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EDITORIAL

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More respect for the CVP

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In 1970, Swan and Ganz introduced the flotation catheter for the measurement of pulmonary artery wedge pressure (PAWP) [1] and assessment of the filling pressure or preload of the left heart. Since that time clinicians have used PAWP to evaluate patients' volume status and to optimize preload to improve cardiac output. It is reasoned that cardiac output ultimately depends on ejection of blood from the left heart and, based on the Frank-Starling relationship, the better the filling of the left heart, the better the forward output. Furthermore, since left heart dysfunction can occur without major right heart dysfunction, it is argued that it is important to evaluate left-sided rather than right-sided pressures when determining optimal cardiac filling. This reasoning, however, ignores some important physiological considerations and can potentially lead to errors in clinical management.

Firstly, in the steady state, cardiac output must equal venous return. Therefore, a decrease in cardiac output means that there is a decrease in venous return. Secondly, it needs to be appreciated that the heart does not control cardiac output by creating an arterial pressure which pushes the blood around the body, but rather maintains cardiac output by lowering right atrial pressure and allowing blood to drain back to the heart so that it can be pumped out again [2–4]. We have recently used the analogy of a bathtub to explain this [5]. The flow out of a bathtub is determined by the height of water in the tub and the characteristics of the drain, but the flow out of the tub is not affected over the short run by the flow of water into the tub. This is because the flow from the tap can only increase the flow out of the tub by increasing the height of water in the tub, and the surface of the tub is very large compared to the area of the tap. The height of water in the tub is the equivalent of the pressure in the small venules and veins. Thus, just as the pressure in the tap does not push water out of a bathtub, arterial pressure does not push blood out of veins and therefore has no effect on venous drainage. The real role of the heart in regulating cardiac output is to lower right atrial pressure and allow better drainage from the compliant veins and venules. This also means that it is not left heart function and left atrial pressure which are the major determinants of cardiac output, but rather right heart function and right atrial pressure. A decrease in cardiac output, and thus venous return, whether it is due to left or right heart dysfunction, must be associated with a rise in right arterial pressure, which decreases venous return. The right arterial pressure, or central venous pressure, is thus a major determinant of cardiac output.

A third point that needs to be considered is that there is a plateau to the cardiac function curve (i.e. relationship of cardiac output to preload) and once that plateau is reached, further increases in cardiac filling pressure will not increase output. For this analysis, overall cardiac function can be examined from the relationship of the output from the left heart (i.e. what goes out) to the pressure in the right atrium (what goes in). Of course, in the steady state, what goes out of the left heart also goes out of the right heart. The right atrial pressure thus provides a measure of the filling pressure for the whole heart [2–4].

The important point is that there is no "left-sided success" without "right-sided success", and once the right heart is on the flat part of its function curve, then filling status of the left heart no longer determines cardiac output. It also needs to be appreciated that the plateau of the right heart function curve occurs normally at a right atrial pressure of between 6 and 12 mm Hg, although the value is sometimes higher [6]. Failure to appreciate that there is a limit to right heart filling, and continued use of fluid therapy to try and reach a target PAWP, results in excess fluid use, and might partly explain the failure of the Swan-Ganz catheter to be useful clinically [7].

An example will help illustrate the significance of these points. A common observation is a patient who has undergone cardiac surgery and comes out with a right atrial pressure of 14 mm Hg, PAWP of 8 mm Hg and cardiac index of only 1.8 l/min per m^2. It may at first seem that the problem is underfilling of the left heart. More often, however, the problem is a limitation of right heart filling. While it is true that the left heart is underfilled, the reason in this case is that there is right heart dysfunction and the right heart is not delivering enough volume to the left heart. This patient is most likely operating on the flat part of the cardiac function curve, and further volume loading will not increase the output from the right heart and therefore cannot alter left heart output. Volume loading may raise PAWP, but only because of transmission of pressures from the right heart to the left heart without any actual change in left-sided volumes. In fact, volume loading could make matters worse, for it will increase the pressure in the ventricular wall and thus impede coronary flow, it will shift the septum to the left and decrease the compliance of the left heart [8] and produce peripheral edema.

It needs to be emphasized that even left heart dysfunction produces a decrease in cardiac output by increasing the right atrial pressure. Thus, an increase in left ventricular end-diastolic pressure results in an increase in left atrial pressure, which increases pulmonary venous pressure, which increases pulmonary arterial pressure, which increases right ventricular end-systolic and end-diastolic pressures, which then results in an increase in right atrial pressure and a decrease in venous return and cardiac output. Thus, any decrease in cardiac output due to a decrease in cardiac function should be associated with a rise in right atrial pressure and even left-sided problems can produce a plateau in the cardiac function curve.

How then does one determine if right heart filling is limited? As noted above, it is first important to appreciate that most patients are volume limited at right atrial pressures of 12 mm Hg or less. The status of an individual patient, however, can be determined by giving a sufficient fluid challenge to increase the right atrial pressure by at least 2 mm Hg and then determining if the cardiac output increased. If it does, then the patient is still volume sensitive and volume therapy can increase cardiac output. If there is no change in output, then the patient is on the flat part of the cardiac function curve and further volume infusions will not increase cardiac output and inotropic therapy is needed to increase cardiac output. We presented another approach in spontaneously breathing patients [6]. Patients who have an inspiratory fall in right atrial pressure still have a compliant right heart and should respond to a fluid challenge, for this test indicates that they are not on the flat part of their cardiac function curve. However, if the patient has no respiratory variation in right atrial pressure, this indicates that the patient is volume limited and will not have an increase in cardiac output with volume loading, and inotropic therapy is needed to increase cardiac output.

What then is the usefulness of the PAWP? The PAWP helps the clinician assess the functional status of the left heart. Thus an elevated PAWP without an elevation in right atrial pressure indicates that the cardiac problem is left-sided and diagnostic possibilities include severe hypertension, aortic or mitral valve disease, or coronary artery disease limited to the left ventricle. Furthermore, PAWP tells the clinician the potential hydrostatic force driving fluid out of the pulmonary capillaries and is therefore useful in the management of pulmonary edema. If both right and left atrial pressures are elevated, then diagnostic possibilities include a cardiomyopathy, diffuse coronary disease, pericardial constriction, tamponade or overdistention of the right heart. If the right atrial pressure is greater than PAWP, then one should consider right ventricular dysfunction or pulmonary vascular disease.

In conclusion, failure to appreciate that there is a limit to right heart filling and continued use of fluid therapy to try and reach a target PAWP results in excess fluid use and might partly explain why the use of Swan-Ganz catheters is sometimes harmful [7]. I would argue that, when the clinical objective is to improve cardiac output, the appropriate value for the optimizing cardiac output is always the right atrial pressure. The PAWP tells you the potential harmful effect of this therapy on the lungs and the status of the left heart and is thus useful for diagnostic purposes. The Swan-Ganz catheter is also essential for the measurement of cardiac output, which is a critical variable in the assessment of cardiac function.

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More respect for the CVP

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Sir: I read with interest the editorial requesting "More respect for the CVP" [1], and applaud the author for highlighting the important role of measuring the CVP in critically ill patients. In an era when the pulmonary capillary wedge pressure (PCWP), but not the CVP, has been accepted as a marker of volume and cardiac function, such an analysis is long overdue.

The discussion on the role of the lowering of the right atrial pressure in controlling cardiac output needs some comment. The author has stated that the real role of the heart in regulating cardiac output is to lower right atrial pressure and allow better drainage from compliant veins and venules. I feel that this overstates the role of the right atrial pressure, as it fails to recognize that the real controlling mechanism of cardiac output is the oxygen requirements of the body. Though the venous system is compliant and forms the major reservoir for the blood volume, it would not freely drain into the right atrium if this venous system did not receive an adequate flow from the arterial tree. The flow from the high pressure arterial tree to the low pressure venous reservoir is determined by the arteriolar and precapillary resistance. This is governed by autoregulatory mechanisms which are, in turn, controlled by the oxygen demands of the tissues they perfuse [2].

It would be more complete to state that the real role of the heart in regulating cardiac output is to pump out whatever venous return it receives. This venous return is almost completely dependent on the oxygen requirements of the body and has little to do directly with the right atrial pressure. This is highlighted by observations that maximal sympathetic stimulation in a healthy person will result in a cardiac output of approximately double normal, while an increase in oxygen requirement induced by maximal exercise in a similarly healthy person could raise the cardiac output to 4–4$^1/_2$ times normal. This difference in cardiac output cannot be explained by changes in right atrial pressure, cardiac performance or arterial blood pressure. The main difference in these two settings is that there is arteriolar and precapillary dilation in the vasculature beds supplying the exercising muscles [2]. It is therefore the overall resistance of the arteriolar and precapillary vaculature that actually controls cardiac output by increasing or decreasing flow for any given state of circulating volume, filling pressure and cardiac performance.

The last two decades have witnessed a lot of comments on delivery-dependent oxygen consumption and delivery-independent oxygen consumption [3] ignoring the normal physiological mechanism, which is actually the reverse. Normally, at the tissue level, it is the oxygen consumption which controls the delivery of oxygen through the autoregulatory mechanisms. Keeping the role of oxygen consumption as the primary determinant of cardiac output in mind, I would argue that either the CVP or the PCWP could be used equally effectively or ineffectively to optimize cardiac output. I concur with the author that the response of the cardiac output or other parameters of perfusion to a fluid challenge gives the best indication regarding the fluid status of a patient at any given time.

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Reply

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Sir: I completely agree with Dr. Kapadia that the major determinant of cardiac output under normal physiological conditions is oxygen consumption. There is a tight linear relationship between cardiac output and oxygen consumption, both for the whole body and in isolated skeletal muscles. The relationship is so tight that, in fact, the oxygen consumption can be used to predict the cardiac output within 5% in subjects with a normal hemoglobin [1]. However, I do not agree that the flow of blood from the arterial side into the large capacitant veins is important for venous drainage. This is the point that I have tried to emphasize with the concept of a bathtub. The amount of volume that is in the arteries is very small and therefore is not an important contributor to the outflow from the large venous capacitant system.

How, then, does an increase in oxygen consumption result in an increase in cardiac output? This is a very complicated issue and a number of factors are operative. Changes in arteriolar tone and arterial resistance can affect venous return and cardiac output, but only when they affect the distribution of blood flow between areas which have veins with a low compliance, i.e. the extremities and areas that have a high compliance, i.e. the splanchnic bed. A redistribution of blood flow to the extremities results in an accumulation of blood in non-compliant beds, a rise in the mean circulatory filling pressure in these regions and an increase in cardiac output. The converse occurs when blood flow is redistributed to the splanchnic bed. However, the changes in distribution flow must be very large to have a significant effect [2].

The other way a change in oxygen consumption regulates cardiac output is by producing changes in capacitance through reflex contractions of small venules and veins as well as by decreases in venous resistance [2]. During exercise, there is an important effect of muscle contractions on the venous capacitant beds which also aids the return of blood to the heart and increases cardiac output [3]. Dr. Kapadia must also appreciate the difference between oxygen consumption under normal physiological conditions and in patients in intensive care units. A very frequent observation