Great Tool or Gold Standard? B-Type Natriuretic Peptide and Congestive Heart Failure

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We would like to thank the contributing authors Manson et al[1] for their article "Identification of Sonographic B-Lines with Linear Transducer Predicts Elevated B-Type Natriuretic Peptide Level."By attempting to validate this easily learned and reproducible ultrasound technique, they have contributed to the growing body of research that suggests bedside thoracic ultrasound can provide rapid point-of-care testing to help guide the diagnosis of congestive heart failure (CHF) in the emergency department (ED).[2]

The rapid diagnosis of CHF in ED patients with undifferentiated dyspnea remains a challenge. Although we have a multitude of clinical information that suggests heart failure, no single aspect of the history, physical examination, echocardiogram, chest radiograph or blood chemistry is specific or sensitive enough to rule in or out the diagnosis.[3] Rather, clinicians must incorporate all of this data into their clinical assessment. Decision scores such as the Framingham and Boston criteria have been developed as systematic ways of weighing such data against the probability of CHF.[4,5]

B-type Natriuretic Peptide (BNP) is an important tool amongst these diagnostic modalities. Multiple studies have demonstrated its effectiveness, particularly its ability to help rule out the diagnosis of CHF when levels are below cutoff values (typically 100). This high negative predictive value is often used in the ED to distinguish dyspnea caused by heart failure from exacerbations of chronic obstructive pulmonary disease.[6]

It is important to remember, however, that BNP is not to be used as a stand-alone test. Multiple factors besides CHF can contribute to an elevated BNP level. These include advanced age, renal failure, acute coronary syndromes and atrial fibrillation.[7,8] BNP levels between 100 and 500 pg/ml fall into a range that are less helpful in excluding or confirming the diagnosis. Therefore, any interpretation of BNP should be made only after the clinician has established the pre-test probability of CHF using all available clinical data.

Just as an isolated BNP value is unacceptable as proof of diagnosis of CHF in clinical practice, so too is its use as a lone diagnostic standard in the research setting. The research submitted by Manson et al. does not attempt to validate bedside ultrasound as a means of differentiating CHF. Rather they test the ability of ultrasound to predict a cutoff value of BNP, a surrogate for the diagnosis. As a result, we are left with a correlation between ultrasound findings and lab values—each one of which must be interpreted with the corresponding patient's clinical presentation if it is to accurately suggest CHF.

If we are to validate such techniques in the future, we must use more accurate methods in establishing the diagnosis of CHF. Because there is no single sufficient diagnostic criterion, the research standard of diagnosing CHF should be the same as those in clinical practice—namely, the judgment of physicians. Prospective studies that include retrospective review by a panel of physicians, considering all presenting signs, symptoms, and diagnostic data of the subject in question, are the most widely accepted and reasonable basis of establishing the diagnosis of CHF. Although such methods are costly and time consuming, they should be considered the criterion standard for further research.

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