Improved Utilization of BNP

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This *Heartbeat* will center on the utilization of brain natriuretic peptide (BNP) for the diagnosis, prognosis and treatment of congestive heart failure (HF). It can be used to stratify ischemic and valvular heart disease patients and may be used to identify congestive heart failure patients who are candidates for urgent heart transplantation. The utilization of BNP will improve risk stratification among a wide range of cardiac patients with a goal of more intensive treatment targeted to higher-risk patients.

Emerging importance of "triage" BNP

Because patients with left ventricular (LV) systolic dysfunction (LVEF <40%) have improved survival and improved quality of life on medications such as ACE-inhibitors, beta-blockers and aldosterone antagonists, it is imperative to make the correct diagnosis as accurately and as early as possible. This will enable both early and optimal maximal therapy. Similarly, the timing of aortic valve replacement is important in improving outcome for asymptomatic critical aortic stenosis patients. Evidence of significant increased LV wall tension could be the "tiebreaker".

B-type (brain) natriuretic peptide, or BNP, is a neurohormone released by the myocytes in the ventricular walls in response to volume expansion and/or pressure overload (tension). This is true for both the right and left ventricles, but obviously higher levels would be formed by the LV because of thicker walls (more mass). The BNP, a relatively inexpensive bedside blood test, correlates BNP level to presence or absence of HF in patients with acute or chronic dyspnea. This is especially true in the emergency room.

BNP along with clinical evaluation improves clinical judgments and triage. Results from the **B**rain Naturetic Peptide for **A**cute **S**hortness of Breath **E**va**L**uation (**BASEL**) trial showed that BNP testing reduces the time to initiation of therapy, the time to discharge and total treatment cost. Later data has additionally shown a 2 day decreased length of stay (LOS).

BNP is used fairly routinely in emergency departments to differentiate patients who have shortness of breath due to non-cardiac causes and patients who have heart failure. It is most helpful when the diagnosis of HF was intermediate. But critics are quick to point out that BNP is at its weakest in this intermediate range and clinical judgment is still the most important part of diagnosis. In a patient presenting with dyspnea, HF is *usually absent* at BNP levels less than 100 pg/mL with 83% accuracy. This improves to 96% if 50pg/ml is used as the cutoff. HF is *possible* between 100 pg/mL and 500pg/mL, and *probable* at levels greater than 500pg/mL. Bad, decompensated HF patients usually present with BNPs in the range of 800pg/mL to 1000 pg/mL.

BNP levels between 100 and 500pg/mL may also be seen in patients with known chronic LV dysfunction, lung disease or pulmonary embolism (BNP produced from the right ventricle), renal failure and MI. BNP is increased in patients with end stage renal disease (pre-dialysis) and in virtually all on dialysis—probably secondary to volume overload.

If a patient does have HF and a BNP less than 200pg/mL, events rates are extremely low, and they can usually be treated on an outpatient basis.

Improved Utility of BNP

Knowing the nuances of BNP can improve your interpretive skills:

- BNP increases with age (decreased LV compliance).
- BNP is higher in men than women (more ventricular mass).
- Obese patients have lower BNP levels, though usually not enough to confuse diagnosis.
- Decreased creatinine clearance (< 60ml/min) increases BNP. (Creatinine of >1.7 in men and > 1.5 in women can increase BNP to 100 to 500pg/mL depending on severity and volume status.
- Many patients with chronic HF secondary to systolic dysfunction just live with chronically elevated BNPs.
- BNP decreases with treatment.
- Most importantly BNP is not the "Holy Grail". It is not a stand alone test and must be used in conjunction with good clinical judgments.

Three BNP determinations per admission are enough. The first assists with diagnosis and gives you a baseline. The second, on the day after admission, documents the patient's response and a third at discharge is your prognosticator. The BNP should decrease by 50% after treatment in the hospital. A BNP at discharge < 350 predicts a 10% rate of death or readmission, and > 750 indicates an 80% chance of readmission. BNP can accurately identify patients at highest risk of death, so this single test can be used to stratify patients for transplant. A BNP of 5,000pg/mL or higher was associated with 60% increased risk for dying within three months of discharge, while a BNP of 1,500pg/mL carried a 37% increased three-month mortality risk. These are patients that should be considered for heart transplant programs.

Outpatient BNP Guide

BNP < 50pg/mL: Echocardiography and cardiac evaluation probably aren't necessary.

BNP >50pg/mL: Consider cardiac evaluation and echo.

BNP can be normalized to < 50pg/mL in patients with low LVEF 50% of the time.

Adjust medical treatment to BNP treatment targets. Beta blockers, angiotensin II blockade (ACE inhibitors, angiotensin receptor blockers) and diuretics (especially aldosterone antagonists) all lower BNP and higher doses decrease BNP further. Maximize all treatment to a BNP target of <50pgmL.

Chronic BNPs (>250pg/mL) are a bad prognosticator.

BNP can help in the differential of shortness of breath in the elderly—most commonly due to age and decreased functional capacity. Here again, clinical judgment is of paramount importance because of some elevation of BNP occurring secondary to age, decreased LV compliance and some azotemia versus HF secondary to diastolic dysfunction.

In summary, BNP can be used *to help* with diagnosis, prognosis and as a guide to optimizing multiple neurohormonal strategies in concert or instituting more aggressive intervention. The results of this assay have to be interpreted cautiously in each setting for each patient and cannot replace or supersede clinical judgment. Unfortunately, this is especially true in that *possible* 100pg/mL to 500pg/mL range (where it would be most valuable), when multiple clinical variables and chronic HF make the differential much more difficult.

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