Letter to the Editor

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B-type natriuretic peptide serum levels in acute heart failure

We read with great interest the article by Cowie et al.1 on clinical applications of the assessment of B-type natriuretic peptide (BNP). This is an important subject and the article also helps to clarify which areas of BNP research need more attention. The authors conclude that in new patients presenting to emergency services with dyspnoea, if the BNP level is <100 pg/mL, then heart failure is highly unlikely to be present.

BNP has been used in many clinical studies to exclude and/or identify congestive heart failure (HF) in patients with dyspnoea,2 however, in most published studies the time from symptom onset to BNP measurement is not reported, and likely BNP levels increase after a few hours. A patient with pulmonary oedema could have a "normal" BNP serum level if he/she presented to the emergency room very soon after symptoms onset. Would the authors advocate for repeated BNP assessments? Acute HF is usually caused by an acute change in LV performance or in structural cardiac integrity (i.e., acute myocardial infarct, papillary muscle rupture or fulminant myocarditis).3 The role of BNP in patients with HF symptoms because of mechanical complications after an acute myocardial infarct and in myocarditis has not yet been evaluated. It appears that the exact role of BNP measurement in the diagnostic algorithm of acute heart failure, a term including many different pathophysiological entities, remains to be fully clarified.

We believe that more limitations of BNP assessment may become evident once it is used in everyday clinical practice. The authors report that BNP levels rise with age, are affected by gender and that several clinical circumstances can alter the clinical interpretation of BNP concentrations. Those states include ischaemia, infarction and renal failure, which are very common problems. What would the authors recommend here?

Finally, we would like to comment that the metabolic effects of BNP include lipolytic effects that are at least as big as, but independent of, the effects of catecholamines.4 BNP may directly contribute to tissue wasting processes, in acute and in chronic heart failure.

References


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Our review1 summarises the evidence of the clinical role of plasma BNP measurements in the management of patients in a variety of clinical settings and situations. As is clearly stated in the paper, and as all experienced physicians will know, BNP should not be used as a 'stand alone' test but should be interpreted in the clinical context. Nevertheless, the results of studies published to date (and further studies currently reporting at scientific meetings but not yet published in full) support the diagnostic utility of BNP measurement, particularly in patients with new symptoms. This is well accepted in the current ESC guidelines on the diagnosis of heart failure.2

Fillipatos and colleagues point out that the definition of ‘acute’ heart failure is still disputed. Indeed this is the case and the forthcoming guidelines from the ESC on acute heart failure are to be welcomed as they are likely to confirm the value of the measurement of plasma BNP in aiding the diagnosis of heart failure. The full evaluation of a patient with acute symptoms of heart failure, whether de novo or as a result of decompensation of chronic heart failure, requires the underlying cardiac abnormality and pathophysiology to be determined. The care of such patients is often poor, and BNP may be a useful aid to the non-expert clinician in raising the suspicion of this diagnosis more rapidly than is often the case. False negatives may occur—particularly if the cardiac dysfunction is sudden or an inappropriately high decision cut-off point for BNP has been selected. However, in usual practice the studies suggest that BNP measurement is a useful diagnostic aid for the clinician and more recent studies suggest that the circulating concentration of BNP can rise within minutes of a cardiac "insult".3

It is true that plasma BNP concentration rises with age and tends to be higher in women than men. Nevertheless, the huge rise of BNP found in most patients with symptomatic and untreated heart failure appears rather small in comparison to this "physiological" rise and is therefore of little importance in clinical practice. Of course, this would be more relevant if BNP were to be used to screen for asymptomatic LV dysfunction, but this is a very different clinical situation as our review points out.

The metabolic effects of BNP await confirmation in human studies. However, should these confirm that BNP does indeed play a role in tissue-wasting the case for monitoring BNP levels in patients with heart failure becomes yet stronger.

The data and our clinical experience continue to support the value of the measurement of plasma BNP in the management of patients with breathlessness and, particularly, heart failure.

Yours sincerely.

References

2. The Task Force for the Diagnosis and Treatment of Chronic Heart Failure, European