Commentary

Pulmonary artery occlusion pressure estimation by transesophageal echocardiography: is simpler better?

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See related research by Vignon *et al.*, http://ccforum.com/content/12/1/R18

Abstract

The measurement of pulmonary artery occlusion pressure (PAOP) is important for estimation of left ventricular filling pressure and for distinction between cardiac and non-cardiac etiology of pulmonary edema. Clinical assessment of PAOP, which relies on physical signs of pulmonary congestion, is uncertain. Reliable PAOP measurement can be performed by pulmonary artery catheter, but it is possible also by the use of echocardiography. Several Doppler variables show acceptable correlation with PAOP and can be used for its estimation in cardiac and critically ill patients. Noninvasive PAOP estimation should probably become an integral part of transthoracic and transesophageal echocardiographic evaluation in critically ill patients. However, the limitations of both methods should be taken into consideration, and in specific patients invasive PAOP measurement is still unavoidable, if the exact value of PAOP is needed.

Vignon and colleagues [1] prospectively assessed the ability of transesophageal echocardiography (TEE) to predict PAOP higher than 18 mmHg in mechanically ventilated patients with an inserted pulmonary artery catheter. In a first group, they analyzed simple Doppler variables derived from transmitral flow (TMF) and pulmonary venous flow (PVF) and performed the usual measurements and calculations (maximal velocity and velocity time integral of E (the maximal velocity of early diastolic TMF) and A (the maximal velocity of late diastolic TMF) wave, E/A ratio, E wave deceleration time (EDT), maximal velocity and velocity time integral of S (the maximal systolic PVF velocity) and D (the maximal diastolic PVF velocity) wave, S/D ratio, atrial filling fraction and systolic fraction of pulmonary venous flow (SFPVF)). TMF recording was inadequate for analysis in 10% of patients. The correlations between Doppler variables and pulmonary artery occlusion pressure (PAOP) were better in patients with depressed left ventricular (LV) systolic function than in those with normal LV systolic function. PAOP could be predicted by E/A >1.4, EDT >100 ms, atrial filling fraction >31% and SFPVF >44%, with similar sensitivity and specificity and acceptable positive and negative predictive values. In a second group these cutoff values were prospectively evaluated for prediction of PAOP higher than 18 mmHg. Additionally, they measured maximal early diastolic velocity of lateral mitral annulus by tissue Doppler (Ea) and color M-mode Doppler flow propagation velocity (Vp). An E/Ea ratio <8 and an E/Vp ratio <1.7 were predictive for PAOP >18 mmHg, but the use of these additional variables did not improve the correct estimation of PAOP.

Elevated PAOP reflects an increase of LV end-diastolic pressure due to LV diastolic and/or systolic dysfunction/failure. PAOP less than 18 mmHg, if measured, supports criteria for the definition of acute respiratory distress syndrome and acute lung injury.

Clinical and radiological estimation of PAOP is uncertain in cardiac patients and almost impossible in intensive care unit patients [2-5]. PAOP measurement by pulmonary artery catheter is, for various reasons, not commonly used in cardiac failure and critically ill patients. On the other hand, TEE and transthoracic echocardiography (TTE) are increasingly used for diagnostic and hemodynamic assessment and in critically ill patients, allowing noninvasive estimation of PAOP by Doppler technique [6]. Basically, two groups of Doppler variables are used. The first group includes relatively simple variables (E, A, E/A , EDT, SFPVF) derived from analysis of diastolic TMF and PVF. The second group includes Ea and Vp; both variables are preload independent and are used to

A = maximal velocity of late diastolic TMF; D = maximal diastolic PVF velocity; E = maximal velocity of early diastolic TMF; Ea = tissue Doppler diastolic velocity of mitral annulus; EDT = E wave deceleration time; LV = left ventricular; PAOP = pulmonary artery occlusion pressure; PVF = pulmonary venous flow; S = maximal systolic PVF velocity; SFPVF = systolic fraction of PVF; TEE = transesophageal echocardiography; TMF = transmitral flow; TTE = transthoracic echocardoigraphy; Vp = color M-mode Doppler flow propagation velocity.

correct the E velocity for relaxation changes (E/Ea and E/Vp ratio).

All variables can be derived by TTE and TEE. In older studies, use of TTE was limited because of inadequate visibility; many patients had to be excluded because of inadequate Doppler signal recordings [7,8]. Technical improvements and the use of harmonic imaging now allow measurement of TMF and PVF in the majority of patients, but TEE is still frequently used, especially in mechanically ventilated critically ill patients.

TMF and PVF variables measured by TTE are accurate for the estimation of LV filling pressure and cardiac index in patients with depressed cardiac function and heart failure, but in patients with normal systolic LV function tissue Doppler derived variables show better correlation with PAOP [9-11]. In patients who have undergone cardiac surgery and in critically ill patients, TEE-derived SFPVF and E/Ea correlate well with left atrial pressure and PAOP [12-14].

The study by Vignon and coworkers shows that in patients with acute lung injury, simple Doppler variables derived from TMF and PVF by TEE predicted elevated PAOP better than atrial filling fraction and EDT and that the use of additional and more advanced variables (Ea and Vp) did not improve the accuracy of prediction. An important practical limitation of the study is the fact that 20% of patients could not be studied because of cardiac problems, and that in a further 10% of patients, some variables could not be recorded.

Concerning the study, the following questions should be considered.

Should we still measure PAOP?

Despite the fact that PAOP is not transmural pressure and does not accurately reflect preload and volume responsiveness, it is still used as a supportive criterion for the diagnosis of acute respiratory distress syndrome and heart failure. PAOP is, therefore, still measured or estimated in routine clinical practice.

Can we estimate PAOP noninvasively?

Noninvasive estimation of PAOP is feasible by using TTE/TEE-derived simple Doppler variables, but not in every patient. Despite technological improvements in past years, adequate Doppler tracing can not be obtained by TTE in many critically ill patients. Also, TEE does not allow adequate recording of Doppler variables in all patients. Additionally, all echo measurements are subjective and require specific operator skill to interpret correctly. It would be interesting to compare TTE and TEE simultaneously for PAOP estimation in a large group of critically ill patients. Besides this, in a certain subset of patients, noninvasive estimation of PAOP is not possible and invasive measurement of PAOP, if needed, is still necessary.

Which variable should we use for noninvasive PAOP estimation?

Taking into account that TTE or TEE should be performed in the majority of intensive care unit patients for initial hemodynamic assessment, the systematic estimation of PAOP by simple analysis of TMF and PVF would undoubtedly increase the overall quality of this. The use of additional variables (Ea, Vp), which are routinely not measured in the intensive care unit setting, is not necessary for PAOP estimation in patients with impaired global systolic LV function, but can improve its estimation in patients with normal systolic function and diastolic dysfunction/failure.

Competing interests

The author declares that they have no competing interests.

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