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NON-INVASIVE IMAGING

Echocardiography in the emergency room

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The evaluation and management of patients who arrive at the emergency department with acute symptoms and/or worrying clinical condition is a daily challenge. Careful history taking—when it is possible—and clinical examination remain the mandatory first steps. It is of the utmost importance to obtain a timely accurate diagnosis and to immediately stratify the risk to the patient's life. Therefore, choice of the correct investigation is essential. Among the possible tools, imaging modalities are frequently required. Echocardiography is the most versatile method. It is non-invasive, can be performed at the bedside, provides rapid results, and avoids contrast injection and exposure of the patient to radiation. Echocardiography is now available in most emergency rooms, allowing immediate, standard transthoracic examination by an emergency department physician. For some indications and some ultrasonic modalities, specialised and trained physicians or sonographers are necessary for performance and interpretation.

This article focuses on the clinical information that can be obtained from echocardiography according to the most frequent presentations suggesting a cardiovascular emergency.

ACUTE CHEST PAIN: SUSPICION OF ACUTE CORONARY SYNDROME

Patients with acute chest pain account for a notable proportion (20–30%) of medical admissions to the emergency department.[1] Acute coronary syndromes can be easily identified in the presence of typical chest pain and a diagnostic ECG, and confirmed by serial changes in cardiac enzymes. However, a vast majority of patients have atypical chest pain and/or normal or non-diagnostic ECG; early determination of serum troponin frequently is negative. Marginal elevations of troponin in this clinical setting result in uncertainty. To avoid unnecessary prolonged hospital observation (which is costly) and mistaken discharge (with the associated potential for litigation), rapid cost effective and accurate risk stratification is important. Numerous alternative strategies are possible (table 1).

Echocardiography can be performed only at rest, conventionally or with implementation of an ultrasound contrast agent. Echocardiography can also be coupled with exercise or pharmacological stress testing.

Rest echocardiography

A brief echocardiographic examination can be performed in <10 min. The primary role of rest echocardiography when performed in the emergency room is to assess the presence and extent of regional wall motion abnormalities. A dyssynergic ventricular region can be ischaemic, stunned, hibernating or necrotic. In particular, the distinction between ongoing ischaemia and previous myocardial infarction may be difficult; in addition, acute ischaemia can occur in segments adjacent to necrotic tissue. On the other hand, the absence of segmental dyssynergy cannot definitely exclude a recent episode of ischaemia in the absence of stunning. Regional wall motion abnormalities can also be present in other conditions, such as myocarditis, right ventricular (RV) pressure or volume overload, pre-excitation, left bundle branch block or the presence of a pacemaker.

The main limitation is that recording of reliable images can be technically difficult if a skilled echocardiographer is not available. The interpretation of subtle wall motion abnormalities requires training and experience. The current development of telemedicine may be helpful; an expert can guide the recording and interpret images accurately.

A major advantage is the versatility of the echocardiogram. Cardiac causes of acute chest pain other than coronary artery disease can be detected (box 1).

Contrast echocardiography

Slow bolus intravenous injections or continuous infusion of an ultrasonic contrast agent containing small microbubbles results in the opacification of the left ventricle, improving endocardial border detection, and permits the observation of myocardial perfusion. The assessment of both regional function and perfusion at the bedside represents a significant advantage. A multicentre study compared contrast echocardiography and gated single photon emission computed tomography (SPECT) in more than 200 patients presenting to the emergency room with chest pain and non-diagnostic ECG. Both methods provided more incremental information for early cardiac events than did the demographic, clinical and ECG assessment.[2] Another study in the same clinical setting used contrast echocardiography in 957 patients. This technique provided accurate short, intermediate, and long term prognostic information. Normal regional function was most efficient for predicting

Table 1 Resting imaging modalities for triage of patients with acute chest pain and non-diagnostic ECG

Methods	Advantages	Limitations
Coronary angiography	High accuracy Low uncertainty	Invasive Limited immediate availability
Resting echocardiography	Widely available Diagnosis of causes other than CAD	Difficult interpretation of regional dyssynergy Normal if no ischaemia at rest
Contrast echocardiography	Improved endocardial detection Regional function and perfusion Rapid examination	Challenging interpretation of perfusion images
Multislice CT	Rapid image acquisition Diagnosis of causes other than CAD Coronary anatomy	Requires apnoea and contrast injection Radiation
Cardiac MRI	Detection of function, perfusion and necrosis Quantitative information	No isotopes Limited immediate availability
SPECT	High negative predictive value Wide validation	Not available "after hours"

CAD, coronary artery disease; CT, computed tomography; MRI, magnetic resonance imaging; SPECT, single photon emission computed tomography.

lowest risk. For a given regional function, normal perfusion was indicative of a very low risk whereas abnormal perfusion identified a high risk for acute coronary syndrome.[3] However, the choice of appropriate technical settings and correct interpretation of images require experience which is not easily obtained by emergency department physicians.

Stress echocardiography

Current guidelines recommend pre-discharge exercise testing in patients with acute chest pain, normal or non-diagnostic ECG, and normal serum troponin values. The diagnostic accuracy of ST segment changes is limited, however.

Exercise echocardiography requires trained physicians, and patients who are capable of exercising. Echocardiograms are performed at rest and either immediately after treadmill or bicycle exercise, or preferably at peak stress during supine bicycle exercise.[4]

Exercise echocardiography and exercise SPECT have been shown to provide comparable short term prognostic information in the triage of chest pain patients, allowing safe early discharge; the negative

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Box 1 Acute chest pain—cardiac causes detectable by rest echocardiography

- ► Acute coronary syndrome
- ► Pericarditis
- ► Aortic dissection (low sensitivity of transthoracic echocardiography)
- ► Hypertrophic cardiomyopathy
- ► Aortic stenosis
- ► Mitral valve prolapse

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predictive value was 97% in both methods, but exercise echocardiography is preferable because of a higher positive predictive value.[5]

Pharmacological stress echocardiography can be used in patients unsuitable for exercise testing. Graded dobutamine infusion with addition of atropine if necessary, or high dose dipyridamole and atropine, can be used as stressors. Early dobutamine stress after admission to the emergency room has been shown to be feasible and safe. Pre-discharge dobutamine stress echocardiography has important and independent prognostic value in low risk, troponin negative, chest pain patients.[6] As compared to exercise ECG, dobutamine stress echocardiography was found to be more cost effective: the mean length of stay in the hospital was lower; and no event occurred in a 2 month follow-up in patients with a normal dobutamine test, whereas the event rate was 11% in patients with normal exercise ECG.[7] Several studies combining patients submitted to exercise echocardiography or dobutamine stress echocardiography have also demonstrated good risk stratification, especially through a high negative predictive value.[8][9]

In summary, echocardiography has an important role in the evaluation of chest pain in the emergency room when the ECG is non-diagnostic. Rest echocardiography alone has diagnostic limitations. Contrast or stress echocardiography have substantial value for the accurate detection of acute coronary syndromes and for prediction or risk. The strength of these methods is their high negative predictive value. The positive predictive value depends on the patient's characteristics, in particular his probability of suffering from significant coronary artery disease.

ACUTE CHEST PAIN: OTHER CAUSES
Pericarditis

Individuals with pericarditis usually present with chest pain, which is classically increased in the lying position and during inspiration. The pain can be transient. The ECG may show ST segment elevation but it is usually widespread and concave upwards. A pericardial friction rub is audible in only one third of patients and frequently transient.[10] Elevated cardiac troponin can be detected in half of the patients.[11] Echocardiography is helpful if pericardial effusion is detected; this occurs in two thirds of cases.[12] However, regional wall motion abnormalities may be present if pericarditis is associated with myocarditis, and small pericardial effusion is a frequent complication of acute myocardial infarction.

In the absence of pericardial effusion, increased pericardial thickness is detectable, more easily from the subcostal window.

Aortic dissection

Dissection of the aorta is a life threatening condition for which rapid diagnosis and correct management are essential.[13] Chest pain is typically
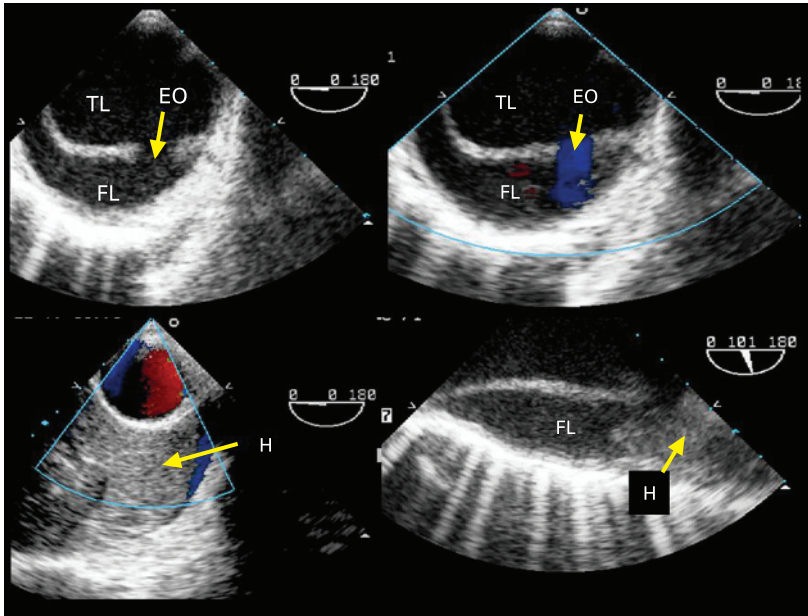
Figure 1 Transoesophageal echocardiographic examination obtained in a patient with acute aortic dissection. EO, entry orifice; FL, false lumen; H, intense spontaneous contrast + thrombus; TL, true lumen.

severe, and immediately of maximum intensity. Clinical suspicion is easier if the pain is migrating and in the presence of specific malperfusion syndrome. Rapid and non-invasive diagnostic imaging is required; the choice of the modality depends on the relative immediate availability.[14] Transthoracic echocardiography can be performed without delay to visualise the aortic root and arch, but the distal ascending aorta and descending aorta are more difficult to evaluate. The specificity is high (90%). The sensitivity has been shown to vary from 55–75%; the proportion of false negative studies decreases when all ultrasonic windows are used, including right parasternal, subcostal, suprasternal, abdominal or even dorsal views. Clearly, a normal transthoracic echocardiographic examination cannot rule out aortic dissection. A major pitfall is the possibility of reverberation artefacts. The presence of a mobile intimal flap separating the true and false lumen can be observed in the proximal ascending aorta. Severe complications can also be identified from the transthoracic approach: acute aortic regurgitation, pericardial effusion, or regional wall motion abnormalities suggestive of dissection involving a coronary artery.

Transoesophageal echocardiography is the preferred ultrasonic modality (fig 1).[15] The blind region is limited to the most distal part of the ascending aorta and the proximal part of the arch. An intimal flap, a large false lumen and a true lumen are diagnostic. The assessment of entry is essential to distinguish between type A and type B, as their management strategies are strikingly different.[16] Several features may be able to differentiate the true and false lumen. The false lumen is usually larger; its diameter increases in diastole; intense spontaneous contrast is frequent in the

presence of blood stasis; and thrombus can be observed. Accuracy of transoesophageal echocardiography requires skilled hands. This method of imaging is the preferred first choice in the emergency room in centres where an experienced cardiologist is readily available.[17] The positive predictive value is of course dependent on the disease prevalence and is lower than magnetic resonance imaging (MRI) in the low prevalence populations.[18]

Other causes of acute aortic syndrome include intramural haematoma and penetrating atherosclerotic ulcers. Although their management remains controversial, proper identification is of clinical importance. Transthoracic echocardiography is of limited value and the transoesophageal approach is more efficient. The semiology of intramural haematoma is well known: >7 mm crescent or circumferential heterogenous thickening of the aortic wall. Longitudinal extension is usually important. Sometimes, an echo-free region may be observed, suggesting haemorrhage or liquefaction of the haematoma. If the diagnosis is questionable, other imaging modalities such as MRI may be necessary.[19] A penetrating atherosclerotic ulcer most frequently occurs in the descending aorta; the transoesophageal window is thus mandatory, but computed tomography and MRI perform better.

Pulmonary embolism

Chest pain is frequent (65%) in patients admitted to the emergency room with a pulmonary embolism, but is usually associated with dyspnoea. Both symptoms and clinical signs are not specific and the differential diagnosis is frequently difficult with acute coronary syndromes or other conditions.[20] If echocardiography is immediately available, important information can be rapidly obtained. The visualisation of a large mobile, serpentine thrombus trapped in the right heart chambers (embolism in transit) is rare but makes the diagnosis evident.[21] Transoesophageal echocardiography is a useful method to visualise pulmonary embolism, especially if it is central. In this case, the sensitivity is 90–95% and the specificity is 100%.[22] In practice, echocardiography provides excellent assessment of the acute haemodynamic consequences. The main findings, although nonspecific, are dilatation of the right heart chambers and of the inferior vena cava, abnormal motion of the interventricular septum, and an abnormal ratio of right ventricular (RV) diameter or area to left ventricular (LV) diameter or area. A ratio between end diastolic right to LV diameter >0.6 and a ratio of end diastolic right to LV area >1.0 are consistent with massive pulmonary embolism associated with RV dysfunction (fig 2).[23]

Another interesting feature is hypokinesis of the mid RV free wall and preserved contraction of the RV apex: the so-called McConnell sign.[24] RV systolic pressure, and thus systolic pulmonary arterial (PA) pressure in the absence of pulmonary valve stenosis, can be estimated from the peak
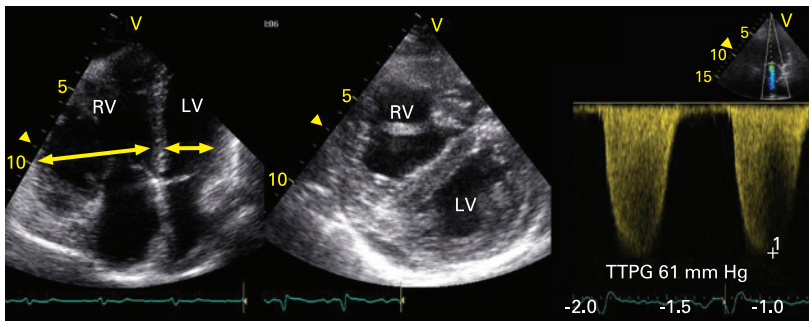
Figure 2 Echocardiographic examination of a patient admitted for a recurrent episode of pulmonary embolism. The right ventricle (RV) was enlarged compared with the left ventricle (LV) and the ratio between end diastolic RV to LV diameter was >0.6. The transtricuspid pressure gradient (TTPG) was notably increased, indicating severe pulmonary hypertension.

velocity of the tricuspid regurgitant jet and application of the simplified Bernoulli equation: PA systolic pressure = right atrial pressure + $4V^2$. Right atrial pressure is estimated by clinical examination of the jugular veins or by the diameter of the inferior vena cava and its respiratory changes. In the presence of a first episode of pulmonary embolism, systolic PA pressure cannot increase to more than 40–50 mm Hg. A chronic process—repetitive episodes of pulmonary embolism or chronic cor pulmonale—is probable if the PA pressure is >60 mm Hg (peak velocity of tricuspid regurgitant jet >3.5 m/s). New ultrasonic modalities such as tissue Doppler imaging, permitting the measurement of RV free wall velocities and strain, can add significant information on RV function.

The parameters cited above not only help in the diagnosis of pulmonary embolism but can also predict prognosis. They could be used and integrated with troponin and B type natriuretic peptide (BNP) elevation to decide upon the therapeutic option—for example, anticoagulation versus thrombolytic therapy.[25 26]

HYPOTENSION AND JUGULAR VEIN DISTENSION

The association of hypotension and elevated jugular venous pressure may be present in different clinical conditions, such as acute heart failure, cardiac tamponade, RV infarction, severe pulmonary embolism, and exacerbation of chronic obstructive pulmonary disease (COPD). Echocardiography is well suited to potentially provide rapid diagnosis in this clinical setting.

Cardiac tamponade

Cardiac tamponade corresponds to acute or subacute compression of the heart. It results from important and/or rapid accumulation of fluid in the pericardial space that increases intrapericardiac pressure above intracavitary pressure. Diastolic filling is reduced and ventricular ejection is compromised. Compensatory tachycardia and vasoconstriction initially maintain a normal cardiac output. At a critical level of intrapericardial pressure, cardiac output and arterial pressure fall. Pulsus paradoxus is present: LV stroke volume decreases during inspiration because of reduced LV preload and paradoxal motion of the interventricular septum resulting from RV volume overload. These clinical features correspond to the echocardiographic findings. The echocardiographic diagnosis of this life threatening condition is usually rapid and simple (fig 3).

Two dimensional echocardiography shows the presence, location and volume of pericardial effusion. The presence of fibrin, thrombus, spontaneous contrast or separated effusion usually implies surgical drainage. The most important aspect in the emergency room is the assessment of haemodynamic consequences.[27] Indeed, the repercussions mainly depend on the rapidity of fluid accumulation rather than on effusion size. The earlier sign indicating a risk of tamponade is right atrial diastolic collapse, but its specificity is moderate. Diastolic RV collapse occurs later, is more specific, and is best appreciated in the parasternal short axis view. Dilatation of the inferior vena cava without change during deep inspiration has a good sensitivity but a moderate specificity for the diagnosis of tamponade. Pulsed wave Doppler is essential. Changes in velocities during inspiration should be recorded at low speed. The most interesting characteristics include inspiratory increase of peak pulmonary ejection velocity (>40%) and of RV inflow velocity (>85%), as well as a parallel reduction during inspiration of LV inflow and aortic ejection velocities.

If the patient's condition requires urgent pericardiocentesis, the procedure can be echocardiographically guided.[27] The subcostal view is important to determine the distance between the patient's skin and the effusion. If necessary, the correct position of the pericardial needle can be visualised and, in the case of doubt, agitated saline contrast can be injected to confirm the intrapericardial position of the needle. Echocardiography can of course be used to verify whether the effusion has been completely drained. Transoesophageal echocardiography is rarely indicated in this setting.
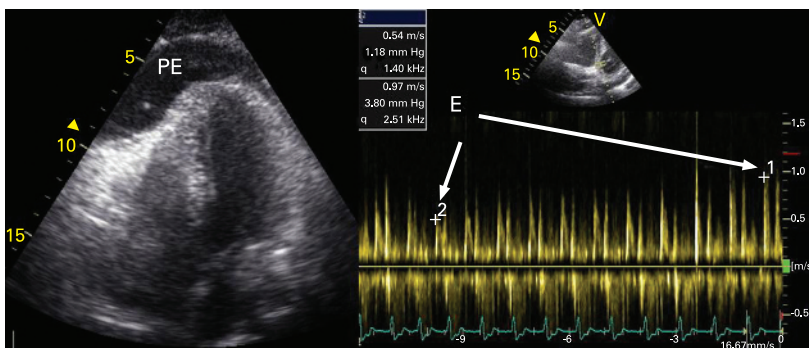

Figure 3 Echocardiographic examination of a patient admitted for cardiac tamponade. The diagnosis was confirmed by the combination of a pericardial effusion (PE), a complete right ventricular collapse, and the significant respiratory changes in mitral E wave velocity.

Figure 4 Echocardiographic examination showing depressed left ventricular (LV) systolic function in a patient admitted for acute dyspnoea.

Acute decompensated heart failure

Numerous patients, especially the elderly, are admitted to the emergency room for acute decompensated heart failure (figs 4 and 5). In addition to hypotension and jugular vein distension, these patients present with dyspnoea and orthopnoea. Pulmonary rales, peripheral oedema and hepatojugular reflux provide evidence of elevated filling pressures. Rapid measures of BNP or NT-proBNP enhance the accuracy of diagnosis. Rapid distinction between heart failure due to systolic versus diastolic dysfunction should be obtained since there are significant differences in treatment. An

Figure 5 Echocardiographic examination showing preserved left ventricular (LV) systolic function in a patient admitted for acute dyspnoea. The diagnosis of hypertrophic cardiomyopathy was made. The spectral tissue Doppler derived E/Ea ratio revealed an increased LV filling pressure confirming the diagnosis of diastolic heart failure. E, early mitral inflow velocity; Ea, diastolic mitral annular velocity.

immediate echocardiogram at the bedside in the emergency room makes it possible to visualise dilatation of the heart chambers, diffuse or regional abnormalities in contraction, functional mitral and/or tricuspid regurgitation or, in contrast, normal LV volume, enlarged left atrium, LV hypertrophy and preserved ejection fraction. Numerous additional measures and detailed evaluation of systolic and diastolic function can be obtained later during the hospital stay.

Severe pulmonary embolism

The role of echocardiography in this clinical setting has been already described.

ACUTE PULMONARY OEDEMA

Acute cardiogenic pulmonary oedema may result from acute events or from acute deterioration of a chronic disease. The main mechanisms include reduced outflow, reduced inflow or backward flow. Some conditions are obvious causes of pulmonary oedema, such as a large acute myocardial infarction or severe tachyarrhythmias. The patients should first be treated with oxygen, nitrates and loop diuretics. Physical examination is insensitive in detecting a cardiac murmur. Thus, Doppler echocardiography can be useful and performed when treatment has been initiated.

Hypertensive crises induce exacerbation of diastolic dysfunction. In this setting, systolic dysfunction is not observed, even transiently; the LV ejection fraction can be normal and similar to that found later after treatment.[28]

Doppler echocardiography can rapidly reveal valvular heart disease, such as severe aortic stenosis, severe acute aortic regurgitation due to endocarditis or rupture of a sinus of Valsalva in the LV, huge mitral regurgitation resulting from papillary muscle or primary chordae rupture, or severe dysfunction of a prosthetic valve. Dynamic exacerbation of chronic ischaemic mitral regurgitation is another condition, probably more difficult to ascertain, because early treatment, especially glyceryl trinitrate, substantially decreases mitral regurgitant volume. Ischaemic mitral regurgitation may appear to be mild or moderate, although it could have been severe at the onset of the syndrome. If this condition is suspected, exercise Doppler echocardiography could reveal it a few days later, showing a dynamic increase in mitral regurgitation resulting in an increase in pulmonary vascular pressure.[29 30]

CONCLUSIONS

Echocardiography should be available in the emergency room for immediate or rapid observation at the bedside of many possible abnormalities that help diagnose cardiovascular syndromes or diseases. This quite versatile imaging method can find a place in the diagnostic algorithm of most clinical acute presentations. This article has only focused on the most frequent or important applications.

Echocardiography in the emergency room: key points

Acute coronary syndrome

► Echocardiography is helpful in patients with non-diagnostic ECG and negative troponin.
► The absence of segmental dyssynergy on resting echocardiography cannot exclude recent ischaemia.
► Rest echocardiography alone has diagnostic limitations.
► Normal perfusion on contrast echocardiography is indicative of very low risk.
► Stress echocardiography is recommended in patients with non-diagnostic ECG and negative troponins.
► The positive predictive value of stress echocardiography is mainly based upon the likelihood of significant coronary artery disease.

Acute heart failure

► Rapid diagnosis in patients presenting with hypotension and jugular vein distension.
► Immediate risk stratification and provides help in clinical decision making.
► Distinction between heart failure due to systolic versus diastolic dysfunction.
► Cardiac tamponade: association of pericardial effusion, right atrial diastolic collapse and respiratory changes (>40% of decrease) in mitral E velocity.
► Severe pulmonary embolism: association of right ventricular dilatation, hypokinesis of the mid right ventricular free wall, significant pulmonary hypertension and elevated BNP.

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