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REVIEW

Replacing the pulmonary artery catheter by less invasive monitoring. Are we ready yet?

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Abstract - The goal of hemodynamic monitoring is to identify cardiovascular pathology, monitor resuscitation and determine when recovery has been achieved. The pulmonary artery catheter (PAC) has been used for over 40 years to accomplish those goals. PAC specific measures include cardiac output, intrathoracic vascular pressures and mixed venous O2 saturation (SvO2). Although newer less invasive devices are now available to monitor specific components of the profile given by the PAC, none can accurately estimate SvO2 or pulmonary arterial or venous pressures (as pulmonary artery occlusion pressure). However, cardiac output can be easily measured by many other devices including arterial wave form monitors and esophageal Doppler catheters and transthoracic echocardiography. To the extent that data useful for diagnosis and/or management can be made by these less invasive means, then the PAC should not be used. But in those settings where accurate measures of SvO2 and pulmonary vascular pressures are needed, the PAC is the only device that can do the job.

Keywords - Hemodynamic monitoring, clinical utility, minimally invasive monitoring, mixed venous oxygen saturation

Introduction

The pulmonary artery catheter (PAC) has been a fundamental hemodynamic monitoring tool in the daily practice of the intensive care units for almost 40 years [1]. During these years, the PAC has been widely used in critically ill patients for diagnostic and therapeutic guidance, and the benefit derived from its use was just assumed. It was difficult to argue that the PAC was not needed when prospective studies documented that bedside estimates of ventricular filling pressures were poor using bedside diagnostic clues [2]. Furthermore, several specific cardiovascular management approaches were developed to use these measured values. However, during the last years, the usefulness of the PAC has been under constant debate [3-5]. Moreover, when outcomes were assessed relative to PAC use, several studies consistently showed that the presence of a PAC did not improve survival of the patients [6-15]. In some of these studies, the presence of a PAC was actually associated with increased mortality [6-9]. In addition, recent technological advances have provided less invasive tools for cardiovascular monitoring, reinforcing the idea that the routine use of the PAC in critically ill patients has passed. However, we have also stressed that the PAC gives uniquely useful hemodynamic data, such as the mixed venous oxygen saturation (SvO_2) [16], and when used for this strength may prove useful in guiding resuscitation [17,18]. Nevertheless, the data on the opposite side is quite strong, but is it strong enough to support the disappearance of the PAC?

Rationale for hemodynamic monitoring

If one is discussing whether the PAC is useful or not, one must first understand the meaning and purpose of hemodynamic monitoring. In the critically ill patient, despite the origin of his disease, maintenance of oxygen supply to the tissues is of utmost importance, while a definitive or specific treatment is provided. Circulatory shock can be arbitrarily divided into four broad categories of hypovolemic, cardiogenic, obstructive and distributive shock. Each is treated different and each has a characteristically different set of hemodynamic values, collectively referred to as the hemodynamic profile [19]. This is one of the fundamental principles for diagnosis and management of acutely unstable patients as treated in the critical care or emergency departments. In this regard the PAC seems ideally suited to monitor shock because it measures cardiac output, SvO_2 and pulmonary artery occlusion pressure (Ppao), the three defining variables in most shock states.

The fundamental concept of hemodynamic monitoring-derived resuscitation is that the rapid reversal of tissue hypoxia by the correct treatment of the underlying process will improve outcome. In our daily acute care practice, we assume that hemodynamic monitoring is a natural extension of physical examination by quantifying the various aspects of the physical assessment. The type and degree of monitoring should depend on multiple aspects of the patient, such as severity, time-point of disease, and location. Essentially, monitoring is used to identify tissue hypoxia and to determine when tissue wellness has been restored. Critical illness results in a rise in metabolic demand, owing to increased catecholamine release, and the ability of the cardiovascular system to meet this demand and avoid tissue hypoxia correlates with survival [20]. Therefore, based on these arguments: the need to define the etiology of cardiovascular insufficiency and monitoring the response to therapy, the PAC has become a cornerstone monitoring device in the management of the patient in the intensive care unit (ICU).

Hemodynamic monitoring should be helpful in three specific ways:

1. Diagnostic tool: Recognition of specific hemodynamic profiles seen in disease processes
2. Assessing and monitoring treatment, specifically fluid-responsiveness and vasoactive drug titration

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3. Hemodynamic goals: The goal-directed therapy, based on physiological end-points.

Thus, the indication and uses of hemodynamic monitoring are varied. Once one has identified the need for hemodynamic monitoring, the choice of monitoring tool now emerges. If we are to remove the PAC from our tool kit, we must inquire which might be the reasons for replacing or discarding a tool that has been used over the past years as a gold standard in critically ill patients. There are two potential reasons to discard PAC use: the data acquired from the PAC is not useful in treating the patient, or the same information can be acquired from a less invasive and safer device. We previously published a list of criteria for the use of any hemodynamic monitoring device, which is duplicated in Table 1 [21]. Let us consider these two issues separately.

1. Is the PAC a useless hemodynamic monitoring tool?

From a clinical point of view, it seems reasonable to assert that the PAC provides useful information about the physiological status of the monitored patient, and thus, it should be useful and helpful to achieve a diagnosis, and guide therapy [22]. However, the lack of data proving that the use of PAC improves outcome in critically ill patients has been central in the debate, even though no other monitoring tool has provided that improvement in survival [23,24]. Clearly, no monitoring tool will be capable to improve patient outcome, unless coupled to a treatment that does. Therefore, it is that treatment, and how the tool helps in guiding that treatment, what will be responsible of the benefits regarding patient's survival [18], and not the tool itself. In fact, the PAC has been overused for many years, and this non-specific use might be responsible, at least in part, for the lack of efficacy pointed out in different studies [25,26].

Some studies have shown that PAC use in goal-directed therapies improves outcome. When resuscitation strategies were driven by PAC-measured hemodynamic variables, such as oxygen delivery (DO_2), cardiac index (CI), or SvO_2 [27-35], survival was increased and hospital length of stay decreased. In surgical patients, for instance, different pre-operative and post-operative hemodynamic optimization protocols, driven according to a DO_2 target, have proved to diminish mortality and post-operative complications [27-29,30-34]. Consequently, it seems that the PAC is, in fact, a useful tool, even capable of improving survival when coupled to specific physiologic endpoints in a treatment algorithm. Therefore, the problem of prior association studies of PAC use and outcome might rely on how, when, where, and on which patients the PAC was used. In the recent FACTT study [15] comparing Ppao vs. central venous pressure (CVP) for fluid management strategies, one would not expect to see a beneficial effect of the PAC because neither Ppao nor CVP are reliable measurements of preload-responsiveness, and might be even harmful when used for that purpose [36]. Similarly, one cannot conclude that PAC-guided therapy has no impact on outcome when a considerable part of the treatment group does not achieve the DO_2 endpoint [37]. The PAC has also shown a lack of benefit when used to guide resuscitation in the late stages of medical conditions, once organ failure has already developed [38-41], or when used in populations with low expected mortality rates [41]. Clearly, once organ injury has developed the potential for rapid reversal of organ dysfunction by increasing DO_2 is minimal.

In addition to the debate regarding outcome, the potential complications derived from the catheter insertion were pointed out to strengthen the arguments against the PAC. These complications can be summarized as: insertion-related local complications (hematoma, arterial puncture, pneumothorax), arrhythmias, pulmonary artery rupture, catheter-related infections, and thrombo-embolic events. Various studies have shown that complications derived from PAC are due primarily from a central vascular access placement (42-44). However, PAC use is associated with increased risk of infections (0.7-1.3% incidence of bacteremia) [44-46] and thrombotic complications during prolonged retention (>48 hours), increased risk of arrhythmias during insertion (although with a low incidence of serious arrhythmias, and without influence on patient outcome) [44,47], and very rare pulmonary artery rupture complications. According to these data, PAC insertion should be performed only in those patients in whom the information derived will be used to diagnose and treat existing or potential cardiovascular insufficiency. And PAC insertion should not be avoided because of safety concerns in critically ill and high-risk surgery patients where precise cardiovascular monitoring is needed. Thus, the PAC is a useful tool for monitoring cardiovascular status, and should represent an acceptable risk of complications when used properly in these specific populations.

2. Are there newer less invasive monitoring devices available that can obtain the same data as the PAC?

The PAC provides information on three different variables: intrathoracic intravascular pressures, blood flow measurements (cardiac output), and a tissue perfusion marker (SvO_2). The PAC allows the accurate measurement of these variables, and can monitor them in a continuous fashion. Furthermore, from cardiac output, SvO_2 and pulse oximetry (SpO_2) measures one can readily calculate DO_2 and VO_2. What devices can replace cardiac output, Ppao and SvO_2 measures?

2.1. Blood flow measurements.

Over the last years, a number of new techniques have developed in the area of cardiovascular monitoring, and the major advances have arrived from the arterial pressure pulse contour analysis.

Table 1. Tests for documentation of effectiveness of invasive monitoring

1. Information received from invasive monitoring can not be acquired from less invasive and less risky monitoring systems.
2. Information received from invasive monitoring allows for improving the accuracy of diagnosis, prognosis, and/or treatment based on known physiological principals.
3. The changes in diagnosis and/or treatment result in an improved patient outcome (morbidity and mortality).
4. The changes in diagnosis and/or treatment result in a more effective use of health care resources.

*from Bellomo R, Pinsky MR. Invasive Monitoring. In: Tinker J, Browne D, Sibbald W (eds.), Critical Care - Standards, Audit and Ethics, Arnold Publishing Company, London, pp. 82-104, 1996 [20].

Several devices use different algorithms to derive continuous flow measurements (CO) from this analysis [48-50]. Currently, the widely available devices are the LIDCO®, the PICCO®, and the FloTrac-Vigileo®. Either, the LIDCO® and the PICCO® require a venous central line, an arterial line and initial calibration (according to the thermodilution principle), whereas the FloTrac-Vigileo® only requires an arterial line to derive the CO, and no initial calibration. Nevertheless, one device, the FloTrac-Vigileo® system, has poor reliability in unstable conditions [51]. In addition to pulse contour analysis, esophageal Doppler monitoring of aortic blood flow has been proposed as a reliable technique to evaluate CO [52,53]. And recently, a new device based on chest bioreactance has demonstrated its reliability for measuring CO non-invasively in post-surgical patients [54]. This is a promising technology, although there is a lack of data regarding its reliability in unstable critically ill patients yet. Therefore, today there exist reasonable less invasive to non-invasive alternatives to the PAC in monitoring cardiac output.

2.2. Intrathoracic intravascular pressures measurement.

The properly positioned PAC provides pressures from three different locations: the right atrium (CVP), the pulmonary artery (Ppa), and the pulmonary veins - the so called "wedge", or occlusion, pressure (Ppao). Since the CVP can be also monitored with a central venous access, we will focus on the pulmonary artery pressure (PAP) and Ppao. Originally, the PAC was developed to allow the measurement of the Ppao, which estimates the pulmonary venous pressure downstream from the alveolar capillary bed. And still today, Ppao remains as the best estimate of the pulmonary venous pressure at the bedside, a useful measurement that has no practical alternative. The main clinical applications of the Ppao are summarized in table 2.

Some of the applications shown in table 2, such as diagnosis of structural cardiac disorders or preload-responsiveness assessment, can be achieved with alternative tools (echocardiography and pulse-contour analysis, respectively). In fact, Ppao displays remarkably poor accuracy in the evaluation of fluid-responsiveness [24]. We do not recommend that one use Ppao values to predict response to fluid resuscitation. Here, variables obtained from the arterial waveform analysis (such as pulse pressure variation) have shown more reliability in predicting preload-responsiveness [36]. However, the Ppao is still the best aid in the diagnosis of the origin of pulmonary hypertension, and the differentiation between cardiogenic and non-cardiogenic pulmonary edema. Measures of Ppao are commonly used to determine the cause of pulmonary edema. It has been stated that Ppao values below 18-20 mm Hg suggest a non-hydrostatic cause, whereas values above 18-20 mm Hg suggest a hydrostatic cause [55]. Nevertheless, these are not hard values, and one can find low values in patients with hydrostatic pulmonary edema if either the Ppao increase had been transient, or if pulmonary capillary pressure exceeds Ppao [56]. Furthermore, increased pleural pressure (hyperinflation, i.e.) can lead to artificially elevated Ppao values in a patient without hydrostatic pulmonary edema, since the Ppao is measured relative to atmospheric pressure [57]. Regarding the application in the differential diagnosis of pulmonary edema, the utility of the PAC is not limited to its diagnostic aid, being also a useful variable in its management. Therefore, the Ppao is a physiological variable not measurable by any other means and useful in the limited cases described above. Thus, to the extent that the accurate diagnosis of pulmonary edema or the management of pulmonary hypertension requires close titration, there is no substitute for the PAC.

Table 2. Clinical applications of the Ppao

1. Diagnosis of cardiovascular disorders		
	.by pressure analysis.	.differentiate pulmonary HT due to increased vascular resistance from pulmonary venous HT (left heart)
	.by waveform analysis.	.Acute Mitral valve regurgitation.
		.Constrictive pericarditis and restrictive cardiomyopathy.
	.by blood gas analysis.	.inter-auricular and inter-ventricular communications.
2. Diagnosis and management of pulmonary edema.		
3. Evaluation of preload and preload-responsiveness.		

2.3. Mixed venous oxygen saturation.

Measurement of SvO_2 has been advocated as a reflection of global tissue perfusion. In several diseases, low values of SvO_2 have been related to poor prognosis [35,58,59]. Similarly, central venous oxygen saturation ($ScvO_2$) has been proposed as a simple method to assess the adequacy of global tissue perfusion in different clinical scenarios [60,61]. However, whether the $ScvO_2$ mirrors SvO_2 has been under constant debate, especially in critically ill patients. Nevertheless, Rivers and coworkers, using $ScvO_2$ to titrate resuscitation efforts in early septic shock, documented an improved outcome [62]. Despite the differences between SvO_2 and $ScvO_2$ during shock, various studies have shown that both measurements tend to change in a parallel manner, with higher values of $ScvO_2$ when compared to SvO_2 when blood flow distribution is unaltered. If flow to the lower body is reduced, then a proportionally higher flow will go to the upper body, which is sampled by $ScvO_2$. In these settings $ScvO_2$ may be very much higher than SvO_2 and give the false impression of perfusion adequacy despite overt shock. Therefore, $ScvO_2$ is a reasonable surrogate of SvO_2 in many clinical settings, and during shock states, if used to define a low SvO_2 not to determine whether resuscitation goals have been achieved [63,64].

Thus, there are less-invasive reliable alternatives to some of the features that PAC provides, mainly regarding blood flow and SvO_2 measurements. However, there is no practical alternative to the Ppao measurement and applications. Therefore, unless specific Ppao values are needed to diagnose disease and guide therapy, the routine use of the PAC cannot be recommended. In these conditions, less invasive monitoring tools should be used. However, one major caveat exists here as well. With the exception of the LiDCO® device

as a post-operative resuscitation guide [65], none of these less invasive monitoring tools has been shown to improve outcome. The PAC may provide a better understanding of the etiology of disease and closer guide to its management in certain clinical situations where cardiac and intravascular pressures are important, such as cardiac surgery, severe heart failure, or ARDS. Although, its benefit is lost in the DO_2-optimization trials, which can be easily run using the less invasive monitoring tools.

In addition, to examine the ability of new technologies to provide variables obtained from the PAC, we should also consider the capability of these new systems to obtain additional variables, such as pulse pressure variation (PPV), stroke volume variation (SVV), extravascular lung water (EVLW), and intrathoracic blood volume (ITBV). These are extra assets available at the bedside that can be useful in the decision-making process. The value of PPV and SVV obtained from pulse-contour analysis systems to predict preload-responsiveness has been widely proven in several conditions [36], and their use has become essential in the fluid management of critical care patients. Moreover, intra-thoracic volume measurements (EVLW, ITBV) obtained from the thermal-dilution technique (PICCO®) might prove useful in selected clinical conditions, such as EVLW in the fluid management of patients with ARDS [66,67]. However, to date, no studies have documented the utility of EVLW and ITBV measures in the diagnosis or management of critically ill patients.

Conclusions

The fact that the use of the PAC has fallen over the last years [5] does not mean that the information it provides is useless. Although clinical practice needed supporting data from well-designed clinical trials, in the absence of such trials, reliance on physiological principles remains the most common rationale for monitoring and treatment. Not always will there be an algorithm showing the path the clinician has to follow to succeed in his patient management to improve patient outcome. That precise resuscitation algorithms benefit in selected patient groups cannot be extrapolated to the rest of the critically ill population. Often the bedside clinician has to deal with complex patients, and has to decide which degree of cardiovascular monitoring is the best for that particular situation. For those patients, the use of the PAC may be warranted if baseline and changing hemodynamic profile data are needed.

Thus, the PAC is a useful hemodynamic monitoring tool in the care of selected critically ill patients. Its use should be limited to those patients in whom a specific rationale for the information derived form the PAC can be given. There are reliable less-invasive alternatives to the PAC placement in many clinical scenarios. Finally, it is important that the clinician becomes familiar with different monitoring tools, and decides the specific monitoring system for a given patient.

References

1.- Swan HJC, Ganz W, Forrester JS, Marcus H, Diamond G, Channette D. Catheterization of the heart in man with the use of a flow-directed balloon tipped catheter. N Engl J Med 1970; 283: 447-451.

2.- Morris AH, Chapman RH, Gardner RM. Frequency of wedge pressure errors in the ICU. Crit Care Med 1985; 13: 705-708.

3.- Robin ED. The cult of the Swan-Ganz catheter. Overuse and abuse of pulmonary flow catheters. Ann Int Med 1985; 103: 445-449.

4.- Dalen JE, Bone RC. Is it time to pull the pulmonary artery catheter? JAMA 1996; 276: 916-918.

5.- Wiener RS, Welch HG. Trends in the use of the PAC in the United States, 1993-2004. JAMA 2007; 298: 423 -429.

6.- Zion MM, Balkin J, Rosenmann D, Goldbourt U, Reicher-Reiss H, Kaplinsky E, Behar S. Use of pulmonary artery catheters in patients with acute myocardial infarction. Chest 1990; 98: 1331-1335.

7.- Gore JM, Goldberg RJ, Spodick DH, Alpert JS, Dalen JE. A community-wide assessment of the use of pulmonary artery catheters in patients with acute myocardial infarction. Chest 1997; 92: 721-727.

8.- Connors AF Jr, Speroff T, Dawson NV, et al: for the SUPPORT Investigators. The effectiveness of right heart catheterization in the initial care of critically ill patients. JAMA 1996; 276: 889-897.

9.- Hayes MA, Timmins AC, Yau EHS, et al. Elevation of systemic oxygen delivery in the treatment of critically ill patients. N Engl J Med 1994; 330: 1717-1722.

10.- Polanczyk CA, Rohde LE, Goldman L, Cook EF, Thomas EJ, Marcantonio ER, Mangione CM, Lee TH. Right heart catheterization and cardiac complication sin patients undergoing noncardiac surgery: an observational study. JAMA 2001; 286: 309-314.

11.- Murdoch SD, Cohen AT, Bellamy MC. Pulmonary artery catheterization and mortality in critically ill patients. Br J Anaest 2000; 85: 611-615.

12.- Richard C, Warszawski J, Anguel N, et al. Early use of the pulmonary artery catheter and outcomes in patients with shock and acute respiratory distress syndrome: a randomized controlled trial. JAMA 2003; 290: 2713-2720.

13.- Rhodes A, Cusack RJ, Newman PJ, et al. A randomized, controlled trial of the pulmonary artery catheter in critically ill patients. Intensive Care Med 2002; 28: 256-264.

14.- Harvey S, Harrison DA, Singer M, et al. Assessment of the clinical effectiveness of pulmonary artery catheters in management of patients in intensive care (PAC-Man): a randomized controlled trial. Lancet 2005; 366: 472-477.

15.- The National Heart, Lung and Blood Institute Acute Respiratory Distress Syndrome (ARDS) Clinical trials Network. Pulmonary artery versus central venous catheter to guide treatment of acute lung injury. N Engl J Med 2006; 354: 2213-2224.

16.- Kandel G, Aberman A. Mixed venous oxygen saturation. Its role in the assessment of the critically ill patient. Arch Intern Med 1983; 143: 1400-1402.

17.- Pinsky MR, Vincent JL. Let us use the pulmonary artery catheter correctly and only when we need it. Crit Care Med 2005; 33: 1119-1122.

18.- Vincent JL, Pinsky MR, Sprung C, Levy M, Marini JJ, Payen D, Rhodes A, Takala J. The pulmonary artery catheter: in medio virtus. Crit Care Med 2008; 36: 3093-3096.

19.- Pinsky MR. Hemodynamic Profile Interpretation. In: Tobin MJ (ed.), Principles and Practice of Intensive Care Monitoring. McGraw-Hill, New York. Pp. 871-888, 1998.

20.- Shoemaker WC, Appel PL, Kram HB. Hemodynamic and oxygen transport responses in survivors and nonsurvivors of high-risk surgery. Crit Care Med 1993; 21: 977-990.

21.- Bellomo R, Pinsky MR. Invasive Monitoring. In: Tinker J, Browne D, Sibbald W (eds.), Critical Care - Standards, Audit and Ethics, Arnold Publishing Company, London, pp. 82-104, 1996.

22.- Wiedemann HP, Matthay MA, Matthay RA. Cardiovascular-pulmonary monitoring in the intensive care unit (Part 1). Chest 1984; 85: 537-549.

23.- Ospina-Tascon GA, Cordioli RL, Vincent JL. What type of monitoring has been shown to improve outcomes in acutely ill patients. Intensive Care Med 2008; 34: 800-820.

24.- Pinsky MR. Hemodynamic evaluation and monitoring in the ICU. Chest 2007; 132: 2020-2029.
25.- Vincent JL, Bihari D, Suter PM, et al. The prevalence of nosocomial infection in intensive care units in Europe. Results of the European Prevalence of Infection in Intensive Care (EPIC) study. EPIC International Advisory Committee. JAMA 1995; 274: 639-644.
26.- Sakr Y, Vincent JL, Reinhart K, et al. Use of the pulmonary artery catheter is not associated with worse outcome in the intensive care unit. Chest 2005; 128: 2722-2731.
27.- Schultz RJ, Whitfield GF, Lamura JJ, et al. The role of physiologic monitoring in patients with fractures of the hip. J Trauma 1985; 25: 309-316.
28.- Shoemaker WC, Appel PL, Kram HB, et al. Prospective trial of supranormal values of survivors as therapeutic goals in high-risk surgical patients. Chest 1988; 94: 1176-1186.
29.- Tuchschmidt J, Fried J, Astiz M, Rackow E. Elevation of cardiac output and oxygen delivery improves outcome in septic shock. Chest 1992; 102: 216-220.
30.- Boyd O, Grounds M, Bennet ED. A randomized clinical trial of the effect of deliberate perioperative increase of oxygen delivery on mortality in high risk surgical patients. JAMA 1993; 270: 2699-2707.
31.- Bishop M, Shoemaker WC, Appel PL, et al. Prospective randomized trial of survivor values of cardiac index, oxygen delivery, and oxygen consumption as resuscitation endpoints in severe trauma. J Trauma 1995; 38: 780-787.
32.- Yu M, Burchell S, Hasaniya N, et al. Relationship of mortality to increasing oxygen delivery in patients > or = to 50 years of age: a prospective randomized trial. Crit Care Med 1998; 26: 1011-1019.
33.- Wilson J, Woods I, Fawcett J, et al. Reducing the risk of major elective surgery: randomized controlled trial of preoperative optimization of oxygen delivery. BMJ 1999; 318: 1099-1103.
34.- Lobo SMA, Salgado PF, Castillo VGT, et al. Effects of maximizing oxygen delivery on morbidity and mortality in high risk surgical patients. Crit Care Med 2000; 28: 3396-3404.
35.- Polonen P, Ruokonen E, Hippelainen M, Poyhonen M, Takala J. A prospective, randomized study of goal oriented hemodynamic therapy in cardiac surgical patients. Anaesth Analg 2000; 90: 1052-1059.
36.- Michard F, Teboul JL. Predicting fluid responsiveness in ICU patients: a critical analysis of the evidence. Chest 2002; 121: 2000-2008.
37.- Sandham JD, Hull RD, Brant RF, et al. A randomized controlled trial of the use of pulmonary artery catheters in high risk surgical patients. N Engl J Med 2003; 348: 5-14.
38.- Gattinoni L, Brazzi L, Pelosi P, et al. A trial of goal-oriented hemodynamic therapy in critically ill patients. N Engl J Med 1995; 333: 1025-1032.
39.- Alia I, Esteban A, Gordo F, et al. A randomized and controlled trial of the effect of treatment aimed at maximizing oxygen delivery in patients with severe sepsis or septic shock. Chest 1999; 115: 453-461.
40.- Yu M, Levy MM, Smith P, et al. Effect of maximizing oxygen delivery on mortality and mortality rates in critically ill patients: A prospective randomized controlled study. Crit Care Med 1993; 21: 830-838.
41.- Kern JW, Shoemaker WC. Meta-anlysis of hemodynamic optimization in high-risk patients. Crit Care Med 2002; 30: 1686-1692.
42.- Boyd KD, Thomas SJ, Gold J, Boyd AD. A prospective study of complications of complications of pulmonary artery catheterizations in 500 consecutive patients. Chest 1983; 84: 245-249.
43.- Ducatman BS, McMichan JC, Edwards WD. Catheter-induced lesions of the right side of the heart. A one-year prospective study of 141 autopsies. JAMA 1985; 253: 791-795.
44.- Hadian M, Pinsky MR. Evidence-based review of the use of the pulmonary artery catheter: impact data and complications. Crit Care 2006; 10: Suppl 3-S8.
45.- Elliott CG, Zimmerman GA, Clemmer TP. Complications of the pulmonary artery catheterization in the care of critically ill patients. A prospective study. Chest 1979; 76: 647-652.
46.- Mermel LA, McCormick RD, Springman SR, Maki DG. The pathogenesis and epidemiology ofcatheter-related infection with pulmonary artery Swan-Ganz catheters: a prospective study utilizing molecular subtyping. Am J Med 1991; 91: 197S-205S.
47.- Sprung CL, Jacobs LJ, Caralis PV, Karpf M. Ventricular arrhythmias during Swan-Ganz catheterization of the critically ill. Chest 1981; 79: 413-415.
48.- Goedje O, Hoeke K, Lichtwarck-Aschoff M, et al. Continuous cardiac output ny femoral arterial thermodilution calibrated pulse contour analysis: comparison with pulmonary arterial thermodilution. Crit Care Med 1999; 27: 2407-2412.
49.- Goedje O, Hoeke K, Goetz AE, et al. Reliability of a new algorithm for continuous cardiac output determination by pulse contour analysis during hemodynamic instability. Crit Care Med 2002; 30: 52-59.
50.- Kurita T, Morita K, Kato S, et al. Comparison of the accuracy of the lithium dilution technique with the thermodilution technique for measurement of cardiac output. Br J Anaesthesiol 1997; 79: 770-775.
51.- Opdam HI, Wan L, Bellomo R. A pilot assessment of the FloTrac cardiac output monitoring system. Intensive Care Med 2007; 33: 344-349.
52.- Singer M, Clarke J, Bennett D. Continuous hemodynamic monitoring by esophageal Doppler. Crit Care Med 1989; 17: 447-452.
53.- Valtier B, Cholley BP, Belot JP, et al. Noninvasive monitoring of cardiac output in critically ill patients using transesophageal Doppler. Am J Respir Crit Care Med 1998; 158: 77-83.
54.- Squara P, Denjean D, Estagnaise P, et al. Noninvasive cardiac output monitoring (NICOM): a clinical validation. Intensive Care Med 2007; 33: 1191-194.
55.- Fishman AP. Pulmonary circulation. In: Handbook of physiology. The Respiratory system. Circulation and nonrespiratory functions, vol 1. American Physiological Society, Bethesda, pp 93-166, 1985.
56.- Bindels AJ, van der Hoeven JG, Meinders AE. Pulmonary artery wedge pressure and extravascular lung water in patients with acute cardiogenic pulmonary edema requiring mechanical ventilation. Am J Cardiol 1999; 84: 1158-1163.
57.- Pinsky MR. Clinical significance of pulmonary artery occlusion pressure. Intensive Care Med 2003; 29: 175-178.
58.- Kasnitz P, Druger GL, Yorra F, Simmons DH. Mixed venous oxygen tension and hyperlactatemia. Survival in severe cardiopulmonary disease. JAMA 1976; 236: 570-574.
59.- Krafft P, Steltzer H, Hiesmayr M, et al. Mixed venous oxygen saturation in critically ill septic shock patients. The role of defined events. Chest 1993; 103: 900-906.
60.- Reinhart K, Radermacher P, Sprung CL, et al. PA catheterization: quo vadis? Do we have to change our current practice with this monitoring device?. Intensive Care Med 1997; 23: 605-609.
61.- Dalen JE, Bone RA. Is it time to pull the pulmonary artery catheter? JAMA 1996; 276: 916-918.
62.- Rivers E, Nguyen B, Havstad S, et al. Early goal-directed therapy in the treatment of severe sepsis and septic shock. N Egl J Med 2001; 345: 1368-1377.
63.- Reinhart K, Rudolph T, Bredle DL, et al. Comparison of central venous to mixed venous oxygen saturation during changes in oxygen supply/demand. Chest 1989; 95: 1216-1221.
64.- Martin C, Auffray JP, Badetti C, et al. Monitoring of central venous oxygen saturation versus mixed venous oxygen saturation in critically ill patients. Intensive Care Med 1992; 18: 101-104.
65.- Pearse R, Dawson D, Fawcett J, et al.: Early goal-directed therapy after major surgery reduces complications and duration of hospital stay. Critical Care 2005; 9: 687-693.
66.- Mitchell JP, Schuller D, Calandrino S, Schuster DP. Improved outcome based on fluid management in critically ill patients requiring pulmonary artery catheterization. Am Rev Respir Dis 1992; 145: 990-998.
67.- Groeneveld ABJ, Verheij J. Methodology and value of assessing Extravascular Lung Water. In: Functional hemodynamic monitoring. Pinsky MR, Payen D (Eds.), Update in intensive care and emergency medicine, Springer-Verlag, Berlin Heidelberg, pp 165-173, 2005.