In providing primary care, general practitioners (GPs) form the backbone of health care systems in most countries. In the last few decades we have witnessed a relentless increase in specialization in all fields of medicine, advances in medical technology (particularly in imaging techniques), budgetary restrictions, and high patient expectations. As a consequence, most GPs now find themselves in a keenly competitive situation (1). The introduction and clinical validation of novel laboratory tests for common diagnostic and prognostic challenges offer valuable help for GPs.

B-type natriuretic peptides (BNP) and N-terminal (NT)-proBNP are quantitative markers of heart failure (HF), summarizing the extent of systolic and diastolic left ventricular dysfunction, valvular dysfunction, and right ventricular dysfunction (2,3). They have been shown to be extremely helpful in the management of patients presenting with dyspnea to the emergency department (ED) (4). Among ED patients with dyspnea, HF is the cause in about one-half.

Most patients with dyspnea, however, will present to their GP rather than to the ED. In comparison with their ED colleagues, GPs are on the whole more experienced and additionally benefit from having detailed knowledge of their patients’ past medical and personal histories. On the other hand, their access to further investigations (for example, chest radiograph, pulse oximetry, spirometry, computed tomographic scanning, and echocardiography) and specialist consultation is more limited. In addition, disease severity may be less and mean age may be greater in patients who present to their GP—2 factors that further increase the diagnostic challenge (3).

The measurement of BNPs may, therefore, be useful for GPs as well as ED physicians, particularly because this can be done relatively cheaply and quickly using simple point-of-care devices. Previous reports suggesting that the use of BNPs might be as useful for the GP as they are for the ED physician (5–8) are confirmed in the excellent report by Mogelvang et al. (8) in this issue of the Journal. In a substudy of 959 patients reporting some degree of dyspnea in the Fourth Copenhagen City Heart Study, plasma proBNP concentrations were shown to be increased in left ventricular dilatation, hypertrophy, systolic dysfunction, or diastolic dysfunction, but were unaffected by pulmonary dysfunction.

Attention to the following 5 critical issues may help put these data into perspective. First, the incidence of HF as the cause of new or worsening dyspnea in patients presenting to their GP is uncertain, and may vary significantly according to the local setting. Available data suggest that HF is the cause of dyspnea in 25% to 35% of cases (5–8). Although this is significantly less than the 50% observed in the ED, it would still leave HF as the most common disorder leading to dyspnea in the primary care setting. Second, it is crucial to note that the identification of cardiac dysfunction using echocardiography as the gold standard is a different matter from determining whether HF is the cause of dyspnea. The latter additionally requires detailed patient history, physical examination, and at times additional tests (e.g., D-dimers and computed tomography scans to exclude or detect pulmonary embolism). One cannot assume that all patients with some degree of cardiac dysfunction on echocardiography do in fact have HF and cardiac dyspnea! Third, this distinction leads to the conclusion that echocardiography as a stand-alone test is an appropriate gold standard for the detection of cardiac dysfunction, but not for determining whether HF is the underlying cause of dyspnea. For example, left ventricular ejection fraction as a single variable is significantly less accurate for the diagnosis of HF as compared with BNP testing (5,9). On the other hand, because BNPs summarize the extent of systolic and diastolic left ventricular dysfunction, valvular dysfunction, and right ventricular dysfunction, their correlation with left ventricular ejection fraction is only modest (10). Accordingly, BNPs and echocardiography should be considered complementary methods, each providing a different window on the heart. Fourth, diastolic dysfunction is still poorly understood and there is considerable uncertainty surrounding its interpretation. For example, there is no consensus regarding which mitral inflow pattern constitutes normal diastolic function in a 75-year-old person. This uncertainty is highlighted in the current article, which reports Doppler echocardiographic findings interpreted as severe diastolic dysfunction in comparable frequency in asymptomatic individuals and in patients with dyspnea (see Table 1 of Mogelvang et al. [8]).
Although tissue Doppler scanning may provide additional insights, this technique is often not routinely applied. When discussing these fundamental questions, it is important to bear in mind that diastolic dysfunction is thought to account for HF in about one-half of all cases. Fifth, it is currently an unresolved question whether the use of BNPs for the evaluation of patients presenting with dyspnea to physicians in primary care requires specific cutoff values or whether the cutoff values validated in large ED studies can be applied. In the absence of a large observational primary care study with dedicated adjudication of the gold standard diagnosis, the predominance of New York Heart Association functional class I to III patients in recent ED studies seems to justify the use of identical cutoff values in patients presenting with new or worsening dyspnea (11,12), irrespective of the setting.

Overall, there is a sound basis for GPs to measure BNPs in patients with dyspnea. Available data suggest that BNPs are also the single most accurate variables in the diagnosis of dyspnea in the primary care setting. In recent studies, the area under the receiver-operator characteristic curve for the ability of BNPs to diagnose HF in primary care was approximately 0.85. Additionally, there is evidence from a large randomized controlled trial conducted by one of the most prestigious groups in this area showing that the use of BNPs significantly improves the diagnostic accuracy of HF by GPs over and above customary clinical assessment (6).

This study included 305 patients age 40 years or older presenting to their GPs with symptoms of dyspnea and/or edema of recent onset. We are currently awaiting the results of BASEL III–Private Practice (B-Type Natriuretic Peptide for Acute Shortness of Breath Evaluation Study III–Private Practice), an international, multicenter, randomized, controlled study of the impact of rapid BNP testing on patient management and resource use in patients presenting with new or worsening dyspnea to doctors in private practice. Enrollment into the study was completed in March 2007, and the results will document whether the increase in diagnostic accuracy offered by BNPs will ultimately improve patient outcome.

B-type natriuretic peptides are simple, rapid, inexpensive, and accurate quantitative markers of HF. All of the current evidence, including the data presented in this issue of the Journal, points us toward strongly encouraging our colleagues in primary care to use BNPs in patients who present with dyspnea.

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