Pa Catheter Controversy Continues, Why?: Part I

J Civetta

Citation

J Civetta. *Pa Catheter Controversy Continues, Why?: Part I*. The Internet Journal of Anesthesiology. 1998 Volume 3 Number 2.

Abstract

In the late 70s, I wrote:

Cardiovascular physiology in the Intensive Care Unit represents one of the areas in which the bedside technology and basic physiologic principles learned in the laboratory can be combined to produce an effect that influences patient care daily. We usually use a very simple analogy derived from direct current electricity in which we look at flow, pressure and resistance in the cardiovascular system as being similar to amperes, volts, and ohms in the direct current electrical analogy. The flow in the circuit is cardiac output, and this produces a specific blood pressure at a particular peripheral resistance. Cardiac output is the product of the volume per beat or stroke volume times the number of beats per minute or heart rate. The patient with a normal heart increases heart rate in order to increase cardiac output. This is effective up to a rate of approximately 150 beats per minute. Above this rate, the stroke volume actually decreases because of the decrease in diastolic filling time, cardiac output no longer increases but falls. However, this mechanism cannot be used by the patient with a failing heart. If the heart rate is increased there is, in fact, a decrease in cardiac output initially because the stroke volume is compromised. So another mechanism must be used to increase cardiac output. This mechanism involves stretching the cardiac muscle fiber and was described as the Frank Starling mechanism which says that the energy of contraction is proportional to the initial length of the cardiac muscle fiber. How can a clinical estimation of myocardial fiber length be made? It is obvious that fiber length cannot be measured directly, so we must resort to some indirect measurement. There is a proportionality between fiber length, left ventricular end diastolic volume and left ventricular end diastolic pressure. This is not an equality but a proportionality. This proportionality is different in normal and abnormal states. How do we now measure filling pressures? The Swan Ganz flow directed pulmonary artery catheter introduced in 1970 indirectly measures left atrial pressure through a balloon-tip catheter introduced via the central venous circulation. This provides one element necessary to construct a ventricular function curve. Diastolic filling pressure or pulmonary capillary wedge pressure can be used as an estimation of fiber length and this is plotted against the stroke work produced when the heart contracts. In order to calculate stroke work, measurement of cardiac output and calculation of stroke volume are required, so the clinical monitoring of cardiovascular competence requires direct measurement of cardiovascular parameters. A normal ventricular function curve shows that at low filling pressures, small increases will produce dramatic increases in the work produced on contraction. However, when the heart becomes stretched, even large increases in filling pressure will only produce small further increases in stroke work. In the intensive care populations these relationships do not hold to quite the same degree. In fact, Ross and Braunwald, some years ago, described patients with cardiovascular function as either being normal, compromised, or failing and we can approximate this in a mathematical way. A failing heart, in other words, describes a patient who has symptoms even at rest: it means that the work required for the basal metabolic functions of the body could not be produced at any filling pressure. However, a compromised heart would be revealed under stress because the increased work needed to compensate for the stress could not be generated at any filling pressure. But the important factor to remember is that stress, surgery, or trauma can produce a situation in which the compromised heart is revealed and the required work can no longer be done. In these cases, interventions are possible with respect to preload, contractility, and afterload. There are limitations using preload as an interventions which can be seen by constructing a ventricular function curve in the patient with a compromised heart. Data has now been collected from patients with acute myocardial infarctions, after cardiac surgery, and in stressed postoperative patients.

There is a very consistent range of filling pressures considered “optimal” - 13 to 17 mm Hg.

The second intervention relates to contractility. Agents that increase contractility increase the force of contraction and eject a larger stroke volume. However, this increase generates increased myocardial oxygen consumption. A balance between the two must be carefully chosen.

The third major intervention that can be manipulated is afterload or the impedance which the heart must face. One can consider afterload in terms of a very simple analogy. In the early days of vasodilator therapy, in fact, this analogy was considered to represent the primary effect of afterload reduction. The heart was similar to a man unable to lift up a very large number of very heavy packages. But if the load is divided, then the man could easily pick up one of these packages. So, in terms of the heart, if the impedance to left ventricular ejection was so high because of increased systemic vascular resistance then, in fact, the heart would fail. If the workload was decreased by vasodilators, the heart then could function more effectively. In fact, afterload has more effects and is one of the most interesting areas of interventions because it is effective in treating critically ill patients and it requires an understanding of so many cardiovascular principles which have become increasingly important as myocardial energenetics have been investigated.

That is an excerpt from an abstract written for a CME meeting. For those of us who received our training before the introduction of the Swan Ganz catheter, this new found ability to estimate left arterial pressure, measure cardiac output and perform various calculations at the bedside seem almost miraculous. I choose that word with care because miracles cannot be explained. I had cared for many patients who were hypotensive and oliguric, giving many fluid boluses, slavishly measuring the CVP, only stopping when the CVP rose to the twenties yet the clinical condition was not improved. Imagine my surprise and delight when many of these patients, after pulmonary artery catheterization became available, turned out to have very low wedge pressures and responded favorably to further fluid administration. In fact, an increase in wedge pressure was often associated with a decrease in the CVP - - but who could have known? Prior to 1970, the diffuse bilateral infiltrates of ARDS were often treated with diuretics. But if the wedge pressure was 2 and the cardiac output was also 2, fluids not diuretics became the treatment of choice. When potent vasodilators were administered, precipitous hypotension resulted – if the clinician did not happen notice that the wedge pressure was very low and that adequate intravascular volume replacement was necessary before treating the increased systemic vascular resistance. Having this new tool forced us to go back and review cardiovascular physiology, whose principles had been learned during second year of Medical School and had not been disturbed since! In fact, my first years as an academic practitioner coincided with the introduction of the Swan Ganz catheter and I amassed many frequent flyer miles reiterating at CME meetings the long established principles of cardiovascular physiology that now had practical applicability at the bedside. There were no controversies except perhaps which was the best route to obtain access, how to keep the site sterile and other details of maintenance. The advent of the new monitoring tool, then, provided a stimulus to study, learn and apply physiology. In this case, it was cardiovascular physiology; however today there are two other monitoring techniques that require the same dedication and study in order to use them effectively. The first is the Bicore pulmonary function monitor and the second is gastric intramucosal pH monitoring. In the first case, the student must wade through the difficult field of work of breathing and in the second, the student quickly becomes overwhelmed by the need to understand gastric physiology, splanchnic circulation, the origins of multiple organ system failure and ischemia-reperfusion injury.

Perhaps the reason for continued PA catheter controversy is the unwillingness of practitioners to become students again, that is, to spend the time and effort to ensure that the tool is used correctly, the information is accurate and the underlying physiology is understood. But no one noticed that the lack of this information was one of the necessary prerequisites for utilizing a new monitoring tool for over 15 years.

References

Author Information

Joseph M. Civetta, M.D.
Professor and Chairman, Department of Surgery, University of Connecticut Health Center