Cross sectional study estimating prevalence of heart failure and left ventricular systolic dysfunction in community patients at risk

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Abstract
Objective—To examine a general practice population to measure the prevalence of signs and symptoms of heart failure (SSHF) and left ventricular systolic dysfunction (LVSD).

Design—Cross sectional screening study in three general practices followed by echocardiography.

Setting and patients—All patients ≥ 50 years in two general practices and ≥ 40 years in one general practice were screened by case record reviews and questionnaires (n = 2158), to identify subjects with some evidence of heart disease. Among these, subjects were sought who had SSHF (n = 115). Of 357 subjects with evidence of heart disease, 252 were eligible for examination, and 126 underwent further cardiological assessment, including 43 with SSHF.

Main outcome measures—Prevalence of SSHF as defined by a modified Boston index, LVSD defined as an indirectly measured left ventricular ejection fraction ≤ 0.45, and numbers of subjects needing an echocardiogram to detect one case with LVSD.

Results—SSHF afflicted 0.5% of quadragenarians and rose to 11.7% of octogenarians. Two thirds were handled in primary care only. At ≥ 50 years of age 6.4% had SSHF, 2.9% had LVSD, and 1.9% (95% confidence interval 1.3% to 2.5%) had both. To detect one case with LVSD in primary care, 14 patients with evidence of heart disease without SSHF and 5.5 patients with SSHF had to be examined.

Conclusion—SSHF is extremely prevalent in the community, especially in primary care, but more than two thirds do not have LVSD. The number of subjects with some evidence of heart disease needing an echocardiogram to detect one case of LVSD is 14.

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Keywords: heart failure; left ventricular systolic dysfunction

Several mortality trials have provided evidence for the use of angiotensin converting enzyme (ACE) inhibitor, β blocker, and spironolactone treatment in patients with congestive heart failure caused by left ventricular systolic dysfunction (LVSD). Although the data supporting the use of these drugs for this indication are compelling, the patients studied in these trials were highly selected and recruited from hospital populations. Serious questions have to be asked regarding whether these results can be extrapolated to primary care.

Epidemiological data suggest that heart failure occurs in about 2% of the population. Prevalence rates of over 10% have been reported in the very old. However, studies on the validity of the diagnosis in primary care suggest that only 20–50% of these have definite cardiac malfunction including the important subgroup with LVSD. One might therefore hypothesise that the number of patients who are eligible for the mortality trial treatment is two to three times less than epidemiological data suggest. At present, we have no data on the number of patients who are actually eligible for treatment in primary care.

Recently two large population based studies used echocardiography to determine the prevalence of LVSD in the community. The prevalence was 2.9% in Glasgow and 3.7% in Rotterdam, but comparisons are hampered by use of different methods and criteria for assessing LVSD. For instance, the Glasgow prevalence rose to 7.7% if a left ventricular ejection fraction (LVEF) limit of 0.35 was used instead of 0.30. About 1.5% in the Glasgow study had symptomatic LVSD. In clinical practice, however, only subjects who present with cardiopulmonary complaints can be examined for LVSD, usually by an echocardiogram or radionuclide/contrast ventriculogram.

Our strategy for detecting LVSD would therefore be to carry out echocardiography in a high risk population—for example, patients with signs and symptoms suggestive of heart failure (SSHF) or of heart disease. The outcome of using this strategy is not known although it is essential for estimating the likely impact of the trial results in clinical practice, the resources required for their implementation, and whether screening for LVSD would be appropriate. The number of patients eligible for the trial treatment can be estimated by investigating such a high risk population, which was the aim of the present study.

We undertook a screening of a general practice population to identify subjects with SSHF or heart disease. They were further evaluated by cardiac assessment including echocardiography. On the basis of this information, we calculated the number of subjects in the community with a suspected diagnosis of heart failure and with LVSD.
Prevalence of heart failure and LV systolic dysfunction

Table 1: Principal cardiac diagnoses according to severity in patients over 50 years of age

<table>
<thead>
<tr>
<th>ICD code</th>
<th>Short description</th>
<th>Non-responder (n=230) (%)</th>
<th>Responder (n=1367) (%)</th>
<th>Nursing home (n=158) (%)</th>
<th>Total (n=1755) (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I25.9</td>
<td>Previous MI and angina</td>
<td>2 (0.9)</td>
<td>32 (3.2)</td>
<td>0 (0.0)</td>
<td>34 (1.9)</td>
</tr>
<tr>
<td>I21.9</td>
<td>Previous MI</td>
<td>1 (0.4)</td>
<td>23 (1.7)</td>
<td>6 (3.8)</td>
<td>30 (1.7)</td>
</tr>
<tr>
<td>I20.9</td>
<td>Angina pectoris</td>
<td>9 (3.9)</td>
<td>31 (2.3)</td>
<td>4 (2.5)</td>
<td>44 (2.5)</td>
</tr>
<tr>
<td>I48.9</td>
<td>Atrial fibrillation</td>
<td>5 (2.2)</td>
<td>21 (1.5)</td>
<td>5 (3.2)</td>
<td>31 (1.8)</td>
</tr>
<tr>
<td>I37.0</td>
<td>Pulmonary valve stenosis</td>
<td>0 (0.0)</td>
<td>1 (0.1)</td>
<td>0 (0.0)</td>
<td>1 (0.1)</td>
</tr>
<tr>
<td>I35.2</td>
<td>Aortic stenosis</td>
<td>0 (0.0)</td>
<td>4 (0.3)</td>
<td>1 (0.6)</td>
<td>5 (0.3)</td>
</tr>
<tr>
<td>I34.0</td>
<td>Mitral insufficiency</td>
<td>0 (0.0)</td>
<td>3 (0.2)</td>
<td>0 (0.0)</td>
<td>2 (0.1)</td>
</tr>
<tr>
<td>I11.9</td>
<td>Hypertensive heart disease</td>
<td>4 (1.7)</td>
<td>79 (5.8)</td>
<td>1 (0.6)</td>
<td>84 (4.8)</td>
</tr>
<tr>
<td>I49.8</td>
<td>Pacemaker</td>
<td>0 (0.0)</td>
<td>9 (0.7)</td>
<td>1 (0.6)</td>
<td>10 (0.6)</td>
</tr>
<tr>
<td>I52.8</td>
<td>Secondary heart disease</td>
<td>2 (0.9)</td>
<td>15 (1.1)</td>
<td>2 (1.3)</td>
<td>19 (1.1)</td>
</tr>
<tr>
<td>I51.7</td>
<td>Cardiomegaly on x ray</td>
<td>1 (0.4)</td>
<td>5 (0.4)</td>
<td>2 (1.3)</td>
<td>8 (0.5)</td>
</tr>
<tr>
<td>I26.9</td>
<td>Pulmonary embolus</td>
<td>0 (0.0)</td>
<td>10 (0.8)</td>
<td>0 (0.0)</td>
<td>3 (0.2)</td>
</tr>
<tr>
<td>I27.9</td>
<td>Cor pulmonale</td>
<td>0 (0.0)</td>
<td>13 (1.0)</td>
<td>0 (0.0)</td>
<td>15 (0.9)</td>
</tr>
<tr>
<td>I47.9</td>
<td>Paroxysmal supraventricular tachycardia</td>
<td>0 (0.0)</td>
<td>8 (0.6)</td>
<td>1 (0.6)</td>
<td>10 (0.6)</td>
</tr>
<tr>
<td>I51.8</td>
<td>Unknown heart disease</td>
<td>9 (3.9)</td>
<td>34 (2.5)</td>
<td>15 (9.5)</td>
<td>58 (3.3)</td>
</tr>
<tr>
<td>Any sign of heart disease</td>
<td>36 (15.7)</td>
<td>280 (20.5)</td>
<td>38 (24.1)</td>
<td>354 (20.2)</td>
<td></td>
</tr>
<tr>
<td>No apparent heart disease</td>
<td>194 (84.3)</td>
<td>1087 (79.5)</td>
<td>120 (75.9)</td>
<td>1392 (79.3)</td>
<td></td>
</tr>
</tbody>
</table>

ICD, International classification of diseases, 10th revision; MI, myocardial infarction.

Methods

STUDY POPULATION

The study population was defined by asking the National Health Insurance Register for names and addresses of all subjects who were connected to three general practices in the Copenhagen municipality on three separate dates from 1993 to 1995. This procedure draws a near random sample independently of previous health contacts because more than 97% of the population have an appointment with their health insurance that connects them to a specific primary care physician with free and unrestricted access to consultations. Practices were chosen because the four primary care physicians working there allowed us to have unlimited access to case notes and because they had used the same computer program for case notes during the previous 2–4 years (Docbase, Roskilde, Denmark). The required sample size was estimated to be around 2200 to obtain a standard error of about 0.5% on the prevalence estimate.

We included all subjects ≥ 50 years of age from all three general practices (n = 1755) and all subjects aged 40–49 years from one of the practices (n = 403). In the Copenhagen municipality 30% of the total population (141 682 of 471 300) is ≥ 50 years of age. The age and sex composition in the study population was not different from that of the entire Copenhagen municipality. There was no difference between the studied districts and the entire Copenhagen municipality with respect to certain socioeconomic parameters (unemployment, social support, and average duration per hospital admission).

CROSS SECTIONAL SCREENING PROCEDURE

Screening was based on case record reviews and questionnaires. One research fellow in cardiology (OWN) reviewed all 2158 general practice case notes for cardiac history. Further information was obtained from hospital discharge letters and in some cases by requesting hospital records. A questionnaire sent to all subjects outside nursing homes (n = 2000) asked about dyspnoea, angina, and previous symptoms, treatment, or hospitalisation for heart trouble. Non-responders received a single reminder. We conducted telephone interviews with almost 500 subjects who had reported cardiac symptoms or disease in the questionnaire. The study was approved by the local ethical committee (appraisal No 01–086/95) and all examined patients gave informed consent.

DEFINITIONS IN CROSS SECTIONAL SURVEY

On the basis of the screening information anyone with past or present signs or symptoms of heart disease was allocated to one of the principal cardiac diagnoses in table 1, after discussion between OWN and a consultant cardiologist (JFH). Classifications used in this study were as follows.

Definite heart disease required objective evidence of heart disease—for example, echocardiography, catheterisation, stress test, cardiac scintigraphy, hospital admission for myocardial infarction, typical angina pectoris in the Rose questionnaire, and atrial fibrillation. Hypertensive heart disease was defined as a history of hypertension combined with a suspected type of heart disease (International classification of diseases, 10th revision (ICD-10) code i51.7, i26.9, i27.9, i47.9, i47.9, or i51.8).

Suspected heart disease appears from ICD-10 codes in table 1. Unknown heart disease (i51.8) was defined as unexplained or unknown reasons for atypical chest pain, SSHF (defined below), palpitations, abnormal ECG, hospital admission to a coronary care unit, and cardiovascular treatment. Secondary heart disease (i52.8) was unknown heart disease in a patient with concurrent chronic diseases.

No apparent heart disease was defined as no definite or suspected heart disease or heart failure.

Signs and symptoms of heart failure (SSHF) were noted if a physician in primary or secondary care had recorded such symptoms and treated or admitted the patient accordingly. The signs and symptoms, as inferred from the case records, should score ≥ 5 points in the modified version of the Boston index (table 2). The index had to be modified because case records did not systematically grade

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Table 2  Boston index for evaluating signs and symptoms of heart failure (SSHF)

<table>
<thead>
<tr>
<th>Category</th>
<th>Point</th>
</tr>
</thead>
<tbody>
<tr>
<td>I: History from questionnaire or case record</td>
<td></td>
</tr>
<tr>
<td>Rest dyspnoea, orthopnoea</td>
<td>4</td>
</tr>
<tr>
<td>Paroxysmal nocturnal dyspnoea</td>
<td>3</td>
</tr>
<tr>
<td>Dyspnoea on walking on level</td>
<td>2</td>
</tr>
<tr>
<td>Dyspnoea on climbing</td>
<td>1</td>
</tr>
<tr>
<td>II: Physical examination from case record</td>
<td></td>
</tr>
<tr>
<td>If heart rate &gt; 91 to 110 beats/min</td>
<td>1</td>
</tr>
<tr>
<td>If heart rate &gt; 110 beats/min</td>
<td>2</td>
</tr>
<tr>
<td>Neck vein distension, hepatomegaly or leg oedema</td>
<td>2</td>
</tr>
<tr>
<td>Lung crackles</td>
<td>2</td>
</tr>
<tr>
<td>Wheezing</td>
<td>3</td>
</tr>
<tr>
<td>Third heart sound</td>
<td>3</td>
</tr>
<tr>
<td>III: Chest radiography from case record</td>
<td></td>
</tr>
<tr>
<td>Alveolar pulmonary oedema</td>
<td>4</td>
</tr>
<tr>
<td>Interstitial pulmonary oedema</td>
<td>3</td>
</tr>
<tr>
<td>Bilateral pleural effusions</td>
<td>3</td>
</tr>
<tr>
<td>Cardiothoracic ratio &gt; 0.49</td>
<td>2</td>
</tr>
<tr>
<td>Upper zone flow redistribution</td>
<td>1</td>
</tr>
</tbody>
</table>

Patients with suspected or definite heart disease were evaluated by this index if they had been treated or referred for heart failure. The sum within each category contributed at most four points to the composite score. SSHF was a composite score of ≥ 5 points.

dyspnoea, neck vein distension, hepatomegaly, or leg oedema. The questionnaire alone could not classify SSHF but it gave supportive data on dyspnoea if these were lacking in the case records. 

Managed in primary care was defined as a diagnosis of and treatment for SSHF in primary care only.

Known LVSD was information of “moderately or severely impaired systolic function” or an LVEF below 0.4 in case notes or discharge letters.

ECHOCARDIOGRAPHY IN STUDY POPULATION

Of 357 patients with definite or suspected heart disease, 126 were included in the echocardiographic study phase. The purpose of the echocardiography was to identify LVSD in heart patients from primary care who were interested in attending, and able to attend, an ambulant cardiological examination. We excluded subjects in nursing homes, non-responders, and patients receiving inpatient or outpatient treatment for advanced heart failure. There was no upper age limit. Patients were offered free transportation and they spent at least four hours at the hospital to complete supplementary investigations. They were advised that all investigations were being performed as part of a research study.

Each patient underwent a comprehensive echocardiographic examination where M mode, cross sectional (two dimensional), and Doppler images including left ventricular diastolic filling parameters were recorded by one of the investigators (OWN). Recorded echocardiograms and videotapes were later analysed by OWN, who was blinded to other patient data, and a sample of echocardiograms was validated by an independent experienced operator (CTL). Definitions were as follows.

LVEF was indirectly estimated from fractional shortening as the median of five cardiac cycles or, in the 38% (48 of 126) where M mode measurements were unattainable, from a nine segment model for assessing wall motion score. This wall motion index score has a positive linear correlation with LVEF where a score of 2.0 approximates LVEF of 0.60 while a score of 1.0 approximates LVEF of 0.30.

LVEF was defined as a wall motion index score ≤ 1.5 or a fractional shortening < 0.26, approximately equal to LVEF ≤ 0.45. The standard deviation of a single LVEF estimate was equal for intraobserver variability and interobserver variability—that is, 0.05 ejection fraction units or a coefficient of variation of 8%.

Other cardiac abnormalities were left ventricular hypertrophy; LVEF between 0.45 and 0.55; valvar defects in the presence of left ventricular hypertrophy, dilated left atrium (> 45 mm), or dilated left ventricle (> 60 mm); and diastolic dysfunction, defined as two or more of three abnormal filling parameters (deceleration time > 0.224 s, ratio of early to late diastolic filling velocity < 0.5, isovolumetric relaxation time > 0.1 s).

DATA HANDLING AND STATISTICAL ANALYSIS

Screening divided the population into one of four distinct groups: group 1, no apparent heart disease; group 2, no SSHF but suspected or definite heart disease; group 3, SSHF in primary care only; and group 4, SSHF in secondary care. Prevalence data are presented as observed figures and calculated figures, as follows.

Observed figures in cross sectional survey refer to total of SSHF (total of groups 3 and 4 in part I of table 3). Observed figures in echocardiographic substudy refer to the percentage with LVSD of groups 2 and 3, and to the subgroup of group 4 that did not receive inpatient or outpatient treatment any more.

Calculated figures describe the prevalence of LVSD in primary and secondary care (part III of table 3). Here observed prevalence data from the cross sectional survey (part I, column f) were multiplied by percentages of LVSD from the echocardiographic substudy (part II, column h). The assumptions were that group 1 had zero prevalence of LVSD; that groups 2 and 3 had a percentage of LVSD as determined in echocardiographic substudy; and that those in group 4 with known LVSD had 100% of LVSD. The rest were ascribed a percentage of LVSD as group 4 in the echocardiographic substudy (part II, column h).

Ranked ICD-10 codes between the three groups in table 1 were compared by the Kruskal-Wallis test and mean LVEF between groups 2 to 4 (part II of table 3) were compared by analysis of variance. The computer package Statistica (Statsoft, Tulsa, Oklahoma, USA) was used for all calculations.

Results

CROSS SECTIONAL SURVEY

The study population comprised 2158 persons or 1.2% (1755 of 141 682) of Copenhagen municipality's population ≥ 50 years of age.

No data were available in 2.5% (55 of 2158) who were coded as healthy. The questionnaire response rate was 86% (1504 of 1757) for subjects ≤ 80 years of age, while 48% (191 of 401)
of subjects ≥ 80 years were either non-responders or nursing home patients.

Definite or suspected heart disease was identified in 354 (20% of 1755) subjects > 50 years of age (table 1) and in three subjects (0.7% of 403) from 40–49 years of age. No difference was documented in cardiac morbidity between non-responders and responders despite differences in data availability (Kruskal-Wallis, p = 0.08), but nursing home patients had a higher prevalence of suspected or unknown type of heart disease (Kruskal-Wallis, p = 0.03).

Part I in table 3 shows how the total prevalence of SSHF rose with age and was 6.4% in subjects ≥ 50 years of age. Of 357 patients with some evidence of heart disease 105 were excluded and 126 of 252 eligible patients underwent echocardiography. The 105 patients were excluded by priority: 38 lived in nursing homes, 36 did not respond to the questionnaire, 21 without definite heart disease were excluded for administrative reasons in an early study phase, and 10 patients had advanced heart failure. Of the invited 252 patients, 126 dropped out: 32 declined the invitation, 1 died, 37 were disabled by various medical and psychosocial conditions, and 56 patients, after various degrees of contact, did not show up. The age of the 126 examined patients ranged from 49–93 years (5th and 95th centiles, 53 and 83 years).

Compared with the 126 patients not examined, those examined were younger (mean 70 v 77 years of age, p < 0.001) and more often had myocardial infarction (30 v 10, p < 0.05) or angina pectoris (37 v 16, p < 0.05), but there was no significant difference (p > 0.05) in the prevalence of atrial fibrillation (11 v 19), hypertension (69 v 62), diabetes (12 v 23), chronic obstructive pulmonary disease (31 v 30), and sex (55 v 50 men). Echocardiography was performed in one patient < 50 and 19 patients > 80 years of age. Of those dropping out 47% were > 80 years of age (59 of 126).

In part II echocardiography showed LVSD in 15 subjects among the 126 examined from groups 2–4 (column g, table 3). Their median LVEF was 0.35 (range 0.22–0.45), and 10 patients had LVEF ≤ 0.40. Mean LVEF decreased steadily from group 2 to group 4 (column I, table 3, analysis of variance, p = 0.002) while the prevalence of LVSD increased (column h).

In patients without SSHF (group 2), the number of subjects needing an echocardiogram to detect one case of LVSD was 14 (83/6). Definite heart disease (n = 69), but not
Figure 1 illustrates the relations between LVSD, SSHF, and management in secondary and primary care. The figure combines data from parts I and III in table 3 (column f and h). LVSD occurred in less than one third of SSHF. One third of SSHF patients had been managed in secondary care. Half of these had LVSD. Asymptomatic LVSD among patients with evidence of heart disease afflicted 1% corresponding to 34% (1/2.9) of all with LVSD. Obviously our strategy did not include subjects who never presented a sign of heart disease. Supplementary analyses revealed that the ratio of LVSD to SSHF differed for older and younger subjects.

Figure 2 highlights the age dependent relation between SSHF and its subgroup with symptomatic LVSD. The figure is constructed by calculating LVSD in groups 3 and 4 while accounting for known LVSD at each age band. Figure 2 suggests that LVSD is more likely to be the cause of SSHF in younger than in older patients. The proportion with LVSD was 43% of SSHF patients < 70 years of age as opposed to 25% of SSHF patients ≥ 70 years of age (p < 0.0001).

Discussion

MAIN FINDINGS

This study examined subjects with some evidence of heart disease in primary care and found that, of subjects ≥ 50 years of age, 6.4% had SSHF; 2.9% had LVSD, and 1.9% had both SSHF and LVSD. The number of subjects with evidence of heart disease needing an echocardiogram to detect one case of LVSD was 5.5. This number was 5.5 in SSHF patients from primary care.

PREVIOUS WORK

Several prevalence studies of heart failure have been reported, but the present one is the first to use a clinically pragmatic screening procedure along with echocardiography. The present study’s SSHF prevalence was 30% higher than in the Framingham heart study, probably because of more liberal criteria. In contrast, the Gothenburg study showed a much higher prevalence rate in 67 year old men (of 13%) by 8-fold and primary care.

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Prevalence of heart failure and LV systolic dysfunction

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is substantially higher than that reported in this
4.4% prevalence at age 80–89 in Framingham
studies with one exception. The calculated
70–79 and 80–89 years of age, respectively.4 A
prevalence of symptomatic LVSD was 2.4%. The
Framingham study reported a 4.9% and 9.1%
prevalence of symptomatic heart failure at
70–79 and 80–89 years of age, respectively.1 A
subsequent study reported that 49% of Fram-
ingham’s patients with heart failure (mean age
73) had LVSD.27 This makes for a 2.4% and
4.4% prevalence of symptomatic LVSD at
70–79 and 80–89 years of age, respectively.
Figure 2 is therefore in concert with previous
studies with one exception. The calculated
4.4% prevalence at age 80–89 in Framingham
is substantially higher than that reported in this
and other studies. The difference may reflect
the prospective design of the Framingham
study, which ensured detection of elderly
patients with a very short survival who cannot
be detected in a cross sectional design with
a retrospective case identification. An alternative
explanation is that patients > 80 years old with
congestive heart failure have less than the mean
49% LVSD. Wheelton and colleagues6 con-
cluded, from a general practice study, that the
population prevalence of combined sympto-
matic heart failure and LVSD was 0.84%. A
comparable figure of 0.6% (0.3 × 1.9%) can be
derived from the present study as 30% of the
population was aged ≥ 50 years and by assum-
ing zero prevalence in subjects < 50 years.

The present study’s prevalence estimates for
total (symptomatic and asymptomatic) LVSD
is the same as that of Morgan and colleagues11
for comparable ages and LVEF values.11 Our
total 2.9% prevalence relates to 2.9% in the
Glasgow and 3.7% in Rotterdam studies.11 12
Comparison is, however, hampered by differ-
ences in the methods and limits used for
assessing LVSD between studies. The Glasgow
study used Simpson’s rule with biplane echo-
cardiography and the Rotterdam study used M
mode echocardiograms and a fractional short-
ening ≤ 0.25 for LVSD. Unlike epidemiologi-
cal surveys, the present study ignored further
assessment of subjects without apparent heart
disease. These methodological aspects also
affect the percentages with asymptomatic
LVSD, which was 34% in the present study,
83% (50 of 60) in the Rotterdam study, and
48% in the Glasgow study using an LVEF limit
of 0.30 but 77% when a limit of 0.35 was used.

STUDY STRENGTHS AND LIMITATIONS

We are confident about the validity of the present study because some of the calculated
figures reproduce data from other studies, such
as the frequency of LVSD in hospitalised heart
failure patients,28 in general practice,1 10 and in a
high risk group similar to our group.29 Only
a few patients with significant symptoms of
heart failure could have escaped our liberal cri-
teria and careful screening procedure. We
aimed for correct classification by having a
research fellow in cardiology scrutinise medical
records, interview subjects, and categorise all
diagnoses, a procedure that would have been
tedious in a larger population and without
electronic records. Heart failure diagnosis is
fraught with difficulties and the present classi-
fication, though careful in each case, was not
validated by an independent observer who
might have classified a few subjects differently.

LVSD prevalence was calculated in four
strata of the general practice clientele to correct
for differences in frequency of heart disease
and SSHF between the screened population
and the echocardiographic sample population.
The calculation assumed that all patients in a
group were comparable with the same risk of
LVSD. This was not proved, and the calcula-
tion may have underestimated the prevalence
of LVSD if the unexamined patients were
more diseased and had more LVSD. There was
no direct adjustment for sex, hypertension, or
diabetes. These are important risk factors for
myocardial infarction at the level of primary
prevention, but may be less important risk fac-
tors for LVSD1 once heart disease has become
clinically apparent. The echocardiographic
substudy showed that all but one case of LVSD
were found in patients with prior definite rather
than suspected heart disease.

We had intended to obtain an echocardiogram
for all with some evidence of heart disease but many, especially elderly, subjects
were unwilling to attend our hospital based
echocardiographic clinic. We did not systemati-
cally examine their reasons for declining but it
is our opinion that physical disability and psy-
chological factors such as anxiety in regard to
disease and hospitals were important. In
contrast Morgan and colleagues31 obtained
echocardiograms in 68% (817 of 1200) of a
random sample of patients aged 70–84 years
when they were examined in their homes.

WHAT IT MEANS FOR PRACTICE

These results are important when discussing
the diagnostic and therapeutic needs of mod-
ern heart failure management. It is still, after
the HOPE (heart outcomes prevention evalua-
tion) study,30 important to try to identify
subjects with LVSD and SSHF because they
have a greater morbidity and poorer prognosis,
and benefit particularly from the trial treat-
ment. About 1.9% of the population ≥ 50
years of age have symptomatic LVSD and
should match inclusion criteria as used in the
mortality trials. However, the number of
patients eligible for trial treatment may be
lower considering the contraindications for
treatment, that an ejection fraction limit of

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0.35–0.40 was used in the mortality trials as opposed to the 0.45 ejection fraction limit in the present study, and that access to echocardiography may be limited in clinical practice.

Thus, the need for echocardiographic service is huge if one is to provide individually guided treatment to those 6.4% of subjects with SSHF ≥ 50 years of age, especially as a service to primary care. The need for this service detecting LVSD in asymptomatic heart patients is even greater because 14 echocardiograms are required to detect one case. Screening high risk patients, especially those with definite heart disease, by simple measurements of ECG, natriuretic peptides, simplified echocardiography, or simple clinical decision rules holds promise, although studies that show the cost effectiveness of this approach are not yet available.

It is our experience that it may be troublesome to detect LVSD in subjects over 80 years of age because they are less willing to participate in a screening programme. Future studies should provide a better understanding of the pathophysiology of heart failure in the elderly and define useful treatment strategies for the many patients with symptoms ascribed to heart failure without LVSD. Meanwhile, studying survival in the various groups of this study would indicate where additional efforts are needed. The sixth to eight year follow up is taking place in 2001.

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Cross sectional study estimating prevalence of heart failure and left ventricular systolic dysfunction in community patients at risk
O W Nielsen, J Hilden, C T Larsen and J F Hansen

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