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Commentary

Balloon Flotation Catheters

Their Use in Hemodynamic Monitoring in Clinical Practice

IN THE past decade, acute myocardial infarction has rightly received increasing attention among the medical community. In many patients, a principal cause of death—primary arrhythmias—can now be prevented, and, in others, the catastrophic consequences are frequently rectified by prompt cardiopulmonary resuscitation and electrical countershock. This major achievement was the outcome of the development of equipment suitable for continuous monitoring of cardiac rhythm, associated with advances in antiarrhythmic therapy. Thus, the oscilloscope, adapted for monitoring applications, allows for the prompt and accurate recognition of a cardiac arrhythmia, its designation as lethal, major, or minor, and permits immediate effective treatment.

See also pp 891 and 893.

However, the other principal pathophysiological consequence of acute myocardial infarction is an alteration of cardiac function. The output of the heart usually decreases, the filling pressure of the ventricle increases, and both contractility and compliance may, but do not necessarily, decrease. The development of an appropriate device, the Swan-Ganz balloon flotation catheter,[1] has practical application to the acquisition of important diagnostic and therapeutic information relative to the severity of cardiac disability in acute myocardial infarction[2] and in a wide variety of other cardiopulmonary conditions. Use of these devices is now receiving wider application in critical and intensive care units, in surgical and postsurgical management,[3] in diagnostic cardiac catheterization,[4] and in other fields of medicine.

The Swan-Ganz Balloon-Tipped Catheter

In its simplest form, the catheter consists of an extruded, double lumen, polyvinylchloride catheter, 100 cm in length, with an outside diameter of 1.6 mm. Different lengths and sizes are now used for specific applications. A balloon is attached at the tip of the catheter and may be inflated through the smaller of the two lumens in the shaft of the catheter. The bursting volume of the balloon at the tip of the catheter is approximately 3.0 ml, but it is recommended that inflation be conducted using a 1-ml syringe to a volume of 0.8 ml. The other lumen of the catheter is adequate in size for the recording of intracardiac and great vessel pressures, with a frequency response uniform to approximately 10 Hertz with the most commonly used electromanometers.

The minor lumen connected to the inflatable balloon is never liquid-filled, but is continuously attached by a one-way stopcock to a 1-ml syringe filled with air. The second lumen is saline-filled before introduction of the catheter into the circulation and is connected to an appropriate pressure-recording device.

In its usual application, the catheter is advanced at the patient's bedside either by percutaneous puncture of a suitable vein in the brachial, subclavian, or femoral region, or by a venotomy in the antecubital fossa. After the catheter is passed centrally, approximately 40 cm as guided by markings on the shaft of the catheter, the patient is requested to cough. Oscillations of pressure indicate the catheter to be within the thoracic cavity.

Pulmonary Artery Catheterization

With the catheter in the superior vena cava, the balloon is inflated with 0.8 ml of air and the catheter advanced gently. The catheter is of sufficient flexibility so that the inflated balloon at its tip will be guided by the stream of blood flowing through the tricuspid valve into the right ventricle and out into the pulmonary artery. The position of the balloon and the pliability of the catheter is such that stimulation of the endocardium of the right atrium or right ventricle is uncommon and, hence, ectopic rhythms are not frequent with the use of this catheter. Experience in our own department and elsewhere has indicated that catheterization of the pulmonary artery may be achieved in between 15 and 20 seconds in the vast majority of instances, without the use of fluoroscopy. With the balloon still inflated, the flowing blood will continue to direct the catheter more distally into the pulmonary tree.

When the balloon-tipped catheter enters a vessel that

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approximates the inflated balloon in size, the system cannot advance further. Hence, there is a static nonflowing column of blood in the pulmonary arterioles, capillaries, and veins of the pulmonary segment to which the artery is occluded by the balloon. This fluid column allows transmission of the pressure wave in the dynamic vascular system immediately downstream from this position, namely, the pulmonary veins and left atrium. Hence, the pressure recorded with the balloon occluded and passed distally into the pulmonary vascular bed ("balloon-occluded pulmonary artery pressure")[5] is similar to what has been known, heretofore, as the "wedge" pressure, and is a phase-delayed and amplitude-damped version of left atrial pressure. The mean values for wedge and left atrial pressures are, for practical purposes, identical.[5]

Indicator of Left Ventricular Function

Knowledge of left atrial mean pressure is of critical significance in the assessment of pulmonary congestion, since it is the principal determinant of the egress of fluid from the vascular to the extravascular space of the lung. In addition, this pressure is closely related to the filling pressure of the left ventricle and, hence, is an indicator of left ventricular function. Knowledge of the pulmonary artery wedge pressure, therefore, allows the physician to know with precision the mean pulmonary distending pressure. Values of pressure (zero reference point, one-half distance from sternum to back at fourth interspace with patient horizontal) in excess of 18 to 20 mm Hg will cause dyspnea, excepting patients in whom left atrial hypertension has existed for a long time, eg, mitral stenosis. Excessive transudation of fluid into the extravascular pulmonary spaces occurs at pressures between 20 and 30 mm Hg, and pulmonary edema is usually present at pressure between 30 and 35 mm Hg.

The use of conventional diuretics of either rapid- or longer-acting characteristics will allow the physician to adjust the pulmonary congesting pressure, measured by means of the balloon-tipped flotation catheter, with a high degree of precision. Diuretics should be used until the mean pulmonary arterial pressure wedge is less than 18 mm Hg and preferably lies at approximately 14 mm Hg. While the physical examination of the chest and serial x-ray films can be extremely helpful in defining the degrees of pulmonary congestion, these clinical findings may follow by several hours the actual and fundamental alterations in circulatory dynamics. Hence, in a rapidly changing state, knowledge of mean pulmonary venous pressure is invaluable. Additionally, the possibility of "overtreating" such patients with excessive diuretics is obviated by direct measurement of the filling pressure in the left side of the heart. In many instances, observation of the level of or changes in central venous pressure are of no value or may be misleading.[6]

While end-diastolic left ventricular pressure represents the precise degree of stretch on the left ventricle immediately prior to systole, the left atrial and left ventricular pressures approximate each other up to the end of the diastolic portion of the cardiac cycle. The "a" wave, sometimes readily recognizable in the pulmonary artery wedge pressure, relates remarkably closely to the magnitude of the "a" wave in the left ventricular pressure contour.

Left Ventricular Filling Pressure

A knowledge of left ventricular filling pressure has practical importance. The observations formulated by Starling into the "Law of the Heart" indicated that a more powerful contraction is elicited in response to a higher degree of stretch of the myocardial fiber, provided physiological limits were not exceeded. It has been found that optimal left ventricular performance can be achieved in the presence of a mean pulmonary artery wedge pressure value of between 14 and 18 mm Hg with use of the reference levels just indicated.[7]

Other workers using different reference levels have established essentially similar data. The "stiffer" ventricle of hypertension may require filling pressures at the upper level of the values indicated, but no specific data to this point are available. A most important phenomenon is the absence of any further increase in left ventricular output at filling pressures above this general range. The preponderance of data available would suggest that a left ventricular filling pressure of 15 to 18 mm Hg is optimal in terms of cardiac performance. In other words, pressures in excess of these values will not result in a further increase in stroke volume and cardiac output. It is possible that under certain conditions, such as extreme ventricular hypertrophy with aortic stenosis, or following acute cardiopulmonary bypass for direct cardiac surgery, values in excess of these would be found optimal. It is also clear that left ventricular filling pressures of less than 12 mm Hg may be associated with depression of cardiac output in the diseased heart. While filling pressures of 6 to 8 mm Hg probably pertain and are adequate for cardiac control in the normal and undiseased heart, similar values may be associated with depression of cardiac function in the presence of disease. Precise adjustments of pulmonary artery wedge pressure to the 15- to 18-mm Hg range result in optimal left ventricle performance (for this control factor) without adverse effects on lung performance.

Summary

Balloon flotation catheterization of the pulmonary artery at the patient's bedside and without use of fluoroscopy allows the measurement of pressures in the right atrium, right ventricle, pulmonary artery, and in the distal "occluded" branch of the pulmonary artery, thus reflecting the levels of pressure in the left ventricle. From these values, estimates of the venous pressure in the lung and the mean filling pressure of the left ventricle may be derived, which are fundamental determinants of both pulmonary congestion and edema on the one hand, and the output of the heart on the other. A knowledge and control of these values places the therapy of heart disease on a more logical footing.

No procedure that introduces a foreign body into the circulation is without potential hazard. In regard to balloon flotation catheters, these hazards are similar to those of other forms of cardiac catheterization, but with a low incidence of serious cardiac arrhythmias because of the re-

lationship of the inflated balloon to the catheter tip, which prevents the concentration of catheter forces that might cause subendocardial injury or irritation.

A particular word of caution is necessary in relation to the balloon-occluded pulmonary segment. For more than 20 years, it has been known that catheters wedged in small pulmonary arteries will result in segmental pulmonary infarction. It is, therefore, recommended that the Swan-Ganz balloon-tipped catheter not be allowed to remain in the distal pulmonary artery for a time in excess of that required to obtain important measurements. The balloon is then deflated and the catheter will ordinarily retract spontaneously into a larger pulmonary artery. Reinflation of the balloon should allow the identification of the pulmonary wedge pressure once more for monitoring purposes. Again, after the relevant data have been obtained, the balloon is promptly deflated and pulmonary artery pressure recorded. These and other complications and recommendations to minimize their occurrence are outlined in detail in the information inserts supplied by the manufacturers of these devices (Edwards Laboratories, Santa Ana, Calif, 1973).

The indications for application of the Swan-Ganz balloon-tipped catheter are those situations in which a critical or potentially critical alteration in cardiac dynamics may occur. I personally believe that the indication for the use of a flotation catheter is "any situation in which a physician would consider placing a central venous pressure line for the purposes of cardiovascular monitoring."

The development of devices based on these principles to measure cardiac output, intra-atrial and intraventricular electrical potential, and to permit atrial, ventricular, or sequential cardiac pacing and other indexes of cardiac function may allow the physician to approach his patient with a degree of understanding of hemodynamics similar to that provided to him in regard to arrhythmias by oscilloscopic monitoring of the electrocardiogram.

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Louis Braille, educator of the blind, was born in Coupvray, France, in 1809. When he was 3 years old, he became blind, and at 10 years of age, he was admitted to the Institute for the Blind in Paris, where he became proficient in science and music. In his day, he was one of the most distinguished organists and cellists in Paris.

When he was 20 years old, he modified Barbier's system of writing with points, making it practical and convenient. This new system—the Braille system—was soon introduced into the Royal Institute, although an account of it was not published until 10 years later. The Braille system quickly spread to the continent and the United States, where it is still successfully used today. In the original system, there were 43 signs, embracing the entire alphabet, all the diphthongs, and marks of punctuation. Ten fundamental signs form the basis of the remainder of the system.

Braille died in 1852 and was honored philatelically on a stamp (Scott No. B222) issued by France in 1948. He also has been honored postally by Russia and Brazil.—R. A. KYLE and M. A. SHAMPO