Monitoring Heart Failure Hemodynamics With an Implanted Device: Its Potential to Improve Outcome*

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The introduction of angiotensin-converting enzyme inhibitors and subsequently beta-blockers marked an important advance in the management of most forms of heart failure (HF). Recently, nonpharmacologic methods have been added to the armamentarium; these include surgical remodeling of the left ventricle, initially for ventricular aneurysm, but later for the severely dilated ventricle. Other recent advances are resynchronization for regional wall motion asynergy and a variety of cardiac restraints to decrease ongoing ventricular dilation and, thereby, transduction of signals responsible for ventricular remodeling. Although the morbidity and mortality of HF remain distressingly high, it is generally agreed that substantial gains have been made in its treatment.

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Until the introduction of methods for assessing ventricular systolic function several decades ago, our ability to diagnose HF remained static, and any but the most gross changes in the clinical course often went unrecognized. Despite an improved ability to assess systolic and particularly diastolic function, it is fair to say that this situation had hardly changed, especially in the case of patients managed in offices and clinics, until the clinical utility of brain natriuretic peptide (BNP) in HF was recognized several years ago and incorporated into clinical practice.

The inability to detect worsened HF (before a patient gains several pounds in weight and edema has greatly increased) has the unfortunate consequence that treatment is likely to be delayed until the patient requires hospital admission. Prophylactic treatment to obviate the necessity for hospitalization would clearly be preferable. Determining the level of BNP was a notable step in diagnosis. The level of this peptide, excreted by ventricular myocardium consequent to volume-overload states and when the heart has remodeled, provides a reliable, rapidly available, and inexpensive means of separating dyspnea of cardiac origin from dyspnea due to other diseases (1), notably chronic obstructive airways disease. General internists know that it is difficult to make this distinction based on clinical grounds. Furthermore, HF significantly alters the results of pulmonary function, somewhat limiting their value in apportioning dyspnea to the lungs of heart patients who have both conditions. Serial determinations of BNP, both short term in the hospital and longer term in the office or clinic, supplement established methods for assessing an improvement in, or a worsening of, HF (2,3). Thus, recently, progress has been made in the management as well as diagnosis. Physicians measure BNP when they suspect a significant change in a patient’s status; therefore, its use for monitoring the course of HF is sporadic and influenced by clinical bias. Parameters that can be accessed any time during the clinical course and under a variety of circumstances might therefore further enhance the management of HF.

In this issue of the Journal, Adamson et al. (4) report the results from data obtained by monitoring right ventricular (RV) pressure using a permanently implanted device in 28 adults ranging in age from young to old, with HF of differing severity. The investigators did not provide the data to the treating physicians for nine months, but from then until the completion of the 17-month-long monitoring period, the physicians managing the patients were allowed to use the data to help them make clinical decisions. The results of this study suggest that a permanently implanted device capable of long-term, continuous measurement of hemodynamic variables may, in the future, find a place in the management of patients with severe chronic HF.

The ability to provide these data constitutes a significant bioengineering accomplishment. If all that such monitoring provided were RV systolic and diastolic pressures, physicians would benefit when they see patients in the hospital, office, or clinic; but, importantly, the device also derives pulmonary arterial diastolic pressure from the micromanometrically measured pressure signal and its first derivative, using an established algorithm. Calibration was stable, baseline drift was negligible, and output was stable up to very high frequencies. Using a traditional lumen catheter attached to an external transducer yields unreliable results because there are hydrostatic errors when the patient’s posture changes, unless the level of the transducer is correctly readjusted. With a solid-state transducer, as used by the authors, changes in posture cause only small changes due to gravity in the measured pressure; hydrostatic errors are minimal. A lower value for ventricular and pulmonary arterial diastolic pressures when the patient is standing would thus be almost entirely physiologic, not a hydrostatic error. One can therefore be confident about the validity of the data and turn to the question of how they might ultimately be used.

Similar to changes in BNP determined sporadically, increased and decreased right heart pressures correlated with volume overload and depletion, respectively. Of particular importance, these changes in right heart pressures occurred

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24 h before the clinicians decided that an intervention, including hospitalization, was necessary. This observation encourages the thought that monitoring of this nature does, in fact, predict deterioration near the time it commences and may trigger earlier intervention. Patients in this study had moderately severe systolic pulmonary hypertension and RV diastolic pressure approaching 30 mm Hg. The data were acquired continually and stored. The device, in addition to its promise in patient care in the future, can provide new information on the pathophysiology of HF, the activity of the circulation to mental and physical stress, and possibly, provide another index of prognosis not otherwise readily obtainable using standard techniques. This information would come from analysis of hemodynamic variation in a manner comparable with, and perhaps complementary to, that provided by analysis of heart rate variability. Most patients undergoing cardiac catheterization to evaluate HF are studied in a postprandial state, supine, and lightly sedated, while they are not experiencing symptoms. A permanently implanted system, by contrast, would yield data under much more normal and variable conditions. It would record the hemodynamic responses when patients pursue normal daily activities or are performing treadmill or bicycle stress testing. Long-term right heart pressure monitoring can be exploited to study hemodynamics during normal sleep, and this monitoring may increase access to hemodynamic changes associated with sleep apnea. The ready availability of high-fidelity RV pressure recordings made during different volume status and patient activities would be of great interest to physiologists and physicians taking care of patients implanted with a cardiac support device. One of the concerns in these patients is the possibility that the restraining device could cause a constrictive pericarditis-like syndrome. This complication has not been found in animals or patients studied under basal conditions (5).

This pilot study establishes the feasibility of long-term hemodynamic monitoring of patients with HF, particularly outpatients who have chronic HF, and that the device furnishes trustworthy data. The method is invasive, and its suitability for clinical application will require rigorous testing in large clinical trials enrolling appropriate patients. The patients would presumably be in New York Heart Association functional class III or IV, as it would not be justified in patients with less severe HF. It would be necessary to demonstrate a statistically significant advantage over such standard measures as the BNP level and a patient's weight, radiologic assessment of pulmonary and systemic venous hypertension, and jugular pressure. One of the quality-of-life instruments that focuses on pathophysiologic findings of HF should be a component of the trial. The cost-effectiveness, calculated by comparing the savings resulting from fewer exacerbations and hospital admissions with the increased costs associated with the device, could then be estimated with acceptable accuracy.

How may this device be made even more valuable in the future to physicians treating HF? Perhaps the first modification to consider would be incorporation of an oximeter or an electrode that measures blood gas tension. The saturation or tension of oxygen in blood in the outflow tract of the RV is an excellent surrogate for cardiac output. It has the great advantage of being a directly measured variable, and it is nearly constant in normal subjects under basal conditions, rather than being calculated using a number of assumptions. It may become feasible to transmit the signal by telemetry to doctors' offices or organizations dedicated to participating in the long-term care of these patients. As a special application, the device so equipped, in association with cardiopulmonary exercise testing, including measurement of peak oxygen consumption, would be ideal for assessing patients for heart transplantation. Ideally, the device of the future might even measure pulmonary wedge pressure. Theoretically, this could be accomplished using a double lumen catheter with the proximal port in the RV outflow region and an end hole in a suitably sized branch of the pulmonary artery, with a balloon inflatable by an external signal. At present, such a device is not possible, but the authors are leading us along an exciting new path.

**REFERENCES**


