Part 1

Hemodynamic monitoring: a personal and historical perspective

H.J.C. SWAN, MD, PH D; WILLIAM GANZ, MD, C SC

It is gratifying that the last half of the 1970s has revealed significant alteration of attitudes toward quantitative assessments of cardiovascular function in the clinical setting. This is a consequence of a compelling recognition of the limitation of clinical findings as accurate indicators of cardiovascular function. The relative insensitivity and unresponsiveness of clinical methods to rapid physiologic changes has resulted in an increasing recognition of the basic accuracy and value of quantitative biologic measurements.

The application of modern technology, which now allows measurements of important phenomena in a manner practical for the clinical situation, is an additional reason for this change of attitude. One basic principle is that common clinical findings, including many clinical laboratory estimates, are "slow" indicators of biologic change. As such, they are of considerable value in clinical situations in which the pathophysiologic changes are "slow". Endocrine disorders, neoplastic disease and even many forms of infectious disease result in deterioration of body function over a matter of hours, days, weeks or years. In cardiovascular disease acute, "fast" phenomena frequently dominate issues of outcome, and clinical findings often lag behind or misrepresent the underlying pathophysiology. For example, a severe reduction of cardiac output does not necessarily result in a corresponding drop in blood pressure but could be masked by pronounced vasoconstriction in the peripheral arterial and venous beds. Pulmonary edema may progress unrecognized by clinical findings until the accumulation of fluid is many, many times the normal pulmonary water content. In the past, physicians tended to assume that such issues were irrelevant to clinical practice. Nevertheless, the strong principles of fundamental biology are the true determinants of outcome of disease, and physicians have traditionally claimed these principles as the scientific basis of medicine. Yet practitioners have tended to downgrade the significance of the fundamentals of physiology and the relevance of the findings of biologic research in favour of clinical "intuition" in diagnosing and treating disease.

In the last 5 to 10 years the principles of hemodynamic monitoring have allowed basic physiological measurement to be applied in a consistent and meaningful manner to the management of the cardiovascular aspects of critical illness. This review is a personal interpretation of the significance of the application of certain physiological measurements in the clinical setting. It also relates the importance of these measurements in decision-making in relation to diagnosis, therapy and prognosis. It is appropriate to regard hemodynamic monitoring as an extension of the basic physiological concepts developed in the early part of this century by Frank and Starling and their predecessors. These were brought into practical application in humans by the development of diagnostic cardiac catheterization between 1942 and 1950 by Cournand and Richards, McMichael and Schaffer, Dexter and others. The specialized laboratory procedures of the past have now been made applicable at the bedside by the introduction of flotation catheters by ourselves and colleagues at Cedars–Sinai Medical Center in Los Angeles. The development of bedside intravascular catheterization procedures has permitted extension of application of the physiological principles established in the early part of this century to the broad population of critically ill patients. The simplicity of the procedures and the emphasis on practical application within the physical constraints of operating rooms, critical care units and outpatient laboratories have been fundamental to the acceptance of hemodynamic monitoring as an important adjunct to the care of such patients.

Historical background

In 1965 one of us (H.J.C.S.) — formerly director of a cardiac catheterization laboratory at a major medical centre — became responsible for the practice of cardiology at an institution (Cedars of Lebanon Hospital) in which ischemic heart disease was the most important cardiac admission diagnosis. Concerned by the lack of understanding of the pathophysiologic process of the disease, he believed that hemodynamic monitoring, particularly the measurement of cardiac output and left ventricular filling pressure, was essential to correct classification of patients with such disorders and assessment of their responses to therapy. One bright fall afternoon in 1967, while observing the motion of sailboats on Santa Monica Bay, he wondered if the attachment of a sail to a cardiac catheter would allow progression to the pulmonary artery and pulmonary artery wedge position with a high frequency of success and few, if any, complications. Although transmural measurement of pressure with the aid of sails was not technically feasible and carried an excessive risk of thromboembolic complications, the idea served as a background and a stimulus for our

From the department of cardiology, Cedars–Sinai Medical Center, Los Angeles

Reprint requests to: Dr. H.J.C. Swan, Department of cardiology, Cedars–Sinai Medical Center, 8700 Beverly Blvd., Los Angeles, CA 90048, USA

close collaborative effort. In a relatively short time this resulted in the development of the balloon-tipped Swan–Ganz catheter.[1]

Initially we specified a double-lumen extruded polyvinylchloride catheter with an outside diameter of 1.6 mm and a length of 110 cm. The extrusion permitted a "minor" lumen approximately 0.4 mm in diameter and a "major" lumen approximately 1 mm in diameter. The minor lumen was used to inflate the balloon to a volume of approximately 0.8 ml. A wound binding ensured retention of the balloon by the catheter and provided an ideal surface, from the standpoint of physical forces, to maximize the drag provided by the undersurface of the inflated balloon. We placed the first flexible balloon-tipped catheter in the superior vena cava of a dog previously used for other experimental purposes. Each time the balloon was inflated the catheter passed with the slightest advancement from the superior vena cava to the right pulmonary artery of the anesthetized dog. Deflation of the balloon resulted in repositioning of the catheter tip in the right atrium. Thus, in one experiment feasibility had been demonstrated.

However, as so often occurs in science, the fundamental nature of our observation turned out not to be original. In 1953 Lategola and Rahn[2] had been interested in isolating each lung by selective endotracheal intubation and the passage of a rather large catheter (outside diameter 3.2 mm) bearing a balloon to occlude the left or right main pulmonary artery and provide the desired experimental preparation. In their report to the Society for Experimental Biology and Medicine they noted that the catheter passed readily and consistently to the pulmonary artery without the need of extensive manipulation, such as characterized clinical cardiac catheterization at that time. This observation of Lategola and Rahn did not gain clinical application, but within a few months of our experiment the value of application in humans had been demonstrated.

Our initial study, the results of which were published in 1970, involved 100 consecutive patients in whom bedside hemodynamic monitoring was performed.[1] We found the following:

- It was possible to catheterize the pulmonary artery at the bedside without the use of fluoroscopy in a very high proportion of patients.
- The frequency of adverse arrhythmias was small — in fact, fractional compared with the accepted frequency of arrhythmias in patients undergoing routine cardiac catheterization with semistiff diagnostic catheters.
- It was possible to catheterize the heart without the skills usually defined and developed in the specialized catheterization laboratory and, therefore, such techniques were applicable in a wide clinical environment.
- The information provided by flotation catheterization proved to be of fundamental significance in the definition of the pathophysiology of a given illness in a given patient. The findings frequently related closely to prognosis. Therapy designed to reverse gross abnormalities of hemodynamics now appeared to have a rational basis, and therapeutic failure could be promptly recognized.

Measurements of pressure

The primary objective of balloon flotation catheterization was the measurement of the pulmonary artery diastolic pressure or the pulmonary capillary wedge pressure or both, in particular (but not exclusively) for the management of patients with acute myocardial infarction. One of the early observations in such patients was the absence of any relation between the filling pressures of the left and right ventricles; this was presumably a natural consequence of the variability of the disease process.[3]

The factors that influence this lack of a relation include the magnitude of involvement of each ventricle, the presence or absence of valvular regurgitation, the state of the pulmonary vascular bed prior to acute myocardial infarction and the degree of hypertrophy of each ventricle. An early publication from our department clearly demonstrated a poor "biologic" correlation between simultaneously measured right atrial ("central venous") and pulmonary capillary wedge pressures.[4] Further, it was impossible to predict the pulmonary capillary wedge pressure from the right atrial pressure alone. Even more surprisingly, changes in one set of pressures were not necessarily associated with changes of similar magnitude or direction in the other. Bedside catheterization stimulated additional studies. In 60% to 80% of patients the pulmonary artery diastolic pressure and the mean pulmonary capillary wedge pressure are reasonably close — within 3 mm Hg. Significantly higher pulmonary artery diastolic pressures (greater than 6 mm Hg) suggest the presence of pulmonary vascular disease associated with cor pulmonale, pulmonary embolus or a primary pulmonary abnormality. Further study demonstrated that the mean pulmonary wedge pressure relates closely to the mean left ventricular diastolic pressure. However, in instances of markedly elevated left ventricular end-diastolic pressures (greater than 35 mm Hg) the mean pulmonary wedge pressure underestimates the end-diastolic pressure.

The pulmonary artery diastolic pressure and the mean pulmonary capillary wedge pressure reveal fundamental information concerning the transfer of fluid into the lung (the hydrostatic outward force of the Starling capillary permeability relationship) as well as the competence of the left ventricle. In regard to transcapillary movement of intravascular water, mean intracapillary pressures of approximately 25 mm Hg are associated with marked pulmonary congestion and dyspnea, and greater values are usually associated with alveolar pulmonary edema. Mean pulmonary capillary wedge pressures of between 14 and 18 mm Hg appear to indicate optimal left ventricular performance, and filling pressures of less than 8 to 10 mm Hg in the acutely injured heart are associated with marked reductions in cardiac output, hypotension and tachycardia. Accordingly, in the critically ill patient pulmonary capillary wedge pressures of 14 to 18 mm Hg are now routinely maintained.

Measurement of cardiac output

It was a fortunate coincidence that both of us had a long-standing

record of cardiac output determination by the indicator dilution principle, one (H.J.C.S.) working with indicator dyes in Dr. Earl Wood's laboratory at the Mayo Clinic, and the other working with Dr. Fronek and the thermal indicator initially in Prague and later at Cedars–Sinai. This association very soon and naturally led to the incorporation of the cardiac output measuring capacity into the existing pressure measuring catheter. Addition of the temperature measuring device — a thermistor — to the distal portion of the catheter and a lumen for injection of cold indicator allowed for the first time measurement of cardiac output on a wide clinical basis.[5,6] In 1971 we demonstrated that the thermodilution technique was applicable at the bedside in critically ill patients and that it measured cardiac output with a high degree of accuracy and precision (variance about 4%). A single injection of cold glucose solution had several practical advantages over the dye indicators then used. Detection of the thermal response by the single thermistor placed on the catheter shaft and positioned in the pulmonary artery allowed the procedure to be conducted without the requirement of blood withdrawal. Minimal recirculation facilitated computation of the cardiac output from the thermodilution curve with a high degree of accuracy. Finally, cardiac output estimations could be obtained frequently; there was no practical limitation on the number of estimates.

From the data thus obtained, certain clinically relevant criteria could be readily developed. In normothermic nonanemic patients suffering from a variety of illnesses the cardiac index usually exceeds 2.2 l/min per square metre. From the cardiovascular standpoint such patients have a favourable prognosis. Moderate depression of the cardiac index (to less than 2.2 but more than 1.8 l/min per square metre) is usually associated with a favourable prognosis but may suggest impending deterioration. Treatment can be effectively directed during this phase of an illness to moderately increasing the cardiac output, if necessary by the use of impedance reduction and manipulation of the heart rate and the left ventricular filling pressure. A cardiac index of 1.7 l/min per square metre or less is usually associated with a poor to an extremely poor prognosis and demands prompt and effective treatment to raise the cardiac index to a level that will provide for the resting needs of the body organ systems.

Electrode catheters

Miniature extrusion and wiring technology has now permitted the development of sophisticated multipurpose flotation catheters. Of these, the most immediately practical and applicable are those that bear surface electrodes for sensing right ventricular and right atrial electrical activity and that provide a site for stimulation in either atrium or ventricle. For ventricular sensing and pacing, an electrode pair separated by 1 cm is positioned at a point of altered stiffness 20 cm proximal to the tip. Differential stiffening of the more proximal portion of the catheter allows for appropriate contact of the electrodes with the inflow tract of the right ventricle. To make atrial sensing and stimulation possible, an array of four electrodes over a distance of approximately 5 cm is provided. The pair of atrial electrodes that provides pacing at the lowest threshold and without stimulation of the diaphragm is selected.[7]

The use of electrode catheters also permits the identification of a wide variety of arrhythmias that cannot be diagnosed from surface leads alone. The diagnosis can be made with a high degree of certainty since the signal originating from the appropriate electrode pairs can be identified as either atrial or ventricular. Thus, such a diagnosis as supraventricular tachycardia with aberration, as distinguished from ventricular tachycardia, can be made promptly and without doubt. Atrial, atrioventricular and ventricular pacing are possible either to achieve an appropriate cardiac output or as an "overdrive" mechanism for suppression of areas of ectopy within the atria or ventricles. These applications are in a developmental stage and appear to have great potential, particularly in circumstances that may require emergency pacing — for example, postoperatively or when the patient is suffering from toxic effects of a drug.

The use of automated techniques for analysis and quantification of cardiac arrhythmias is likely to be greatly facilitated by these techniques and their further development in application.

Conclusion

Hemodynamic monitoring has come of age. It is accepted as standard practice in many medical centres throughout the world. Although such techniques do not supplant clinical skills, they open the door to major advances in the science and art of medicine. As with all invasive techniques, complications can occur. (Of these the most serious is rupture of a pulmonary artery branch in patients with pulmonary hypertension in whom repeated balloon inflation has been performed.) Thus, in anesthesiology accurate monitoring techniques, particularly in the elderly, appear to allow for more efficient management of patients undergoing hazardous surgical procedures such as genitourinary, orthopedic and vascular repairs.[8] Patients undergoing cardiac operations appear particularly to benefit from careful hemodynamic monitoring; monitoring in such patients may aid in minimizing subsequent morbidity and mortality.

The disciplines of surgery and anesthesia appear to be those most likely to use hemodynamic monitoring, but there appears to be an increasing role for this technique in the respiratory care and medical intensive care units and in patients with acute ischemic cardiac syndromes.

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Physiologic significance of hemodynamic measurements and their derived indices

BRIAN W. GILBERT, MD; ERNEST M. HEW, MD

The fundamental value of hemodynamic monitoring is that it provides direct physiological measurements. The appropriate use of these requires an understanding of normal cardiac physiology and its alterations in certain disease states. An appreciation of the determinants of cardiac output, coronary blood flow and myocardial oxygen consumption will improve the interpretation and subsequent manipulation of these values when one is confronted with them clinically.

L'utilité fondamentale de la surveillance hémodynamique réside dans le fait qu'elle procure des mesures physiologiques directes. L'utilisation appropriée de celles-ci exige une compréhension de la physiologie cardiaque normale et de ses perturbations dans certains états pathologiques. Une appréciation des déterminants du débit cardiaque, de la circulation sanguine coronaire et de la consommation d'oxygène du myocarde améliorera l'interprétation et les manipulations subséquentes de ces valeurs lorsqu'on leur est confronté cliniquement.

Hemodynamic monitoring is an important aspect of contemporary care of critically ill patients. The fundamental value of this technique is that it provides direct physiological measurements, the appropriate use of which is predicated by an understanding of normal cardiac physiology as well as its alterations in the critically ill patient. The measurement of raw hemodynamic data and the subsequent calculation of derived indices allow quantitative analysis of certain factors that affect cardiac performance and tissue perfusion. Once these factors are known, they can be thoughtfully manipulated to optimize both cardiac output (CO) and blood pressure, and thereby improve tissue perfusion. In this presentation we will define the determinants of CO, coronary blood flow (CBF) and myocardial oxygen consumption ($M\dot{V}O_2$), identify the values that can be measured and discuss how this information may be of help in the care of the critically ill patient. The appendix details the calculation of appropriate hemodynamic indices for this paper and others in this symposium.

From the division of cardiology and the department of anesthesia, Mount Sinai Hospital, University of Toronto

Reprint requests to: Dr. Brian W. Gilbert, Mount Sinai Hospital, 600 University Ave., Toronto, Ont. M5G 1X5

Determinants of cardiac output

One of the important variables that can be measured by means of contemporary hemodynamic monitoring techniques is the CO — that is, the volume of blood pumped by the heart, expressed in litres per minute. The stroke volume (SV) is the amount of blood ejected from the ventricle during each systole; the SV is the output of the heart per beat and is equivalent to the CO divided by the heart rate. The SV also represents the difference between the end-diastolic volume (EDV) and the end-systolic volume (ESV) of the ventricle; when it is expressed as a percent of the EDV it is referred to as the ejection fraction (EF). For comparison of measurements between individuals of different sizes, flow is often expressed in terms of square metres of body surface area; that is, instead of comparing the CO in absolute numbers of litres per minute, one can express the output of the ventricle as the cardiac index (CI), the units of which are litres per minute per square metre of body surface area.

The performance of the heart as a pump, as reflected by the SV of the intact ventricle, has been shown to be determined by three main factors: (a) preload, the length of the muscle at the start of contraction, or the ventricular EDV; (b) afterload, the tension that the muscle is called upon to develop during contraction; and (c) contractility, the inotropic state of the muscle, expressed as the velocity of fibre shortening for any given preload or afterload.

Preload

Preload can be defined as the