

CARDIOGENIC VS PULMONARY DYSPNEA  
A DIFFERENTIAL APPROACH WITH BRAIN NATRIURETIC PEPTIDE (BNP)

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Today it is always more frequent in our hospital settings, to see new daily used devices, that let us have a lot of information previously obtained only by means of highly invasive techniques or the collaboration of biochemical laboratories with high specific characteristics and for this reason, often very expensive.

Among these there is a new technique for dosing the "B natriuretic peptide" (BNP) .

This paper aims to describe a dosing technique for BNP that in a very short time gives a quantitative determination, useful in cardiovascular monitoring, at the bedside and without needing complex and expensive typical devices of analysis laboratories. Its use is simple: BNP is dosed collecting a 2-3 cc sample of whole blood or plasma into a tube containing potassium EDTA, by means of an apposite kit read by a mechanical lector. In about 15 minutes is able to give a quantitative result expressed in pg/ml.

BNP values < 100 pg/ml suggest a non cardiac dyspnea, BNP > 100 pg/ml suggests an increasing in pressure or volume in cardiac atria and/or ventricles. BNP higher than 1000 pg/ml suggests a with high specificity and sensitivity cardiac source of dyspnea.

**Physiologic summary:** One of the regulation systems of cardiovascular homeostasis is claimed by the action of the natriuretic peptides, belonging to a family of cytoplasmatic and membrane peptides that activates the receptors of guanylyl cyclase A in separate tissues causing an increase in c-GMP concentration. This action mechanism has as end effect the interaction with the renin-angiotensin-aldosterone system, with antagonism to the action of the angiotensin II on the vascular tone, of the secretion of aldosterone, of the tubular reuptake of the sodium and of the growth of the endovasal endothelial cells. The end result is the electrolytes and water elimination from the kidney. With the elimination of electrolytes and water in the kidney as end result.

This mechanism occurs every time that the heart is submitted to increasing volume or pressure with the acquisition by cardiac myocytes of endocrine properties that rule the secretion of the natriuretic peptides in order to modulate the antihypertensive and antihypervolemic action.

The BNP, is mainly produced in the ventricles but it is dosable also in the atria, increasing subsequently to an increase of pressure and volume of one or more cardiac chambers.

The normal range in which the BNP varies in normal subjects is universal known, in the numerous studies about this argument, between 0 to 100 pg/ml. Actually, whereas there is no doubt in the clinical approach to be maintained concerning the values found within this interval, it is easy to find interpretative doubts about the higher values, but for those extremely high (> 1000).

Really there are some variables that may influence the basal dosage of BNP making the interpretation of the quantitative datum not always clear.

The renal failure seems to be an important factor to be taken into account in evaluating the gold standard baseline and defining the normal range.

Also age and sex seem to influence the basal values of BNP, even if not in all the studies the Authors agree about the influence of these two parameters on the interpretation of dosage values.

**Clinical applications:** The rapid response in the secretion in the ventricle of BNP in a very high concentration, makes this peptide a good marker for the individuation of the dilatation of one or more cardiac chambers.

One of the first use in a clinical setting of BNP has been the possibility of an early differential diagnosis between a pulmonary or cardiac disease in the early and sudden development of dyspnoea.

The pure pulmonary disease (pneumonia, pneumothorax,...), having as mechanism an alteration of the pulmonary tissue and of its being able to change respiratory gases, does not appear, at least

initially, as a circulation congestion with subsequent dilatation of the cardiac chambers. It is important to note that in all the published studies about this argument, there is an agreement about the possibility to exclude the cardiac overload as the cause of the acutedevelopment of dyspnoea if the at bedside dosage of BNP is lower than 100 pg/ml. Similarly, with a very high dosage (over 1000 pg/ml) there is no doubt that it is of vital importance to consider as the primary cause a cardiac failure.

However, the clinical cases do not always appear so clear because there is a large grey zone with BNP values greater than 100 pg/ml but lower than 1000 pg/ml. For these cases the literature has not yet arrived to an interpretation agreement.

- **Maisel, McCullough, Burnett, Peacock, Yancy**  
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MONITORING AND THERAPEUTIC ROLES OF NATRIURETIC PEPTIDES IN CARDIOVASCULAR  
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**Congestive Heart Failure**