Do patients with suspected heart failure and preserved left ventricular systolic function suffer from "diastolic heart failure" or from misdiagnosis? A prospective descriptive study

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Abstract

Objectives To characterise the clinical features of patients with suspected heart failure but preserved left ventricular systolic function to determine if they have other potential causes for their symptoms rather than being diagnosed with "diastolic heart failure."

Design Prospective descriptive study.

Setting Outpatient based direct access echocardiography service.

Participants 159 consecutive patients with suspected heart failure referred by general practitioners.

Main outcome measures Symptoms (including shortness of breath, ankle oedema, and paroxysmal nocturnal dyspnoea) and history of coronary heart disease and chronic pulmonary disease. Transthoracic echocardiography, body mass index, pulmonary function tests, and electrocardiography.

Results 109 of 159 participants had suspected heart failure in the absence of left ventricular systolic dysfunction, valvular heart disease, or atrial fibrillation. Of these 109, 40 were either obese or very obese, 54 had a reduction in forced expiratory volume in 1 second to ⩽70%, and 97 had a peak expiratory flow rate ⩽70% of normal. Thirty one patients had a history of angina, 12 had had a myocardial infarction, and seven had undergone a coronary artery bypass graft. Only seven patients lacked a recognised explanation for their symptoms.

Conclusions For most patients with a diagnosis of heart failure but preserved left ventricular systolic function there is an alternative explanation for their symptoms—for example, obesity, lung disease, and myocardial ischaemia—and the diagnosis of diastolic heart failure is rarely needed. These alternative diagnoses should be rigorously sought and managed accordingly.

Introduction

With the recent wide availability of non-invasive assessments of left ventricular function it has become apparent that many patients diagnosed as having heart failure have preserved left ventricular systolic function.[1 2] It has been proposed that these patients have abnormalities of ventricular filling in diastole, and the term "diastolic heart failure" has been coined.[1–3] It does, however, seem likely that given the non-specificity of the symptoms and signs used to diagnose heart failure at least some of these patients may not have abnormalities of diastolic ventricular function but other causes of their symptoms altogether. We studied consecutive patients who were referred with a diagnosis of suspected heart failure by general practitioners to our direct access transthoracic echocardiography service and were found to have preserved left ventricular systolic function. We defined their clinical characteristics and considered the alternative diagnoses of obesity, respiratory disease, and ischaemic heart disease.

Methods

Patients—We studied patients with preserved left ventricular systolic function who were referred with suspected heart failure to an outpatient based direct access cardiography service. Patients with left ventricular systolic dysfunction (according to qualitative "eyeball" assessment, see below), valvular heart disease, and atrial fibrillation were not studied further. The study was approved by our local committee for medical ethics. Each patient gave written informed consent.

Medical history, drug history, and symptoms—A full clinical history was taken. Symptoms of shortness of breath at rest or on exertion, paroxysmal nocturnal dyspnoea, and ankle swelling were specifically recorded. A past or current history of angina was also specifically elicited. The severity of angina was graded according to the Canadian Cardiovascular Society.[4] Current medication was recorded.

Body mass index—Body mass index was calculated in the usual way (weight (kg)/height $(m)^2$). Participants with a body mass index of less than 18.5 were defined as underweight, 18.5-24.9 as normal weight, 25.0-29.9 as overweight, 30.0-39.9 as obese, and ⩾40 as extremely obese.[5]

Respiratory function—All patients had peak expiratory flow rate measured and had spirometry performed—that is, forced expiratory volume in 1 second (FEV_1) and forced vital capacity.

Electrocardiography—A standard, resting 12 lead electrocardiogram was recorded in each patient.

Editorial by Hobbs

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Table 1 Characteristics of patients referred for investigation of suspected heart failure. Figures are number (percentage) of patients unless stated otherwise

	Men (n=34)	Women (n=75)	All (n=109)
Mean (SD) age (years)	70 (13)	72 (11)	71 (11)
Mean (SD) heart rate (beats/min)	72 (18)	75 (15)	74 (16)
Mean (SD) systolic blood pressure (mm Hg)	148 (19)	154 (24)	152 (23)
Mean (SD) diastolic blood pressure (mm Hg)	84 (9)	85 (11)	85 (11)
Symptoms†			
Short of breath at rest	11 (32)	16 (21)	2 (25)
Short of breath on exertion	29 (85)	71 (95)	100 (92)
Paroxysmal nocturnal dyspnoea	8 (24)	17 (23)	25 (23)
Ankle swelling	14 (41)	60 (80)	74 (68)
Concomitant conditions*:			
Hypertension	16 (47)	35 (47)	51 (47)
Myocardial infarction	6 (18)	6 (8)	12 (11)
Angina	12 (35)	19 (25)	31 (28)
CABG	5 (15)	2 (3)	7 (6)
Pulmonary disease	9 (26)	17 (23)	25 (23)
Cigarette smoking			
None	4 (12)	29 (39)	33 (30)
Current	12 (35)	17 (23)	29 (27)
Former	18 (53)	29 (39)	47 (43)
Medication†			
Diuretics	28 (80)	57 (76)	85 (78)
β blockers	9 (26)	9 (12)	18 (17)
Nitrates	10 (30)	12 (16)	22 (20)
Calcium channel blockers	7 (21)	15 (20)	22 (20)
ACE inhibitors	2 (6)	4 (5)	6 (6)
Aspirin	14 (41)	19 (25)	33 (30)
Inhaled β_2 agonists	7 (21)	15 (20)	22 (20)
Inhaled corticosteroids	4 (12)	4 (5)	8 (7)
Hypoglycaemic agents	2 (6)	4 (5)	6 (6)
Lipid lowering drugs	2 (6)	1 (1)	3 (3
NSAIDs	3 (9)	6 (8)	9 (8)
Digoxin	1 (3)	3 (4)	4 (4)
Warfarin	1 (3)	1 (1)	2 (2)

*Not mutually exclusive, patients can be in more than one category. CABG: coronary artery bypass grafting; ACE: angiotensin converting enzyme; NSAID: non-steroidal anti-inflammatory drug.

Pathological Q waves were taken as evidence of previous myocardial infarction. ST/T changes, previous myocardial infarction, or left bundle branch block were considered to be consistent with a diagnosis of coronary heart disease.

Transthoracic echocardiography—Each patient underwent transthoracic echocardiography with an Acuson 128XP10c, with the patient recumbent in the left lateral decubitus position. The same operator (LC) performed all examinations. Qualitative assessment of left ventricular systolic function was made by the "eyeball" technique, with two dimensional images.[6 7] Quantitative assessment was made by measurement of ejection fraction and fractional shortening by using M mode echocardiography. Ejection fraction was also measured using the Simpson's biplane method.[8] For diastolic function the E:A ratio was measured as described previously.[1 9] Left ventricular hypertrophy was assessed by M mode echocardiography.

Results

Patients—One hundred and fifty nine patients with suspected heart failure were referred, and 34 had left ventricular systolic dysfunction (18 out of 61 men (30%; 95% confidence interval 19% to 41%) and 16 out of 98 women (16%; 9% to 23%)). Ten patients had atrial fibrillation, two patients had valvular disease, and four patients had both atrial fibrillation and valvular heart disease. One hundred and nine patients had suspected heart failure in the absence of left ventricular systolic function, valvular heart disease, or atrial fibrillation. Details of these 109 patients is shown in table 1. In keeping with findings of epidemiological studies, the patients were elderly and usually female. Most patients had been prescribed diuretics. Thirty three patients were non-smokers, 29 were current smokers, and 47 were former smokers.

Dyspnoea and ankle swelling—Most men and women reported dyspnoea on exertion, and about a quarter reported either dyspnoea at rest or at night (table 1). Men and women differed in their reporting of ankle swelling, however, with twice as many women (80%) complaining of this symptom.

Angina, myocardial infarction, and coronary artery bypass graft—Thirty one (28%; 20% to 36%) patients had a history of angina though only 11 (10%; 4% to 16%) currently had symptoms (seven were classified as grade I, four as grade II). Twelve (11%; 5% to 17%) patients gave a history of myocardial infarction, and seven had undergone a coronary artery bypass graft. In total, 33 (30%; 21% to 39%) patients either had a history of angina or myocardial infarction or had undergone a coronary artery bypass graft. Eight patients had electrocardiographic evidence consistent with coronary heart disease.

Body mass index—Of the 109 patients with suspected heart failure in the absence of left ventricular systolic dysfunction, valvular heart disease, or atrial fibrillation, 108 had their body mass index calculated. Details are

Table 2 Prevalence of abnormalities of body mass index, forced expiratory volume in one second (FEV_1), and electrocardiographic abnormalities. Figures are number (percentage) of patients

	Men	Women	All
Body mass index (kg/m²)			
Underweight	0	1 (1.3)	1 (1)
Normal	8 (24)	15 (20)	23 (21)
Overweight	16 (47)	32 (43)	48 (44)
Obese	8 (24)	23 (31)	35 (32)
Extremely obese	2 (6)	3 (4)	5 (5)
FEV_1 (%) of predicted			
<50%	8 (24)	16 (21)	24 (22)
50-70%	14 (12)	16 (21)	30 (28)
71-90%	8 (24)	32 (43)	40 (37)
>91-100%	1 (3)	4 (5)	5 (5)
>100%	2 (6)	5 (7)	7 (6)
Electrocardiography			
Normal	17 (50)	53 (71)	70 (64)
Abnormal	17 (50)	22 (29)	39 (36)
Abnormalities on electrocardiography*			
Left bundle branch block	0	2 (3)	2 (2)
Left ventricular hypertrophy	2 (6)	1 (1)	3 (3)
Myocardial infarction	4 (12)	1 (1)	5 (5)
ST/T changes	4 (12)	9 (12)	13 (12)
Right bundle branch block	2 (6)	3 (4)	5 (5)
Paced	0	0	0
Bradycardia	2 (6)	1 (1)	3 (3)
Conduction abnormality	2 (6)	3 (4)	5 (5)
Right ventricular hypertrophy/right axis deviation	0 (0)	2 (3)	2 (2)

*Categories not mutually exclusive.

shown in table 2. Thirty five (32%; 23% to 41%) were obese, and five (5%;1% to 9%) were extremely obese.

Respiratory function tests—Of 109 patients, 106 had their respiratory function measured. Table 2 shows the results of these measurements. Fifty four patients (50%; 41% to 59%) had FEV_1 less than 70% of that predicted, and 97 (92%; 86% to 98%) had a peak expiratory flow rate ⩽70% of normal.

Electrocardiography—Seventy (64%; 55% to 73%) patients had a normal results on electrocardiography. More men than women had abnormalities. These are shown in table 2. As left bundle branch block, myocardial infarction, and ST/T changes are typical findings in coronary heart disease, 24% of men and 16% of women had electrocardiographic evidence of possible coronary heart disease. Taken in conjunction with evidence from the clinical history (33 patients), eight additional patients had either clinical or electrocardiographic evidence of possible coronary heart disease—that is, a total of 41 (38%; 29% to 47%) patients had either clinical or electrocardiographic evidence of possible coronary heart disease.

Transthoracic echocardiography—Table 3 shows measurements of left ventricular systolic function. By using the E:A ratio, 67% (74% men and 64% women) had "diastolic dysfunction." Left ventricular hypertrophy was detected in about one quarter of patients.

Table 3 Electrocardiographic parameters in patients referred with suspected heart failure

	Men	Women	All
Mean (SD) fractional shortening	29 (8)	30 (7)	30 (7)
Mean (SD) ejection fraction (M mode)	55 (12)	56 (10)	56 (11)
Mean (SD) ejection fraction (Simpson's biplane)	44 (11)	46 (9)	45 (10)
No (%) with left ventricular hypertrophy:			
Mild	4 (12)	18 (24)	22 (20)
Moderate	2 (6)	3 (4)	5 (5)
Severe	1 (3)	0 (0)	1 (1)
No (%) with E:A ratio <1.0	20/27 (74)	45/70 (64)	65/97 (67)

Assessment of the overlap of obesity, respiratory disease, and cardiac abnormalities—Tables 4 and 5 show the overlap between abnormalities of body mass index (overweight, obesity, or extreme obesity), FEV_1 (less than 70% of predicted), cardiac structure (left ventricular hypertrophy), and evidence of coronary heart disease (clinical or electrocardiographic). Table 4 compares the overlap of obesity and angina with the presence of FEV_1 less than 70% of predicted and left ventricular hypertrophy. Table 5 differs from table 4 in using abnormal body mass index (overweight, obese, and extremely obese) in place of obesity and coronary heart disease (history of angina (past or present), myocardial infarction, coronary artery bypass graft, or ECG changes consistent with coronary heart disease) in place of angina. Of 109 patients for whom complete data were available for all parameters, 106 were included. Only nine (9%; 4% to 14%) patients were of normal weight and had a FEV_1 greater than 70% predicted. Of these, two had clinical or electrocardiographic evidence of coronary heart disease. Consequently, only seven patients (7%; 2% to 12%) had no evidence of abnormalities of body mass index, respiratory disease, or coronary heart disease.

Table 4 Overlap between FEV_1, left ventricular hypertrophy, angina, and obesity in 106/109 patients with complete data

	FEV_1 ⩽70%		FEV_1 ⩾70%	
	Obese	Not obese	Obese	Not obese
Angina				
Left ventricular hypertrophy:				
Yes	1	2	1	1
No	6	13	1	4
No angina				
Left ventricular hypertrophy:				
Yes	4	11	4	3
No	11	22	7	15

Table 5 Overlap between FEV_1, left ventricular hypertrophy, coronary heart disease, and abnormal body mass index (BMI ⩾25) in 106/109 patients with complete data

	FEV_1 ⩽70%		FEV_1 ⩾70%	
	Abnormal BMI	Normal BMI	Abnormal BMI	Normal BMI
Coronary heart disease*				
Left ventricular hypertrophy:				
Yes	2	1	6	0
No	12	6	10	2
No coronary heart disease				
Left ventricular hypertrophy:				
Yes	6	2	8	2
No	17	7	21	4

*History of angina (past or present), myocardial infarction or CABG, or ECG changes typical of coronary heart disease.

Discussion

Along with others, we have found that many patients presenting with heart failure have preserved left ventricular systolic function.[1–3 10–15] While these patients may have "diastolic dysfunction" it is also possible that there are other explanations for their symptoms. The problem about making a diagnosis of "diastolic" heart failure non-invasively is that there is no agreement on how this should be done, and different criteria for diastolic dysfunction give enormously differing prevalences.[2 16] One of the most commonly used criterion, an E:A ratio of <1, showed that most of our patients with heart failure and preserved left ventricular systolic function could be said to have diastolic dysfunction. Rather than examine the vexed issue of how one defines diastolic dysfunction with echocardiography we have examined an alternative—that is, could there be another explanation for these patients' symptoms?

The most obvious alternative diagnoses are obesity, respiratory disease, and myocardial ischaemia. We found that the first two of these were common. A third of patients were either obese or very obese. Half of the patients had a considerable reduction in FEV_1 (to 70% or less) and 89% had a peak expiratory flow rate less than or equal to 70% of normal. Remarkably, only nine patients were of normal weight and had FEV_1 greater than 70% predicted. Though we sought to identify myocardial ischaemia only by recording a history of angina, 31 patients admitted to this symptom, 12 had a history of myocardial infarction, and seven had undergone coronary artery bypass surgery. Furthermore, at least 20 patients had electrocardiographic changes consistent with myocardial ischaemia or infarction. If we had undertaken exercise stress electrocardiography even more patients with myocardial

What is already known on this topic

Patients with suspected heart failure but preserved left ventricular systolic function are commonly said to have "diastolic heart failure"

What this study adds

Most of these patients have an alternative explanation for their symptoms, such as obesity, pulmonary disease, and myocardial ischaemia

Complete investigation of these patients requires more than an echocardiogram

Improved patient care should result from recognition of the true cause of a patient's symptoms as there are appropriate management strategies for these alternative diagnoses; this is preferable to ascribing symptoms to diastolic heart failure for which there is no evidence based treatment

ischaemia would probably have been identified. Of the nine patients with normal weight and FEV_1 greater than 70%, a further two had clinical or electrocardiographic evidence of coronary heart disease. In other words, only seven patients in this study with a diagnosis of heart failure but preserved left ventricular systolic function lacked a recognised explanation for their symptoms—that is, a diagnosis of diastolic heart failure was inappropriate. The important message for clinicians is that an echocardiogram suggesting diastolic dysfunction on the basis of an abnormal E:A ratio is not diagnostic and represents insufficient investigation.

We clearly need to improve differentiation of breathlessness due to isolated diastolic dysfunction from that with other causes. It would seem that a rigorous search for non-cardiac causes of breathlessness must be pursued, with pulmonary function testing, calculation of body mass index, resting and exercise electrocardiography, and, probably, chest radiography. Even if these other causes are excluded it may still be difficult to be sure that a patient's breathlessness is definitely cardiac in origin. One possibility is that measurement of plasma natriuretic peptide concentrations might further refine the diagnostic process. It might be expected that increased left ventricular mass, wall stress, or filling pressures would increase secretion of atrial or brain natriuretic peptides. This possibility needs to be tested further.

Of course, it is also possible that patients may have more than one cause of their dyspnoea. This real diagnostic dilemma reinforces the need for better means of determining whether or not there really is a non-systolic cardiac contribution in such cases. We believe that improved patient care should result from recognition of the true cause of a patient's breathlessness as appropriate management of the correct alternative diagnoses can improve presenting symptoms.

In summary, we have shown that in most patients with a diagnosis of heart failure but preserved left ventricular systolic function there are alternative explanations for their symptoms—for example, obesity, lung disease, and myocardial ischaemia. For that reason the diagnosis of diastolic heart failure is probably unnecessary, even though a high proportion of these patients will have echocardiographic evidence of diastolic dysfunction.[16]

Contributors: JJVM had the original idea for the study. JJVM, APD, and LC were involved in the study design. LC collected the data and performed the echocardiograms. MCP and JJVM analysed and interpreted the data. The paper was written jointly by JJVM and MCP. JJVM is the study guarantor.

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Science commentary

Science commentary: Echocardiography

Echocardiography uses very high frequency sound waves (ultrasound), which are inaudible to human ears. Sound waves are forms of pressure wave; they can travel through any medium. In echocardiography these waves are generated and detected by means of a piezoelectric crystal contained within a transducer.[1] A simple mechanical scanner comprises a single crystal that is oscillated by a small motor, while the newer ("phased array") systems comprise multiple crystal elements that are pulsed in a rapid and controlled sequence. In cardiac imaging the ultrasound beam arises from a small source and is directed, in a pyramidal sector, across the heart.

In the body, echoes are generated when ultrasound waves reach tissue, where the acoustic properties (den-

sities) change, and are then reflected back. This usually happens at organ boundaries, tissue interfaces, and cellular boundaries. The transducer detects echoes before being converted into electrical signals that are represented on an oscilloscopic display. Dense structures, such as the pericardium and calcified valves, appear bright (white), whereas blood filled cavities (atria, ventricles) are almost echo free (black). Multiple reflections occur when the ultrasound beam reaches an object with greatly differing transmission characteristics from those of the surrounding tissues. For example, prosthetic valves may produce multiple parallel echoes and associated "ghost" images.

Ultrasound waves are sinusoidal fluctuations in pressure; the size of each pressure wave is termed the amplitude, the distance between the two waves is the wavelength, and the number of waves per second is the frequency. For medical applications, the frequency of ultrasound tends to lie within the range of 2-20 million cycles per second (MHz), and echocardiography sits at the lower end of this spectrum (2.5-10 MHz). During transmission through tissues, energy is lost due to absorption and scattering, and thus the reflected energy of the echo is invariably lower than the original ultrasound. Higher frequency waves may provide better image quality, although tissue penetration is not as good.

Complete echocardiographic examination entails the use of three different imaging modes: two dimensional, M mode (a graph of motion against time), and Doppler studies. Two dimensional imaging enables the operator to make a subjective assessment of the size of the cardiac chamber and ventricular function and allows detailed assessment (morphology/mobility) of valvar structures. M mode is useful for making accurate measures of chamber size and wall thickness at specific points in the cardiac cycle. Doppler techniques are divided into three types: pulsed wave, continuous wave, and colour flow. Pulsed wave and continuous wave Dopplers are used to detect the direction and velocity of blood flow across heart valves (this allows calculation of valve gradients). Colour flow imaging provides a pictorial coloured representation of blood flow in the heart and across valves. This is a useful screening tool as the direction of blood flow and the presence of turbulence can be identified. It is particularly useful for detecting evidence of regurgitation across valves or abnormal patterns of blood flow (for example, in a ventricular septal defect).

Abi Berger *science editor, BMJ*

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Not such an angel?

Easter Monday was a relaxing day, working in the garden and greenhouse after a weekend on duty. All went well until late afternoon when I stopped for a cup of tea. I suddenly realised that my vision was blurred. I closed one eye, then the other, and it quickly became clear that my left eye was at fault. Calmly I went in and looked in the mirror—only to find that my left pupil was grossly dilated, the right one being normal and reacting. Then I became less calm. Thoughts of vitreous haemorrhage, Homer's syndrome, retinal vein thrombosis, etc, went through my mind. Nothing for it but to contact our out of hours cooperative, where one of my partners happened to be on duty at a surgery session. At least this wasn't the middle of the night.

Having checked my fundus, visual fields, blood pressure, etc, and found all normal, he was not sure what was wrong. Reassured by the normal fundus I began to wonder if I had been in contact with something in the garden. The next step was down to the district general hospital, some 23 miles away, to see the ophthalmic senior house officer. He checked the eye thoroughly with the slit lamp, although by now the vision was beginning to improve, and the pupil was showing some reaction to light. He contacted the consultant, who came and again examined the eye. He was not sure what was going on but proposed a review in the eye clinic if it did not improve. The suggestion that some chemical from the garden was responsible was regarded as a possibility, although he was more concerned that mydriatics from my medical bag may have somehow found their way into my eye.

Next day in surgery, my partners were relieved to find that my vision was better, so I was able to drive and work. The left pupil was slightly smaller, but remained larger than the right one.

After morning surgery, while we were all busy with prescriptions and telephone queries, our registrar used the internet to search for "unilateral dilated pupil." We were both surprised at how many entries there were, but on scanning through them I noticed mention of "angel trumpet"—a paper in German without an abstract[1]. Knowing that I had two of these plants in the greenhouse and may have brushed against them, we looked for similar papers. We found a further paper on angel trumpet referring to its hallucinogenic properties and the danger of ingesting any part of it.[2] Apparently a group of youths died in the summer of 1997 after ingesting flowers from the plant. Analysis showed scopolamine among the poisons identified, almost certainly explaining my experience. This paper was also in German, but fortunately had an English abstract. Further searches revealed a paper confirming the presence of scopolamine in this plant.[3] Atropine was looked for, but not found.

By that evening the pupil was back to normal, but I shall be treating my datura (angel trumpet) with great care in future. These plants are widely available. Perhaps they should carry a health warning.

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We welcome articles of up to 600 words on topics such as *A memorable patient, A paper that changed my practice, My most unfortunate mistake*, or any other piece conveying instruction, pathos, or humour. If possible the article should be supplied on a disk. Permission is needed from the patient or a relative if an identifiable patient is referred to. We also welcome contributions for "Endpieces," consisting of quotations of up to 80 words (but most are considerably shorter) from any source, ancient or modern, which have appealed to the reader.