B-type Natriuretic Peptide (BNP) and N-terminal pro B-type Natriuretic Peptide (NT-proBNP)

Description: These peptides are released in response to ventricular volume expansion and pressure overload.
Clinical Use: Elevated levels indicate heart failure and help differentiate it from other causes of dyspnea

B-type natriuretic peptide, commonly referred to as brain-type natriuretic peptide (BNP), was initially identified in 1988 following its extraction from porcine brain tissue. However, subsequent research revealed that its primary source is the heart, classifying it as a cardiac hormone. BNP is part of the natriuretic peptide family, which includes other structurally related peptides such as atrial natriuretic peptide (ANP), C-type natriuretic peptide (CNP), and urodilatin. These natriuretic peptides share a distinctive biochemical structure characterized by a 17 amino-acid ring and a disulfide bond between two cysteine residues. The ventricular myocardium is the principal site for BNP synthesis and secretion. In contrast to ANP, which is stored in granules for immediate release upon stimulation, BNP is stored in minimal quantities, with its secretion primarily regulated by rapid gene expression and de novo synthesis. BNP is produced as a prehormone known as proBNP, consisting of 108 amino acids. Once released into the bloodstream, it is cleaved into two equal parts: the biologically active 32 amino acid BNP, which is the C-terminal fragment, and the biologically inactive 76 amino acid N-terminal fragment (NT-proBNP). Both fragments are continuously released and can be measured in the blood. The primary trigger for the increased synthesis and secretion of BNP and NT-proBNP is myocardial wall stress, although factors such as myocardial ischemia and endocrine modulation by other neurohormones and cytokines also play significant roles. In the systemic circulation, BNP exerts various biological effects by binding to the natriuretic peptide receptor type A (NPR-A), leading to the production of intracellular cGMP. The physiological effects of BNP are diverse, including promoting natriuresis and diuresis, inducing peripheral vasodilation, and inhibiting the renin–angiotensin–aldosterone system (RAAS) as well as the sympathetic nervous system (SNS).
BNP is removed from plasma primarily by its interaction with the natriuretic peptide receptor type C (NPR-C) and through the action of neutral endopeptidases that facilitate proteolysis. In contrast, NT-proBNP is predominantly eliminated via renal excretion. However, recent research indicates that there may be additional significant mechanisms involved in the clearance of NT-proBNP. The half-life of BNP is approximately 20 minutes, while NT-proBNP has a half-life of around 120 minutes. This difference accounts for the fact that serum levels of NT-proBNP are roughly six times greater than those of BNP, despite both peptides being released in equimolar amounts.

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