Commentary

Estimation of pulmonary capillary pressure: different methods for different pathophysiological processes?

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See related research by Souza *et al.* in this issue [http://ccforum.com/content/9/2/R132]

Abstract

In the absence of a direct method with which to measure pulmonary capillary pressure in humans, various methods for analyzing the pulmonary artery pressure decay following balloon occlusion have been described. In this issue of *Critical Care*, Souza and coworkers investigate the adequacy of these methods for assessing various pathophysiological processes. They studied patients presenting with pathologies characterized by different distributions of pulmonary vascular resistance. Their findings suggest that no single method for estimating pulmonary capillary pressure is adequate for all disease processes.

In healthy individuals pulmonary vascular resistance is low and is mostly located within the pulmonary arterial bed. In the absence of significant venous resistance, pulmonary capillary pressure (PCP) is very close to the pulmonary arterial occlusion pressure. Under these circumstances, pulmonary arterial occlusion pressure may be used as a surrogate for the pressure determining filtration from the capillaries to the interstitium (PCP). However, this is no longer the case when pulmonary venous resistance is increased, as may occur in pulmonary hypertension of various aetiologies, acute lung injury (ALI) and acute respiratory distress syndrome (ARDS). In these patients PCP must be estimated by analyzing the pulmonary arterial pressure decay after pulmonary artery occlusion. Souza and coworkers [1] used this technique to estimate PCP by different methods in patients with idiopathic pulmonary arterial hypertension and with ARDS. These authors addressed the fundamental issue of whether the methods used to calculate PCP are adequate in different disease processes.

In idiopathic pulmonary arterial hypertension, the massive increase in pulmonary arterial resistance accounts for most of the pulmonary resistance, even though some degree of venous resistance is also present [2]. In these patients, the use of a biexponential approach for the analysis of the pulmonary arterial pressure decay after occlusion appears to be more suitable than a monoexponential approach. A biexponential fit provides an estimation of both the fast and slow components of the pulmonary pressure drop, and therefore avoids assumptions regarding the time needed for emptying of the arterial compartment, which is markedly prolonged in this pathophysiological situation [3]. A monoexponential fit starting from 200 ms after occlusion may still include part of the pressure trace resulting from emptying of the pulmonary arterial compartment, as was suggested by the findings of Souza and coworkers [1]. As discussed by those authors, extrapolation to 150 ms after occlusion for the calculation of PCP may for the same reasons also be inaccurate, with values up to 199 ms having been described in the experimental literature as being more accurate in these circumstances [4].

Monitoring of both extravascular lung water and PCP may help one to estimate the severity and avoid worsening of the hyperpermeability lung oedema in ALI and ARDS [5,6]. However, the three-compartment model of the pulmonary vasculature used by Baconnier and coworkers [7] as a basis for estimating PCP may be inadequate in this clinical context because of an increase in the resistance of the capillary compartment, which is not taken into consideration by the model. Moreover, this capillary resistance is not constant and may vary throughout the course of the disease because of the pathophysiological changes that characterize the disease process [8]. Taking these limitations into consideration, Collee and coworkers [9] reported successful use of this model in severe ALI. However, data on the behaviour of pulmonary artery pressure decay after balloon occlusion in different stages of ALI/ARDS are scarce [10]. It is reasonable to conclude that the three-compartment model may not be

ALI = acute lung injury; ARDS = acute respiratory distress syndrome; PCP = pulmonary capillary pressure.

suitable at every stage of disease and/or that the methods needed to estimate PCP may need to vary according to the changing patophysiological characteristics of the pulmonary vascular tree.

Estimation of the exact time of occlusion is a challenge during measurements of PCP in humans. Respiratory noise is easily avoided in artificially ventilated patients by maintaining an end-expiratory pause during pulmonary artery occlusion. A short period of apnoea may be possible in cooperative spontaneously breathing patients. The variability due to the cardiac cycle may be minimized by performing several occlusions and choosing only those that occur during systole [1]. Estimates of PCP based on these tracings have been shown to be more accurate than those derived from occlusions that occur during diastole [4]. However, the steepness of the pressure decay after occlusion during systole may lead to overestimation of PCP [9,11]. Although the start of occlusion can easily be identified during systole, the inflation of the pulmonary arterial catheter balloon is always relatively slow, and accuracy (at least with respect to the starting point of balloon inflation) could still be enhanced by the use of a superimposed nonoccluded pulmonary arterial pressure curve [9].

It is clear from the issues discussed above that estimation of PCP in clinical practice cannot be based on a single method. Further development of experimental models that are better able to mimic clinical conditions may help in identifying the most accurate methods with which to estimate PCP in each clinical condition. More extensive clinical data on the behaviour of the pulmonary vascular tree throughout different disease processes are also needed. Until then, estimations of PCP may still help with clinical decision making, but caution is needed while interpreting individual findings.

Competing interests

The author(s) declare that they have no competing interests.

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