Ten Things to be Considered in Practicing Critical Care Echocardiography

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Varieties of critical scenarios, such as hypotension, hypoxemia, dyspnea, cardiac arrest, and acute kidney injury (AKI), which affect multiple organs, are always encountered in critically ill patients. Recently, with the noninvasive, convenient, and unique value of quick diagnosis and dynamic monitoring during the course of treatment, critical ultrasonography has been widely used in critical care medicine, which makes the bedside and real-time evaluation practical.[1] As an important part of critical ultrasonography, critical care echocardiography (CCE) can provide information about the circulatory system from two aspects of morphology and function, which can be an effective tool to save lives in critically ill patients with hemodynamic instability. CCE can quickly identify potential causes of shock and provide definitive information to physicians to choose the correct treatment strategy.[2-5] Therefore, CCE has become a milestone in the hemodynamic monitoring of critically ill patients. In this article, we summarize the following ten principles of CCE, which may be helpful in clinical applications.

Qualitative Assessment of Cardiac Function is Useful in the Intensive Care Unit

Qualitative CCE can provide sufficient information in the treatment of hemodynamically unstable patients.[6] Melamed *et al.*'s study[7] showed that after only 6 h of training on image acquisition and interpretation, critical care physicians using handheld ultrasound could effectively assess the left heart function of patients with an accuracy above 80%. In addition to the left ventricular (LV) systolic function, qualitative evaluation was easily able to distinguish patients with significant or nonsignificant superior vena cava (SVC) respiratory changes and nondilated or dilated right ventricle (RV).[8] Actually, qualitative information on LV dimensions, systolic function, RV function, volume status, pericardial effusion, gross signs of chronic heart disease, and gross valvular abnormalities are all targets of CCE,[4] which can not only assist in early shock diagnosis but also alert clinicians to underlying physiologic disturbance.[9]

Quantitative Heart Assessment is Fundamental to the Timing and Targeting of Hemodynamic Therapy

Timing and targeting are two basic features of hemodynamic therapy. Different types of shock coexist, and mutual, transformational, continuous measurement of the hemodynamic variables is preferable; in these circumstances, therefore, quantitative cardiac assessment is also very important.[10] Unlike traditional echocardiography, CCE focuses on several key points, such as cardiac output, systolic function, volume status, and cardiac filling pressures, instead of comprehensive assessment.

Cardiac output is the product of stroke volume and heart rate, and the stroke volume can be obtained by measuring the area and velocity time integral of the LV outflow tract (LVOT), and the value is close to the result obtained by the pulmonary artery catheter.[11,12] Left systolic function can be assessed by LV ejection fraction,

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which can be calculated indirectly from the LV fractional shortening by measuring the LV end-systolic diameter and end-diastolic diameter in patients without regional dysfunction. In patients with positive pressure ventilation, the absolute value of the inferior vena cava (IVC) diameter at the end of expiration can indicate the patient's volume status, and variation of the IVC diameter according to the heart–lung interaction can provide useful information of fluid responsiveness.[13] In addition, the quantitative measurement of filling pressure of the heart is of great significance for determining the pressure target of the hemodynamic therapy. Right atrium (RA) pressure is most commonly estimated by IVC diameter and the presence of inspiratory collapse.[14] Systolic pulmonary arterial pressure estimated by tricuspid regurgitation can not only assess the extent of pulmonary embolism (PE) and pulmonary hypertension but also be widely used in the evaluation of acute cor pulmonale (ACP) in acute respiratory distress syndrome (ARDS).[15] Left atrium pressure (LAP) is also valuable in the diagnosis of weaning failure of cardiac origin.[16]

Preexisting Cardiac Diseases Should be Recognized First

A variety of abnormal signs can be found when CCE is performed in critically ill patients. These signs could be clues for the intensivists to search for not only the causes of hemodynamic instability but also the consequences of preexisting cardiac diseases. For example, RV dilatation is a typical sign of acute RV dysfunction, which may be induced by PE, a severe disease that should be managed immediately; however, in patients with chronic pulmonary hypertension, RV dilatation may be a common sign without any clinical symptoms. Thus, when the findings of the CCE were used in the diagnosis, it was very important to recognize whether these findings were caused by the patients' preexisting cardiac disease.

As the RV is the only significantly acutely dilatable chamber, an easy way to recognize preexisting cardiac disease is observing the morphology of the chambers. In addition, RV and LV hypertrophy are also signs of preexisting diseases as they are usually caused by chronic pressure overload. LV and LA dilation may indicate the existence of dilated cardiac myopathy; a marked LV hypertrophy can exist in hypertrophic obstruction cardiac myopathy, severe aortic stenosis, and hypertension; and RA dilatation or RV dilatation and hypertrophy can be the signs of chronic pulmonary hypertension.

Furthermore, pericardial effusion and severe valvular diseases should also be noticed, as they not only may be the signs of preexisting cardiac diseases but can also be the causes of the hemodynamic instability, as tamponade and severe aortic stenosis are both common causes of obstructive shock.

Echocardiographic Assessment of the Hemodynamics of Critically Ill Patients Starts from the Inferior Vena Cava

Volume status and fluid responsiveness are important parts of hemodynamic assessment in critically ill patients and should also be initially considered when CCE is introduced into this procedure. IVC is a highly collapsible major vein whose diameter has not been found to be affected by the body's compensatory vasoconstrictive response to volume loss,[17] so it reflects volume status more closely than other parameters based on the arterial system. Studies showed that IVC diameter could be a reliable and quick mode of volume status assessment and guide fluid resuscitation[18] and that the respiratory variation in the IVC diameter could detect fluid responsiveness in mechanically ventilated patients with septic shock,[19] even in those patients with atrial fibrillation.[20] The IVC diameter can be measured in the subcostal area and the right mid-axillary line, and the two results cannot replace each other.[21] Furthermore, the shape change index can provide morphological information about the IVC.[22]

The size and dynamics of the IVC can also be used as an index of right-side cardiac function.[23] When factors increasing the right heart pressure exist, the IVC will be dilated and fixed; thus, it is a useful tool in the diagnosis of right heart failure, which can be caused by PE, pulmonary hypertension, and so on.

Echocardiographic Assessment of Cardiac Function Starts from the Right Heart in Critically Ill Patients

In critically ill patients with circulatory shock, the role of the LV has long been appreciated. RV, in contrast, is considered "forgotten", perhaps because it is thinner walled, more difficult to image, and coupled indirectly to the systemic circulation. The ascendance of intensivist-conducted echocardiography has forced the intensivist to pay more attention to the RV.[24] The right side of the heart, comprising the RA and RV, accepts the entire cardiac output and pumps it through the pulmonary circulation to the left side of the heart.[25]

The RV and LV share the same septum, so the diastolic function of the LV can be influenced by the increased pressure load of the RV, which is common in the critically ill patient, as the thin-walled RV is much more sensitive to increase in afterload. Therefore, putting the right heart into first place in the cardiac function assessment and identifying the influence of the right heart on the left heart or even the whole circulation system are very important.

Acute RV dysfunction causes and exacerbates many common critical illnesses, which can be divided into four categories, including acute pressure overload, acute decrease in contractile function, acute volume overload, and acute decrease in diastolic filling.[25] ARDS is one of the most

common conditions to challenge the RV. The incidence of acute RV failure is approximately 25% even with lung protective ventilation.[15,26] Pulmonary vascular dysfunction, defined by an elevated transpulmonary pressure gradient, occurs in 73% of ARDS patients, can lead to an abrupt increase in pulmonary artery pressure, and may induce ACP.[27] ACP can also be induced by high positive end-expiration pressure in several ways, including biventricular-reduced venous return and increased RV afterload.[28] In unselected patients with PE, echocardiographic features of RV dysfunction are present in between 29% and 56%, which is correlated with an increased risk of in-hospital mortality. RV infarction and postcardiotomy RV failure can also increase the risk of complication and death.[29] RV function can also be injured in septic shock on several aspects, which is one of the most common diseases in the Intensive Care Unit (ICU). First, the pathophysiology mechanism of septic shock includes decreasing of the systemic vascular resistance and increasing of the pulmonary vascular resistance; second, septic shock is often accompanied by myocardial depression, which influences both the LV and RV;[30] at last, AKI is also common in septic shock. AKI often induces volume overload and consequently influences the right heart. Therefore, RV dysfunction may occur in many critically ill patients and is associated with the prognosis. The assessment of cardiac function in critically ill patients should start from the right heart.

Echocardiographic Assessment of the Right Heart Starts from the Right Ventricular Size

The free wall of the RV is composed of transverse muscle fibers, which are thinner than the LV. This unique anatomical structure makes the RV more sensitive to pressure and volume load. The RV dilates in response to chronic volume and/or pressure overload, and with RV failure, the RV/LV end-diastolic diameter ratio was shown to be a predictor of adverse clinical events and/or hospital survival in patients with acute PE.[31]

RV enlargement is best evidenced on an apical four-chamber view. Normally, the RV/LV end-diastolic area ratio (RVEDA/LVEDA) is <0.6, a ratio ranging from 0.6 to 1.0 indicates mild RV dilation, whereas a ratio >1 denotes severe RV dilation.[32] RV pressure overload also distorts the normal circular short-axis geometry of the LV by shifting the septum leftward away from the center of the RV and toward the center of the LV, resulting in the flattening of the ventricular septum and D-shaped short-axis LV cavity profile.[14] Many experts have defined ACP as an RVEDA/LVEDA ratio in the long axis >0.6 associated with septal dyskinesia in the short axis.[33] Conversely, the compressed RV is often associated with pericardial tamponade or pneumothorax of the right lung, accompanied by the dilated IVC and elevated central venous pressure. Therefore, RV enlargement or compression is a manifestation of RV involvement; the assessment of the RV should initiate from the size.

Echocardiographic Assessment of the Left Ventricular Function Starts with the Diastolic Function

Diastole is a sensitive part of the cardiac cycle. Normal aging is associated with a slowing of LV relaxation, which may lead to diastolic dysfunction. The earliest abnormality in any cardiac pathology is diastolic dysfunction. Thus, it is natural that any cardiac disease can cause LV diastolic dysfunction to a variable extent. Doppler can only detect LV diastolic dysfunction at later stages.[34] The heart being a pump, diastolic function is often ignored as systolic function is critical. In septic shock patients, >40% of patients had diastolic dysfunction,[35] and early diastolic dysfunction is a strong and independent predictor of mortality in cancer patients, presenting with septic shock.[36] There is a large difference between treatment of diastolic dysfunction and systolic dysfunction; determining the existence of diastolic dysfunction is very important in critically ill patients.

Diastolic function can be quickly recognized in a qualitative way with CCE. First, patients with cardiac hypertrophy and LA enlargement are usually accompanied by diastolic dysfunction; second, patients with atrial fibrillation are also combined with diastolic dysfunction due to the lack of regular atrial contraction; in addition, when systolic dysfunction occurs, diastolic dysfunction is usually also involved.

LV filling pressures are important parts of the evaluation of LV diastolic function. The diastolic filling pressure comprises LV end-diastolic pressure and LAP, while the LAP is the pressure that relates better to the mean pulmonary capillary wedge pressure.[37] Diastolic dysfunction usually manifests as the decrease in relaxation time and compliance, which leads to the increase in filling pressures in the same volume state. The E/e' ratio can be used to predict LV filling pressures, which is feasible and reproducible. Values of average E/e' ratio <8 usually indicate normal LV filling pressures, values >14 have high specificity for increased LV filling pressures, while the "gray zone" includes the values in which LV filling pressures are indeterminate.[36] This ratio is also helpful in predicting weaning failure of cardiac origin.[16]

Differentiation between a Diffuse Hypokinesia and a Regional Wall Motion Abnormality Must be Determined when Assessing the Left Ventricular Systolic Function, and the Differentiation between a Coronary-Related Segmental Wall Motion Abnormality and a Stress-Induced Takotsubo Cardiomyopathy Must also be Determined

There are various causes of cardiac dysfunction in critically ill patients, such as severe infections, acidosis, cardiac

arrest, acute coronary syndrome, and administration of negative inotropic drugs. Per the regions involved, the LV systolic dysfunction is divided into diffuse hypokinesia and regional wall motion abnormality (RWMA). Acute coronary syndrome (ACS) and Takotsubo cardiomyopathy are two common diseases that can cause RWMA and are usually treated by the recanalization of the criminal vessels or eliminating the stress factors. Diffuse hypokinesia is usually caused by systemic factors, such as infections, drugs, and acidosis, whose recovery depends on the improvement of systemic factors.

Although there are no significant differences in the length of ICU stay and duration of mechanical ventilation, the mortality of day 28 in patients with diffuse hypokinesia is significantly higher than that of patients with RWMA.[38] Therefore, determining the type of LV systolic function is of great importance in choosing the correct treatment strategy and predicting the prognosis.

In patients with LV RWMA, whether the coronary arteries are involved should be determined. The patients with coronary artery disease are prone to present ACS due to hypotension and hypoperfusion in the ICU; in addition, factors such as high fever, tachycardia, agitation, and excessive use of vasoconstrictive drugs can also lead to coronary spasm and myocardial ischemia. Takotsubo cardiomyopathy is not rare in critically ill patients as another type of RWMA; the pathophysiological mechanism and clinical features are different from those of ACS. Previous studies have suggested that Takotsubo cardiomyopathy is preceded by emotional or physical triggers, even without any evident preceding trigger. Takotsubo cardiomyopathy is divided into four forms: apical, midventricular, basal, and focal.[39] As this condition is usually accompanied with LVOT obstruction (LVOTO), reversible mitral regurgitation, RV dysfunction, and thrombosis, early diagnosis and classification are of great importance.

Since patients with Takotsubo cardiomyopathy commonly present with symptoms similar to those among patients with an ACS,[40] and the treatment strategies are quite different, initial differential diagnosis is necessary when LV RWMA is detected.

Dynamic Left Ventricular Outflow Tract Obstruction is Not Unusual in Critically Ill Patients: An Assessment with Critical Care Echocardiography is Necessary for This Phenomenon

LVOTO was first described in patients with hypertrophic cardiomyopathy (HCM). LVOTO usually occurs because of systolic anterior movement of the anterior leaflet of the mitral valve and is characterized by a saber-shaped Doppler flow curve with late acceleration.[41] However, many clinical case reports indicate that critically ill patients without HCM may also develop LVOTO.[42] Recently, findings showed that LVOTO is not unusual in ICU patients, particularly in septic shock patients.[43-45]

LVOTO is currently considered to be a dynamic phenomenon, and the onset of LVOTO requires the coexistence of two elements: predisposing anatomical factors and a physiological condition that induces this type of phenomenon.[46] The anatomical substrates include LV hypertrophy in patients with HCM, hypertension or aortic stenosis, myocardial infarction, mitral valve replacement or repair, steep aortic root angle, and abnormalities of the mitral subvalvular apparatus, ACP, or AF. However, in a significant number of patients, no anatomical predisposition is identified. Functional abnormalities, such as decreased afterload, preload, or increased heart rate or contractility, can result in a small and hypercontractile LV, which predisposes to LVOTO. Clinical situations, such as hypovolemia, bleeding, and surgery with blood loss can cause a decreased preload with a small LV; pain arrhythmias, inotropic agents, and fever can lead to tachycardia; septic shock or anesthetic drugs can lead to vasoplegia; all the above are the precipitating factors of LVOTO. Catecholamines such as dobutamine, which increases LV contractility, reduce LVOT during systole and can also induce LVOTO.[47]

In conclusion, LVOTO is an underestimated dynamic phenomenon in ICU patients. Hypotension and low cardiac output syndrome caused by LVOTO do not respond to typical treatment or may even worsen following administration of positive inotropic and vasodilating agents. When the presence of LVOTO is confirmed, inotropic drugs should be stopped, or at least reduced, and fluid infusion may be useful particularly in septic shock. In addition, β-blockers should be considered if the clinical situation has not been improved.[48] Echocardiography with evaluation of LVOT is essential to diagnose LVOTO in ICU patients with shock.

Evaluation of Cardiac Function is the Basis for Assessment of Fluid Responsiveness

Volume expansion is the first-line treatment in the majority of cases of acute circulatory failure. Fluid is administered with the expectation that it will increase cardiac preload and cardiac output to a significant extent. Nevertheless, this can occur only if cardiac output is dependent upon cardiac preload, i.e., if both ventricles operate on the ascending limb of the cardiac function curve.[49] It is worth noting that the cardiac function curve greatly changes among different cardiac function statuses. When the ventricular function enhances, the same increase in cardiac preload induced by volume expansion can result in a significant increase in stroke volume, and in patients with poor ventricle function, the increase can be negligible depending upon the shape of the curve. The concept of fluid responsiveness is actually to assess preload dependency by observing the effects on cardiac output of changes in cardiac preload.[50]

Cardiac functional status can be quickly and correctly assessed by CCE and can predict the fluid responsiveness in the early

stage. Patients with good ventricular function are more likely to be fluid responsive and are more tolerant to volume, while patients with poor ventricular function are less likely to be fluid responsive and are prone to exert adverse effects after volume expansion. At this point, improving heart function by inotropic agents to recover the fluid responsiveness of patients should be an important consideration.

In addition, the popular indices for prediction of fluid responsiveness, such as arterial pulse pressure variation (PPV) and stroke volume variation, can also be influenced by cardiac function. PPV has been reported to be falsely positive, especially in patients with RV dysfunction,[51] and it is not a valid predictor of fluid responsiveness in patients with elevated LV filling pressure.[52] Furthermore, the variation of the IVC is unable to predict fluid responsiveness in the first 6 h after cardiac surgery, and complexity of physiologic factors modulating cardiac performance may be responsible.[53] These conditions indicate that the echocardiographic assessment of LV or RV function should be performed before the prediction of fluid responsiveness.

In conclusion, when using CCE to assess hemodynamic problems, we suggest following the direction of blood flow into and out the heart. As all the blood flows into the heart through the SVC and the IVC, the diameter and the respiratory variation of the IVC can indicate the volume status and the volume responsiveness. The right heart is the end point of the venous return, which is more sensitive to increase in afterload. The dilation of the RV can influence the ejection of the LV, which can be easily diagnosed by ultrasound using the RVEDA/LVEDA ratio and septal dyskinesia in the short axis. Any cardiac disease can cause LV diastolic dysfunction to a variable extent, and CCE can be used to either qualitatively recognize the diastolic dysfunction or assess the LV filling pressures indirectly. The LV systolic dysfunction in critically ill patients can be divided into diffuse hypokinesia and RWMA according to the regions involved. The etiology of LV systolic dysfunction can be differentiated correctly and fast, which can lead to proper treatment. LVOTO is not usual in critically ill patients, and echocardiography with the evaluation of LVOT is essential to diagnose LVOTO in ICU patients with shock. From here, we can see that with the aid of CCE, the blood flow in the heart becomes visual, the relationship between the volume status, right heart, left heart, LVOT, and blood vessels becomes clearer, and this will greatly promote the further development of hemodynamic therapy.

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Conflicts of interest

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