

Bedside ultrasonography: Applications in critical care: Part I

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Abstract

There is increasing interest in the use of ultrasound to assess and guide the management of critically ill patients. The ability to carry out quick examinations by the bedside to answer specific clinical queries as well as repeatability are clear advantages in an acute care setting. In addition, delays associated with transfer of patients out of the Intensive Care Unit (ICU) and exposure to ionizing radiation may also be avoided. Ultrasonographic imaging looks set to evolve and complement clinical examination of acutely ill patients, offering quick answers by the bedside. In this two-part narrative review, we describe the applications of ultrasonography with a special focus on the management of the critically ill. Part I explores the utility of echocardiography in the ICU, with emphasis on its usefulness in the management of hemodynamically unstable patients. We also discuss lung ultrasonography - a vastly underutilized technology for several years, until intensivists began to realize its usefulness, and obvious advantages over chest radiography. Ultrasonography is rapidly emerging as an important tool in the hands of intensive care physicians.

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Introduction

Point of care ultrasonography is emerging as an important bedside tool to enable expeditious decision making in critically ill patients. Ultrasonography has spread beyond the realms of conventional radiology to assist intensive care physicians answer specific clinical questions in day-to-day practice. Although computerized tomography remains the gold standard imaging tool for many disease processes, the hazards of transferring a sick patient out of the Intensive Care Unit (ICU) and the delays involved have prompted ICU physicians to seek innovative ways to use ultrasound, such as in lung imaging. Besides, ultrasound guidance has enhanced the safety of invasive interventions in the ICU, including insertion of vascular catheters and drainage of collections. Clinical examination is perhaps being increasingly complemented by the use of ultrasonography as an “extension of the examining

hand” to help find quick answers in the care of the rapidly deteriorating patient in the ICU. This two-part review focuses on the use of ultrasonography with emphasis on seeking crucial information by the bedside to optimize care in critically ill patients. In Part I, we describe the applications and utility of echocardiography and chest ultrasonography in the ICU setting.

Echocardiography in the intensive care unit

It is being increasingly realized that point of care echocardiography carried out by intensive care physicians could be a valuable tool that helps with the diagnosis and hemodynamic management of critically ill patients.^[1-3] Referral to specialist, “third party providers” may entail delays and perhaps suffer from lack of adequate correlation with the clinical situation. A more detailed study, if required, may be performed by specialists in the echocardiography laboratory later on. Jensen *et al.* were able to obtain relevant hemodynamic parameters for optimizing care by following a protocolized approach using standard echocardiographic windows in 97% of critically ill patients (Focus Assessed Transthoracic Echo).^[4] A transthoracic examination is easier to learn and do,

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and preferred more often. Transesophageal studies may be required if transthoracic windows are poor, or for detailed evaluation of vegetations, thrombi, and prosthetic valves. This review focuses on transthoracic examination.

Basic echocardiographic views

Basic echocardiographic views are depicted in Figure 1. The apical views are obtained by placing the probe at the site of the apex beat with the probe marker directed towards the left axilla. A four chamber view including the atria, ventricles, the interventricular and the interatrial septa are seen by moving the probe along the intercostal space. The apical five chamber view includes the left ventricular out flow tract (LVOT) and the aortic valve. This view is obtained by gradually tilting the probe upwards from the apical four chamber position. The parasternal views are obtained by positioning the probe on the second or third intercostal space close to the left sternal border. The parasternal long axis (PLAX) view is obtained with the probe marker pointing towards the right shoulder. For the short axis view, the probe is rotated clockwise through about 90° from the PLAX position to point the probe marker towards the head. Tilting the patient to the left lateral position improves the view. In mechanically ventilated patients, the expanding lung may obscure views. If appropriate, reducing the inspiratory pressures and positive end expiratory pressure (PEEP) level for a brief period might improve visualization. If conventional views are unobtainable, the subcostal view may be an option. To acquire this view, the probe is placed to the right of the xiphoid notch with the probe marker pointing to the left hip. Pushing down with gentle pressure would enable the ultrasound beam to travel under the sternum and the rib cage to view the heart. In some patients, this may be the only accessible echo window.

Assessment of ventricular function

Assessment of global LV function is crucial in the hemodynamically unstable patient. "Eyeball" estimates of LV function by experienced observers may be comparable to quantitative measures such as ejection fraction (EF).^[5,6] Moreover, such quantitative estimates may be misleading in the presence of preload or afterload changes. For instance, in the vasodilated, septic patient, the EF may be overestimated due to unloading of the LV resulting from low systemic vascular resistance.^[7] Quantitative measurement of EF is commonly performed in the PLAX view by measuring the LV internal diameters in systole and diastole on M mode (Teicholz method). Poor LV function in the hypotensive patient is important to diagnose,

as this may necessitate inotrope administration once fluid resuscitation is optimized. Echocardiographic assessment of LV function may compare favorably with cardiac output measurements using a pulmonary artery catheter.^[8] It must be emphasized that frequent echocardiographic studies are required to assess the effect of therapeutic interventions.

The size of the right ventricle (RV) may be assessed by measurement of the area from the apical four chamber view. The ratio of the RV: LV area is used to estimate RV dilatation. The normal ratio is <0.6; dilatation is considered moderate if it is 0.6-1.0 and critical if more than 1.0.^[9] The RV function may be assessed visually, as with the LV. The interventricular septum must be closely examined; in case of RV overload due to acute respiratory distress syndrome (ARDS), acute pulmonary embolism (PE) or high ventilation pressures, the septum shifts toward the left. In this situation, the LV loses its circular shape on the parasternal short axis view and assumes a "D" shape.^[10]

Pulmonary embolism (PE)

Acute PE may present with dramatic clinical deterioration; diagnosis is often difficult due to nonspecific clinical signs. Although contrast-enhanced Computerized tomography (CT) scan of the lungs has excellent sensitivity for PE, it may not be feasible to transfer an unstable patient to the radiology suite. Echocardiography is an excellent tool to diagnose massive PE by the bedside.^[11] The characteristic finding is RV dilatation [Figure 2] and dysfunction; however, this finding is not specific for acute PE. The pattern of RV dysfunction in PE is typically confined to the mid septum with apical sparing. This finding may help differentiate from other causes of RV dilatation and dysfunction.^[12] A

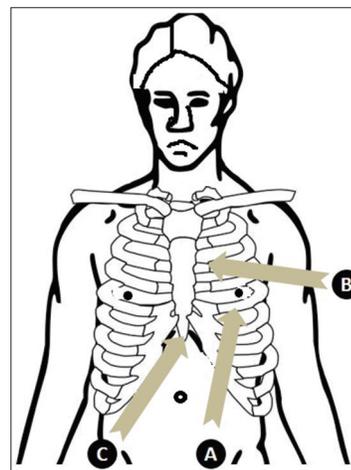


Figure 1: Probe location for basic echocardiographic views. (a) Apical, (b) parasternal, (c) subcostal

tricuspid insufficiency pressure gradient of 60 mm Hg or less is also suggestive of acute PE.^[13] Centrally located clots may be visible on transthoracic echocardiography; however, more distal emboli cannot be seen.^[14]

Pericardial effusion

Fluid accumulation in the pericardial space is seen as an echo-free space around the heart, and is easily diagnosed on transthoracic echocardiography [Figure 3]. If more conventional windows are not obtainable, the subcostal view usually offers a good alternative to visualize pericardial fluid. Cardiac tamponade is an immediately life-threatening emergency that presents with characteristic features on echocardiography. Diastolic collapse of the right atrium (RA) and RV is typically seen with tamponade; RA collapse occurs in early diastole, while RV collapse occurs later. Besides, RA collapse that lasts for more than one-third of the R-R interval suggests hemodynamically significant effusion. Although right-sided collapse is highly sensitive for tamponade, it is less specific, being present in 34% of patients without clinical evidence of tamponade in one study.^[15] Besides confirming the diagnosis, a hemodynamically significant pericardial effusion may be safely drained under real time echocardiographic guidance.^[16]

Assessment of hypovolemia

There is increasing awareness that overzealous fluid resuscitation and untitrated fluid challenges may lead to adverse outcomes.^[17] It is crucial to assess hypovolemia and “volume responsiveness” precisely and a more judicious approach to fluid resuscitation seems appropriate. Static indices of volume status, such as central venous and pulmonary capillary wedge pressures have been evaluated, but found to be imprecise tools.^[18,19] The inferior vena cava (IVC) size and its variability with respiration is easily assessed by the bedside. The IVC is viewed in the long axis from the epigastrium and measurements are performed on M mode [Figure 4]. An IVC diameter of 2 cm or more in a spontaneously breathing subject indicates high right sided pressures. With mechanical ventilation, the IVC diameter is usually larger than with spontaneous ventilation. In mechanically ventilated patients with septic shock, a with respiration variation of IVC diameter of more than 12% predicted an increase in cardiac output with volume loading.^[20] IVC diameter also correlated with central venous pressure (CVP), extravascular lung water index and intrathoracic blood volume index as measured by transpulmonary thermodilution technique in septic patients.^[21] Obliteration of the LV cavity in systole at the level of the papillary muscle on the PLAX view, commonly referred to as the “kissing sign”, is

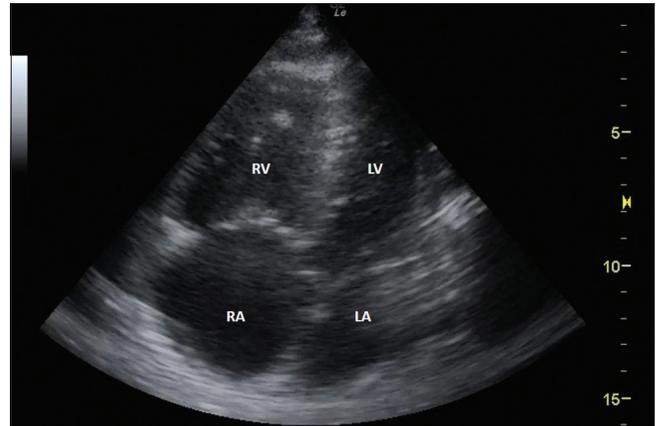


Figure 2: Right atrial and right ventricular dilatation in acute pulmonary embolism

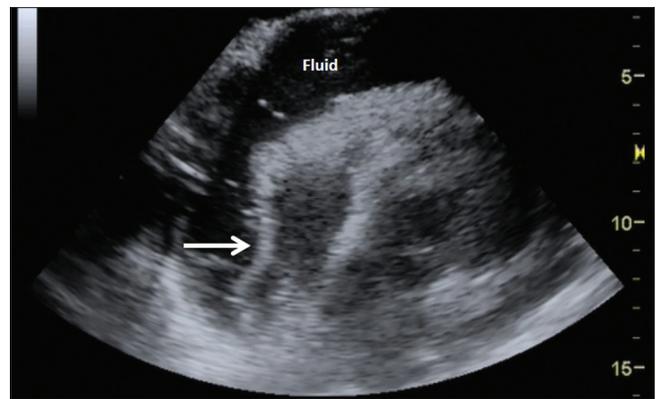


Figure 3: Pericardial effusion seen as an echo-free space around the heart. Arrow depicts right atrial collapse

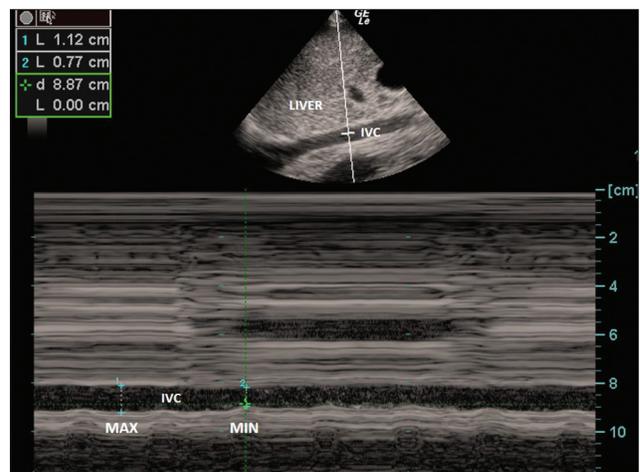


Figure 4: Diameter variation of the inferior vena cava with respiration. Maximum and minimum diameters are measured on M mode (IVC: Inferior vena cava; Max: Maximum diameter; Min: Minimum diameter)

suggestive of hypovolemia. More objective assessment by measurement of the LV end diastolic area may be performed by tracing the endocardial border; however, this may be difficult to carry out due to poor echo windows and is time-consuming.

Measurement of cardiac output

According to the basic hydraulic formula, flow across a fixed orifice is equal to the product of the velocity of flow and cross-sectional area of the orifice. Flow across the LVOT per heart beat (stroke volume) may be calculated using this formula. However, since velocity of flow across the LVOT varies with time throughout systole, individual velocities across the Doppler spectrum need to be integrated as the velocity time integral (VTI). Pulse wave Doppler is applied with the sample volume placed at the LVOT on an apical five chamber view and the resulting waveform is traced [Figure 5]. The built-in software package calculates VTI. The LVOT diameter is measured on the PLAX view at the point of attachment of the aortic leaflets, when the valve is fully open in systole.

$$\text{LVOT area} = \pi \times r^2 \text{ or, } \pi \times (d/2)^2$$

$$\text{Stroke volume} = \pi \times d^2 / 4 \times \text{VTI}$$

$$\text{Cardiac output} = \text{Stroke volume} \times \text{Heart rate}$$

Variation of VTI with respiration may be indicative of volume responsiveness and has been validated in patients undergoing coronary artery surgery, under closed chest conditions.^[22]

Measurement of chamber pressures

With Doppler echocardiography, pressure within cardiac chambers may be measured indirectly. Pressure gradients may be calculated by measurement of velocity of flow, using the Bernoulli equation:

$$\text{Pressure gradient} = 4 (\text{velocity})^2$$

To calculate RV or pulmonary artery systolic pressure,

the pressure gradient across the RV and the RA is measured from the tricuspid regurgitation jet.

$$\text{Pressure gradient across the tricuspid valve} = \text{RV systolic pressure} - \text{RA pressure.}$$

$$\text{RV systolic pressure (equal to pulmonary artery systolic pressure)} = \text{Pressure gradient} + \text{RA pressure (CVP).}$$

Similarly, LA pressure can be measured from the mitral regurgitation jet.

$$\text{Pressure gradient across the mitral valve} = \text{LV systolic pressure} - \text{LA pressure.}$$

$$\text{LA pressure} = \text{LV systolic pressure (systolic BP)} - \text{pressure gradient across mitral valve.}$$

Echocardiography during cardiopulmonary resuscitation

In a cardiac arrest situation, echocardiography may be used to identify the underlying cause, and detect mechanical activity during cardiopulmonary resuscitation.^[23] Presence of mechanical activity may be associated with improved outcomes, while absence of activity is a poor prognostic sign.^[24] Among the underlying causes, hypovolemia, cardiac tamponade, PE, coronary artery disease, aortic dissection and pneumothorax may be diagnosed by ultrasound examination.^[25] The American Heart Association suggests that echocardiography be considered to diagnose any underlying cause that may have led to the arrest and guide further management.^[26]

Assessment of right to left shunts

Intractable hypoxemia may occur in mechanically ventilated patients due to right to left shunts, either across

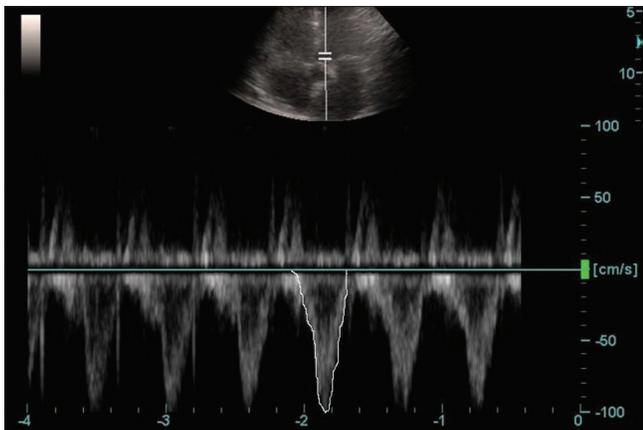


Figure 5: Measurement of the velocity-time integral (VTI). The cursor is placed at the left ventricular outflow tract and interrogated with pulse wave Doppler. The resulting waveform is traced down to measure VTI

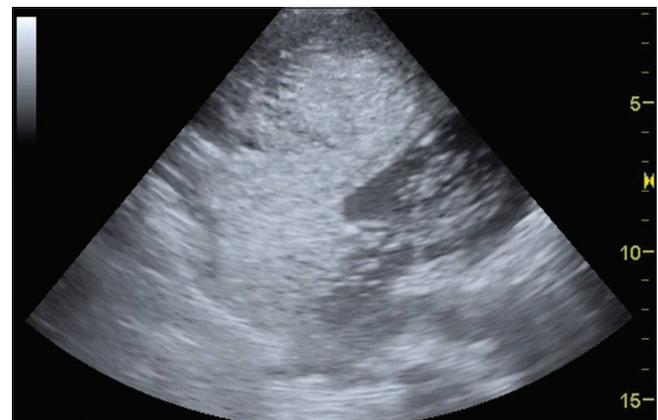


Figure 6: Bubble contrast echocardiographic study, showing a right to left shunt at the atrial level. Opacification is visible in the left atrium and left ventricle

the heart, or through abnormal pulmonary arteriovenous shunts.^[27] A patent foramen ovale is present in up to 25% of normal subjects. With high ventilation pressures and PEEP levels, pulmonary artery and right heart pressures may rise, leading to intermittent right to left shunt across the foramen ovale. A bubble contrast echocardiography helps to detect right to left shunts across the heart or in the lungs. Rapid injection of 10–20 ml of agitated saline intravenously, preferably through a central venous catheter, results in immediate opacification of the left atrium (LA) and the LV in case of a right to left shunt across an open foramen ovale or an atrial septal defect [Figure 6]. If a right to left shunt occurs across pulmonary arteriovenous fistulae, opacification of the LA and the LV occurs typically after 3–5 cardiac cycles.^[14] Intrapulmonary shunts are often seen in patients with end stage liver disease.

The hypotensive patient

Although firm evidence is lacking, a systematic, algorithmic approach using ultrasonography could help assess and guide therapy in the hemodynamically unstable patient. A suggested approach is outlined in [Figure 7]. The first step is echocardiography, looking specifically for cardiac tamponade and LV systolic function. Eyeball estimate of LV function is quick and probably as precise as formal measurements of EF. Dilatation of the RA and RV might suggest acute massive PE, depending on the clinical context. In case of hypovolemia, the heart chambers look underfilled—suggested by a hyperdynamic LV with approximation of the papillary muscles on the PLAX view. If hypovolemia is suspected, the next logical step would be to look for significant variation of IVC diameter with respiration. The IVC size might vary significantly with spontaneous breathing even in normovolemic patients; in this situation, the VTI of the LV outflow tract may be measured before and after performing a passive leg raising test. This maneuver involves raising both the lower limb to an angle of 45° from the semi-recumbent position, for a period of 2 min. A difference in VTI of more than 12–15% is indicative of volume responsiveness. The final step is ultrasound scan of the lung. A normal scan shows less than three B lines in an intercostal space, suggesting that a fluid challenge may be appropriate. On the other hand, in the absence of variation of IVC diameter or VTI, and abnormal B lines (more than three per intercostal space), excessive vasodilatation may be assumed.

Thoracic ultrasound

Advances in CT technology over the years has led to generation of excellent quality images of the

lungs. Conventional X-rays of the chest lag far behind CT imaging in regard to the extent of obtainable information. Given the clear-cut advantage of CT, it was perhaps natural that ultrasound technology failed to draw enough attention by radiologists for viewing the lungs. Intensive care physicians, out of necessity, have pioneered ultrasonography of the chest given the benefits of bedside availability, easy reproducibility, and ability to carry out repeated examinations—an important requirement in critically ill patients.

Probe selection and technique

A linear probe of 8–15 MHz frequency is ideally suited to obtain good resolution images of the pleura. A lower frequency curvilinear or micro-convex probe enables imaging of the lung as well, at the cost of lower resolution of more superficial structures. The lung is viewed in the supine position, starting from anteriorly just below the clavicle, and moving downwards step-by-step. Similarly, a lateral scan is performed from the axilla downwards until the diaphragm is seen. Most often, in critically ill patients, consolidation and pleural effusion occur early at the lung bases. To view the lung bases, the patient will need to be tilted to the opposite side to allow access to the posteroinferior part of the thorax. A systematic approach to ultrasonographic examination of the lung with emphasis on specific areas has been described.^[28]

Pneumothorax

Conventionally, pneumothorax is suspected on the basis of clinical examination and confirmed by chest radiography. Bedside diagnosis of pneumothorax in the ICU can be difficult. In the mechanically ventilated patient, an abrupt decrease in lung compliance, fall in oxygen saturation and raised airway pressures may alert the clinician, but these are relatively nonspecific signs. Antero-posterior X-rays in the supine position are poorly sensitive and often leads to a missed diagnosis.^[29,30] CT confirms the diagnosis and assesses the extent of a pneumothorax, but it entails unavoidable delays, transport of potentially unstable patients out of the ICU, increased costs and exposure to radiation. Ultrasonography of the chest is being increasingly recognized as a reliable tool to diagnose pneumothorax.

To detect a pneumothorax, the probe is placed on the anterior chest wall, below the midpoint of the clavicle, in the second or third intercostal space. The probe marker should point towards the head. The first step is to identify the longitudinal shadows cast by adjacent ribs of an intercostal space. The pleural line is seen as a thick, white, horizontal line seen about ½ cm below the rib line. The space formed by the periosteum of the ribs

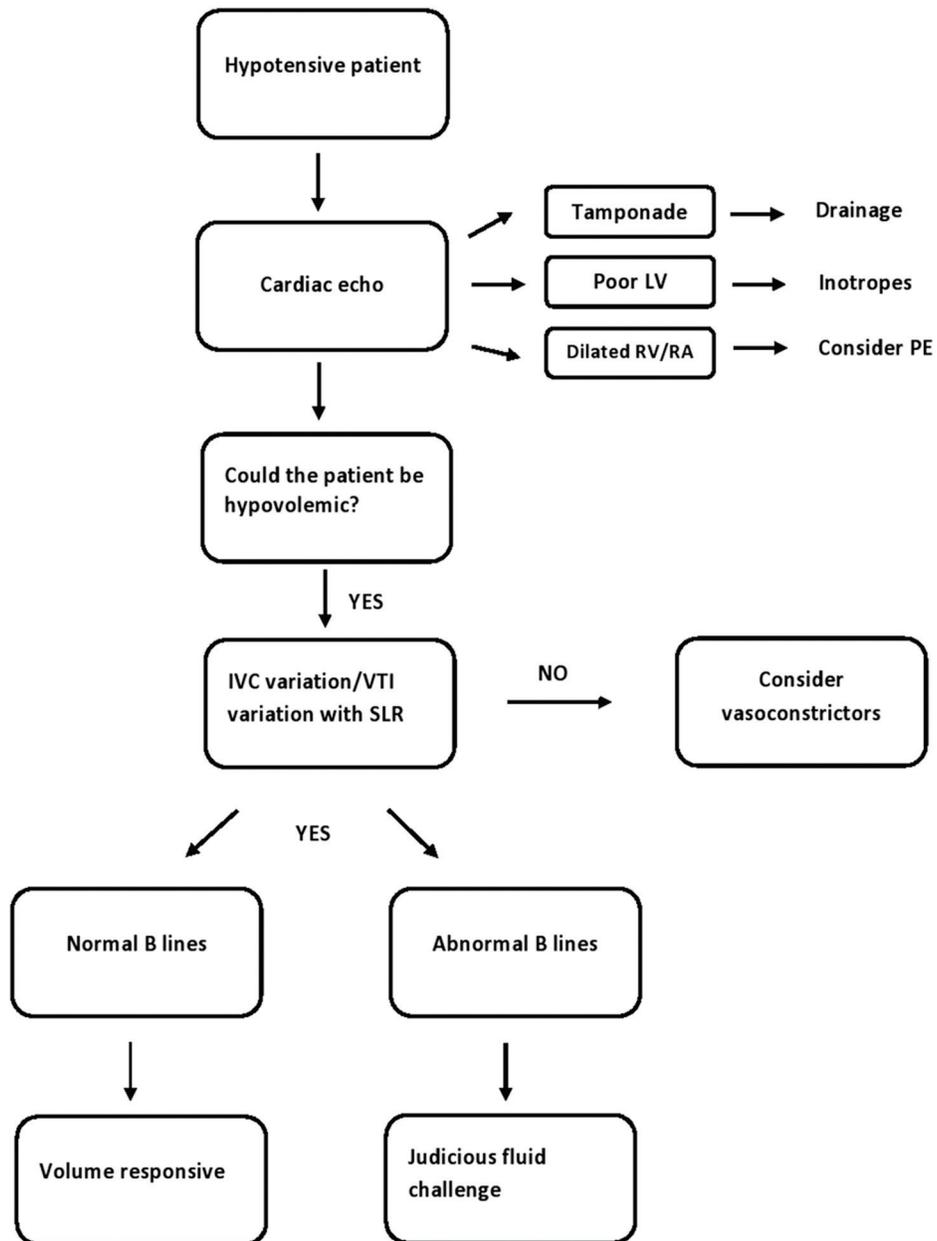


Figure 7: Suggested algorithm for the evaluation and management of a hypotensive patient. LV: Left ventricle; RV: Right ventricle; RA: Right atrium; PE: Pulmonary embolism; IVC: Inferior vena cava; VTI: velocity time integral of the LV outflow tract; SLR: Straight leg raising test

on either side and the pleural line in between appears like a flying bat with its wings spread out and is referred to as the “bats sign”.^[28] With each breath, the visceral pleura slides on the parietal pleura, clearly seen as a side to side, “creeping” movement. On the M-mode, a distinctive pattern appears. The upper part, up to the pleural line is formed by multiple horizontal lines one above the other and the lower part, corresponding to the lung, has a grainy appearance. This has been described as the “seashore” sign, with the upper part of the image mimicking the sea while the lower part appears like the shore.

Pneumothorax is characterized by the absence of pleural sliding. This occurs because the lung collapses down as air gets interspersed between the pleural layers. The visceral pleura lies deeper down and cannot be visualized as ultrasound penetrates poorly through air. On M mode, the “seashore” sign is replaced by a series of horizontal lines extending from top to bottom, referred to as the “bar code” or “stratosphere” sign. Presence of pleural slide effectively rules out a pneumothorax.^[31] However, loss of pleural slide is a nonspecific sign and may be seen in ARDS,^[3] pleural adhesions, emphysematous bullae^[32]

and endobronchial intubation.^[33] The specific feature of pneumothorax on ultrasound is the “lung point” sign [Figure 8]. When the lung is partly collapsed down, the junction at which the visceral pleura meets the parietal pleura during lung expansion is called the “lung point.” The pleural slide is seen to appear during inspiration and extends only partly across the image frame.^[34,35] The size of a pneumothorax may be assessed by the position of the “lung point,” with a more lateral location suggesting a bigger pneumothorax.^[36] Ultrasound has been extensively studied for the diagnosis of pneumothorax in polytrauma and found superior to chest radiography with sensitivity and specificity of more than 90% using CT diagnosis as the reference standard.^[9,32,33]

Alveolar interstitial syndrome

In the normal lung, ultrasound beams bounce off layers of fluid-air interphase, resulting in characteristic “B” lines. These hyperechoic lines arise from the pleural line and fan out to reach the bottom of the frame and move with the pleura as it slides. “B” lines have been described as “lung comets” because of their typical comet-tail like appearance.^[3] The presence of three or more “B” lines between two adjacent ribs has been suggested as indicative of excess fluid in the lung interstitium [Figure 9a and b].^[37] In a prospective observational trial, the comet-tail artifact had a sensitivity of 85.7% and a specificity of 97.7% in the diagnosis of alveolar interstitial syndrome when compared to chest X-rays.^[38] The number of lung comets has shown a positive correlation with extravascular lung water, pulmonary capillary wedge pressure and radiological lung water score in post cardiac surgical patients.^[39] The ultrasonographic “lung comet” count correlates with the New York Heart Association class, EF and LV diastolic function.^[40] Ultrasound findings in ARDS is characterized by non homogenous distribution of “B” lines, anterior sub-pleural consolidations, reduced or absent lung sliding, areas of parenchymal sparing and irregularly thickened and fragmented pleural line.^[16] Apart from cardiogenic pulmonary edema and ARDS, diffuse or localized “B” lines may also be seen in pulmonary fibrosis^[41] interstitial pneumonias, lung contusion and atelectasis.^[42]

Consolidation

The ultrasound signs of consolidation have been described in detail.^[3] The echogenicity of the consolidated lung assumes a “tissue-like” pattern that resembles the liver and is referred to as “hepatization” [Figure 10].^[43] The lung tissue may look like shredded paper with the “shred line” lying deep to the pleural line. Air bronchograms may be seen

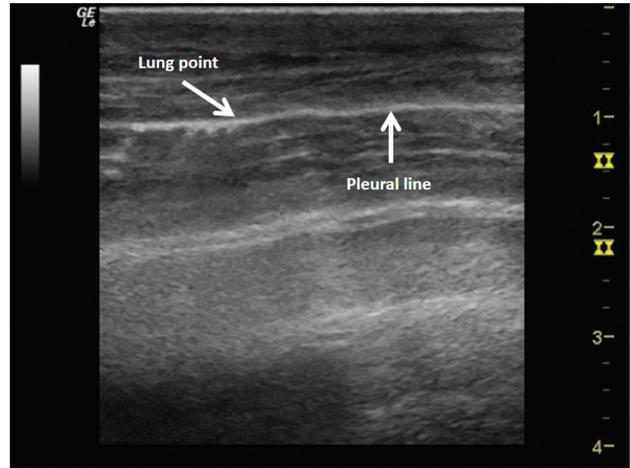


Figure 8: The “lung point” sign seen at the confluence of the visceral and parietal pleura

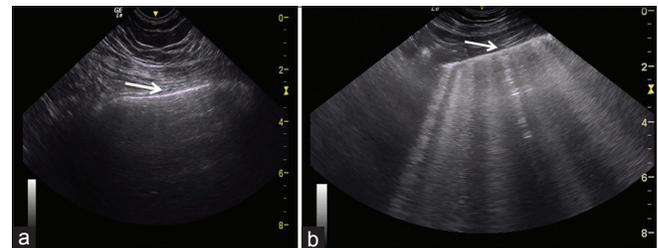


Figure 9: (a) Pleural line (arrow) with normal lung below, (b) alveolar interstitial syndrome with several characteristic “B” lines seen radiating from the pleural line



Figure 10: Consolidated lung with “tissue like” echogenicity, resembling the liver. Arrows point to air bronchograms that brighten up during inspiration

within the consolidated lung, in the form of linear, hyperechoic areas.^[44] These linear streaks turn brighter with inspiration, as air passes through them. In a prospective study, using the “shred sign” as diagnostic criterion, Lichtenstein *et al.* found a sensitivity of 90% and specificity of 98% for the diagnosis of consolidation by ultrasound.^[43] These results were corroborated in a more recent study.^[45]

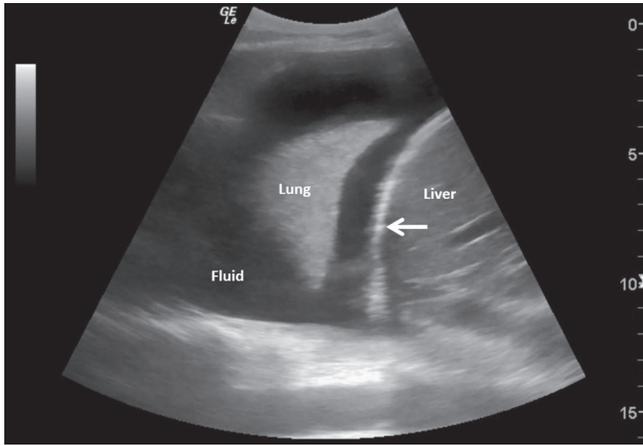


Figure 11: Pleural effusion with the lung floating in fluid like “jelly fish”. Arrow points to the diaphragm

Pleural effusion

Chest radiographs perform poorly in the detection of pleural effusions; besides, distinction from parenchymal opacities is often difficult,^[46] and may be inferior to auscultation.^[47] Ultrasound helps to diagnose, quantify and guide drainage of pleural fluid. Small sized pleural effusions are common in the ICU; occasionally, it might be large enough to necessitate drainage.^[48] Pleural effusions appear on ultrasound as hypoechoic areas between the parietal and visceral pleura. If the fluid collection is sufficiently large, the lung may be seen floating on it like a “jelly fish” [Figure 11].^[3] Effusions may be echogenic and septate if exudative in nature; hemothorax and pyothorax are typically hyperechoic on ultrasound.^[49,50] The approximate volume of pleural effusion may be estimated by measuring the distance between the parietal and visceral pleura at the lung base with the breath held in mid expiration, using the formula:^[51]

$$\text{Volume of effusion (ml)} = \text{inter - pleural distance (mm)} \times 20$$

Drainage of pleural fluid is best performed under real time ultrasound guidance, avoiding the liver, spleen, lungs, and heart that may lie in close proximity. A pig-tailed catheter using the Seldinger technique would suffice for most transudative effusions.

Lung ultrasound was used to determine the cause of acute respiratory failure in 260 dyspneic patients. A rapid bed side diagnosis was possible in more than 90% of cases using a protocolized pathway (The blue protocol).^[52]

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