Clinical Utility of Daily Patient Weights in the Coronary Care Unit

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It has been estimated that upward of 50 percent of all patients with an acute myocardial infarction (AMI) experience some degree of left ventricular failure (LVF). With the refinement of arrhythmia detection and treatment, pump failure is now the major cause of death in patients admitted to the coronary care unit. Because early detection and treatment of LVF impacts significantly on patient morbidity and mortality, the recognition and communication of changes in patients' hemodynamic status is an integral part of coronary care nursing. The completeness of the data base is fundamental to the planning of care and thus to the quality of cardiovascular nursing practice.

In past years, daily weighing of patients in the coronary care unit was an important parameter in the detection of fluid retention with LVF; assessment of weight gain and loss is also included in the Standards of Cardiovascular Nursing Practice. However, six more recent critical care nursing texts addressing assessment of cardiovascular function did not include daily patient weights in the suggested parameters to be routinely monitored. Several reasons for this can be cited, including the increasing popularity of the flow-directed balloon-tipped pulmonary artery catheter and the ready availability of intake and output records.

The flow-directed balloon-tipped pulmonary artery catheter has proven to be a major clinical tool for the early detection of LVF and pulmonary vascular congestion. Yet, not all patients with an AMI can or should undergo intrapulmonary vascular pressure monitoring. Monitoring fluid balance with intake and output records has been shown to be grossly inaccurate. According to Grant and Kubo, "body weight, measured daily is the most accurate bedside measurement of a patient's hydration status." The logistic difficulty of actually weighing a bedfast patient connected to a vast array of tubes and wires causes one to stop and consider the desirability and clinical utility of daily weighing in the bedside assessment of cardiovascular function. Wells has implored nurses to know the basis of their clinical activities—not what they do, but why. Thus, the question of whether or not patients should be weighed on a daily basis in the coronary care unit can best be answered by reviewing the physiology of fluid retention with AMI and looking at patient weight patterns after admission to the coronary care unit.

MECHANISMS OF FLUID RETENTION WITH ACUTE MYOCARDIAL INFARCTION

With AMI, there is a loss of viable contractile muscle mass and therefore of contractile force; left ventricular ejection fraction per beat is reduced. The vasomotor center becomes "aware" of this problem through the aortic and carotid artery baroreceptors and sympathetically mediated vasoconstrictive mechanisms are activated to redistribute the circulating blood volume and augment left ventricular filling prior to contraction. It must be remembered that the somatic experience of myocardial infarction and the psychologic impact of admission to the coronary care unit produce anxiety in the patient. The ensuing state of arousal can have profound effects on the sympathetic nervous system and thus on the total body response to the AMI (see Figure 1).

With activation of the sympathetic nervous system and renal arteriolar vasoconstriction, blood flow is shunted away from the kidney. Perfusion pressure at the glomerulus is lower and glomerular filtration is decreased. Receptors within the juxta-glomerular apparatus are sensitive to both the decreased perfusion pres-
sure within the afferent arteriole and the decreased sodium load subsequently delivered to the macula densa, and reflexly cause an increased release of renin. The renin-containing granular cells of the juxta-glomerular apparatus are also directly stimulated by sympathetic post-ganglionic fibers to secrete renin.\(^{12}\)

Once renin is secreted, it triggers the conversion of circulating angiotensinogen from the liver to angiotensin I. In the lungs, angiotensin I is converted to angiotensin II, which stimulates the adrenal cortex to secrete aldosterone. Aldosterone then acts on the distal tubules and collecting ducts of the kidney to increase reabsorption of sodium, and consequently, water. In addition, psychological arousal stimulates the secretion of ACTH, another hormone that stimulates the adrenal cortex to secrete aldosterone.

Angiotensin II is a powerful vasoconstrictor and probably intensifies the perfusion changes already mediated by the sympathetic nervous system. The catecholamines and angiotensin II also act directly on the renal tubular cells to stimulate sodium reabsorption.\(^ {13}\) Psychological arousal and angiotensin II stimulate secretion of antidiuretic hormone (ADH), which in turn increases the reabsorption of water from the distal tubules and collecting ducts of the kidney. ADH also has vasopressor effects, though not as powerful as angiotensin II. There is also evidence that atrial and ventricular baroreceptors can modify renal function and secretion of ADH and are probably involved in the regulation of blood volume.\(^ {14}\)

**CLINICAL SIGNIFICANCE OF FLUID RETENTION AFTER AMI**

In the normal myocardium, the increased blood volume and venous return would serve to increase the diastolic stretch on the myocardial fibers, increasing the force of systolic contraction and thus the ejection fraction per beat. But the increased force of contraction, greater diameter of the ventricular chamber, and elevated arterial resistance also increase myocardial oxygen consumption, aggravating the initial problem of myocardial ischemia. The subsequent decrease in left ventricular ejection fraction intensifies the vasomotor and neuroendocrine responses, setting up a vicious cycle of vasoconstriction, fluid retention, and increasing myocardial ischemia (see Figure 2).

It has been reported that in patients admitted to the coronary care unit with an AMI, there is an almost immediate rise in serum catecholamines and cortisol that persists for one to two days.\(^ {15,16}\) The rise in serum catecholamines is believed to explain the temporary rise in patient blood pressures and heart rates after admission to the coronary care unit. It may also contribute to any fluid retention seen after admission as well. Because of the changes in muscle wall compliance and cardiac output with myocardial ischemia, it is reasonable that the body would activate mechanisms to increase left ventricular filling pressure in order to maintain a more normal left ventricular ejection fraction.
It is not known at what point sympathetically mediated mechanisms might adversely affect the ischemic myocardium and what role psychologic activation of these mechanisms may play in tipping the balance. Karlsberg, et al. found that serum levels of catecholamines correlate with patient survival from AMI, though it is unknown if they reflect the extent of myocardial injury incurred or contribute to the extent of the injury ultimately incurred. The relationship between psychologic arousal and sympathetic nervous activity led Gibson to postulate that perhaps “the simplest unloading therapy in early uncomplicated AMI is reassurance, meticulous attention to relief of pain, and adequate sedation.”

CLINICAL PATTERNS OF FLUID RETENTION AFTER AMI

It would seem that the detection of fluid retention would be useful both in the early detection of left ventricular failure as well as the identification of patients at greater risk by virtue of their total body response to the psychophysiologic experience of myocardial infarction. The question remains as to how best to detect it. In an attempt to answer this question, charts of 29 patients admitted to the coronary care unit for an AMI were reviewed. Of the 12 charts with fairly complete weight and fluid intake and output records, 10 patients demonstrated a weight gain or greater intake of fluid than output in the first 48 hours after admission. Of 5 patients who did not develop clinical signs of LVF, all 5 had a weight gain of at least 0.5 kg. The retention of fluid was also evident in laboratory measurements of hemoglobin and hematocrit which showed hemodilution in the same time period. In one patient with an inferior wall and right ventricular infarction who received large amounts of fluid to achieve a higher pulmonary capillary wedge pressure, weight rose 3.0 kg before any signs of peripheral edema were noted.

There was no identifiable relationship between weight changes and intrapulmonary vascular pressures. This was probably because pressure monitoring lines were put in if the patient’s condition was deemed unstable, and nonoptimal pressures were immediately manipulated with fluids, vasodilators, and/or diuretics. Weight was done only on a 24-hour basis, and intrapulmonary pressures could change many times over in that time period. There was no instance of a detectable weight gain immediately preceding or coinciding with an elevation in pressures.

To further assess the clinical utility of daily weights in the detection of fluid retention with AMI, agreement between weight and recorded intake and output of fluids was checked. The calculated fluid balance differed from that indicated by the weight change per 24 hours by more than 0.5 liter 72 percent of the time. When the recorded output was adjusted to allow 500 cc per day insensible fluid loss, they differed 60% of the time. These observations support those of Pflaum.

SUMMARY

The development of left ventricular failure is of concern to cardiovascular nurses in their endeavors to detect early and possibly prevent some of the problems besetting the patient with an acute myocardial infarction. One can see from a psychophysiologic perspective that fluid retention is part of the total body response to an AMI and admission to the coronary care unit. Not all patients with an inferior left ventricular failure will develop fluid retention, and it is not always the case that fluid retention will progress to pulmonary edema. However, it is clear that fluid retention is a significant factor in the clinical course of AMI and should be closely monitored.

DECREASED CORONARY ARTERY BLOOD FLOW

MYOCARDIAL ISCHEMIA

DECREASED LEFT VENTRICULAR EJECTION FRACTION

INCREASED SYMPATHETIC NERVOUS ACTIVITY

increased levels of renin, angiotensin, aldosterone

increased peripheral vascular resistance

fluid retention

INCREASED MYOCARDIAL OXYGEN CONSUMPTION (INCREASED CARDIAC OUTPUT)

increased heart rate and contractility

Figure 2. Physiologic Cycle to Increase Cardiac Output.
will develop LVF nor will undergo intrapulmonary vascular pressure monitoring to ensure early detection of it. From this survey of clinical records, it does appear that there is a fluctuation in fluid balance that warrants attention, whatever its implications for care might be. If indeed fluid retention should be a patient problem, intake and output records may inaccurately reflect the patient's true hydration status, a good proportion of the time. Fluid intake and output records should be accompanied by daily weighing to alert caregivers to fluid shifts and calculation inaccuracies. Finally, a clinical study should be done to establish the frequency and extent of weight gain and fluid retention in patients with both a complicated and uncomplicated clinical course of recovery.

REFERENCES

13. Ibid., 111.

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