The right ventricle under acute and chronic overload: early detection of right ventricular dysfunction
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Citation for published version (APA):

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UTILITY OF A BRAIN NATRIURETIC PEPTIDE AS A MARKER FOR RIGHT VENTRICULAR DYSFUNCTION IN ACUTE PULMONARY EMBOLISM

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{J Am Coll Cardiol 2002; 89:2080}
We read with interest the study by Morrison et al\(^1\), in which it was shown that rapid measurement of brain natriuretic peptide (BNP) concentrations in blood is a sensitive and specific test for differentiating patients with heart failure from those with primary pulmonary causes of dyspnea in acute care settings. The authors stated that correct, accurate and early diagnosis of left ventricular (LV) dysfunction is imperative for improved survival. However, recent data indicate that this statement also applies to right ventricular (RV) dysfunction\(^2,3\). The clinical diagnosis of RV dysfunction, however, is usually more complex than that of LV dysfunction. RV function plays an important role in conditions of increased pulmonary vascular resistance (such as pulmonary embolism), resulting in increased workload of the RV and eventually in RV dysfunction and failure. RV function may deteriorate with increase in RV afterload accompanied by an increase in neurohormonal activation, but the increase of RV systolic pressure will be limited\(^2,3\). A suddenly increased pressure load on the RV is poorly tolerated because of the inability of the normally thin-walled RV to develop and sustain high wall tension and stress\(^1\).

In two recent studies\(^2,3\) we showed that there was no correlation between RV systolic pressure and BNP levels. In our opinion (unpublished results involving 114 patients with acute pulmonary embolism), patients with relatively low RV systolic pressure and high BNP (above 180 pg/ml) are at imminent risk for RV failure.

Concerning prognosis and management of patients with acute pulmonary embolism BNP concentrations between 80 and 300 pg/ml are of particular interest\(^1\). Indeed, low BNP levels have excellent negative predictive value and most patients with high BNP levels probably have already clinically manifest heart failure, leaving patients with intermediate BNP levels in “difficult to interpret” zone. Serial BNP measurements in combination with echocardiography should help solving this important clinical problem.

We found significantly higher plasma BNP levels in patients with RV dysfunction due to pulmonary embolism compared to patients with pulmonary embolism and normal RV function\(^1\). Therefore, BNP is of clinical importance as a supplementary tool for assessment of RV function and discrimination between patients with normal RV function, RV dysfunction and RV failure under circumstances of acute RV pressure overload. This might add to the purpose of the Morrison et al. study and differentiate heart failure from dyspnea of pulmonary etiologies even more accurate.

REFERENCES