Hemodynamic Monitoring:

Invasive Techniques

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PROMPT RECOGNITION and accurate assessment of serious circulatory changes in patients gravely ill or undergoing major surgical interventions is of critical importance. Although monitoring heart rate, arterial blood pressure, and central venous pressure has been a valuable guide, these values do not provide a sufficient basis for accurate diagnosis and proper management. The value of central venous pressure is limited by the fact that it basically reflects the functional state of the right ventricle, which frequently does not parallel that of the left ventricle[1-6] (fig. 1). Information about the function of the left heart is, however, often essential for proper evaluation. In recent years, techniques that allow easy monitoring and analysis of the function of both ventricles have become available. This paper describes these techniques and demonstrates how their use enables proper diagnosis and therapy of commonly encountered clinical situations.

Monitoring Techniques

SYSTEMIC ARTERIAL BLOOD PRESSURE

In most situations the sphygmomanometer accurately determines blood pressure. However, in some low-cardiac-output states, pulses may be poorly palpable and Korotkoff sounds hard to hear while the intra-arterial pressure may be only moderately reduced. Monitoring of intra-arterial pressure is readily accomplished by percutaneous insertion of an 18- or 20-gauge sheath into a radial, brachial

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or femoral artery. With appropriate display systems continuous pressure monitoring is obtained. Long-term patency is facilitated by intermittent flushing with 2–5 ml of heparinized 5 per cent dextrose in water.

RIGHT HEART AND PULMONARY VASCULAR PRESSURES

While the right atrial (central venous) pressure can easily be measured at the bedside, catheterization of the pulmonary artery with semirigid catheters requires fluoroscopic guidance and substantial skill. Even in experienced hands, a risk of serious complications exists when severely ill patients are catheterized. These problems have been largely overcome by the introduction of balloon flotation catheters,[7] which allow for rapid and relatively safe catheterization of the pulmonary artery without fluoroscopy.

CATHETERIZATION WITH BALLOON FLOTATION CATHETERS

Catheterization can be performed in any hospital location where appropriate support devices are available for effective detection and therapy of arrhythmias and for recording hemodynamic data. Catheterization is performed during continuous electrocardiographic monitoring. The basilic, brachial, femoral, subclavian and internal jugular veins are used as insertion sites, the latter two being particularly preferred by anesthesiologists and surgeons. After entry into the selected vein, the catheter is advanced until the tip is in or near the right atrium. This usually occurs after advancement for approximately 15 cm when the jugular or subclavian vein is used, after 40 cm with use of a vein in the right antecubital fossa, after 50 cm with use of a vein in the left antecubital fossa, and after about 30 cm when a femoral vein is used. Increase in respiratory fluctuation confirms that the catheter tip is in the thorax. At this time the balloon is inflated to the recom-

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mended volume and the catheter advanced further. The catheter-tip pressure is continuously recorded as the catheter proceeds from the right atrium into the right ventricle, pulmonary artery, and finally into a "wedge" position (fig. 2). At this point the diameter of the balloon (11 or 13 mm) slightly exceeds that of the pulmonary artery. In the "wedge" position the tip senses the pressure transmitted with some delay and damping from the left atrium retrograde through the pulmonary veins and capillaries. With deflation of the balloon pulmonary arterial pressure will reappear. Reinflation will cause the balloon to float into "wedge" position again.

As the catheter material (polyvinylchloride) softens with time, the transcardiac catheter loop tends to diminish, and this may result in migration of the catheter tip into smaller branches and into "wedge" position. Continuous or frequent (every 15 to 30 minutes) monitoring of pulmonary arterial pressure is, therefore, recommended. Inflation of the balloon to full capacity when the catheter tip is in a small branch of the pulmonary artery will result in a spuriously high pulmonary wedge pressure reading, caused by compression of the catheter lumen (fig. 3). Reinflation of the balloon should, therefore, be performed slowly, adding increments of 0.1 to 0.2 ml air until a change in pressure contour from pulmonary arterial to pulmonary wedge pressure is seen. If the pulmonary wedge pressure is obtained at a volume substantively less than the recommended inflation volume, the catheter should be gradually withdrawn (several cm) until the volume required for wedging is equal or nearly equal to the full inflation volume. When the balloon is deflated, the ideal catheter position is with the tip in one of the primary branches of the pulmonary artery.

One of the most important applications of the balloon flotation catheter is in the recording of the pulmonary capillary wedge pressure obtained when the inflated balloon impacts into a slightly smaller branch of the pulmonary artery. The pulmonary capillary wedge pressure is of great significance in clinical practice in that it provides information about two important determinants of cardiopulmonary function. First, the level of this pressure is a basic factor in pulmonary congestion and in the shift of fluid from the
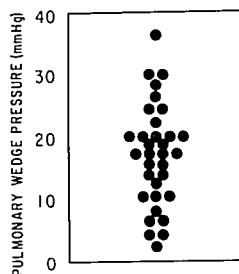

FIG. 1. Data from 33 patients, showing the wide range of pulmonary capillary wedge pressure values measured in the presence of normal central venous pressure (1–5 mm Hg).

pulmonary capillaries into the interstitial tissue and alveoli. Second, the pulmonary capillary wedge pressure closely reflects left atrial pressure[8,9] (fig. 4) and can, therefore, serve as an index of left ventricular filling pressure.

The mean pulmonary capillary wedge and left atrial pressures closely approximate left ventricular end-diastolic pressure in patients who have normal left ventricular[10,11] and mitral valve function. In left ventricular failure, the elevated left ventricular end-diastolic pressure may significantly exceed the mean left atrial and accordingly, pulmonary capillary wedge pressure.[12,13] Nevertheless, in clinical practice the mean pulmonary capillary wedge pressure has proved to be a reliable and useful index of left ventricular filling, and as such provides highly relevant information on the function of the left ventricle.

Since the pulmonary vasculature is a low-resistance circuit, the pulmonary arterial end-diastolic pressure is normally only slightly higher (1 to 3 mm Hg)[12,14] than the mean pulmonary capillary wedge pressure and can, therefore, be used as an index of left ventricular filling,[3,11,14-16] when the pulmonary capillary wedge pressure is not obtainable. However, in states associated with high pulmonary vascular resistance, the pulmonary arterial end-diastolic pressure may markedly exceed the mean pulmonary capillary wedge pressure[3,8-12,14-18] (fig. 5).

As noted above, in the absence of tricuspid valve disease, the mean right atrial pressure
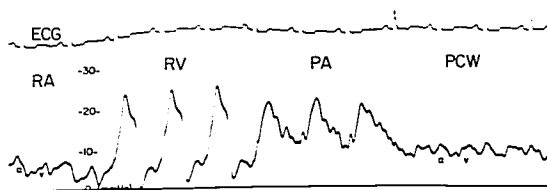
FIG. 2. Pressure tracings from the tip of the flotation catheter during passage from the right atrium (RA) into the right ventricle (RV), pulmonary artery (PA) and pulmonary wedge (PCW) position. Electrocardiogram = ECG.

is sufficiently close to the right ventricular end-diastolic pressure to serve as an index of right ventricular filling and a basis for evaluation of right ventricular function.

MEASUREMENT OF CARDIAC OUTPUT

In the past, the direct Fick procedure and the dye-dilution technique were the methods commonly used for determination of cardiac output. These methods are, however, not suitable for rapid, serial measurements at the bedside of the critically ill. In recent years, a modification of the indicator-dilution principle has greatly facilitated application at the bedside of the critically ill. The method, originally described by Fegler,[17] uses (negative) heat, rather than dye, as indicator.

The theory and practical steps of the determination have been reviewed.[19] Briefly, if a known quantity of (negative) heat is introduced into the circulation, the resulting cooling curve recorded at a position sufficiently downstream to permit even distribution of the injected (negative) heat in the flowing blood allows computation of cardiac output. Adequate mixing of blood with the cold injectate has been found to occur during passage of the mixture through two valves and one cardiac chamber.[18] In practice, 10 ml of 5% glucose cooled to 0 to 5 C or room temperature is injected into the superior vena cava or high right atrium or into the inferior vena cava (when the catheter is inserted from the femoral vein). The decrease in temperature is
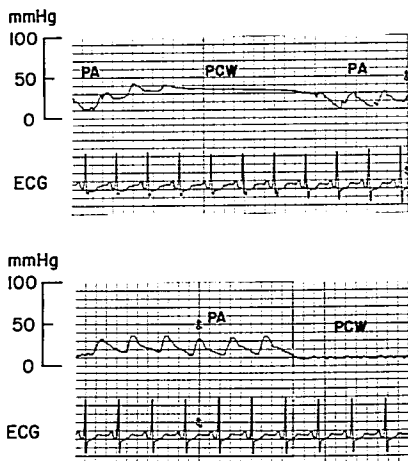

FIG. 3. A (above), the pulmonary capillary wedge pressure is spuriously high, due to overinflation of the balloon in a small branch of the pulmonary artery. B (below), the pulmonary capillary wedge pressure is now correct. Inflation of the balloon was stopped when the change in pressure tracing from pulmonary arterial to pulmonary capillary wedge was noted.

FIG. 4. A simultaneous recording of left atrial and pulmonary capillary wedge pressures demonstrates the similarity of waveforms and near identity of mean pressures.
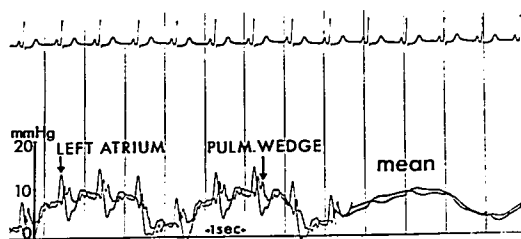
detected in the pulmonary artery. A single balloon flotation catheter is used for both the injection of the cold solution and detection of temperature change by a thermistor located 4 cm from the catheter tip.[19] From the thermodilution curve so obtained, cardiac output can be computed by manual planimetry or by electronic computational devices that allow "on-line" determination of cardiac output from the given quantity of injected (negative) heat.

In contrast to dye-dilution, the thermodilution technique does not require withdrawal of blood from the arterial system or removal of blood for calibration. Repeat measurements can be performed at short intervals, about twice per minute. In our experience, triplicate cardiac-output determinations have a reproducibility (coefficient of variation) of 4 per cent using 10 ml of cold injectate and a bedside computer.‡ Somewhat greater variability (5.5 per cent) has been observed when room-temperature injectate has been used. Since the signal reflecting the temperature change is two to three times smaller when solutions at room temperature rather than at 0 to 5 C are injected, the use of room-temperature injectate is not recommended in patients who have significant respiratory fluctuation in the pulmonary arterial temperature, such as patients who have severe shortness of breath or are mechanically ventilated. Also, under circumstances associated with very high cardiac outputs, the use of cold injectate is preferable.

The position of the thermistor in the pulmonary artery has no effect on the accuracy of cardiac output measurement except when

‡ Edwards Laboratories, Model 9510.

the catheter tip becomes wedged, as indicated by the pressure tracing.

Clinical Applications

With knowledge of the above mentioned modalities, clinical states can be defined in pathophysiologic terms, the most appropriate therapy chosen, and the progress of treatment monitored.
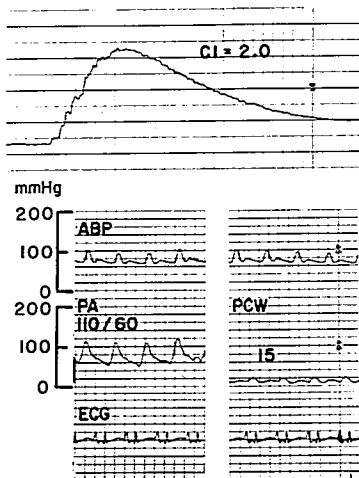

FIG. 5. Hemodynamic findings in a patient with a history of recurrent pulmonary embolism. There is severe pulmonary hypertension. The marked discrepancy between the pulmonary artery end-diastolic (60 mm Hg) and the pulmonary capillary wedge pressures (15 mm Hg) is typical of a primary abnormality in the pulmonary vascular bed.
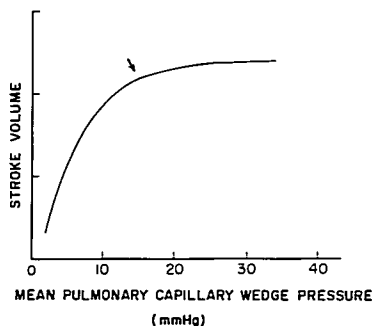
FIG. 6. Representation of Starling ventricular function curve. The curve indicates that between 0 and about 15 to 18 mm Hg, small changes in mean pulmonary capillary wedge pressure will cause marked changes in stroke volume (steep portion of Starling curve). In contrast, at levels exceeding 18 mm Hg, even marked changes in pressure will have little effect on stroke volume (flat portion of Starling curve).

NORMAL CENTRAL HEMODYNAMICS

Normal central hemodynamics are characterized by a cardiac index (cardiac output/body surface area) of 2.5 to 3.5 l/min/m², mean right atrial pressure between 1 and 5 mm Hg, and mean pulmonary capillary wedge pressure between 6 and 12 mm Hg.

HYPOVOLEMIA

In uncomplicated hypovolemia there are decreases in cardiac index, right atrial and pulmonary capillary wedge pressures, and usually also systemic arterial pressure. The progress of volume replacement can be guided by changes in pulmonary capillary wedge pressure and cardiac output. Return of pulmonary capillary wedge pressure to the normal range will usually suffice. Further elevation of pulmonary capillary wedge pressure to between 15 and 18 mm Hg will result in additional increase in cardiac output. Increase in pulmonary capillary wedge pressure to more than 18 mm Hg will, however, produce pulmonary congestion, but no significant further increase in cardiac output[20].

The relationship between left ventricular filling pressure and cardiac output is best characterized by the Starling curve (fig. 6). As filling pressure increases along the ascending portion of the curve, the stroke volume increases rapidly. When the flat portion of the curve is reached, at levels of pulmonary capillary wedge pressure of around 15 to 18 mm Hg in our experience,[20] further increase in filling pressure will not be associated with a significant increase in stroke volume.

PULMONARY CONGESTION

Pulmonary congestion, whether resulting from fluid overload or from left ventricular failure, is characterized by a pulmonary capillary pressure in excess of 18 mm Hg, and as high as 30 mm Hg and more in pulmonary edema. The purpose of treatment with diuretics or phlebotomy is to reduce the pulmonary capillary wedge pressure to about 15–18 mm Hg. Such a reduction in pulmonary capillary wedge pressure will relieve pulmonary congestion without reducing cardiac output, since these changes occur on the flat portion of the Starling curve. Removal of more fluid will lower the pulmonary capillary wedge pressure (filling pressure) to the steep portion of the Starling curve, resulting in a decrease in cardiac output.

Transudation of fluid across the pulmonary capillaries occurs when the pulmonary capillary pressure exceeds the plasma colloid osmotic pressure, which is normally 25 to 30 mm Hg. Pulmonary edema is, therefore, usually observed at pulmonary capillary pressures in excess of 30 mm Hg. When the plasma colloid osmotic pressure is reduced, pulmonary edema may occur at pulmonary capillary pressure levels substantively lower than 30 mm Hg or even in the presence of a normal pulmonary capillary pressure, e.g., in patients who have severe hypoproteinemia or following administration of large amounts of crystalloids.[21]

HEART FAILURE

Left ventricular failure is characterized by a decrease in stroke volume and increases in left ventricular end-diastolic, left atrial, and pulmonary capillary wedge pressures.

Pulmonary capillary wedge pressures of more than 18 mm Hg are associated with increasing pulmonary congestion, dyspnea, and pulmonary edema.

In mild heart failure, cardiac output may be normal despite a lower stroke volume due to a baroreceptor-induced increase in heart rate. In such cases, signs of pulmonary congestion dominate the clinical picture (fig. 7).

When heart failure is severe and the cardiac output markedly depressed, signs of peripheral hypoperfusion and shock (obtundation, oliguria, cold and cyanotic skin) dominate the clinical picture. Hemodynamically, cardiogenic shock is characterized by a very low cardiac index (about 1.5 l/min/m² or less) and a high pulmonary capillary wedge pressure (more than 20 mm Hg) (fig. 8). Pulmonary arterial pressure is also elevated, while right atrial pressure may be normal or increased.

Right ventricular failure is characterized

FIG. 8. Hemodynamic data from a patient with acute myocardial infarction and clinical signs of shock and pulmonary congestion. The cardiac index (CI) and arterial blood pressure (ABP) are low despite an increase in heart rate to 96 beats/min. The pulmonary capillary wedge pressure (PCW) is high.

by an increase in right ventricular end-diastolic and right atrial pressures and a decrease in stroke volume. In primary right heart failure, for instance, in the presence of a right ventricular myocardial infarction with no significant involvement of the left ventricle, the pulmonary capillary wedge and the pulmonary arterial end-diastolic pressures may be normal or low. If right heart failure is secondary to left heart failure, the pulmonary capillary wedge and pulmonary arterial end-diastolic pressures are also elevated.

PULMONARY EMBOLISM

In significant pulmonary embolism, mechanical obstruction and other factors combine to increase pulmonary vascular resistance. The hemodynamic findings are characteristic: the pulmonary arterial systolic and end-diastolic pressures and usually the right ventricular end-diastolic and right atrial pressures are elevated.[23] Importantly, the pul-
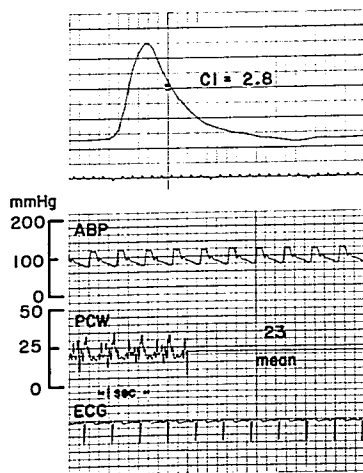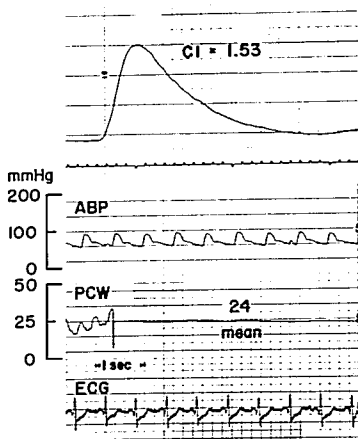

FIG. 7. Hemodynamic data from a patient with acute myocardial infarction and signs of pulmonary congestion. The elevation of pulmonary capillary wedge pressure (PCW) and reduction in stroke index (25 ml/beat/m²) indicate left ventricular failure. Due to increase in heart rate to 110 beats/min, the cardiac index is normal and there are no signs of peripheral hypoperfusion.
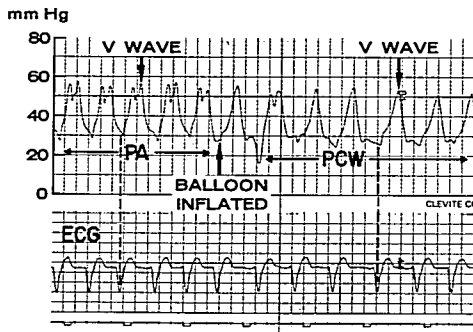
monary capillary wedge pressure is normal or low. The discrepancy between the pulmonary arterial end-diastolic pressure and the pulmonary capillary wedge pressure (5 mm Hg or more) indicates an increase in pulmonary vascular resistance.

CHRONIC DIFFUSE PULMONARY DISEASE

Pulmonary vascular resistance is frequently elevated in the presence of severe diffuse pulmonary disease, producing pulmonary hypertension and secondary hypertrophy of the right ventricle. The term "pulmonary hypertension" implies an elevation of the pulmonary arterial pressure to levels above the accepted limits of normal, *i.e.*, 35/15 mm Hg. In the absence of left ventricular abnormality the pulmonary capillary wedge pressure is normal. Failure of the overloaded right ventricle will result in an increase of the right ventricular end-diastolic and right atrial pressures.

ACUTE MITRAL REGURGITATION

Acute mitral regurgitation may occur as a complication of myocardial ischemia, acute myocardial infarction, or severe left ventricular failure of any origin. Acute mitral valve incompetence as the mechanism underlying the sudden development of a systolic murmur can be clearly demonstrated by the large and peaked "v" waves recorded in the pulmonary wedge position (fig. 9).

CARDIAC TAMPONADE

In cardiac tamponade the rising intrapericardial pressure interferes with the diastolic filling of the heart, causing the pulmonary capillary wedge, pulmonary arterial end-diastolic, right ventricular end-diastolic, and right atrial pressures to rise markedly to a similar level, so that the usual difference between the pulmonary capillary wedge pressure and the right atrial pressure practically disappears (fig. 10). Stroke volume is reduced. A similar hemodynamic picture can be seen in constrictive pericarditis and restrictive cardiomyopathy.

Complications

COMPLICATIONS OF BALLOON CATHETERIZATION

The flexibility of the flotation catheter shaft and the protective function of the inflated balloon when the catheter passes through the right ventricle tend to minimize the occurrence of complications. However, the simplicity of the catheterization procedure itself leads sometimes to underestimation of potential hazards, associated with any invasive procedure, and to unnecessary complications.

Balloon Rupture

Rupture of the balloon is not uncommon in catheters that have been subjected to many uses or used continuously over an extended

mm Hg

FIG. 9. Pressure tracing from a patient with severe mitral regurgitation. Transmitted V waves are seen deforming the pulmonary arterial pressure tracing (PA). The pulmonary capillary wedge (PCW) tracing obtained by inflation of the balloon shows giant V waves, characteristic of mitral regurgitation.

FIG. 10. Pressure tracings from a patient with cardiac tamponade. Characteristically there is equalization of right atrial (RA), right ventricular (RV) end-diastolic, pulmonary arterial (PA) end-diastolic, and pulmonary capillary wedge (PCW) pressures, with values of about 20 mm Hg.
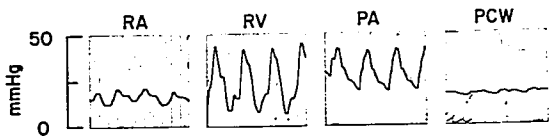
period (several days). Leakage of 0.8 or 1.5 ml of air into the circulation normally has no detectable consequence. However, penetration of air into the left side of the circulation may result in cerebral or coronary embolization. For this reason, carbon dioxide and not air should be used as the inflation medium in patients in whom the possibility of a right-to-left shunt exists, or when balloon flotation catheters are applied for catheterization of the left heart and aorta, particularly in pediatric cardiology.

Pulmonary Infarction

Balloon flotation catheters may migrate peripherally and become wedged in a small branch of the pulmonary artery. If undetected, prolonged wedging may result in pulmonary infarction which, however, is usually small, asymptomatic, and detectable only by a roentgenogram of the chest.[24]

More severe pulmonary infarction can be induced by keeping the balloon inflated in wedge position, thereby occluding a larger branch of the pulmonary artery, for an extended period. Pulmonary capillary wedge pressure should, therefore, be measured only intermittently, for not more than 1 or 2 minutes at one time.

Rupture of Pulmonary Artery

When the catheter tip is in a small branch, inflation of balloon to its full capacity can damage the pulmonary arterial wall,[25] particularly in patients who have pulmonary hypertension and structural changes in the vessel wall.[26]

To avoid damage to the pulmonary artery by overdistention, it is recommended that in the absence of fluoroscopic control, inflation of the balloon be performed slowly and stopped immediately when the change from pulmonary arterial to pulmonary capillary

wedge pressure is detected. If the inflation volume is substantially less than the maximum recommended, the catheter should be pulled back to a position in which wedge pressure is obtained at near-maximum inflation volumes.

Knotting

Knotting of the catheter is more likely with the small catheters and less likely with the larger ones, in which the shaft's flexibility is limited. If a knot forms, the balloon should be deflated, the catheter gently withdrawn to the site of insertion, and if necessary, a venotomy performed for removal of the tightened knot.[27] Successful unknotting with the use of an appropriate guidewire has also been reported.[28]

To minimize the likelihood of knotting by coiling of the catheter in the right atrium or right ventricle, advancement of the catheter should be discontinued if the right ventricle is not reached within 60 cm from the right antecubital fossa, 70 cm from the left antecubital fossa, 35 cm from the internal jugular and subclavian veins, or 50 cm from the femoral vein. Once in the right ventricle, the catheter tip should reach the pulmonary artery after it has been advanced no more than 15 cm. If the right ventricle and pulmonary artery are not reached within the anticipated distances, the catheter should be pulled back and insertion attempted again. Stiffening of the shaft by injection of 10–20 ml of a cold solution may facilitate entry into the pulmonary artery.

Rhythm Disturbances

Although cardiac arrhythmias of significance are rare due to the protective effect of the inflated balloon and the flexibility of the catheter shaft, instances of ventricular fibrillation and short episodes of ventricular tachycardia have

been reported. The catheter should not be advanced into the right ventricle unless the balloon is inflated. Catheterization must be performed during continuous electrocardiographic monitoring, and equipment for cardiovascular resuscitation must be at hand.

Thromboembolic Complications

Whenever a foreign body exists in the cardiovascular system, it may serve as a focus for build-up of clots.[29,30] The likelihood of thrombosis increases with the severity of the patient's illness. Under usual circumstances, anticoagulation is not necessary; however, if a hypercoagulable state exists, or if prolonged monitoring is needed, anticoagulation should be considered.

Infections

Infections and localized thrombophlebitis associated with manipulation of the catheter and inadequate vein sterilization techniques, have been reported. These necessitate removal of the catheters. If continued monitoring is necessary, a new catheter should be inserted from a different vein.

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