

Response to questions at ACCA Webinar on Echocardiography in Critical Care

The participants in the Webinar were Bernard Cosyns [Professor of Cardiology at the Free University, Brussels, Belgium, and Chairman of the Echocardiography Section of the European Association of Cardiovascular Imaging] and Susanna Price [Consultant Cardiologist and Intensivist, Royal Brompton Hospital, London, and Chairman of the Education Committee of the Acute Cardiac Care Association], and it was chaired by Alan Fraser [Professor of Cardiology at Cardiff University, U.K.]. Both Associations are part of the European Society of Cardiology, and each provides educational material, educational courses, and scientific recommendations, which can be accessed through the ESC website.

Here are answers from all three participants to the questions submitted to this webinar:

Case presentation by SP

- What is non-injurious ventilation?

In intensive care settings, mechanisms that induce lung injury during positive pressure ventilation have been widely studied. In patients with acute respiratory distress syndrome (ARDS), high tidal volumes and airway pressures are associated with increased mortality. Protective ventilatory strategies are now used to protect the lung against volutrauma and barotrauma; these limit plateau pressures and tidal volumes to 6ml/kg ideal body weight. More aggressive ventilation is known to be injurious to the lung. Higher plateau pressures in ARDS are also associated with increased acute cor pulmonale, and mortality. In patients with normal lungs, using a lower tidal volume (not more than 6ml/kg ideal body weight), and PEEP of 6-12 cm H₂O, can decrease the development of ARDS, and reduce pulmonary infection and atelectasis, but not mortality (in the perioperative period and the ICU) [see *Critical Care* 2014, 18:211].

- Why you did not use ECMO?/Why you did you not use ECMO instead of pacemaker?

In the case described, the patient had a recent posterior fossa cerebral infarct. VA ECMO demands full anticoagulation, and therefore strategies were used to try to avoid ECMO if at all possible, as the risk of haemorrhagic transformation existed. As the patient improved over the subsequent hours, as described in the webinar, ECMO was not required. If he had continued to deteriorate, he would have continued to fulfil INTERMACS I criteria and so he would have been supported with ECMO – albeit at risk of stroke. [see *Circulation* 2012; 126: 1401-6].

- Would you compare previous images? Of course the clinical history is important.

Where available, previous images should be reviewed. In this case it was the patient's first medical presentation, so no previous images were available for review. In the ICU, recording and storing of images to allow sequential review are vital – in the same way as chest radiographs and CT scans are reviewed sequentially.

- In my opinion cardiopulmonary exercise stress testing may add important information from Doppler echocardiography in this patient. What is your opinion?

In a patient in cardiogenic shock, where they are already receiving moderate doses of beta adrenergic agents, and with a heart rate of 122-124, I do not believe this would add anything to the immediate management.

- Was this a fulminant myocarditis?

In this patient, no definitive diagnosis was made, but it was considered possible that he had left ventricular failure due to a myocarditis. It was not fulminant, in the sense in which this word is usually used (relentlessly progressive and severe disease culminating in early death).

There is an extensive literature on the role and utility of early endomyocardial biopsy, and more recent publications on the use of magnetic resonance imaging. These are summarised in a recent review [Biesbroek PS et al, *Int J Cardiol.* 2015;191:211-219].

Chest pain

- Is it believable to find normal wall motion by echo during chest pain of MI?

Yes, this may happen in some patients with non-transmural necrosis. Very localized regional wall motion abnormalities may be missed on subjective visual assessment and the detection of hypokinesia is challenging, especially for inexperienced echocardiographers; there is a learning curve just as with stress echo interpretation. In many circumstances, deformation imaging has been shown to be more sensitive to detect subtle myocardial dysfunction. It demonstrates changes in regional myocardial function within seconds of the onset of severe ischaemia and it shows features such as post-systolic shortening and reduced early diastolic lengthening of a myocardial segment that cannot be recognised visually.

- What is the most ideal timing of the usage of speckle tracking in a patient suspected for myocardial infarction?

Within 48 hours after the onset of MI, it has been shown to add prognostic value to the commonly used clinical, biological and echocardiographic parameters [Antoni ML et al, *Eur Heart J.* 2010;31:1640-7].

- Do you think we must do a TTE in every patient with chest pain?

Accurate assessment of chest pain in the emergency department requires a thorough knowledge of the differential diagnosis and appropriate use of diagnostic tools. It is essential not to miss an aortic dissection, pulmonary embolus, or acute myocardial infarction, and to avoid over-treating pericarditis or musculoskeletal pain. In this setting, it is actually recommended to use echocardiography in the following conditions:

- For diagnosis of underlying cardiac disease in patients with chest pain and clinical evidence of valvular, pericardial, or myocardial disease
 - For evaluation of chest pain in patients with suspected acute myocardial ischemia, when baseline ECG and other laboratory markers are non-diagnostic and when the study can be obtained during pain or within minutes after its abatement
 - For evaluation of chest pain in patients with suspected aortic dissection.
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- Would you advise to use speckle tracking in the E&A department due to chest pain? For example to rule out NSTEMI?

If available and appropriately interpreted, it could be helpful. It may add value to make the difference between myocarditis and coronary artery disease, as pointed out during the webinar.

Sepsis

- How do you see the evolving role of bedside echo as mentioned in the latest Sepsis bundles update after the 3 major trials?

There have been several trials recently evaluating early goal-directed therapy (EGDT) in sepsis (ProMISe, ProCESS, and ARISE) with seemingly disappointing results when compared with the 2001 Rivers study - no benefit (or harm) with EGDT vs “usual care”. However, if echocardiography (or focused cardiac ultrasound) is used appropriately as part of “usual care” then this should not be dismissed. In expert hands, the greatest strength of echocardiography (rather than assessing volume status) is its higher diagnostic yield, more accurate differential diagnosis, and earlier diagnosis when compared with clinical judgement and/or pulmonary artery catheterisation alone.

Right heart

- How to differentiate between pulmonary hypertension due to acute pulmonary embolism or other chronic causes like COPD?

In severe acute pulmonary hypertension the PA pressure may not be particularly high (probably not more than about 60 mmHg) because the RV is not hypertrophied, but RV stroke volume and cardiac output will be low. On echocardiography, the RV will be dilated with poor global function, and with secondary tricuspid regurgitation. In more chronic

pulmonary hypertension, related to COPD or to idiopathic pulmonary hypertension, the PA pressures may be much more elevated (even to supra-systolic levels) and the RV will be hypertrophied. Of course, these conditions are mostly identified from the overall clinical presentation, history, and other investigations, in addition to echocardiographic features.

- Comment about PAH grading: suggest correction for CI as low cardiac output will affect PVR assessment.

The determinants of pressure are flow and resistance and there are situations whereby stroke volume and pulmonary vascular resistance contribute unequally to pressure. When stroke volume is markedly decreased, the sPAP may artificially be underestimated in these pathological clinical situations. PVR can be estimated using $((TVR/RVOT\ VTI) \times 10) + 0.16$ with a good estimate compared with invasively measured PVR, up to approximately 8 WU (Wood units). Other formulae are available but none have not been validated for clinical use and so they should not replace invasive measurements.

- Do you use tissue Doppler to assess right ventricular diastolic function in ventilated patients?

Yes – but interpretation is more difficult because of the multiple influences of altered loading and drug therapy.

- Is it useful to measure pulmonary vascular resistance in critical patients?

Yes, but it rarely adds much to other parameters (right heart VTI (velocity time integral, a measure of cardiac output), pulmonary acceleration time, TR volume and velocities, RV systolic function, PA Doppler, and evidence of RV restriction) in particular when directing management in intensive care. It is also potentially limited by image quality – especially when working on a cardiothoracic ICU where many patients may be post-operative and the RV wall difficult to image to an adequate quality.

- Any specific measurements for the RV?

Tricuspid annular systolic excursion (TAPSE) measured by M-mode echocardiography or from displacement on tissue Doppler, is a good index of RV systolic function, although it is affected by changes in preload. RV relaxation time measured with pulsed tissue Doppler at the tricuspid annulus can demonstrate when RV diastolic pressure is elevated. RV regional deformation measured by speckle tracking or myocardial velocity imaging gives useful information about RV function for example in pulmonary hypertension.

LV filling pressure

- Can we trust echocardiographic measures to establish left ventricular end-diastolic pressure (LVEDP)?

The duration of retrograde flow in the pulmonary veins during atrial systole (compared with antegrade flow) is an excellent guide to LVEDP, that can be trusted, but obviously this is not available in patients in atrial fibrillation. The E/e' index was first developed as a non-invasive guide to mean pulmonary capillary wedge pressure, but in some studies in patients with severe heart failure it has been shown not to correlate with directly measured LV filling pressures, so its use as a single test is unwise. In patients with poor global LV function (low ejection fraction) who do not have severe mitral regurgitation, a short deceleration time of mitral inflow indicates a high filling pressure and a poor prognosis in patients in atrial fibrillation as well as those in sinus rhythm.

Pericardial effusion / tamponade

- What about PW Doppler to assess flow variation (in RV and LV) in case of suspicion of cardiac tamponade, when we have doubt about the severity of the effusion?

Of course, this is mandatory. It is important not to rely on the measurement of only one single parameter. It has been shown that the amount of effusion is not an appropriate parameter to exclude tamponade; the rate of accumulation is much more critical. Moreover, loculated effusion may also be responsible for tamponade. In other words, the severity of the effusion is not a sufficient criterion. An integrative approach is recommended, including PW Doppler flow variation > 30 % of the AV inflow.

Cardiac output

- Can we trust echocardiographic measures to establish cardiac output?

There are challenges when using TTE for evaluating CO – TOE has been shown to be superior, and in experienced hands to correlate well with PA catheter thermodilution methods. A major issue is image quality when measuring LVOT dimensions in the critically ill, as well as the presence of arrhythmias. Changes in VTI (velocity time integral; from the left and/or right heart) are useful when evaluating response to therapeutic interventions, however, the echocardiographer/sonographer must be absolutely rigorous in application of the technique.

Lung comets

- Do we need to perform lung US exam simultaneously with heart echo exam?

This is an interesting and challenging question. The short answer is that it can be helpful – in particular when excluding interstitial oedema, or in a cardiac arrest situation to exclude pneumothorax, but the usefulness and level of evidence depends on the specific scenario. This excellent review gives an in-depth discussion: *Cardiovascular Ultrasound* 2011;9:6.

Arterial function and ventricular-arterial coupling

- When is left ventricular systolic elastance more appropriate in ICU patients to evaluate systolic function?
- Could you please comment on the usefulness of ventriculo-arterial coupling evaluation in the acute setting.

Estimation of the LV end-systolic pressure volume relationship (ESPVR) and its slope (LV elastance) is determined by contractility (and inotropy) as well as a number of other important variables. Despite the theoretical attractiveness of such measurements, in the acute setting, a number of concerns exist regarding using non-invasive assessment (as a single beat assessment in echocardiography), as assumptions include that elastance is not influenced by loading conditions or heart rate. Theoretically attractive with respect to both the left and the right heart, these assumptions plus the lack of evidence for their validity in terms of directing interventions/supportive measures and improving outcomes, and lack of specificity of any abnormal findings in the rapidly changing situation of the CICU make them challenging for routine use. As part of an integrated echocardiographic haemodynamic approach to monitor effects of changing interventions, I believe they warrant further attention. [see *Critical Care* 2013;17:213.]

The assessment of CV function by evaluation of the E_a/E_{lv} ratio can offer additional information to understand the pathophysiology of altered haemodynamic profiles and guide therapeutic strategies but this concept still needs to be tested in large trials.

Further information

- Can you please give us a list of basic literature for those engaged in acute echo?
- What do you suggest as a textbook for helping to understand echocardiography in the ventilated patient?

Daniel De Backer et al: *Hemodynamic monitoring using echocardiography in the critically ill*. Springer (2011). •ISBN 978-3-540-87956-5

See the EACVI/ACCA paper on “The use of echocardiography in acute cardiovascular care: Recommendations of the European Association of Cardiovascular Imaging and the Acute Cardiovascular Care Association”. Lancellotti P et al, *Eur Heart J Cardiovasc Imaging*. 2015; 16(2):119-46. doi: 10.1093/ehjci/jeu210.

Who should perform echocardiography and how?

- What do you think about critical care echo training? What should the cardiologist in training do to increase skills in this field?

- Spend formal time on the intensive care unit to learn about critical care interventions (ventilation, inotropes, filtration) that will change echo findings.
- Spend some time with an anaesthetist accredited to perform intraoperative transoesophageal echocardiography, to understand mechanical circulatory support, acute haemodynamic changes during separation from cardiopulmonary bypass, and response to cardiac filling.
- Spending a day with a perfusionist is always excellent value to understand right and left heart haemodynamics better.
- Attend the ICU ward rounds on a regular basis – including the discussion regarding the patient you have been asked to image.
- Spend some time training with those who do high-level critical care echo on a regular basis.
 - All new techniques shown are interesting but in emergency we have TTE..
 - Can we do only portable transthoracic echocardiography in the ER or would you recommend complete echocardiography?

A complete and comprehensive echocardiographic study is always preferable but sometimes, depending on time constraints, material and specialist availability, a portable echocardiographic study can provide precious information.