

Cardiac Limited Ultrasound Examination Techniques to Augment the Bedside Cardiac Physical Examination

Bruce J. Kimura, MD, David J. Shaw, MD, Stan A. Amundson, MD, James N. Phan, RDCS, RVT, Daniel G. Blanchard, MD, Anthony N. DeMaria, MD

The current practice of physical diagnosis is dependent on physician skills and biases, inductive reasoning, and time efficiency. Although the clinical utility of echocardiography is well known, few data exist on how to integrate 2-dimensional screening “quick-look” ultrasound applications into a novel, modernized cardiac physical examination. We discuss the evidence basis behind ultrasound “signs” pertinent to the cardiovascular system and elemental in synthesis of bedside diagnoses and propose the application of a brief cardiac limited ultrasound examination based on these signs. An ultrasound-augmented cardiac physical examination can be taught in traditional medical education and has the potential to improve bedside diagnosis and patient care.

Key Words—continuing medical education; graduate medical education; hand-carried ultrasound; echocardiography; physical examination; physician training; point-of-care ultrasound; screening; stethoscope; vascular ultrasound

Received September 3, 2014, from Scripps Mercy Hospital, San Diego, California USA (B.J.K., D.J.S., S.A.A., J.N.P.); and University of California, San Diego Medical Center, San Diego, California USA (D.G.B., A.N.D.). Revision requested October 14, 2014. Revised manuscript accepted for publication November 29, 2014.

We thank Dudie Keane for administrative support, Tanya Wolfson for statistical support, and the cardiac sonographers at Scripps Mercy Hospital for their work.

Address correspondence to Bruce J. Kimura, MD, Department of Noninvasive Cardiology, Scripps Mercy Hospital, 4060 Fourth Ave, 206, San Diego, CA 92103 USA.

E-mail: kimura.bruce@scrippshealth.org

Abbreviations

CHF, congestive heart failure; CLUE, cardiac limited ultrasound examination; IVC, inferior vena cava; LA, left atrium; LV, left ventricular; RA, right atrial; RV, right ventricular

doi:10.7863/ultra.15.14.09002

For centuries, physicians have taught examination of the heart and lungs, as the physical findings of these organs are often critical in formulating medical diagnoses and managing the course of both cardiac and noncardiac disease. The recent advent of pocket-sized ultrasound devices has made the application of ultrasound to enhance the accuracy of the physical examination not only possible but convenient. However, in current cost-conscious and evidence-driven practice, any future expansion of bedside examinations must be time-efficient, demonstrate value in patient care or in the prediction of outcomes, and be feasibly taught.¹⁻⁴

In this article, we address the novel concept of adding specific, evidence-based 2-dimensional “quick-look” ultrasound techniques to conventional bedside assessment of the cardiovascular system. A simplified cardiac limited ultrasound examination (CLUE) can be created, which searches for only a few ultrasound “signs” indicating the presence of left ventricular (LV) systolic dysfunction, pulmonary edema, pleural and pericardial fluid, right ventricular (RV) enlargement, and elevation of central venous pressure: traditional targets of the cardiac physical examination that have been firmly embedded in bedside formulative reasoning. The CLUE imaging procedure was structured in accordance with practical requirements that the examination can be completed within a few minutes, can be routinely applied to all patients by all physicians, and requires only basic skills and equipment such that training can be broadly incorporated into medical school or postgraduate curricula on physical examination.

CLUE: 6 Ultrasound Signs of Cardiac Disease

In CLUE, the basic 2-dimensional views are designed to integrate physiologic assessment with the acquisition of structural data using a standard 2–3-MHz ultrasound transducer. Sequential imaging is performed from the LV to the left atrium (LA) to the lungs to the RV and finally to the inferior vena cava (IVC). By working backward against the flow of blood, the user can deduce the extent of disease encountered (Table 1) and more easily remember the examination (Figure 1).

Cardiac (LV) Systolic Dysfunction Sign

The detection of LV systolic dysfunction has long been a goal of the cardiac examination, as its presence has prognostic, diagnostic, and therapeutic implications in both acute and chronic settings. In CLUE, the cardiac systolic dysfunction sign is present when the anterior leaflet of the mitral valve fails to approach the ventricular septum to within 1 cm by subjective estimation of its opening motion from the parasternal long-axis view (Figure 1, site 1). This so-called E-point septal separation distance has been justified by echocardiographic⁵ and magnetic resonance imaging⁶ studies with sensitivity of 65% and specificity of 92% for detection of systolic LV dysfunction. Teaching this single-view parameter to novice users improves their diag-

nostic accuracy for LV dysfunction beyond the physical examination alone⁷ and elevates their bedside estimates of LV function to that of the visually estimated LV ejection fraction by experts.⁸

In the CLUE protocol, the parasternal long-axis view, a view obtained by placing the transducer against the left side of the patient's sternum in the third or fourth intercostal space and aiming its directional marker toward the right shoulder, was chosen over other windows due to its relative ease of acquisition and display of multiple cardiac disorders. The parasternal long-axis view can be obtained on supine, restrained, or intubated patients and preserves modesty by exposing only the sternal region during outpatient examinations.

In a “real-world” assessment of 1016 consecutive echocardiograms, the cardiac dysfunction sign had sensitivity of 69% and specificity of 91% for an LV ejection fraction 40% or lower and showed a univariate relationship with in-hospital mortality.³ As with the detection of an S3 gallop, the clinician must recognize that the presence of this ultrasound sign needs confirmation by standard echocardiography and a search for its cause. Furthermore, as is true when any abnormal finding is discovered, early termination of the diagnostic process should be resisted, as the presence of LV dysfunction does not necessarily imply a sole cardiac etiology in an episode of shock or dyspnea.

Table 1. Diagnostic Patterns in CLUE

Disorder	C	L	U	E	S _R	S _I
LV systolic dysfunction, compensated	•	++/-				
Diastolic dysfunction, compensated		•				
Atrial fibrillation (paroxysmal or chronic)		•				
Severe mitral regurgitation/stenosis, compensated		•				
Severe multivessel CAD, compensated	+/-	•				
Symptomatic aortic valve disease, compensated		•				
CHF exacerbation, HFpEF		•	++/-	•		•
CHF exacerbation, HFrEF	•	•	++/-	•		•
Cardiogenic pulmonary edema		++/-	•	++/-		++/-
ARDS, noncardiogenic pulmonary edema			•	+/-		+/-
Interstitial lung disease (acute or chronic)			++/-		+/-	
COPD with cor pulmonale			+/-		•	•
Pneumonia or small pulmonary embolism			+/-	+/-		
Submassive pulmonary embolism			+/-	+/-	•	•
Cardiogenic shock	++/-	•	•	+/-		•
Tamponade				•		•
RV myocardial infarction					•	•
Chronic right heart failure or severe TR					•	•
Septic or hypovolemic shock						•

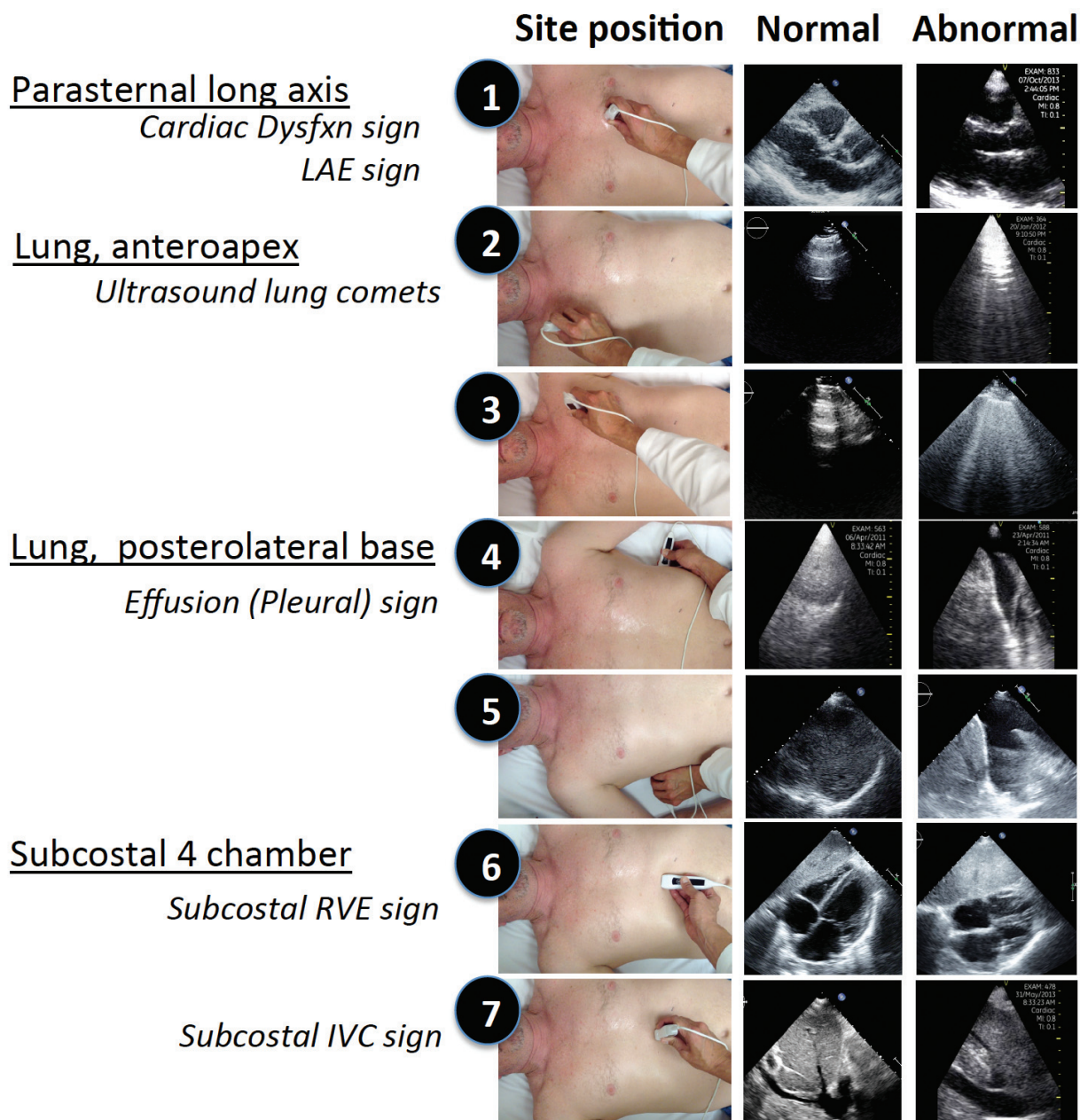
ARDS indicates adult respiratory distress syndrome sign; C, cardiac dysfunction; CAD, coronary artery disease; COPD, chronic obstructive pulmonary disease; E, pleural (or pericardial) effusion sign; HFpEF, heart failure with preserved ejection fraction; HFrEF, heart failure with reduced ejection fraction; L, LA enlargement sign; S_I, subcostal IVC plethora sign; S_R, Subcostal RV enlargement sign; TR, tricuspid regurgitation; U, ultrasound lung comet tail sign; •, present; ++/-, frequently present; +/-, commonly present; and +/-, occasionally present.

Pitfalls of this sign include false-negative results from acute LV dysfunction without symmetric dilatation, as in acute apical ischemia or stress-induced (Takotsubo) cardiomyopathy, and false-positive results due to off-axis imaging or abnormal anterior mitral valve leaflet motion, as in eccentric aortic insufficiency or leaflet calcification.

Left Atrial Enlargement Sign

Although traditional physical examination techniques are insensitive in the detection of clinically relevant LA enlargement, the presence of even mild LA enlargement likely signifies sustained or repeated elevations of LA pressure and brain natriuretic peptide, atrial arrhythmia, and substantial

Figure 1. CLUE protocol, hand position, and normal versus abnormal findings. The 6 CLUE signs and 7 hand positions and probe sites are shown with resultant views when the sign is absent (normal) or present (abnormal). Longitudinal images are oriented with cranial to the right. LAE indicates LA enlargement; and RVE, RV enlargement.



structural findings on echocardiography^{9,10} and has outpatient prognostic value.¹¹ The detection of LA enlargement can have utility in the assessment of unexplained dyspnea or palpitations, improve referral for echocardiography, and be recognized by novice users.⁹ Despite its importance in both acute and chronic cardiac disease, relatively few data exist regarding evaluation of LA enlargement by bedside ultrasound imaging.

The easily recognized LA enlargement sign is defined from the same parasternal long-axis view as the cardiac dysfunction sign after assessment of the LV ejection fraction and is present when the anteroposterior diameter of the LA appears larger than the overlying aorta throughout the cardiac cycle (Figure 1, site 1).¹⁰ The reported sensitivity and specificity of this sign for LA enlargement as indicated by an LA volume index of greater than 28 mL/m² are 59% and 79%, respectively¹⁰ and 75% and 72% when compared to the reporting of any LA enlargement on standard echocardiography.³ The absence of the LA enlargement sign in the setting of the cardiac dysfunction sign can suggest a well-compensated or well-diuresed state, whereas its presence in the setting of normal LV systolic function can suggest diastolic dysfunction, substantial left-sided heart valve disease, or atrial fibrillation (Table 1). Pitfalls of the LA enlargement sign include interpretation errors due to a bright far-field side-lobe artifact generated from the normal right atrial (RA)–pericardial border, asymmetric LA elongation caused by space limitations within the anteroposterior dimension of the chest cavity due to right heart enlargement or, less commonly, skeletal deformity (eg, pectus excavatum or ankylosing spondylitis), and the presence of an aortic root aneurysm.

Ultrasound Lung Comet Tail Sign

Examination of the lung for rales or effusions remains a vital component in the cardiac physical examination. Ultrasound imaging of the lung parenchyma can identify a B-line artifact manifest as a linear full-field reverberation artifact generated from subpleural tissue or interlobular septa that has been thickened by extravascular lung water, inflammation, or fibrosis. The appearance of at least 3 B-lines in a single view constitutes the ultrasound lung comet tail sign.³ Although there is limited standardization and pathologic confirmatory data, the ultrasound lung comet tail artifact has become an accepted finding¹² akin to a sonographic form of rales, showing similar clinical behavior and implications.

In CLUE, after detection of LA enlargement, the lungs are examined for evidence of elevated LA pressure from cardiac decompensation, manifesting as pulmonary edema

or pleural effusions. In the absence of LA enlargement, the ultrasound lung comet tail sign can also be attributed to a capillary leak in adult respiratory distress syndrome, localized lung edema or inflammation, or diffuse primary interstitial lung disease (Table 1). B-lines, either when tabulated as a total score by mapping the lung¹³ or when grouped in ultrasound lung comet tail signs and found in the lung apices,³ are independent predictors of mortality that are more powerful than the LV ejection fraction. Although multiple imaging protocols exist to evaluate the chest for B-lines, a recent study confirmed high specificity of 89% (99% confidence interval, 74%–97%), albeit low sensitivity of 40% (99% confidence interval, 21%–61%) for congestive heart failure (CHF) of 2 anteroapical views.¹⁴ In CLUE, the anteroapices of each lung are interrogated as a sagittal image in the midclavicular line in approximately the third intercostal space (Figure 1, sites 2 and 3). The specificity of these 2 views for CHF can be made particularly effective when complemented with the 85% to 90% sensitivity afforded by elevated N-terminal pro–brain natriuretic peptide measurement or the presence of effusions on basal lung imaging.¹⁵ Imaging for ultrasound lung comet tails is generally easy to learn^{16,17} and likely has device variability and minimal acquisition errors. Ultrasound lung comet tails, like rales, can vary by patient position and are gravity and lung volume dependent, can be present in the lung bases in healthy individuals, and can disappear rapidly with diuresis or dialysis.¹⁸ In addition, the presence of ultrasound lung comet tails under the probe virtually excludes the presence of pneumothorax at that site, as does the presence of pleural sliding,¹⁹ a normal motion best seen with a higher-frequency (5–7-MHz) probe. An important clinical pitfall can occur when ultrasound lung comet tails are chronic and due to fibrosis but are mistakenly attributed to acute edema or inflammation, an occurrence that can be common in chronic lung disease and could be avoided by prior documentation of the finding.

Effusion (Pleural) Sign

Ultrasound imaging has the ability to show as little as a few milliliters of fluid present in the costophrenic angle and has more sensitivity than physical examination or standard anteroposterior chest radiography,¹⁵ which typically require an effusion volume of at least 200 mL before detection.²⁰ Commonly, bilateral effusions with right-sided predominance suggest CHF,²⁰ whereas the presence of a unilateral effusion, particularly when complicated by septation or particulate matter, suggests the possibility of parapneumonic causes, pulmonary embolism, or malignancy.

After determination of the presence or absence of LA enlargement and the apical ultrasound lung comet tail sign with CLUE, a search for fluid in the lung bases can help differentiate CHF from primary interstitial lung disease (Table 1). Pleural fluid, an anechoic region between the lung and diaphragm, is sought with the transducer in a longitudinal (coronal) view in the posterolateral axillary line at the costophrenic angle (Figure 1, sites 4 and 5). In the acutely ill patient, it is important to note that blood, a thrombus, or purulence may cause an effusion to appear nearly isoechoic. A posterior probe position is critical in detecting small layering effusions in the supine intubated patient and also when estimating the volume. False-positive errors can occur if ascites or gastric fluid is mistaken for fluid but can be avoided by identification of the diaphragm and the cephalad location of pleural fluid. Outcome studies of patients with CHF, community-acquired pneumonia, and cancer that was complicated by pleural fluid on chest radiography or echocardiography have generally reported a negative prognostic significance,^{20–22} perhaps indicating that the presence of pleural effusion denotes more severe disease or results in additional mortality due to respiratory compromise or refractory infection.

Subcostal RV Enlargement and IVC Plethora Signs

The subcostal view is a vital complementary or surrogate window to the parasternal long-axis view and can similarly assess LV contraction or LA enlargement and show pericardial fluid or RV enlargement. The initial parasternal long-axis view can be of poor quality in 15% to 20% of cases when obtained by the recently trained⁷ and may be technically difficult for even experienced sonographers in supine or intubated patients with large chests, emphysema, or limited access to the parasternal site due to dressings, pads, or chest compressions. Although the subcostal window is often of good quality in patients with a poor parasternal long-axis view, the subcostal 4-chamber view can be technically challenging, particularly in the obese, in whom a voluntary deep breath hold is often necessary to visualize the heart. When image quality is adequate, pericardial fluid can be easily recognized in this view as an anechoic or hypoechoic region anterior to the right heart and superior to the left lobe of the liver. The presence of a pericardial effusion must be differentiated from the anterior epicardial fat pad, which is comparatively thin and small, or pleural fluid in the right thorax, which collects posterior to the RA and should have been noted in the preceding lung view.

In CLUE, after evaluation of the lungs, the subcostal 4-chamber view can assess right heart performance (Figure 1,

site 6). As the normal RV size is considered to be two-thirds of the area of the LV from the apical or subcostal views, the RV enlargement sign has been defined as the subjective recognition of an RV area approaching or equating to the LV area and is likely a specific sign of acute or chronic RV dilatation. The diagnosis of RV enlargement has clinical utility in the management of acute pulmonary embolism, RV dysfunction in adult respiratory distress syndrome, cor pulmonale, and RV myocardial infarction.^{23,24} As the estimation of RV size is problematic on the physical examination as well as by echocardiography, validation of this sign has been difficult, and few data exist on its application by recently trained physicians. Improper foreshortening of the LV due to off-axis imaging can falsely make the RV appear larger than the apparent LV.

Finally, after a quick look at the RV, estimates of RA or central venous pressure are obtained through a longitudinal view of the intrahepatic IVC from the subcostal position (Figure 1, site 7). The diameter and respiratory variation of the IVC have become parameters of interest in central venous pressure estimation and determination of fluid responsiveness in ventilated septic patients.²⁵ The quick-look IVC plethora sign is present when the anteroposterior IVC diameter is dilated, nearly equaling that of the neighboring aorta, and fails to show respiratory variation of greater than 50%. Dilatation of the IVC without an inspiratory diameter reduction of greater than 50% is associated with both increased in-hospital mortality in patients who undergo echocardiography during admission³ as well as readmission in patients admitted with heart failure.²⁶ In addition to its prognostic importance in left-sided heart failure, the presence of IVC plethora is virtually a requirement in the diagnosis of tamponade or shock attributed to acute right heart failure. The most important pitfall of IVC interpretation for the novice is mistaking the neighboring intra-abdominal aorta for the IVC in the setting of a small, overlooked IVC and can be avoided by mandatory identification of both vessels. The aorta is differentiated from the IVC by its position to the patient's left, and deep to the liver within hyperechoic tissue, its lack of hepatic veins, and often its pulsatile, parallel, and often atheromatous walls. In addition, visual or ultrasound analysis of the jugular vein²⁷ may be of use in the estimation of central venous pressure.

Examination Considerations

Equipment

Since 2007, 4 pocket-sized, battery-operated ultrasound devices have been approved by the US Food and Drug

Administration. These devices share the common goal of 2-dimensional cardiac imaging but with differences in technology (Figure 2). All 4 are equipped with low-frequency (2–3-MHz) transducers for cardiac imaging. Two devices use mechanical transducer technology that allows the interchanging of high-frequency probes (5–7-MHz) to the same base device or to a computer. A 5-MHz vascular probe can add the capability to screen for subclinical carotid atherosclerosis,^{2,28,29} evaluate jugular venous distension and deep vein thrombosis, assist central or peripheral line or pacer lead placement, and search for any subse-

quent iatrogenic pneumothorax. A rudimentary Doppler capability can help differentiate arterial from venous flow. At a current price of approximately \$8000, a pocket-sized ultrasound device is the most expensive item a physician would routinely carry but could provide cost-effective outcomes afforded by more accurate initial diagnostic impressions. In addition, the merger of these devices with smartphone technologies has the potential to both globalize health care delivery and dramatically reduce production costs. It must also be recognized that low-frequency cardiac and abdominal probes and higher-frequency vascular

Figure 2. Currently available pocket-sized devices. Top left, Vscan, 1.7–3.8-MHz phased array transducer (GE Healthcare, Wauwatosa, WI; approved 2009). Top right, Acuson P10, 2–4-MHz phased array transducer (Siemens Medical Solutions USA, Inc, Malvern, PA; approved 2007). Bottom left, Sonimage P3, 3–5-MHz mechanical interchangeable transducer (Signostics Ltd, Thebarton, South Australia, Australia; approved 2013). Bottom right, MobiUS SP1, 7.5–12-MHz mechanical interchangeable transducer, smartphone connected (Mobisante, Inc, Redmond, WA; approved 2011).



probes for carotid or deep venous thrombosis imaging are already available on small laptop ultrasound machine platforms, as well as many older cart-based platforms. Therefore, a simplified 2-dimensional examination such as CLUE can be practiced on machines that are already available at most hospitals, emergency departments, and clinics and does not require the newer, more convenient, technologies.

Procedure

In contrast to standard echocardiographic practice, the basic 6-site CLUE can be performed in less than 2 minutes. The ultrasound device is explained to the patient as a “modern stethoscope,” to avoid a patient expectation of a formal ultrasound examination. During the examination, a general attempt is made to minimize gel reapplications and maintain control and cleanliness of the device by not placing the unit down. The physician starts at the parasternal long-axis view and proceeds to interpret “on the spot,” moving from transducer site to transducer site (Figure 1) around the lung views and finishing in the subcostal view of the IVC. Image or video loop recordings are not mandatory and left to physician discretion, but they can be useful at times to later compare with prior examinations or to show the findings to the patient. Afterward, the probe, its cord, and the patient are wiped clean.

The presence of abnormal CLUE signs can be recorded as a part of the cardiac physical examination for ease of reference. Using the acronym CLUE, as listed in this article’s subheadings, where cardiac dysfunction, left atrial enlargement, ultrasound lung comets, effusions (pleural or pericardial), and subcostal (RV and IVC) signs maintain the “working backward” mnemonic, charting of the presence or absence of the CLUE signs is simplified and easily categorized. Alternatively, the reporting of ultrasound and traditional physical signs can be comingled in the traditional charting of the examination by organ system.

Current Terminology and Practice

The act of incorporating ultrasound into the physical examination remains poorly defined as a medical procedure and is a source of controversy in current practice. Similar to the physical examination, CLUE is a search for specific signs to form an initial diagnostic impression or follow the patient serially and does not require measurements, calculations, or Doppler recordings. Compared to recent definitions, CLUE is not a form of limited echocardiography but does share one characteristic of a limited or focused cardiac ultrasound examination³⁰ in that the user is not interpreting all data manifested in each view. However, distinct from the practice of a focused cardiac

ultrasound examination, CLUE is not a specific medical test based on a “focused” intent to answer a clinical question that arose after the initial evaluation. As in the performance of a traditional physical examination, the practice and accuracy of quick-look ultrasound imaging are subjective and dependent on physician skill at eliciting signs by the proper technique. Diagnostic biases during the bedside physical examination, some brought forward from the patient history and some due to the physician’s own convictions about the data acquired by his or her own technique, are unavoidable, have a variable effect on subsequent decision making, and are an integral part of the responsible practice of bedside examinations.

Training

The acquisition of the skills in the performance, interpretation, and integration of CLUE into the physical examination has been reported in the 10-year experience of an internal medicine residency program.⁴ Over the 3-year residency, attainment of the simplified CLUE skills occurred in 80% of residents, required 50 hours of study and 60 cases of imaging, and was not related to the resident’s academic performance. Ultrasound methods such as CLUE enhance the detection of entities that manifest time-honored physical signs, such as S3 and S4 gallops, rales, egophony, parasternal heaves, and jugular venous distension, and therefore can be easily integrated into the physical examination curriculum in traditional medical education. Ultrasound methods that enhance bedside detection of traditional pathophysiologic observations but do not disrupt bedside thinking could be considered fundamental training for all clinicians and could bridge recent generations of bedside practice by linking both traditional and ultrasound physical examination methods in a natural continuum of learning.

References

1. Kimura BJ, Willis CL, Blanchard DG, DeMaria AN. Limited cardiac ultrasound examination for cost-effective echo referral. *J Am Soc Echocardiogr* 2002; 15:640–646.
2. Kimura BJ, Shaw DJ, Agan DL, Amundson SA, Ping AC, DeMaria AN. Value of a cardiovascular limited ultrasound examination using a hand-carried ultrasound device on clinical management in an outpatient medical clinic. *Am J Cardiol* 2007; 100:321–325.
3. Kimura BJ, Yogo N, O’Connell CW, Phan JN, Showalter BK, Wolfson T. Cardiopulmonary limited ultrasound examination for “quick-look” bedside application. *Am J Cardiol* 2011; 108:586–590.
4. Kimura BJ, Amundson SA, Phan JN, Agan DL, Shaw DJ. Observations during development of an internal medicine residency training program

- in cardiovascular limited ultrasound examination. *J Hosp Med* 2012; 7:537–542.
5. Lew W, Henning H, Schelbert H, Karliner JS. Assessment of mitral valve E-point septal separation as an index of left ventricular performance in patients with acute and previous myocardial infarction. *Am J Cardiol* 1978; 41:836–845.
 6. Silverstein JR, Laffely NH, Ritkin RD. Quantitative estimation of left ventricular ejection fraction from mitral valve E-point septal separation and comparison to magnetic resonance imaging. *Am J Cardiol* 2006; 97:137–140.
 7. Kimura BJ, Amundson SA, Willis CL, Gilpin EA, DeMaria AN. Usefulness of a hand-held ultrasound device for the bedside examination of left ventricular function. *Am J Cardiol* 2002; 90:1038–1039.
 8. Secko MA, Lazar JM, Saliccioli LA, Stone MB. Can junior emergency physicians use E-point septal separation to accurately estimate left ventricular function in acutely dyspneic patients? *Acad Emerg Med* 2011; 18:1223–1226.
 9. Kimura BJ, Fowler SJ, Fergus TS, et al. Detection of left atrial enlargement using hand-carried ultrasound devices to screen for cardiac abnormalities. *Am J Med* 2005; 118:912–916.
 10. Kimura BJ, Kedar E, Weiss DE, Wahlstrom CL, Agan DL. A hand-carried ultrasound sign of cardiac disease: The left atrium-to-aorta diastolic ratio. *Am J Emerg Med* 2010; 28:203–207.
 11. Bouzas-Mosquera A, Broullón FJ, Álvarez-García N, et al. Left atrial size and risk for all-cause mortality and ischemic stroke. *CMAJ* 2011; 183:E657–E664.
 12. Volpicelli G, Elbarbary M, Blaivas M, et al. International evidence-based recommendations for point-of-care lung ultrasound. *Intensive Care Med* 2012; 38:577–591.
 13. Frassi F, Gargani L, Tesorio P, Raciti M, Mottola G, Picano E. Prognostic value of extravascular lung water assessed with ultrasound lung comets by chest sonography in patients with dyspnea and/or chest pain. *J Card Fail* 2007; 13:830–835.
 14. Liteplo AS, Marill KA, Villen T, et al. Emergency thoracic ultrasound in the differentiation of the etiology of shortness of breath (ETUDES): sonographic B-lines and N-terminal pro-brain-type natriuretic peptide in diagnosing congestive heart failure. *Acad Emerg Med* 2009; 16:201–210.
 15. Kataoka H, Takada S. The role of thoracic ultrasonography for evaluation of patients with decompensated chronic heart failure. *J Am Coll Cardiol* 2000; 35:1638–1646.
 16. Bedetti G, Gargani L, Corbisiero A, Frassi F, Poggianti E, Mottola G. Evaluation of ultrasound lung comets by hand-held echocardiography. *Cardiovasc Ultrasound* 2006; 4:34.
 17. Mai TV, Shaw DJ, Amundson SA, Agan DL, Kimura BJ. Learning to apply the pocket ultrasound device on the critically ill: comparing six “quick look” signs for quality and prognostic values during initial use by novices. *Crit Care* 2013; 17:448.
 18. Noble VE, Murray AF, Capp R, Sylvia-Reardon MH, Steele DJ, Liteplo A. Ultrasound assessment for extravascular lung water in patients undergoing hemodialysis: time course for resolution. *Chest* 2009; 135:1433–1439.
 19. Alrajhi K, Woo MY, Vaillancourt C. Test characteristics of ultrasonography for the detection of pneumothorax: a systematic review and meta-analysis. *Chest* 2012; 141:703–708.
 20. Wong CL, Holroyd-Leduc J, Straus SE. Does this patient have a pleural effusion? *JAMA* 2009; 301:309–317.
 21. Roguin A, Behar D, Ben Ami H, et al. Long-term prognosis of acute pulmonary oedema: an ominous outcome. *Eur J Heart Fail* 2000; 2:137–144.
 22. Ercan S, Davutoglu V, Altunbas G, et al. Prognostic role of incidental pleural effusion diagnosed during echocardiographic evaluation. *Clin Cardiol* 2014; 37:115–118.
 23. Frémont B, Pacouret G, Jacobi D, Puglisi R, Charbonnier B, de Labriolle A. Prognostic value of echocardiographic right/left ventricular end-diastolic diameter ration in patients with acute pulmonary embolism: results from a monocenter registry of 1416 patients. *Chest* 2008; 133:358–362.
 24. Lainscak M, Pernat A. Importance of bedside echocardiography for detection of unsuspected isolated right ventricular infarction as a cause of cardiovascular collapse. *Am J Emerg Med* 2007; 25:110–114.
 25. Barbier C, Loubières 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; 30:1740–1746.
 26. Goonewardena SN, Gemignani A, Ronan A, et al. Comparison of hand-carried ultrasound assessment of the inferior vena cava and N-terminal pro-brain natriuretic peptide for predicting readmission after hospitalization for acute decompensated heart failure. *JACC Cardiovasc Imaging* 2008; 1:595–601.
 27. Siva B, Hunt A, Boudville N. The sensitivity and specificity of ultrasound estimation of central venous pressure using the internal jugular vein. *J Crit Care* 2012; 27:315.e7–315.e11.
 28. Spence JD, Eliasziw M, DiCicco M, Hackam DG, Galil R, Lohmann T. Carotid plaque area: a tool for targeting and evaluating vascular preventive therapy. *Stroke* 2002; 33:2916–2922.
 29. Kimura BJ, Fowler SJ, Nguyen DT, Amundson SA, DeMaria AN. Detection of early carotid arterial atherosclerosis by briefly trained physicians using a hand-held ultrasound device. *Am J Cardiol* 2003; 92:239–240.
 30. Spencer KT, Kimura BJ, Korcarz CE, Pellikka PA, Rahko PS, Siegel RJ. Focused cardiac ultrasound: recommendations from the American Society of Echocardiography. *J Am Soc Echocardiogr* 2013; 26:567–581.