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Detection of left ventricular dysfunction after acute myocardial infarction: comparison of clinical, echocardiographic, and neurohormonal methods

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Abstract

***Objective*—The SAVE study showed that captopril improves mortality in patients with left ventricular dysfunction after myocardial infarction and that this benefit occurred even in patients with no clinically overt heart failure. On the basis of this, it seems important to identify correctly which patients have left ventricular dysfunction after a myocardial infarction. The objective was to compare various methods of identifying patients with left ventricular dysfunction (left ventricular ejection fraction, LVEF, $\leqslant 40\%$) after acute myocardial infarction. The methods compared were echocardiography (quantitative and qualitative visual assessment), clinical evaluation (subjective assessment and three clinical score methods), and measurement of plasma concentrations of cardiac natriuretic peptide hormones (atrial and brain natriuretic peptides, ANP and BNP).**

***Design*—Cross sectional study of left ventricular function in patients two to eight days after acute myocardial infarction.**

***Setting*—Coronary care unit of a teaching hospital.**

***Patients*—75 survivors of a recent myocardial infarction aged 40 to 88 with no history of cardiac failure and without cardiogenic shock at the time of entry to the study.**

***Main outcome measures*—Sensitivities and specificities of the various methods of detecting left ventricular dysfunction were calculated by comparing them with a cross sectional echocardiographic algorithm for LVEF.**

***Results*—Clinical impression was poor at identifying LVEF <40% (sensitivity 46%). Clinical scoring improved this figure somewhat (modified Peel index sensitivity 64%). Qualitative visual assessment echocardiography was a more sensitive method (sensitivity 82%) for detecting LVEF <40%. Plasma BNP concentration was also a sensitive measure for detecting left ventricular dysfunction (sensitivity 84%) but plasma ANP concentration was much poorer (sensitivity 64%).**

***Conclusion*—Left ventricular dysfunction is easily and reliably detected by echocardiographic measurement of LVEF and also by a quick qualitative echocardiographic assessment but is likely to be missed by clinical assessment alone. High concentrations of plasma BNP maybe another useful indicator of left ventricular dysfunction, particularly in hospitals where not all patients can be screened by echocardiography or radionuclide ventriculography after myocardial infarction.**

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After acute myocardial infarction, the feature that most adversely affects long term survival is left ventricular dilatation, which in some studies ranks even higher than the severity of coronary artery disease as a prognostic feature.[1] Therefore a main objective in managing patients with an acute myocardial infarction is the prevention of infarct expansion, ventricular dilatation, and the ultimate progression to chronic heart failure. Early experimental and clinical studies have shown that angiotensin converting enzyme (ACE) inhibitors attenuate ventricular dilatation after an acute myocardial infarction.[2–5] Consequently several large trials have been initiated to determine their effect on mortality and the results of two of these trials have been published in full. The second cooperative new Scandinavian enalapril survival study (CONSENSUS II) gave enalapril to all patients after acute myocardial infarction irrespective of baseline ventricular function and found no benefit.[6] By contrast, in the survival and ventricular enlargement trial (SAVE), captopril was given to a selected group of patients, who had left ventricular ejection fractions (LVEF) $\leqslant 40\%$, within three to 16 days after an acute myocardial infarction and a 19% reduction in mortality was found.[7] The reasons for the conflicting outcome of these two studies are uncertain. One likely possibility is that treatment with ACE inhibition after an acute myocardial infarction may only be beneficial when given to patients who already have left ventricular dysfunction.[8–10]

A further important consideration from the SAVE study is that the mortality benefit was seen equally in those without clinically overt heart failure (Killip class I) as in those with overt failure. Therefore it seems logical that after a myocardial infarction all patients with an LVEF $\leqslant 40\%$ are identified irrespective of whether they have clinical failure or not. In

the SAVE trial, radionuclide ventriculography was used to assess left ventricular function and LVEF. Radionuclide ventriculography is costly, involves giving radioactivity to the patient, and is not widely available especially in district general hospitals in the United Kingdom. In practice clinical impression by the attending physician is often the first and sometimes the only form of assessment used. Clinical scores of a variety of clinical variables have been devised as prognostic indices and may be applicable to the detection of left ventricular dysfunction.[11–13] Echocardiography is also commonly used to assess left ventricular function providing both qualitative and quantitative information.[14 15] The sensitivity of this technique, however, depends on both the operator and the nature of the measurement. More recently, there has been interest in the measurement of plasma neurohormones as a measure of left ventricular dysfunction.[16 17] The cardiac natriuretic peptide hormones, atrial natriuretic peptide (ANP) and brain natriuretic peptide (BNP), are both high after an acute myocardial infarction[18 19] and in chronic heart failure.[20] The role of ANP in the detection of left ventricular dysfunction has previously been investigated with conflicting predictive accuracies.[18 21 22] Despite its name, BNP is mainly a cardiac hormone in humans and by contrast with ANP, is synthesised and secreted mainly in the cardiac ventricle.[23] It may, therefore be a more sensitive index of ventricular function. There have been no previous direct comparisons of all these methods in the same group of patients after an acute myocardial infarction. The aims of our study were to evaluate and compare clinical, echocardiographic, and neurohormonal (ANP, BNP) methods of identifying left ventricular dysfunction in an unselected and heterogenous group of patients who had survived the first 48 hours after an acute myocardial infarction.

Patients and methods

PATIENTS

From October 1992 to February 1993, patients admitted to the coronary care unit in our hospital with an acute myocardial infarction were randomly selected for study once they had survived the first two days. Diagnosis of an acute myocardial infarction required two of the following three criteria: (*a*) clinical history consistent with ischaemic pain lasting for a minimum of 30 minutes; (*b*) evolving ST-T wave changes on electrocardiography; and (*c*) increased creatinine kinase MB fraction >6%, or a significant increase in total creatinine kinase for more than two days without other explanation. The criteria for exclusion were a history of chronic heart failure or cardiogenic shock at the time of entry to the study. All patients gave their informed verbal consent to this study, which was approved by the local hospital ethics committee.

ECHOCARDIOGRAPHY

Qualitative and quantitative assessment by cross sectional echocardiography was attempted in all patients with a Hewlett Packard Sonos 1000 and Hewlett Packard calculation programme.

Qualitative assessment

Left ventricular function was assessed visually by echocardiography by an independent operator who was unaware of the clinical history and physical examination of the patient. Each echocardiographic evaluation was classified as "good" (no left ventricular dysfunction); fair (mild to moderate left ventricular dysfunction) or poor (severe left ventricular dysfunction).

Quantitative assessment

When possible the following measurements in diastole and systole were obtained: long axis left ventricular internal diameter; short axis left ventricular area at the mitral valve and papillary muscles; apical two chamber left ventricular area; and longitudinal length. Echocardiographic assessment of LVEF was calculated by the Hewlett Packard calculation programme according to the modified Simpson's rule method,[14] the Bullet method,[24] and the single[25] and biplane[26] ellipse methods, depending on available measurements. When more than one method of calculation was possible, the modified Simpson's rule method was taken as the best estimate.

CLINICAL ASSESSMENT

Clinical impression

An experienced independent clinician assessed all patients for the presence of left ventricular dysfunction on day 2 after acute myocardial infarction, from clinical history, physical examination, cardiac enzyme concentrations, as well as electrocardiographic and radiological findings.

Clinical scores

Besides a clinical impression, three different clinical score methods (Killip score, Peel index, and modified Peel index) were used to indicate the presence of left ventricular dysfunction.

The Killip score is based on clinical findings alone.[12] Patients in Killip class I had no evidence of chronic heart failure; patients in class II had evidence of left ventricular dysfunction manifested by an S_3 gallop, rales, or both; patients in class III had evidence of frank pulmonary oedema manifested by an S_3 gallop, rales, and tachypnoea. Patients in class IV (cardiogenic shock) were excluded by the study protocol.

The Peel index is a weighted clinical score[11] and comprised the following variables: historical variables comprising previous acute myocardial infarction angina after infarct, hypertension, diabetes, angina before infarct; clinical variables comprising the presence and degree of cardiogenic shock, the presence of a gallop rhythm, jugular venous distension, pulmonary rales, sinus tachycardia, and arrhythmias (atrial flutter, fibrillation, frequent extrasystoles, heart block); electrocardio-

Table 1 Modified Peel index

Variables	*Score*
History:	
Previous myocardial infarction	6
Hypertension	1
Exertional dyspnoea	1
Angina only	1
Diabetes	1
No cardiovascular disease	
Shock:	
Absent	0
Mild—transient at onset	1
Moderate—present on admission but subsiding with rest and sedation	5
Failure:	
Absent	0
Few basal rales only	1
Dyspnoea, acute pulmonary oedema, orthopnoea, gallop rhythm, oedema or jugular venous distension	4
Electrocardiogram:	
Normal, RT, Twaves changes only	1
QR complexes	3
QS or BBB	4
Rhythm:	
Sinus	0
Atrial fibrillation, flutter, sinus tachycardia frequent extrasystoles, nodal or heart block	4
Chest x-ray film:	
Normal	0
Venous congestion, interstitial oedema, aveolar/consolidation, oedema, increased cardiothoracic ratio	4
Angina: after infarction	
No	0
Yes	3
Maximum creatine kinase (CR) ($\times 10^{-3}$ U/l)*:	
$1< CK \leqslant 2$	1
$2< CK \leqslant 3$	2
$3< CK \leqslant 4$	3
$4< CK \leqslant 5$	4
$5< CK$	5
Thrombolysis:	
Yes	0
No	2
Site of infarction:	
Inferior/lateral/posterior	0
Anterior/anteroseptal/ anterolateral	3

*The height of CK is 1–2 × normal or 2–3 × normal etc.

graphic variables included were bundle branch block, QS or QR complexes, and RT or T wave changes. As originally described, a score of 10 or more is associated with poor prognosis and hence was taken to indicate left ventricular dysfunction. Derivation of the modified Peel Index included all the variables mentioned, and also incorporated radiological findings, site of infarct, maximum creatinine kinase concentration, and whether thrombolytic treatment was given (table 1).

CARDIAC HORMONES

Venous blood (20 ml) was sampled for measurement of plasma ANP and BNP on the day of echocardiographic assessment. It was collected from patients who had rested in a semirecumbent position for 30 minutes and was collected into 10 ml chilled tubes containing EDTA and 4000 kallikrein inhibitory units of aprotonin (Bayer, Newbury, Berkshire). All samples were placed on ice, centrifuged at 4°C at 3000 rpm for 10 minutes, and the plasma separated off and stored at −20°C until assayed. All samples were assayed blind in a single batch. Plasma BNP was assayed after plasma extraction with a commercially available radioimmunoassay (Peninsula Laboratories, Belmont, CA, USA) as previously described by our group.[27] The human BNP-32 antibody used was generated against human BNP-32 (Peninsula Laboratories) and showed the following cross reactivities: human BNP-32, 100%; rat BNP-32, 0·04%; rat BNP-45, porcine BNP-26, 0%; α-human ANP (1-28), 0%. The range of percentage recoveries for ^{125}I labelled human BNP-32 with this assay was 57·4%-81·4%. Plasma ANP was also measured by radioimmunoassay (Amersham International, Buckinghamshire) after plasma extraction by the method of Richards *et al.*[28]

RADIONUCLIDE VENTRICULOGRAPHY

Radionuclide ventriculography was performed whenever possible at the Department of Medical Physics, at our hospital, which provides diagnostic services for all specialities in the Tayside region and serves a population of 390 000.

Multigated equilibrium blood pool imaging was performed after in vivo labelling of red cells with 800 MBq technetium-99m sodium pertechnetate. Twenty four frame image sets were acquired with a General Electric (GE) 400 XCT gamma camera (GE, Wisconsin, Illinois, USA) in a modified left anterior oblique position with a caudal tilt to provide good ventricular separation. The LVEF was calculated by a semiautomated technique with a GE 3000 computer system.

STATISTICAL ANALYSIS

Differences between groups were compared by Student's *t* test. The strength of relations between values for LVEF calculated from radionuclide ventriculography and cross sectional echocardiography were evaluated by determination of the mean (SD) differences between the techniques (bias) and its standard deviation by the Bland and Altman method. Sensitivity for each method was calculated as the number of true positives identified divided by the sum of true positives and false negatives. Specificity was calculated as the number of true negatives divided by the sum of true negatives plus false positives. True positives and false negatives were defined as those having an LVEF $\leqslant$40% by cross sectional echocardiography. Similarly true negatives and false positives were defined as those with LVEF >40% by cross sectional echocardiography.

Results

Seventy five patients (51 men, 24 women) were studied. The mean (SD) age was 62·6 (9·8) (range 40 to 88). Thirty eight (51%) had anterior and 30 (40%) had inferior infarctions (table 2). A previous myocardial infarction had occurred in 21 (28%) patients and 57 (76%) had Q waves on the electrocardiogram. Thrombolysis was given to 51 (68%) patients, according to our usual clinical practice. One important point is that 53% of the patients who had a reduced LVEF had no clinical evidence of failure (Killip class I, table 2). This figure is similar to that found in the SAVE study, and in that study those patients still benefited greatly from ACE inhibitor treatment.

Table 2 Clinical characteristics of patients with reduced and with normal LVEF

	LVEF ⩽ 40% (n = 28)	*LVEF > 40% (n = 29)*
Mean age (range) (y)	63 (40 to 84)	61 (46 to 88)
M/F	21/7	18/11
Mean LVEF (SD)†	30 (9)	50 (8)
Anterior myocardial infarction (%)	20 (71)	12 (41)
Q wave infarction (%)	20 (71)	22 (76)
First infarct (%)	19 (68)	22 (76)
Thrombolysis (%):		
Yes	19 (68)	22 (75)
No	9 (32)	7 (25)
Killip class (%):		
I	15 (53)	24 (83)
II	3 (11)	4 (14)
III	10 (36)	1 (3)
Mean Peel index (SD)	13 (9)	6 (4)
Mean modified Peel index (SD)	18 (10)	9 (4)
Mean atrial natriuretic peptide (SD)	41·1 (35·9)	25·7 (17·5)*
Mean brain natriuretic peptide (SD)	28·3 (18·4)	16·4 (10·2)**

*P < 0·05; **P < 0·01. †LVEF was calculated from cross sectional echocardiographic algorithms.

ECHOCARDIOGRAPHY

Echocardiographic assessment was done two to five days after the acute myocardial infarction. Image quality was considered good or fair in 54 (72%) and poor in 21 (28%). More men (40%) than women (20%) had good quality images.

Qualitative assessment

Visual echocardiographic assessment was possible in all but one patient, and graded as good (no left ventricular dysfunction) in 35 (48%) patients, fair (mild to moderate left ventricular dysfunction) in 19 (26%), and poor (severe left ventricular dysfunction) in 19 (26%). Table 3 shows that visual assessment of echocardiography was both sensitive (82%) and specific (86%) in predicting an LVEF of ⩽40%.

Quantitative assessment

Calculation of the LVEF by echocardiographic algorithm was possible in 57 (76%) patients, with the modified Simpson's rule model in 45 (80%) patients, the Bullet model in three (5%), the biplane ellipse method in three (5%), and the single plane ellipse method in six (10%). The median calculated echocardiographic LVEF was 41% (range 15% to 69%). Twenty eight patients (50%) had an LVEF ⩽ 40%. This echocardiographically calculated LVEF is used in table 3 as the reference by which the clinical criteria, the neurohormonal criteria, and the visual assessment echocardiography are judged. This gold standard was used for practical reasons as the echocardiography and the hormone samples were taken on the same day, whereas this was not the case with the radionuclide ventriculography. Furthermore, cross sectional echocardiography is available in all hospitals, whereas this is not true of radionuclide ventriculography.

CLINICAL ASSESSMENT

Clinical impression

The clinician's assessment was more accurate in predicting normal rather than abnormal left ventricular function. Table 3 shows that patients with poor left ventricular function were noticeably underdiagnosed by clinical impression when evaluated against the calculated echocardiographic LVEF criteria for left ventricular dysfunction. Indeed, its sensitivity was only 46%.

Clinical score

The sensitivities of the Killip score (46%) and the Peel index (54%) were also poor predictors of LVEF ⩽ 40%. The modified Peel index was better. A threshold value of ⩾13 in the modified Peel index had a sensitivity of 64% (table 3).

CARDIAC NATRIURETIC PEPTIDE HORMONES

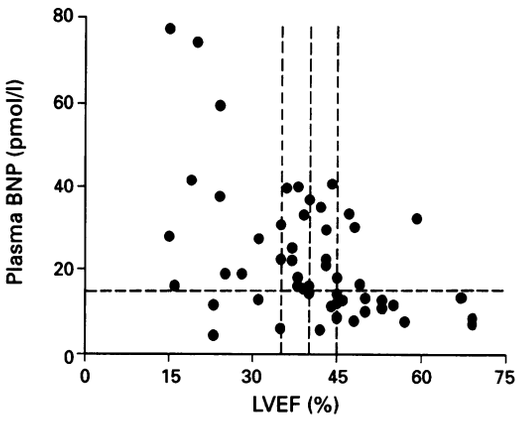
In our laboratory, normal age matched salt replete subjects have mean (SD) plasma ANP and BNP concentrations of 7 (4) and 3·2 (0·4) pmol/l. In our group of patients after an acute myocardial infarction, plasma ANP was 33·8 (27·4) pmol/l and plasma BNP was 22·6 (15·9) pmol/l. The mean plasma BNP concentration was significantly higher in the group of patients with LVEF ⩽ 40% compared with the group with LVEF > 40% (P = 0·002; figure; table 2). There was a small significant difference in mean plasma ANP concentration between those with normal and reduced LVEF (p = 0·04). A plasma ANP value of 20 pmol/l had only a 64% sensitivity and a 45% specificity in identifying an LVEF ⩽ 40% calculated from echocardiographic measurements (table 3). Plasma BNP performed better with a value of 15 pmol/l, having an 84% sensitivity.

RADIONUCLIDE VENTRICULOGRAPHY

For logistic reasons, it was not possible to carry out radionuclide ventriculography in all patients. Data were only available for 34

Table 3 Sensitivity and specificity of various methods used to detect left ventricular dysfunction after acute myocardial infarction compared with LVEF calculated from cross sectional echocardiographic measurements

	LVEF ⩽ 40%		*LVEF ⩽ 35%*		*LVEF ⩽ 45%*	
Method	*Sensitivity*	*Specificity*	*Sensitivity*	*Specificity*	*Sensitivity*	*Specificity*
Clinical assessment:						
Clinical impression	46	89	53	82	40	90
Killip score	46	83	47	74	46	91
Peel index (score ⩾10)	54	81	59	72	51	86
Modified Peel index (score ⩾13)	64	86	71	71	54	86
Cardiac Hormones:						
Brain natriuretic peptide > 15 pmol/l	84	62	77	47	81	73
Atrial natriuretic peptide > 20 pmol/l	64	45	65	41	66	48
Echocardiography:						
Quick qualitative assessment	82	86	88	69	71	91



Relation between plasma BNP and echocardiographic calculations of LVEF. Cut off points are shown for a plasma BNP of 15 pmol/l at LVEFs of 35%, 40%, and 45%.

patients who underwent radionuclide ventriculography within three days of their clinical, echocardiographic, and neurohormonal assessment. These patients were identical in all clinical aspects to those who did not undergo radionuclide ventriculography. The radionuclide ventriculography LVEF ranged from 10% to 68% with a median value of 45%. These radionuclide ventriculography data serve mainly as reassurance that our echocardiographically calculated LVEFs were accurate as there was a reasonably good relation between the LVEFs calculated by both methods in those patients (n = 23) who underwent both procedures. The echocardiographic calculations systematically underestimated the LVEF compared with radionuclide ventriculography by 6·2% (6·9%) (mean (SD)) as has been reported before.[14] Part of this difference in our study may be the three day gap between the two measurements.

DIFFERENT LVEF CRITERIA

Table 3 shows how various methods perform if the cut off point for ACE inhibitor treatment is moved from LVEF $\leqslant$ 40% to either LVEF $\leqslant$ 35% or LVEF $\leqslant$ 45%. This information may be useful because the LVEF 40% cut off point in the SAVE study was arbitary. Physicians and cardiologists would probably prefer to overtreat these patients with ACE inhibitors rather than undertreat them so that the data for LVEF $\leqslant$ 45% are of more interest than the data for LVEF $\leqslant$ 35%. At the cut off point of an LVEF $\leqslant$ 45%, the overall results are similar to those of an LVEF $\leqslant$ 40%—that is, qualitative assessment of the echocardiogram—and plasma BNP are the most sensitive methods available, with sensitivities of 71% and 81%.

Discussion

Several different methods are available to detect left ventricular dysfunction. Radionuclide ventriculography is a validated method of assessing left ventricular function but as a screening procedure it has limitations in terms of availability, cost, and the need to give a radioactive dose.[29–32] Echocardiography is widely available and may be done at the bedside although it is less objective, requires trained personnel, and would need to be expanded considerably in most district general hospitals in the United Kingdom if it were to be used for screening of all patients after a myocardial infarction. A formal echocardiographic calculation of LVEF takes 10–45 minutes and is further limited by the fact that some patients are poorly echogenic: in this study the figure was 24%, which compares favourably with other reports.[14] On the other hand, a quick qualitative echocardiogram is rapid and much less limited by poor echogenicity. As well as these advantages, we have now shown that a quick qualitative echocardiogram is both sensitive and specific at detecting left ventricular dysfunction. Hormonal assays such as ANP and BNP are not widely available at present but the technology and expertise are available to do these assays in all hospital laboratories. Their main advantage is their objectivity and their cheapness. In comparing the techniques, sensitivity is more important than specificity as overtreating these patients with ACE inhibitors is preferable to undertreating them. This preference is partly based on the finding from both the SOLVD and SAVE studies that ACE inhibitors also reduce episodes of reinfarction and unstable angina, although this is controversial. It is also based on the fact that ACE inhibitors produce few side effects when given to patients with milder versions of left ventricular dysfunction.

In the clinical assessment of left ventricular dysfunction, both clinical impression and clinical scores were studied. The Killip score, which is based entirely on physical findings alone, clearly is of limited value in this group of patients. The Peel index was originally designed as a prognostic indicator in patients with acute myocardial infarction rather than as an indicator of left ventricular dysfunction but as it assesses the haemodynamic derangement produced, we thought that it might also reflect left ventricular dysfunction after an acute myocardial infarction. The Peel index itself does not include site of infarction, the maximum rise in creatine kinase, or radiological findings, which may well contribute to left ventricular function after an acute myocardial infarction.[33–35] Thrombolysis is a new entity since the original Peel index was devised and this is clearly another main influence on left ventricular dysfunction. Therefore we included thrombolysis along with the other variables into our modified Peel index. By incorporating these variables, we were able to improve its level of sensitivity. None the less, our data showed that clinical assessment or a clinical scoring system are still unreliable and likely to overlook up to half of the patients who would benefit from ACE inhibitor treatment. Indeed, our findings support previous studies showing that clinical evaluation is inadequate in detecting left ventricular dysfunction after an acute myocardial infarction.[33 34 36] The reason for this may be that patients with depressed left ventricular ejection fractions initially have an increase in end systolic rather than end diastolic volume and

it is thought that only when both volumes are increased does haemodynamic decompensation become clinically apparent.[34 37]

The cardiac hormone ANP is synthesised and released mainly from the atria in response to atrial distension, and produces natriuresis, vasodilation, and diuresis in humans. Pathological increases are found both after an acute myocardial infarction and in patients with chronic heart failure, but reports of its value as a marker of left ventricular function have been conflicting.[21 38] In patients with an acute myocardial infarction, ANP tends to rise early and peak two to four days later.[22 39] Some increase is seen even in the absence of any left ventricular dysfunction or increased left ventricular pressure.[39 40] Indeed, a basal release of ANP may occur as a general response to stress or even as a leak from infarcting or ischaemic tissue. This may be why plasma ANP was such a poor indicator of left ventricular dysfunction after a myocardial infarction.

Although originally isolated from the porcine brain as a putative neurotransmitter, BNP is mainly a cardiac hormone like ANP.[20 23 41] By striking contrast with ANP, BNP is predominantly synthesised and secreted in the cardiac ventricle and it may therefore be a more sensitive index of ventricular function.[42] Indeed, Mukoyama *et al* have reported increased plasma concentrations of BNP that correlated strongly with the severity of disease in patients with chronic heart failure.[20] Two previous studies have examined BNP secretion after acute myocardial infarction. In a small group of 13 patients, Mukoyama *et al* found that BNP but not ANP correlated inversely with cardiac index.[19] More recently, we found that BNP correlated better than ANP with LVEF although that study was performed in a highly selected group of patients with anterior, Q wave first acute myocardial infarction.[43] In our present study, BNP secretion was evaluated in a larger and totally heterogenous group of patients and we still found that BNP was a better predictor of a reduced LVEF $\leqslant$ 40% than ANP. There were, however, more false positives than in our previous study—that is, in this study there was a subgroup of patients with normal LVEFs but with unexplained high BNP concentrations. The reasons for this difference are probably multiple including the heterogenous nature of these patients. Also, the presence of chronic obstructive pulmonary disease,[44] diastolic dysfunction,[45] and renal impairment[46] are known to be associated with high plasma BNP concentrations. These indices did not seem to explain the false positives. One could speculate that these patients might have ongoing ischaemia induced diastolic dysfunction that releases BNP or perhaps BNP is really measuring something different from LVEF itself—that is, BNP may be a sensitive marker of regional wall stress whereas LVEF is a relatively crude measure of left ventricular systolic contractility. The use of LVEF as a gold standard is due to its use in the SAVE study and not because it has any intrinsic merit in choosing patients. Clearly further studies are required to investigate why BNP is high in some patients with relatively normal LVEFs.

In conclusion, we have shown that clinical assessment alone is likely to miss about half of the patients who have asymptomatic left ventricular dysfunction. Echocardiography including a quick qualitative assessment is a reliable and sensitive method in the detection of left ventricular dysfunction and should be performed in all patients after a myocardial infarction. If echo facilities are too limited to screen all such patients, the measurement of BNP is another sensitive method of identifying left ventricular dysfunction. Alternatively, the echocardiographic burden could be reduced by only performing echocardiography in those without clinical heart failure as these patients benefit from ACE inhibitors anyway,[47] and because in our study the presence of clinical failure (Killip II, III) was fairly sensitive at identifying an LVEF < 40%. According to this study, such an approach would halve the number of echocardiograms needed for this group of patients.

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