# Bedside Assessment of Left Ventricular Function in the Respiratory Intensive Care Unit*

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The measurement of the central venous pressure is a widely used technique for gaining information concerning the relationship of the blood volume as it relates to cardiac function. This concept has been widely popularized, and the central venous pressure measurement is extremely useful; however, it has too frequently been thought to be a direct measurement of cardiac function.

The normal value of the central venous pressure is 6 to 12 cm of water as measured vertically from the midaxillary line, preferably with the patient in a supine position. The tip of the catheter, if properly placed, is either in the superior vena cava or the right atrium. The pressure measured is obviously the mean value of the pressure in that particular vascular compartment. If we are to use the central venous pressure as a measurement of left heart function, we are really interested in the relationship of the superior vena cava or right atrial pressure to the left ventricular end diastolic pressure or the pulmonary capillary wedge pressure.

It is known that the mean right atrial pressure does not always accurately predict the mean left atrial pressure, and it is in reality the left atrial pressure that constitutes the filling pressure for the left ventricle and is the more important measurement. It is known that acute left ventricular failure may give rise to increased pulmonary capillary wedge pressure with pulmonary edema in the presence of a normal central venous pressure.

The cardiac output is partially determined by the amount of fluid that is available to the cardiac pump. Obviously, if only a small amount of fluid is placed into the pump, it follows that only a small amount of fluid can be ejected. It has been found that the central venous pressure measurement gives a good indication of the peripheral venous capacity and the state of the blood volume. If there is a low central venous pressure or low mean right atrial pressure, it generally indicates underfilling of the peripheral circulation which gives rise to a lowered cardiac output. An elevation of the central venous pressure usually indicates a full peripheral circulation, and this should lead to an increased cardiac output. An elevation of the central pressure in the presence of a normal to reduced peripheral volume generally indicates either a decrease in pump efficiency or an increased resistance to flow through the pulmonary vasculature.

The indications for monitoring the central venous pressure are:

1) Shock: In hypovolemic shock the central venous pressure is an invaluable guide to effective fluid replacement. The measurement of the CVP often provides the means by which we are able to wean patients off vasopressors after restoring adequate blood volume. In septic shock, there is frequently reduced blood volume as well as impaired cardiac efficiency. The central venous pressure measurement enables us to give the patient an adequate or optimal blood volume, and if this does not restore adequate blood pressure, we then add Isuprel® or massive steroids to increase pump efficiency and to dilate the peripheral vascular beds. In cardiac shock the monitoring of

central venous pressure has lesser value than hypovolemic or septic shock. It has been shown that elevation of the central venous pressure may be a relatively late sign of cardiac failure.

2) To monitor fluid therapy in patients with cardio-respiratory disorders: Patients with histories of congestive heart failure or chronic obstructive pulmonary disease are at relatively greater risk when exposed to the hazards of trauma, major surgery, or prolonged illness with protracted vomiting or blood loss. One of the primary reasons for this much greater risk is the difficulty in optimally replacing the blood volume without overloading the circulation and causing diminution in cardiac function or interstitial edema, thus reducing pulmonary function.

3) Acute renal failure: In the presence of acute renal failure it is essential to rule out hypovolemia as a cause. This is a completely reversible cause, but it must be discovered and treated early in order to avoid acute tubular necrosis. Whenever there is any doubt as to the adequacy of the peripheral vascular volume in the presence of acute renal failure, the central venous pressure should be measured and monitored in an effort to eliminate prerenal factors as a cause of the renal failure.

The physician who is asked to see a patient with an elevated central venous pressure and a cardio-respiratory problem has to consider many factors. Pulmonary hypertension, that is, a pressure in the pulmonary artery greater than 35/15 mm Hg, can cause elevation of the central venous pressure.

As you look at the causes of pulmonary hypertension (Table 1) and note the variety of disorders and the variety of treatments that would be necessary, it is obvious that the mean right atrial pressure may at times be insufficient physiological data to enable us to arrive at a correct diagnosis.

Bedside clinicians have been measuring the pulmonary capillary wedge pressure for years by listening for the presence of fine inspiratory rales at the bases of the lungs which indicate the presence of pulmonary edema. It is known that rales and pulmonary edema do not occur unless the wedge pressure has approached 25 mm Hg. However, it must be kept in mind that pulmonary edema or pulmonary congestion may exist on a basis of a pulmonary disease giving rales in the lung bases that may be indistinguishable from those rales heard in congestive heart failure, in which

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**TABLE 1.**

**PULMONARY HYPERTENSION**
(Greater than 35/15 mm Hg)

1. Elevation of pulmonary capillary pressure and/or left atrial pressure.
   a. Increased volume, fluid overload.
   b. Elevated left ventricular end diastolic pressure.
   c. Mitral valvular disease.
   d. Obstruction proximal to the mitral valve.
2. Decrease in total cross sectional area of the pulmonary vascular bed.
   a. Pulmonary emboli.
   b. Medial hypertrophy of small arterioles.
   c. Functional reduction of the pulmonary vascular bed due to hypoxia and/or acidosis.
3. Increase in pulmonary arterial blood flow with left to right shunts.

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case the pulmonary capillary wedge pressure may be normal or may even be reduced. Therefore, it is obvious that a means for determining the pulmonary capillary wedge pressure at the bedside would be useful in the management of the more difficult cardio-respiratory problems. Formal cardiac catheterization is certainly of great help; however, it is frequently difficult to have use of the cardiac catheterization facilities on an emergency basis, and it is frequently very difficult to transport the patient from the intensive care unit to the cardiac catheterization laboratory. Bedside right heart catheterization is now a practical procedure using the recently perfected Swan-Ganz catheter.

The Swan-Ganz balloon tipped catheter is a number 5 French double lumen catheter with a small balloon at its tip that can be inflated with 0.8 cc of air or carbon dioxide. The Swan-Ganz catheter can be inserted into the vein either percutaneously through a 12 gauge needle or via a cutdown, and is advanced to the right atrium much as a central venous pressure catheter is inserted. When the pressure tracing curves, typical of the catheter with its tip in the thoracic cage, are seen, the balloon is inflated. The catheter is carried by the current of blood and quickly passes through the right ventricle into the pulmonary artery. In the original report concerning this catheter, the average time for passage from the right atrium to the pulmonary artery was 35 seconds, and the ease of passage has been borne out by our use of the catheter in the respiratory intensive care unit.

Once the catheter is in the pulmonary artery, the balloon is left blown up, and it is then slowly advanced to the wedge position at which time the pressure contour will damp considerably. At this point, the balloon is deflated and the catheter is advanced approximately another two to three centimeters. The tip now lies in a small peripheral pulmonary artery, and with the balloon down we can measure the pulmonary artery pressure. When the balloon is inflated the pulmonary artery pressures are damped, and the pressure recording then is essentially that of the pulmonary capillary wedge pressure. This procedure does not require the use of fluoroscopy and can be done quite safely if the pressure pulse contours and the electrocardiogram are monitored. It has been determined that the pulmonary capillary wedge pressures as determined by the Swan-Ganz catheter, when compared with simultaneous direct left atrial pressure measurements, have been approximately equal. There have been no serious cardiac arrhythmias reported as yet with the Swan-Ganz catheter. There appear to be fewer cardiac arrhythmias when using the Swan-Ganz catheter than there are when using the regular cardiac catheter under direct fluoroscopic control. The explanation for this probably lies in the fact that the tip of the catheter is cushioned by the balloon. Other complications would be those persuant to any indwelling catheter left in position for a prolonged period of time.

I would like to describe several cases in which the Swan-Ganz catheter and bedside measurement of pulmonary capillary wedge pressure were invaluable in helping us to direct therapy. The first case is that of a 59-year-old man who was referred to the Medical College of Virginia due to severe dyspnea. He had been hospitalized approximately two weeks prior to his admission because of congestive heart failure and chronic obstructive pulmonary disease. He had been treated with digoxin, diuretics, and bronchodilators with improvement. Following his discharge his condition deteriorated and he developed a cough with purulent sputum and was transferred to MCV where he arrived with severe tachypnea and cyanosis. His past history revealed admission to MCV three years previously with congestive heart failure, atrial flutter, and chronic obstructive pulmonary disease. With cardioversion, digoxin, and the care of the lung disease, he had improved and no specific cardiac diagnosis was made. On physical examination on admission, his temperature was 100.8°, his pulse was

106, his respiratory rate was 36, his blood pressure was 120/80, and there was neck vein distention at 40°. Chest examination revealed that his lungs were hyperresonant to percussion, that there was an increased anterior-posterior diameter, and that there were bilateral diffuse wet fine inspiratory rales. Examination of the heart revealed a grade III holosystolic murmur maximal at the xyphoid radiating to the axilla. There was an S-3 gallop and there was a question of an opening snap. His liver was enlarged and was somewhat tender. The extremities showed 1$^+$ pitting edema. The arterial blood gases on room air on admission showed a $P_{O_2}$ of 29, $P_{CO_2}$ of 75, pH of 7.22, with a bicarbonate of 29. Following treatment with a 24% ventimask the $P_{O_2}$ rose to 45, the $P_{CO_2}$ slightly improved at 70, the pH was improved at 7.31, and the bicarbonate was 34. It was felt initially that the elevation of his neck veins and his obvious cor pulmonale could be explained almost exclusively by chronic obstructive pulmonary disease. However, his chest x-ray revealed cardiomegaly, and there was a hazy pulmonary infiltrate with increased blood flow to the apices. This led to speculation that there was something present other than chronic obstructive pulmonary disease. A Swan-Ganz catheter was inserted, and it was found that his pulmonary artery pressure was 75/37 with a mean pressure of 42, and his wedge pressure had a mean of 20. The wedge pressure remained elevated even after his arterial blood gases were improved. This provided sufficient stimulus to get a formal cardiac catheterization. Significant mitral stenosis was found and mitral commissurotomy was performed. The patient has done quite well following this operation.

The second case involved a 23-year-old man admitted due to a progressively worsening cough productive of bloody, foamy sputum. He had first noted the cough six weeks prior to admission and had received Lincocin® with slight improvement. However, he later became worse and began to cough up bloody sputum and was referred to MCV. He delayed his admission one week and became much worse and was admitted *in extremis*. Physical examination showed blood pressure was 94/60, pulse was 120, respiratory rate was 40, and temperature was 102°. The patient was acutely ill, sitting straight up in bed, and was exceedingly short of breath. He had palpable cervical axillary and inguinal nodes. The neck veins were distended, and he was coughing up a foamy, pink sputum which was thought to be characteristic of pulmonary edema. He had rales throughout his

lung fields and an enlarged tender liver. Review of his chest x-rays showed a widespread pulmonary infiltrate, and there was a question as to whether his cardiac silhouette had enlarged. Blood gases showed that the $P_{O_2}$ was 42, the $P_{CO_2}$ was 29, and the pH was 7.38 with a bicarbonate of 16. The initial diagnosis was pulmonary edema with cause unknown. In this particular case, we were unable on clinical grounds to adequately determine whether this was pulmonary edema on the basis of lung disease or pulmonary edema on the basis of cardiac disease. He was transferred to the respiratory intensive care unit and a Swan-Ganz was passed; this showed that the pulmonary artery pressure was 45/35 mm Hg, and the pulmonary capillary wedge pressure was quite low at 0 to 5 mm Hg. This information led to an open lung biopsy which revealed noncaseating granulomas and an interstitial pneumonia. He was treated with isoniazide, streptomycin, ethambutal, and solu-medrol. He was ventilated with a volume respirator via a tracheostomy performed at the time of lung biopsy. Over the next week his pulmonary congestion rapidly cleared and his blood gases returned to normal. This case demonstrates quite graphically that pulmonary edema is not necessarily on a cardiac basis and it may be difficult, if not impossible, to tell at the bedside which is the primary physiological disturbance.

The third case in which the Swan-Ganz catheter was helpful concerns that of a young man who was brought to our emergency room unconscious with pinpoint pupils and irregular slow respirations. He was immediately intubated, placed on a respirator, and given 10 mg of nalline intravenously. He became more responsive and was coughing up foamy sputum. His blood gases on admission revealed a $P_{O_2}$ of 35, a $P_{CO_2}$ of 70, and a pH of 6.94 with a bicarbonate of 14. The chest x-ray showed diffuse pulmonary edema with a normal cardiac silhouette. The patient at this time had a Swan-Ganz catherization performed and revealed a low pulmonary capillary wedge pressure. This finding allowed us to push fluids to increase his blood pressure, even in the presence of pulmonary edema. He was continued on the ventilator, and over the next 18 hours he was able to be extubated; within three days he was completely normal. At that time cardiac consultation was ob-tained and it was felt that there was no cardiac basis for the pulmonary edema. We were unable to elicit a positive history of heroin or other drug abuse.

In summary, central venous pressure measurement has greatly extended our ability to care for severely ill patients with cardiopulmonary disorders and difficult fluid balance problems. However, the central venous pressure measurement is essentially a measurement of the mean right atrial pressure. It may not be a reliable indicator of the mean left atrial pressure, which is essentially the filling pressure of the left ventricle that determines the cardiac output and the blood pressure. There is now available, and in use at this institution, a bedside method for determination of the pulmonary artery and pulmonary capillary wedge pressures. In selected cases, the data obtained with this catheter have been of great aid in management of the patient. The ease and safety of the procedure seems to be well documented and should make it accepted both by the doctor and the patient.

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