

ECHO EDUCATION

CRITICAL CARE ECHO ROUNDS

Haemodynamic instability

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Abstract

The use of echocardiography, whilst well established in cardiology, is a relatively new concept in critical care medicine. However, in recent years echocardiography's potential as both a diagnostic tool and a form of advanced monitoring in the critically ill patient has been increasingly recognised. In this series of Critical Care Echo Rounds, we explore the role of echocardiography in critical illness, beginning here with haemodynamic instability. We discuss the pathophysiology of the shock state, the techniques available to manage haemodynamic compromise, and the unique role which echocardiography plays in this complex process.

Case:

A 69-year-old female presents to the emergency department with a fever, confusion and pain on urinating. Her blood pressure on arrival was 70/40, with heart rate of 117 bpm. Despite 3 l of i.v. fluid she remained hypotensive. A central venous catheter was inserted and noradrenaline infusion commenced, and she was admitted to the intensive care unit for management of her shock state. At 6 h post admission, she was on high dose of noradrenaline (0.7 µg/kg per min) but blood pressure remained problematic. An echocardiogram was requested to better determine her haemodynamic state.

Shock state

Shock is defined as acute circulatory failure with inadequate or inappropriately distributed tissue perfusion resulting in generalised cellular hypoxia (1). Originally, a French term 'Choc' was translated into English as description of a collapse state following trauma, but its usage has been expanded to cover a syndrome resulting in inadequate tissue perfusion and oxygenation (2).

Management of the shock state constitutes one of the biggest challenges in critical care medicine. Classically, shock is classified into four broad aetiological

categories: hypovolaemic, cardiogenic, obstructive and distributive (Table 1). Whilst this provides a useful means of determining the principal underlying mechanism, it is somewhat of an oversimplification. Multiple mechanisms may co-exist (e.g. as is often the case in the of severe sepsis); furthermore, interventions undertaken to correct one aspect of pathophysiology often have detrimental consequences on other haemodynamic parameters (e.g. the introduction of inodilator agents to improve myocardial contractility often precipitates hypotension via vasodilation). Modern intensive care management of shock therefore involves real-time identification and

Table 1 Classification of shock, mechanism and causes.

Mechanism	Potential causes
Hypovolaemic Reduction in circulating volume leads to decreased left ventricular end-diastolic volume and subsequently decreased stroke volume	Haemorrhage Gastrointestinal loss Burn
Cardiogenic Impaired ventricular systolic function leads to reduced stroke volume	Acute cardiac Ischaemia Myocarditis Valvular dysfunction
Obstructive Obstruction to flow (either due to stenosis or elevated cardiopulmonary pressures) leads to reduced left ventricular end-diastolic volume and decreased stroke volume	Cardiac tamponade Pulmonary embolus Tension pneumothorax Valvular stenosis LVOT obstruction
Distributive Impaired vascular tone leads to reduced blood pressure and inappropriate distribution of cardiac output	Sepsis Anaphylaxis Post-arrest syndrome Post-operative

correction of the underlying pathophysiological derangement coupled with continuous titration of multiple haemodynamic variables (Table 2) with a view to optimising oxygen delivery and utilisation.

To guide this physiological manipulation of contractility, flow and resistance, numerous forms of haemodynamic monitoring have been developed including the pulmonary artery catheter, oesophageal Doppler and various forms of arterial waveform analysis. Yet, despite the wealth of information which these devices provide, none offers the diagnostic capability nor the subtle appreciation of cardiovascular performance of echocardiography. Echocardiography plays a role in diagnosis of the underlying aetiology whilst allowing evaluation of other pathophysiological parameters contributing to the haemodynamic instability (e.g. fluid status). To this end, echo is integral to the optimal management of the shocked patient.

Pragmatically, the approach to echo in shock differs from a standard echo laboratory study. First, the purpose is not to perform a comprehensive formal examination, but rather to obtain a real-time appreciation of cardiovascular function. Second, the dynamic nature of critical illness requires serial studies to observe progress and the effect of any intervention on cardiovascular physiology. Finally, the study findings are useless if they are not communicated directly and immediately with the clinical team as this allows immediate intervention and real-time

observation of the effect. The dynamic nature of both critical illness and its management makes a delayed data communication poorly representative of the patient's rapidly changing condition.

In the aforementioned case, the contribution of heart failure, relative hypovolaemia and peripheral vasodilatation is not known. An echo in addition to other haemodynamic monitoring is fundamental in guiding the treating physician.

A systematic approach to the shocked patient requires the team to answer four pertinent questions (Fig. 1):

- i) is there evidence of major obstruction to blood flow?
- ii) Will the patient respond appropriately to fluid resuscitation?
- iii) Is the systemic vascular resistance (SVR) low necessitating a vasopressor?
- iv) Is there evidence of myocardial dysfunction necessitating an inotrope?

Role of echocardiography in haemodynamic management

Obstruction

Echocardiography is the gold standard means of diagnosing cardiac tamponade. A small acutely accumulating fluid in the pericardium can have more haemodynamic effect than a large chronic one. Echo allows visualisation and quantification of pericardial effusion and determination of the impact upon physiology – i.e. is there evidence of tamponade. Moreover, it has a major role in guiding emergency pericardiocentesis (3). Echo findings suggestive of tamponade are respiratory variability of the mitral and tricuspid maximum *E* velocity (by more than 25 and 40% respectively); diastolic right atrial collapse and diastolic right ventricular (RV) collapse (Video 1). Typically, the inferior vena cava (IVC) is dilated with reduced respiratory variation (<50%). It should be noted that cardiac tamponade can present also by left ventricular (LV) dysfunction rather than the RV, either due to localised pericardial effusion or circumferential effusion in patients with pulmonary hypertension (4).

An acute rise in RV afterload (acute cor pulmonale (ACP)) may precipitate or aggravate shock. Classically, ACP is the consequence of pulmonary embolism, which obstructs pulmonary blood flow. However, the profound oxygenation and ventilation problems – and destruction of lung architecture – associated with severe lung disease (e.g. acute respiratory distress syndrome (ARDS)) may also give rise to elevations in pulmonary artery pressures

Table 2 Outline of haemodynamic parameters frequently manipulated on the intensive care unit.

Haemo-dynamic parameter	Preload	LV after-load/SVR	RV after-load/PVR	Myocardial contractility	Heart rate
Increased by	Fluid administration Reduction in ventilation pressures Vasopressor drugs	Vasopressor drugs (e.g. noradrenaline)	Mechanical ventilation	Catecholamines (adrenaline and dobutamine) Phosphodiesterase inhibitors (e.g. Milrinone and Enoximone) Calcium sensitizers (Levosimendan) Mechanical support (e.g. intra-aortic balloon pump, ventricular assist device and extra-corporeal life support (ECLS)) β blockers (e.g. Esmolol and Metoprolol)	Catecholamines (e.g. adrenaline and dobutamine) Anti-cholinergic agents (e.g. Atropine) Electrical pacing (permanent or temporary devices)
Decreased by	Diuretic drugs Haemodialysis or haemofiltration Positive pressure ventilation	Vasodilator drugs (e.g. GTN and sodium nitroprusside) Intra-aortic balloon pump	Reduction in ventilator pressures Improvement in oxygenation Inhaled nitric oxide Phosphodiesterase inhibitors (e.g. Milrinone)	Acute myocardial ischemia Myocarditis	Drugs (e.g. β blockers and calcium-channel blockers)

LV, left ventricle; RV, right ventricle; SVR, systemic vascular resistance; PVR, pulmonary vascular resistance; GTN, glyceryl trinitrate.

and increased RV afterload, particularly if associated with high-pressure mechanical ventilation (5, 6). It is rare for the RV systolic pressure (RVSP) to increase beyond 60 mmHg unless there is underlying pre-existent pulmonary hypertension. ACP is represented on echo as RV dilatation (most commonly identified by measuring RV diameter from the parasternal long axis view (PLAX), RV outflow (RVOT) and apical four chamber view on transthoracic echocardiography (TTE) or as an RV:LV area ratio >0.6 on transoesophageal echocardiogram (TOE)) (6). Other signs include D-shaped left ventricle (shift of interventricular septum (IVS) from the centre of the RV) on parasternal short axis (PSAX) and rarely used a shortened pulmonary acceleration time (AcT) (<105 ms in PSAX view or RV outflow view) (Video 2). The timing of the IVS shift (D-shaped LV) is beneficial in the differentiating RV volume overload (e.g. tricuspid regurge (TR)) from pressure overload (IVS shift is maximum at end-diastole in RV volume overload while in pressure overload it is marked both in diastole and most marked at end-systole) (7). Pulmonary artery pressures may be quantified by means of the velocity of the TR jet.

Pressure is the product of multiplying flow by resistance (pressure = flow × resistance), therefore the decrease in pulmonary flow as in massive pulmonary embolism associated with shock state can lead to underestimation of echo-derived RVSP. To overcome this, measurement of the pulmonary vascular resistance (PVR) may be more helpful. Classically, right heart catheterisation with measurements of the cardiac output (CO) and the pulmonary artery pressures are used to calculate the PVR, and this technique remains the gold standard. Non-invasive means of measuring PVR using the TTE have been suggested (8). One method utilises the ratio between the TR velocity and time velocity integral (TVI) measured in the RVOT:

$$PVR = 10 \times TR \text{ velocity} / TVI_{RVOT}$$

Video 1

Apical four-chamber view showing pericardial effusion with RA collapse (considered an echocardiographic early sign of cardiac tamponade). Download Video 1 via <http://dx.doi.org/10.1530/ERP-14-0008-v1>

Video 2

PSAX showing dilated and impaired systolic function of the LV. Download Video 2 via <http://dx.doi.org/10.1530/ERP-14-0008-v2>

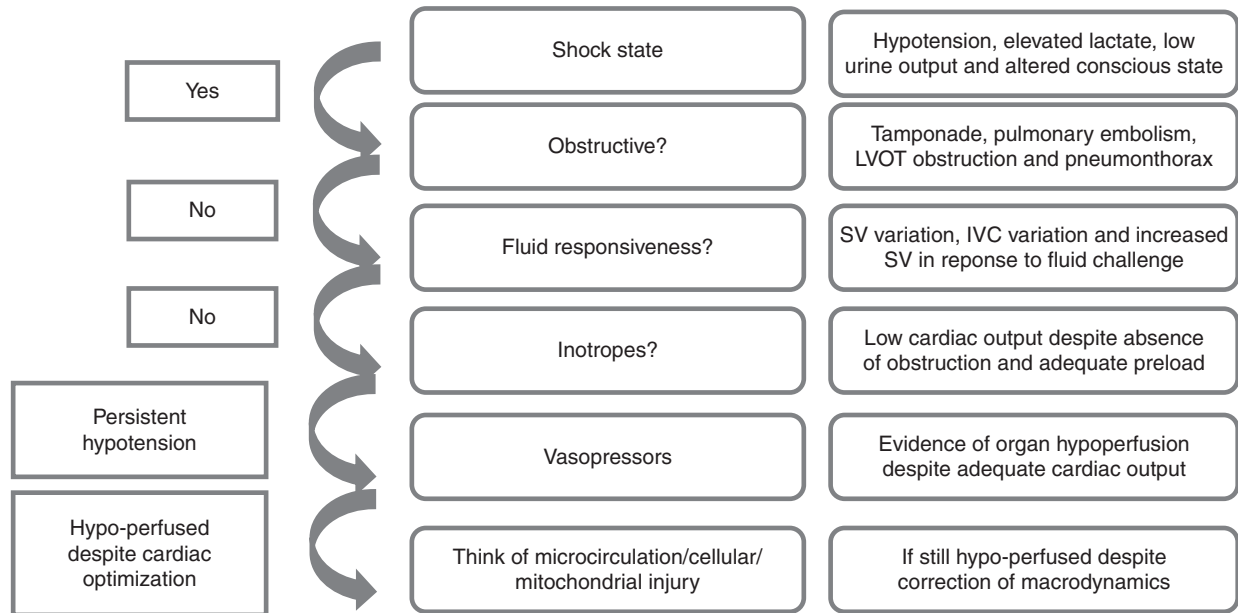


Figure 1
Proposed algorithm of simplified step-wise haemodynamic approach.

A TRV/TVI_{rvot} ratio <0.2 has a sensitivity and specificity of 70 and 94% respectively to detect $PVR <2$ Woods Unit (which equates to normal PVR) (9). Another method suggests an index integrating the pre-ejection period (time between the onset of TR and the onset of pulmonary flow), pulmonary AcT and total systolic time (the sum of the pre-ejection period and pulmonary ejection time) (10). A third study suggested an index of pulmonary artery systolic pressure to heart rate (HR) times the RVOT time TVI. The benefit of the latter is the inclusion of the HR and the right atrial pressure (RAP) (11). These methods have not, however, been validated in the critically ill patients. In our experience, acquiring optimum Echo windows to accurately obtain those measurements is challenging in an intensive care unit (ICU) patient.

Obstruction to flow may also occur due to stenosis. This may be related to a long standing, fixed valvular or membranous lesion (e.g. aortic stenosis) exacerbated by superimposed critical illness, in which case echo is the diagnostic modality of choice. Alternatively and more commonly, the obstruction may be dynamic: a relatively under recognised phenomenon in the critically ill. Dynamic obstruction occurs most commonly in the LV outflow tract (LVOT). In the outpatient setting, LVOT obstruction (LVOTO) is typically associated with hypertrophic cardiomyopathy but can occur in structurally normal hearts or in cases of LV hypertrophy in the context of hypovolaemia

and inappropriate inotrope use (12). Its incidence increases post-cardiac surgery and myocardial infarction (13, 14). Clinically, LVOTO appears as a low CO state refractory to inotropes. Echo is the only available tool which allows its definitive identification. LVOTO is frequently the result of systolic anterior motion of the mitral valve, which can be identified in 2D or by placing the M-mode cursor across the mitral valve leaflets in the PLAX. Continuous wave Doppler interrogation of the LVOT from the AP5C typically shows a dagger shape, late peaking waveform; when haemodynamically significant it is associated with a high-peak gradient. Pulsed wave (PW) can be used to track the gradients from LVOT towards the apex.

Fluid management: hypovolaemia and fluid responsiveness

Exclusion of obstructive shock may be followed by assessment of volume status and fluid responsiveness. The administration of fluid increases LV end-diastolic volume, thereby increasing myocardial stretch, thus improving myocardial performance as described by the Frank-Starling law. This improvement occurs only up to a point, beyond which further fluid may be detrimental to cardiac performance (approximately half of the ICU patients do not respond to fluid challenge) (15). When considering fluid status, it is important to appreciate the distinction between the terms hypovolaemia and fluid

responsiveness. Hypovolaemia is defined as a decrease in circulating volume; fluid responsiveness is defined as $\geq 15\%$ increase in CO or stroke volume (SV) after fluid administration. (<http://pact.esicm.org/media/HaemMon%20and%20Mgt%208%20April%202013%20final.pdf> as accessed on 1st March 2014). Whilst an overtly hypovolaemic patient will almost certainly be fluid responsive, a patient need not to be overtly hypovolaemic to demonstrate increase CO in response to fluid. Therefore, even in the absence of overt hypovolaemia (e.g. euvolaemic or even hypervolaemic patients can be fluid responsive), it is a common practice to assess for fluid responsiveness, with a view to optimise filling status and in doing so improve CO and oxygen delivery. Moreover, the fluid responsiveness is more simple and practical to test in comparison with a real intravascular hypovolaemia.

The role of echo in fluid management is therefore first to diagnose overt hypovolaemia as a precipitant of the shock state, and second to identify those patients in whom fluid will improve CO and optimise haemodynamics, regardless of the primary pathology. Hypovolaemia is represented on echo as a small, hyperdynamic left ventricle: LV end diastolic and end systolic volumes are decreased, as is IVC diameter. Such patients are, by definition, fluid responsive.

In determining fluid responsiveness in an apparently normovolaemic patient, a number of echo techniques may be employed (16).

SV variation Changes in intra-thoracic pressure throughout the respiratory cycle impacts upon cardiac filling: left sided venous return is less during inspiration (as blood is sequestered in the pulmonary vessels) and greater during expiration, this is reflected by variation in the SV measured throughout the respiratory cycle. SV variation (SVV) is more marked in the presence of inadequate hypovolaemia; an SVV $> 9.5\%$ is predictive of fluid responsiveness (17). Variation of the LVOT VTI is the easiest mean of identifying SVV; when variation alone is sought, the LVOT diameter is not required (18).

IVC respiratory variation IVC size and variation are commonly used in ultrasound to estimate RAP. Only one small study ($n=40$) evaluated this measurement in spontaneously ventilated patients and showed sensitivity of 70% and specificity of 80% of fluid responsiveness in those with IVC variability $> 40\%$ (19). However, the use of IVC variability as a marker of fluid responsiveness has been better validated in ventilated patients. In this group, IVC variation is more subtle; a threshold of 12% change

in diameter (maximum diameter—minimum diameter/mean diameter) is widely used as a predictor of fluid responsiveness (20).

There are, however, several limitations to the techniques based upon IVC respiratory variation: inconsistent respiratory tidal volumes, cardiac arrhythmia, low lung compliance (as in ARDS), raised intra-abdominal pressure and open chest all render these techniques less useful (21).

An alternative test for fluid responsiveness is to observe the impact of a fluid bolus on SV and CO. This may be achieved by means of the passive leg raising (PLR) test: in the supine position the patient's legs are raised, ~ 300 ml of blood is transferred from the legs to the thorax. The effect of this 'virtual' and reversible fluid challenge on SV may be assessed by measuring LVOT TVI before and after the PLR; an increase of $> 10\%$ is suggestive of fluid responsiveness (22). The technique involves moving the patient from a semi-recumbent 45° position to supine whilst simultaneously raising the legs to 45° (23). The fluid responsiveness should be assessed preferably within a minute of performing the test. Increased intra-abdominal pressure and starting the test from a horizontal position limit the test value (23).

Fluid administration should continue in shocked patients till the resolution of the shock state, patients become fluid unresponsive or when they reach a raised left atrial pressure, risking the development of pulmonary oedema especially if they are not mechanically ventilated.

Need for inotropy If obstructive shock and severe valvular dysfunction have been excluded and filling status optimised, low CO could be due to myocardial dysfunction. Myocardial dysfunction may be addressed in a number of ways (Table 2).

Cardiac dysfunction is common in critical illness and importantly even in the absence of a history of cardiac pathology; the incidence of LV systolic dysfunction in severe sepsis is reported to be as high as 60% (24). Cardiac dysfunction commonly co-exists with other mechanisms of shock. Consequently, echocardiographic assessment of myocardial function is increasingly utilised in the critically ill even if a cardiac pathology is felt clinically to be unlikely.

Takotsubo cardiomyopathy – a condition associated with excess circulating catecholamines and which manifests as non-ischaemic wall motion abnormalities – has a higher incidence within the intensive care (25); the increasing use of echo in critical illness has increased the detection of this condition. The ability to diagnose

Takotsubo as a contributing factor in haemodynamic instability is a further example of the unique role of echocardiography over other cardiac monitors within the ICU.

In the outpatient environment, quantification of systolic function most commonly takes the form of LV ejection fraction (EF). The dynamic loading conditions encountered in critical illness make the EF less useful. Of greater importance in critical care is the cardiac index (CI; which represents the end point of overall cardiac function). CI is most commonly determined by multiplying HR by the product of LVOT TVI and LVOT cross-sectional area: this determines CO which is subsequently divided by body surface area to give the standardised CI. Frequent echo studies with repeated quantification of CI and LV function allows both the effectiveness of interventions to be assessed and therapy to be titrated. In cases of low CO/CI and signs of hypoperfusion, the detection of decreased LV contractility should orient the treating physician to start inotropic support (pharmacological or mechanical). On the other hand, when interpreting the echo study, the existence of any inotropic support should be considered as it affects the EF, CO and CI. Serial studies should give a dynamic picture about both the pathophysiological changes and the inotrope effect on the cardiac function.

Need for vasopressors

Persistent hypotension despite exclusion of significant cardiac pathology and optimisation of cardiac physiology suggests distributive shock (Fig. 2). Blood pressure is determined by both CO and SVR:

$$\text{Mean arterial blood pressure} = \text{CO} \times \text{SVR}.$$

Hence inappropriate reduction in SVR (as commonly occurs in severe sepsis, anaphylaxis and with many sedative drugs) leads to hypotension. In pure distributive shock, the LV is hyperdynamic with supranormal EF and CO. Management involves the use of vasoconstrictors, the most commonly used agent being noradrenaline.

Commonly distributive shock coexists with both hypovolaemia and cardiac dysfunction. The routine use of echocardiography is therefore a key to ensure the optimum balance of fluids, inotropes and vasoconstrictors.

Effect of inotropes, vasopressors and mechanical ventilation on the echo data: Interpretation of echo in the haemodynamically unstable critical care patient must take into account a number of factors not

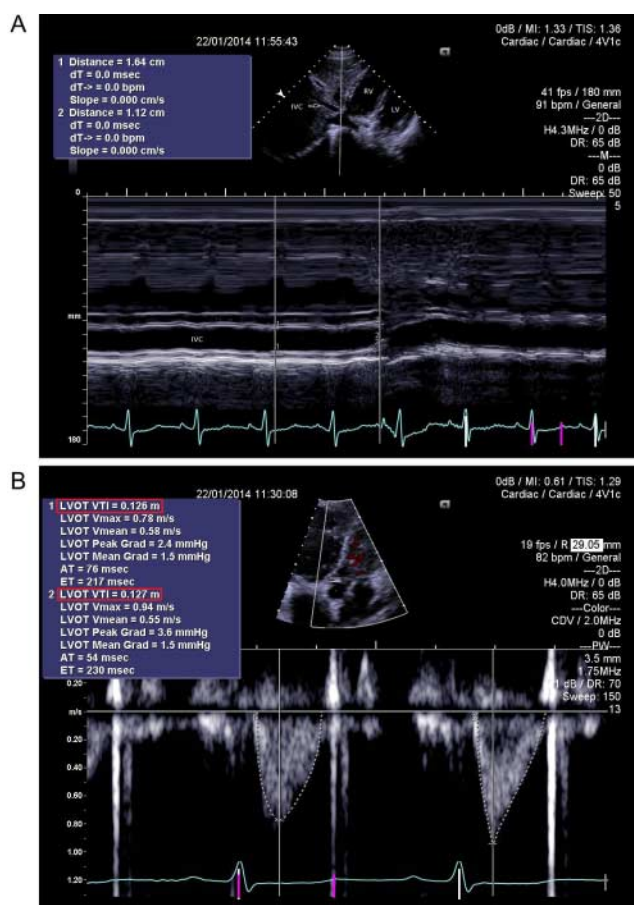


Figure 2

(A) Subcostal view of the IVC. Note the measurement during inspiration and expiration. (B) Apical five-chamber view with PWD in the LVOT. The measured LVOT VTI at the aortic valve level can be used for calculation of the cardiac output.

encountered in the outpatient setting. First, the degree of current haemodynamic support must be considered: in the patient receiving no inotropic support a CO of four litres may be acceptable; the same CO in a patient receiving high-dose adrenaline is a very different prospect.

The conflicting effects of haemodynamic interventions must be considered. In general, inodilators increase EF but decrease SVR; vasoconstrictors increase SVR and in so doing, increase LV afterload; fluids improve LV function to a point but are detrimental in excess. Furthermore, most inotropes are chronotropic as well leading to increase in cardiac oxygen consumption. In an increasingly aged ICU population with underlying coronary artery disease, this can precipitate myocardial ischaemia. Repeated assessment to allow regular titration of therapy is the key.

Mechanical ventilation adds additional complexity, increasing RV afterload. Mechanical ventilation can be

titrated in response to echo findings if RV dysfunction is evident.

Limitations of echocardiography in the ICU

- i) Environmental factors within the intensive care make the physical act of echo challenging. Transoesophageal echo is often a feasible alternative in the frequently sedated ICU population.
- ii) Echo cannot provide continuous monitoring. Repeated scanning places a significant burden on resources. This may be ameliorated to some extent by training critical care staff in basic echo and utilised focused scanning. Recent technological advances can offer a solution (e.g. disposable TOE probes) in the future.
- iii) Despite the use of standardised measurements, there is undoubtedly a degree of subjectivity and operator variability in critical care echocardiography.
- iv) Finally, if not appropriately decontaminated, the echo probe can be a source of infection transmission in the ICU.

Summary

Echocardiography is becoming a standard of care in the ICU. We believe echo to be essential in the diagnostic workup of shock. We believe it constitutes the best tool to assess the haemodynamic state as a whole, thus guiding the array of available haemodynamic interventions. An understanding of critical illness and its management is a key to maximising the benefit of echo in the ICU.

Declaration of interest

The authors declare that there is no conflict of interest that could be perceived as prejudicing the impartiality of the research reported.

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