Echocardiography in a Patient on Mechanical Ventilation

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Abstract

Cardiopulmonary interactions or effects of spontaneous and mechanical ventilation (MV) were first documented in the year 1733. Stephen Hales showed that the blood pressure of healthy individual fell during spontaneous inspiration and he later went on to discover the ventilator. A year later Kussmaul described pulsus paradoxus (inspiratory absence of radial pulse) in patients with tubercular pericarditis. Echocardiography can help to diagnose a wide variety of cardiovascular diseases and can guide therapeutic decisions in patients on mechanical ventilation.

Introduction

Spontaneous and mechanical ventilation induce changes in intrapleural or intrathoracic pressure or lung volume. These changes can independently affect the cardiovascular performance i.e. atrial filling or preload, the impedance to ventricular emptying or afterload, heart rate or myocardial contractility. Echocardiographic examination is warranted in hemodynamic instability either due to ventricular failure, pulmonary embolism, hypovolemia, valvular dysfunction, cardiac tamponade or in patients on MV after cardiothoracic surgery. Other issues which need a good echocardiographic evaluation in such clinical setting include infective endocarditis, aortic dissection or rupture, cardioembolism and hypoxemia.

Influence of Ventilation on Cardiac Function

Changes in intrathoracic pressures are transmitted to the heart and pericardium, the great arteries and vains. Spontaneous inspiration produces a negative pleural pressure, the reduction in intrathoracic pressure is transmitted to right atrium. In contrast, intermittent positive pressure ventilation (IPPV) produces inspiratory increase in intrathoracic pressures thereby increasing the right atrial (RA) pressures and if positive end-expiratory pressure (PEEP) is added, these pressures will remain greater than atmospheric pressure throughout the respiratory cycle. Major compromise in cardiac output by impeding venous return may be seen in cases of septic shock and hypovolemia.¹

Hemodynamic Changes During Weaning

Cardiac-related weaning failure can be assessed by echocardiography. High risk weaning failure can be detected by low ejection fraction (LVEF), diastolic dysfunction and elevated LV filling pressures. LV filling pressures and progression of diastolic failure can be followed by transthoracic echocardiography (TTE). In patients with weaning failure, significantly lower heart rate and E/E’ ratio and higher LVEF are observed before spontaneous breathing test (SBT). During SBT, significant increase in cardiac output, systolic arterial pressure, E/A ratio relation and non-significant increase in E/E’ are observed with significant shortening of deceleration time (DTE). Percentage of weaning failure goes on increasing as LVEF declines. In simple terms, the patients with weaning failure are tachycardic, have a depressed systolic and diastolic function (shorter DTE) with elevated LV filling pressures (higher E/E’ ratio) before SBT and during SBT a further increase in LV filling pressure (increase E/A and E/E’ ratio) and deterioration of diastolic dysfunction is observed. Transthoracic echocardiography (TTE) is able to identify patients who are at risk of cardiac related weaning failure by measuring LVEF, E/E’ and DTE before SBT.² During SBT, hemodynamic changes i.e. increase in pulmonary capillary wedge pressure (PCWP) and changes in stroke volume can be followed up by TTE (Figure 1) Limitations include poor window, patients in atrial fibrillation and paced rhythm. Practical approach is to do a TTE before SBT, second SBT in cardiac-related weaning failure.

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should be closely monitored by pulmonary artery catheterization (PAC). Along with brain natriuretic levels (BNP), TTE can help to monitor and treat pulmonary edema induced by weaning.

Echocardiographic Assessment of Fluid Responsiveness During Mechanical Ventilation

A smaller LV cavity, systolic obliteration of LV cavity (kissing ventricles) signifies underfilling of heart and hypovolemia, and predicts augmentation of cardiac output (CO) in response to fluid challenge. LV end-diastolic area (LVEDA) in short axis at level of papillary muscle can be traced and can be followed up to trend the changes in response to fluid boluses where one is uncertain about the central venous pressure (CVP). Increase in the inferior vena cava (IVC) (Figure 2) diameter by 12-18% (variability and distensibility index) predicts positive response to fluid challenge and 36% distensibility of superior vena cava (SVC) with respiratory cycle on transesophageal echo (TEE) accurately predicts increase in CO with volume expansion in septic ventilated patients.\(^3\)

Distensibility index of IVC (Divc) is calculated by measuring the IVC at end-expiration (Dmin) and at end inspiration (Dmax) in subcostal view.

Divc is ratio of Dmax-Dmin / Dmin expressed in percentage.

Variability index is ratio of Dmax-Dmin /Dmean expressed in percentage.

Assessment of Right Ventricle in Mechanically Ventilated Patients

Direct measurement of right ventricle (RV) is not recommended. The RV should be smaller than LV. The RV:LV end-diastolic area ratio >0.6 indicates dilatation consistent with volume or pressure overload.\(^4\) RV dilatation with increase in tricuspid regurgitation from baseline echo and pulmonary arterial hypertension (PAH) may be seen in patients on positive pressure ventilation and should be considered as a differential diagnosis while evaluation for other causes like pulmonary embolism etc. RV function can be depressed in pulmonary embolism and acute respiratory distress syndrome (ARDS). Increased RV afterload may be seen in positive end-expiratory pressure (PEEP) or increased pulmonary vascular resistance from vascular, cardiac, metabolic or pulmonary causes. Inferior with RV myocardial infarction, acute sickle cell crisis, fat or air embolism, myocardial contusion and sepsis can lead to acute RV dysfunction.

Cardiac Output

Cardiac output varies by 50% with respiration in mechanically ventilated patients. Therefore, end-expiratory phase measurements should be taken into account. Maintaining a high CO has not shown to improve any outcomes but
a CO <5 L/min and cardiac index (CI) <2 L/min/m² significantly increase the mortality and suggest a poor prognosis5 (Figure 3).

Cardiac output (CO) = Stroke volume x Heart rate.

Stroke volume= End-diastolic volume (EDV) – End-systolic volume (ESV).

**Transthoracic vs. Transesophageal Echo in MV Patients**

Transesophageal echocardiography (TEE) is performed when TTE does not solve clinical problems and when TTE has shown some unsuspected findings which required to be confirmed and therapeutic decision needs to be taken. TEE is well tolerated imaging technique in MV patients for assessment of LV function and pericardial effusion, however TTE continues to be excellent diagnostic tool even when PEEP is present.

**Advantages vs Disadvantages: Echo vs Invasive Monitoring**

The advantages and disadvantages of echocardiography comparing invasive monitoring are discussed in Table 1.

**Echo Protocols in ICU**

• RACE (rapid assessment by cardiac echo)
  I. What is LV function?
  II. What is RV function?
  III. Is there any evidence of pulmonary embolism or cardiac tamponade?
  IV. What is fluid status?

• F A T E (f o c u s e d a s s e s s e d t r a n s t h o r c a c i c echocardiography)
  I. Rapid and systemic protocol for cardiopulmonary screening and monitoring.

The above two echocardiography protocols are widely accepted in high volume centers in ICU setting including mechanically ventilated patients. The RACE protocol will only answer the above questions while the FATE protocol is used when only a particular question by the intensivist needs to be answered and it need not look into the other aspects.

**Observations**

A larger left atria (LA) size has been observed in patients with repeated weaning failures. Patients with successful weaning have smaller sized cardiac chambers. Not much difference has been observed in echocardiographic parameters in patients with pressure support ventilation (PSV) and T-piece breathing. Unlike spontaneous breathing patient, IVC diameter and response to respiration is unvalidated in MV patients and some amount of ST-T changes and cardiac ischemia occurs during weaning which also contributes to weaning failure.

**Conclusion**

Hemodynamics in patient on MV are much different from physiological state. Echocardiography can help in both prognosis and follow up of patients on MV. Fluid responsiveness, CO, LV systolic and diastolic parameters with patients on positive pressure ventilation can well be followed up by a non-invasive method using echocardiography.

**References**