pulseless electrical activity: the diagnosis of an acute abdominal aortic aneurysm rupture. *J Emerg Med* 2001;21:141–4.
86. Dent B, Kendall RJ, Boyle AA, et al. Emergency ultrasound of the abdominal aorta by UK emergency physicians: a prospective cohort study. *Emerg Med J* 2007;24:547–9.
87. Costantino TG, Bruno EC, Handly N, et al. Accuracy of emergency medicine ultrasound in the evaluation of abdominal aortic aneurysm. *J Emerg Med* 2005;29:455–60.
88. Tayal VS, Graf CD, Gibbs MA. Prospective study of accuracy and outcome of emergency ultrasound for abdominal aortic aneurysm over two years. *Acad Emerg Med* 2003;10:867–71.
89. Kuhn M, Bonnin RL, Davey MJ, et al. Emergency department ultrasound scanning for abdominal aortic aneurysm: accessible, accurate, and advantageous. *Ann Emerg Med* 2000;36:219–23.
90. Nevitt MP, Ballard DJ, Hallett JW Jr. Prognosis of abdominal aortic aneurysms. A population-based study. *N Engl J Med* 1989;321:1009–14.
91. Moore CL, Holliday RS, Hwang JQ, et al. Screening for abdominal aortic aneurysm in asymptomatic at-risk patients using emergency ultrasound. *Am J Emerg Med* 2008;26:883–7.
92. Kodolitsch Y, Krause N, Spielmann R, et al. Diagnostic potential of combined transthoracic echocardiography and x-ray computed tomography in suspected aortic dissection. *Clin Cardiol* 1999;22:345–52.
93. Budhram G, Reardon R. Diagnosis of ascending aortic dissection using emergency department bedside echocardiogram. *Acad Emerg Med* 2008;15:584.
94. Fojtik JP, Costantino TG, Dean AJ. The diagnosis of aortic dissection by emergency medicine ultrasound. *J Emerg Med* 2007;32:191–6.
95. Blaivas M, Sierzenski PR. Dissection of the proximal thoracic aorta: a new ultrasonographic sign in the subxiphoid view. *Am J Emerg Med* 2002;20:344–8.
96. Clevert DA, Rupp N, Reiser M, et al. Improved diagnosis of vascular dissection by ultrasound B-flow: a comparison with color-coded Doppler and power Doppler sonography. *Eur Radiol* 2005;15:342–7.

97. Kearon CK, Julian JA, Math M, et al. Noninvasive diagnosis of deep venous thrombosis. *Ann Intern Med* 1998;128:663–77.
98. Pezullo JA, Perkins AB, Cronan JJ. Symptomatic deep vein thrombosis: diagnosis with limited compression US. *Radiology* 1996;198:67–70.
99. Blaivas M. Ultrasound in the detection of venous thromboembolism. *Crit Care Med* 2007;35(5 suppl):S224–34.
100. Jolly BT, Massarin E, Pigman EC. Color Doppler ultrasound by emergency physicians for the diagnosis of acute deep venous thrombosis. *Acad Emerg Med* 1997;4:129–32.
101. Eskandari MK, Sugimoto H, Richardson T, et al. Is color flow duplex a good diagnostic test for detection of isolated calf vein thrombosis in high risk patients? *Angiology* 2000;51:705–10.
102. Poppiti R, Papinocolau G, Perese S. Limited B-mode venous scanning versus complete color flow duplex venous scanning for detection of proximal deep venous thrombosis. *J Vasc Surg* 1995;22:553–7.
103. Bernardi E, Camporese G, Buller H, et al. Serial 2 point ultrasonography plus d-dimer vs. whole leg color coded Doppler ultrasonography for diagnosing suspected symptomatic deep vein thrombosis. *JAMA* 2008;300:1653–9.
104. Baarslag HJ, Van Beek EJ, Koopman MM. Prospective study of color duplex ultrasonography compared with contrast venography in patients suspected of having deep venous thrombosis of the upper extremities. *J Intern Med* 2002; 136:865–72.
105. Frazee BW, Snoey ER, Levitt A. Emergency department compression ultrasound to diagnose proximal deep vein thrombosis. *J Emerg Med* 2001;20:107–11.
106. Jang T, Docherty M, Aubin S, et al. Resident performed compression ultrasonography for the detection of proximal deep vein thrombosis: fast and accurate. *Acad Emerg Med* 2004;11:319–22.
107. Burnside PR, Brown MD, Kline JA. Systematic review of emergency physician-performed ultrasonography for lower-extremity deep vein thrombosis. *Acad Emerg Med* 2008;15:493–8.
108. Kline JA, O'Malley PM, Tayal VS, et al. Emergency clinician-performed compression ultrasonography for deep venous thrombosis of the lower extremity. *Ann Emerg Med* 2008;52:437–45.
109. Blaivas M, Lambert MJ, Harwood RA, et al. Lower extremity Doppler for deep venous thrombosis: can emergency physicians be accurate and fast? *Acad Emerg Med* 2000;7:120–6.
110. Rose JS, Bair AE, Mandavia DP. The UHP ultrasound protocol: a novel ultrasound approach to the empiric evaluation of the undifferentiated hypotensive patient. *Am J Emerg Med* 2001;19:299–302.