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# Review Article

# The epidemiology of heart failure

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## Introduction

Cardiovascular disease remains one of the most important causes of morbidity and mortality in Western society. Although much is known about the epidemiology of coronary heart disease, less is known about the epidemiology of heart failure despite its considerable economic impact on health services due to the cost of long-term drug treatment and frequent hospitalization. This burden may increase further as the prognosis of patients with heart failure is improved by medical and surgical interventions[1–3], as the proportion of the elderly within the population increases[4] and as survival from myocardial infarction improves with widespread and prompt administration of thrombolytic therapy[5,6]. In this review we will draw almost exclusively on data collected in the last 40 years. Prior to this time the classification and understanding of heart failure bears little resemblance to current knowledge and practice.

## Methods

The Medline Literature Database from January 1966 to June 1995 was searched using the medical subject headings heart failure (congestive), cardiomyopathy (congestive), epidemiology, diagnosis, prognosis, incidence, prevalence and mortality. The search was extended using lateral references, personal communications with investigators and presentations at recent conferences.

**Key Words:** Heart failure, epidemiology.

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## The Syndrome

There is no universally agreed definition of heart failure[7]. Heart failure is a syndrome which develops as a consequence of cardiac disease, and is recognised clinically by a constellation of symptoms and signs produced by complex circulatory and neurohormonal responses to cardiac dysfunction[8]. The syndrome may occur as the end-result of damage caused by a number of disease processes e.g. coronary artery disease, hypertension, valvular defects, alcohol misuse or viral infection. The presence and severity of heart failure can be assessed by questionnaires, physical and radiographic examination, and by measures of ventricular performance and exercise capacity. All these methods, however, have major limitations when used independently[9]. Most diagnostic difficulties arise in individual cases where the syndrome is mild[10].

Only a small number of studies have addressed the diagnostic value of symptoms and signs for the presence of heart failure[11–17]. Three were carried out in small, highly selected groups of patients awaiting heart transplantation[11,14,17], and another three studies took place in larger groups of patients who had been referred for evaluation of ejection fraction[12,13,16]. A study evaluating patients in whom general practitioners suspected heart failure was developing for the first time[15] reported that only 34% of such patients were found to have heart failure when assessed by a cardiologist before the initiation of treatment. Obesity, unrecognised myocardial ischaemia and chronic obstructive pulmonary disease often led to a false-positive diagnosis of heart failure in general practice. In general, breathlessness appears to be moderately sensitive but poorly specific for the presence of heart failure, whereas physical examination findings have a high specificity but low sensitivity (Table 1). The Dutch Transition project, a study of 22 general practices covering 40 574 patient years from 1985–1988[18,19] provides figures for the predictive value of shortness of

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*Table 1 Sensitivity, specificity, and predictive value of symptoms, signs and chest X-ray findings for the presence of heart failure (defined as ejection fraction <0·40) in series of 1306 patients with coronary artery disease undergoing cardiac catheterization[12])*

| | Sensitivity (%) | Specificity (%) | Positive predictive value (%) |
|---|---|---|---|
| Medical history | | | |
| Shortness of breath | 66 | 52 | 23 |
| Orthopnoea | 21 | 81 | 2 |
| Nocturnal dyspnoea | 33 | 76 | 26 |
| Oedema by history | 23 | 80 | 22 |
| Physical examination | | | |
| Tachycardia (>100 . min$^{-1}$) | 7 | 99 | 6 |
| Rales | 13 | 91 | 27 |
| Oedema on examination | 10 | 93 | 3 |
| Ventricular gallop sound (S3) | 31 | 95 | 61 |
| Neck-vein distension | 10 | 97 | 2 |
| Chest X-ray | | | |
| Cardiomegaly | 62 | 67 | 32 |

breath and dependent oedema on examination for detecting heart failure in general practice. The positive predictive value of breathlessness for heart failure in the presence of coronary artery disease was 25% for individuals aged 65–74 years and 44% for those over 74 years of age. The corresponding figures for oedema were only 11% and 27%.

Echo Doppler cardiography, nuclear studies or cardiac catheterization provide insight into the diastolic and systolic function of the heart but do not determine the presence or absence of heart failure: abnormal function may or may not be accompanied by the syndrome of heart failure[20]. Ventricular dynamics may also vary in one individual over time[21,22] and the effects of exercise or stress on systolic and diastolic function can be marked (especially in patients with coronary artery disease). Many of the indices used in clinical practice are markedly sensitive to changes in the loading of the ventricle, heart rate or drugs[23] and may change with age even in the absence of disease[24]. However, some abnormality of cardiac function will be demonstrable in all patients with heart failure[25], if not at rest, at least on stress.

There is increasing evidence that heart failure can be present in patients without valvular abnormality or systolic dysfunction, but as a result of isolated diastolic dysfunction preventing adequate ventricular filling at normal filling pressures[26–29]. This condition has been termed diastolic heart failure by several authors[27,29,30]. The distinction between systolic and diastolic heart failure is difficult to make using symptoms, signs and radiographic examination alone[27,29,30]. As non-invasive assessment of diastolic function is fraught with difficulties it is not surprising that little is known about the epidemiology of diastolic heart failure: it may be more frequent as age increases[31,32] and its natural history and treatment may differ from that of systolic heart failure[29,33,34].

Exercise testing is of limited value in the diagnosis of heart failure even when accompanied by measurement of oxygen consumption and anaerobic threshold[35], but does provide an objective assessment of the functional severity of the syndrome, and may have prognostic implications. The 6 minute walk test provides a useful alternative to treadmill exercise testing in the assessment of the severity and prognosis of heart failure[36,37]. The New York Heart Association Classification[38] is frequently used to classify the functional severity of heart failure. Although conceptually simple there is lack of inter-observer agreement in classification[39], and often a patient's own assessment of their functional state differs markedly from that of their doctor. Natriuretic peptides (and other measures of neurohormonal activation) are elevated in individuals with left ventricular dysfunction[40,41] but further work is necessary to clarify the role of such measures in the diagnosis of heart failure.

Because heart failure is a clinical diagnosis no one investigation can be considered the 'gold standard' for confirming the diagnosis. Scoring systems that combine several of the measures discussed above have been developed for use in population-based studies[42–44] and in cardiovascular drug research[45–47]. The various symptoms, signs and investigative findings that present in a patient with suspected heart failure are scored, and if the total score is greater than a predetermined number, the patient is classified as having heart failure (Table 2 and Appendix). None of the six scores for heart failure encompass a direct measure of cardiac function, e.g. by means of cardiac catheterization, nuclear ventriculography or Doppler echocardiography. Attempts have been made to validate the Framingham and Boston Scores against such measures as ejection fraction and left ventricular end-diastolic pressure at cardiac catheterization[12,13,47]. Individuals with depressed ejection fraction are not clearly differentiated from those with a normal

*Table 2 Scores for the classification of heart failure (for detailed explanation see appendix)*

| | Framingham[43] | Men Born 1913[44] | Gheorghiade[46] | Boston[47] | NHANES[42] | Walma[45] |
|---|---|---|---|---|---|---|
| History | | | | | | |
| Paroxysmal nocturnal dyspnoea | X | X | X | X | | X |
| Orthopnoea | X | | | X | | |
| Rest dyspnoea | | | | X | | |
| Dyspnoea on exertion | x | | X | X | X | X |
| Dyspnoea (WHO 1–4)[151] | | X | | | | |
| Night cough | x | | | | | |
| Myocardial infarction or angina pectoris[151] | | X | | | | |
| Weight loss | X/x* | | | | | |
| Swollen legs at end of day | | X | | | | |
| Physical examination | | | | | | |
| Neck-vein distension | X | | X | | X | |
| Increased jugular venous pressure | X** | | | X | | X |
| Rales | X | X | X | X | X | X |
| S3 gallop | X | | X | X | | X |
| Hepatojugular reflux | X | | | | | X |
| Hepatomegaly | x | | | X | X | X |
| Oedema | x | | X | X | X | X |
| Wheezing | | | | X | | |
| Circulation time >=25 s | X | | | | | |
| Electrocardiogram | | | | | | |
| Tachycardia | x*** | | X | X | X | X |
| Atrial fibrillation | | X | | | | |
| Chest X-ray | | | | | | |
| Cardiomegaly | X | | X¶ | X | | |
| Acute pulmonary oedema | X | | | | | |
| Pleural effusion | x | | X | X | X | |
| Interstitial oedema | | | X | X | X | |
| Pulmonary venous hypertension | | | X | | | |
| Alveolar changes | | | X | X | X | |
| Redistribution | | | | X | X | |
| Pulmonary function | | | | | | |
| Vital capacity | x**** | | | | | |

ejection fraction[13], with both sensitivity and specificity of the Framingham score to detect an ejection fraction $\leq 40\%$ being 0·63. The corresponding figures for the Boston score were 0·50 and 0·78. Both scores appear moderately useful at identifying individuals with high left ventricular end-diastolic pressure at rest[12,47] — representing, of course, the more severe grades of heart failure.

Echocardiography has revolutionized the non-invasive assessment of cardiac structure and function in cardiological practice[48,49]. Nevertheless, very few published studies have evaluated the use of echocardiography for the detection of heart failure in a non-hospitalised population[50,51]. In a group of 26 men and 44 women (aged 45 to 74 years) in whom a general practitioner diagnosed heart failure for the first time, a combination of several M-mode echocardiographic measurements (E-point septal separation, fractional shortening and peak lengthening rate) had a positive predictive value of 75% for detecting heart failure as determined by a cardiologist; if abnormalities in these three measurements were not present the probability of heart failure was only 7%. The potential of ultrasound to confirm cardiac abnormality has expanded greatly with the more recent introduction of two-dimensional, Doppler and colour flow imaging methods. The development of echo Doppler cardiography has made large scale epidemiological studies realistic, but it is important to recognise the necessity to record and interpret echocardiograms in a standardized and appropriate manner[52–54].

The lack of agreement on a definition of heart failure, as well as the lack of a gold standard to confirm the diagnosis have both resulted in considerable heterogeneity in the diagnosis of heart failure in clinical trials[55] and epidemiological studies. The clinical and epidemiological approaches are distinct: in the latter individual misclassification can be accepted provided disease estimates for the population remain precise, but in clinical practice every effort is made to correctly diagnose the individual. The epidemiological approach is valid to estimate incidence or prevalence, but becomes problematic when describing the patients clinical characteristics and subsequent clinical course and prognosis. The inclusion of patients who do not have heart failure in this description will introduce a number of biases in relation to aetiology, clinical characteristics and investigation results, and the prognosis will appear more benign than is actually the case. For this reason population-based studies of heart failure must employ

the same rigorous diagnostic criteria as used in clinical practice if the purpose of such studies is more than to estimate frequency. There is a widely recognised need for consistent criteria for the diagnosis and assessment of heart failure in clinical practice, epidemiological research and clinical trials to enable comparisons to be made between different studies. The Task Force on Heart Failure of the European Society of Cardiology recently published guidelines for the diagnosis of heart failure[25]. To satisfy the Task Force's definition of heart failure both symptoms and objective evidence of cardiac dysfunction need to be present. Reversibility of symptoms on appropriate treatment was considered desirable. Echocardiography was recommended as the most practical tool to demonstrate cardiac dysfunction.

## Availability of epidemiological data on heart failure

Epidemiological information on heart failure can be obtained from general practice and community surveys, drug prescribing surveys, national death statistics and hospital morbidity and mortality registrations. Information from these sources cannot be readily compared because of differences in ascertainment and classification of heart failure. For studies in general practice the International Classification of Health Problems in Primary Care (ICHPPC) is commonly used[56]. The World Health Organisation (WHO) International Classification of Diseases codes and guidelines on their application are usually employed for mortality (death certification) and hospital discharge data[57], whereas many population-based studies use classification systems that have been specifically designed for those studies[42–44]. Reliability and comparability of mortality and morbidity statistics is significantly limited by variation in data collection and coding and by differences in the approach to diagnosis of cardiovascular diseases within and between countries, and over time[58]. The MONICA investigators recently reported a lack of evidence to support the diagnosis of coronary heart disease in one in four deaths attributed to coronary heart disease[59] and a study of autopsies in a Dutch nursing home revealed that often no evidence is present for a diagnosis of heart failure as mentioned on the death certificate[60]. The reliability of a diagnosis of heart failure in hospital records and hospital discharge forms may also be poor[61].

## Prevalence

The methodology and results of prevalence studies using data collection from medical records supplemented by direct questioning and/or examination of individuals within the general population, drug prescription data analysis, and general practitioner monitoring are summarized in Tables 3, 4 and 5. Prevalence estimates vary widely which reflect the differences in methodology and timing rather than true differences between populations, and the crude prevalence (unadjusted for age) ranges from 3 to 20 individuals per 1000, with a prevalence of 30 to 130 individuals per 1000 for those aged over 65 years.

Analysis of drug prescription data relies upon use of diuretics, with or without abstracting data from the relevant medical records to estimate the prevalence of heart failure[62,63]. This method assumes that all individuals with heart failure have been prescribed a diuretic: this is probably true for those with more than mild degrees of fluid retention, but is likely to miss individuals with mild heart failure, and may therefore underestimate the true prevalence. Overestimation is also possible, as many individuals prescribed a diuretic do not have heart failure.

In prevalence studies relying upon data obtained from general practitioners[18,64–68] no attempt has been made to confirm the diagnosis of heart failure. The validity of the diagnosis of heart failure in general practice is known to be relatively poor[15,48], and this may therefore reduce the value of such studies.

## Incidence

Incidence can be determined by two approaches: re-examining individuals within a cohort at intervals to identify those who have developed heart failure; or by a population-based surveillance system in which subjects developing heart failure for the first time are identified. The Framingham Heart Study and the Study of Men Born in 1913 in Sweden are good examples of the first approach[69–71]. The Finnish Study[72], U.K. and Dutch general practice studies[18,67,68], the Rochester study[73] and the United States Two Counties Study[64] are examples of the second approach which has the advantage that incident cases of heart failure presenting to the health care system (either in primary or higher levels of care) are identified prospectively and may therefore be fully characterized at the time of diagnosis rather than retrospectively. Table 6 summarizes the results of these studies. The crude incidence (unadjusted for age) in the general population ranges from 1·0 to 5·0 cases per 1000 population per annum, with a steep increase with advancing age: the incidence rate for those aged over 75 years is reported to be as high as 40 cases per 1000 population per annum in some studies[18,68].

## Aetiology

Heart failure is the common end-result of many different disease processes that impair cardiac function[74]. Coronary heart disease and hypertension (either singly or together) account for the vast majority of cases of heart failure within the developed world. Valvular heart disease is a much less frequent underlying aetiology of heart failure than previously[75]. Rheumatic valvular

***Table 3 Population based studies of congestive heart failure***

| Study | Population |
|---|---|
| United States | |
| Tecumsen, Michigan 1959–60[152] | Prevalence study of a complete community (90% participation rate). 8641 persons (49% men), heart failure by clinical criteria. 64 cases. |
| Evans County, Georgia 1960–62[153] | Prevalence study of population of Evans County (92% participation rate), 1840 persons (48% men) 45–75 years, interview and physical examination Heart failure by clinical criteria. 39 cases. |
| Vermont/North Carolina, 1962–64[64] | Physician surveillance of own practice in two counties, 22 758 (50% men) and 13 820 (48% men), 183 and 82 prevalent cases, and 19 and 7 incident cases, respectively. |
| NHANES-I, 1971–75[42]* | Prevalence study of sample of non-institutionalized persons throughout the U.S. Estimates based on self report by 14 407 persons (25–74 years, 158 cases) and on clinical examination of a subsample of 6913 persons, 138 cases. |
| Rochester, Minnesota 1981–82[73] | Review of medical records of Rochester residents <75 years using clinical criteria, 46 incident cases in 1981, 113 prevalent cases on 1 January 1982. |
| Rochester, Minnesota 1986[154] | Stratified random sample of Rochester resident 35 years or older, 2122 persons (48% men). Heart failure by clinical criteria using questionnaire and review of medical records, 41 prevalent cases. |
| Framingham, Massachusetts 1948–88[92] | 9405 participants (47% men), biannual examination and interview. 652 incident cases. |
| CHS, 1989–90[155]** | Prevalence study of a cohort of 5201 persons 65 years and older. Presence of definite heart failure based on interview and information from additional sources (physician/medication use/hospital discharge diagnosis). 104 definite cases, 35 cases of possible heart failure (self report not confirmed by information from additional sources) |
| Europe | |
| Sheffield, U.K. 1950[156] | Prevalence study by clinical examination of a random sample of 476 retired men and women (40% men) living at home. 14 cases. |
| Göteborg, Sweden 1963–80[69] | 855 men (88% of sample) born in 1913. An additional sample was drawn in 1970. Heart failure based on findings from medical interview and examination. 18 prevalent cases at age 50 years, 84 cases at age 67 years. Incidence estimated from increase in prevalence with age of sample. |
| Göteborg, Sweden 1971–77[157] | 973 persons (46% men, 70 years) representing 85% of a sample of 70 year olds, examined in 1971/72 and 5 years later. Heart failure by clinical criteria: interview and physical examination of 743 persons attending both examinations (19 and 28 prevalent cases at age 70 and 75 years, respectively). |
| Eastern Finland, 1986–88[72] | Surveillance of 11 035 persons 45–74 years in rural communities. Examination of persons suspected of heart failure. Heart failure by Framingham and Boston criteria, 51 incident cases. |
| London, U.K. 1988[62] | Prevalence study by review of medical records in 3 general practices (30 204 patients), heart failure by clinical criteria in patients who were using diuretics. 117 cases. |
| Transition project, The Netherlands 1985–88[18] | Surveillance of 22 general practices (40 796 patients, 47·3% men), heart failure classified by general practitioner on clinical criteria. 459 prevalent cases, 245 incident cases. |
| Nijmegen, The Netherlands 1987–91[68] | Surveillance of 22 general practices (60 691 patient years), heart failure classified by general practitioner on clinical criteria. 701 prevalent and 197 incident cases. |
| Castel Franco, Italy 1992/94[158,159] | Prevalence study by self report of majority of population in a community combined with verification of prescribed medication. 6529 persons aged 20–64 years (130 cases); 2254 persons aged over 64 years (187 cases). |
| Denmark, 1994[160] | Prevalence study by questionnaire survey of patients >40 years in one general practice (n=963). Heart failure based on history of heart disease combined with breathlessness on exertion and use of diuretics, digoxin or angiotensin converting enzyme inhibitors. 25 severe and 37 mild prevalent cases. |
| Nottinghamshire, U.K. 1994[63] | Prevalence estimates based on prescription data from one county in U.K. |
| Gen Practice, U.K. 1991/92[67] | Prevalence and incidence study by general practitioner monitoring of a 1% sample of population of England and Wales registered with 60 volunteer practices throughout the country. 4166 prevalent cases, 1076 incident cases. |

*NHANES-I. First National Health and Nutrition Examination.
**CHS, Cardiovascular Health Study, carried out in four countries (in North Carolina, Maryland, California and Pennsylvania).

heart disease and nutritional cardiac disease are much more common in the developing world[76,77] and consequently the prevalence and incidence of heart failure in younger age groups is higher. In a substantial minority of cases the aetiology is unknown, and the term 'cardiomyopathy' is applied[78,79] qualified only by a descriptive physiological term such as dilated, hypertrophic or restrictive. The relative frequencies of each pathology vary from one series to another. Often the criteria used to determine aetiology are poorly defined and autopsy and hospital-based data tend to be rather selected, and produce a different league table from that based on community studies[75,80,81] (Table 7). Patients with a history of hypertension may be normotensive at the time of presentation with heart failure ('burnt out' hypertension) and may therefore be misclassified in terms of aetiology unless blood pressure levels were recorded prior to the development of heart failure.

The Framingham Heart Study reported hypertension as the sole or contributory cause of heart failure

***Table 4 Prevalence of heart failure, U.S. studies***

| Study | Prevalence (per 1000 population) | | |
|---|---|---|---|
| | Men | Women | Total |
| United States | | | |
| Tecumseh[152] | | | |
| All ages | 5·9 (3·6–8·2) | 8·9 (6·1–11·6) | 7·4 (5·6–9·2) |
| Evans County[153] | | | |
| 45–74 years | 24·8 (14·6–35 0) | 17·8 (9·5–26·1) | 21·2 (14·6–27·8) |
| Vermont[64] | | | |
| All ages | 8·6 (7 0–10·2) | 11·7 (9·7–13·7) | 10 0 (8·8–11·2) |
| North Carolina[64] | | | |
| All ages | 8·4 (7·7–9·1) | 9·0 (7·9–10 1) | 8·8 (7·3–10·3) |
| NHANES-I[42] | | | |
| self report | | | |
| 25–74 | 11 | 10 | 11 (9·3–12·7) |
| Clinical score | | | |
| 25–54 | 8 | 13 | 11 |
| 55–64 | 45 | 30 | 37 |
| 65–74 | 49 | 43 | 45 |
| 25–74 | 19 | 20 | 20 (17·7–22·3) |
| Rochester 1981/82[73] | | | |
| 45–49 | 1 | 1 | |
| 50–54 | 1 | 2 | |
| 55–59 | 7 | 3 | |
| 60–64 | 12 | 7 | |
| 65–69 | 26 | 11 | |
| 70–74 | 28 | 27 | |
| 0–74 | 3·3# | 2·1# | 2·7# |
| Rochester 1986[154] | | | |
| 35–54 | 0 | 2 | 1 |
| 55–64 | 5 | 5 | 5 |
| 65–74 | 23 | 0 | 12 |
| 75+ | 69 | 80 | 76 |
| All (35+) | 17·6 (16·8–18·4) | 20·9 (20·1–21·7) | 19·3 (18·7—19·9) |
| Framingham*[92] | | | |
| 50–59 | 8 | 8 | |
| 80–89 | 68 | 79 | |
| >=45 | 24 | 25 | |
| all ages | 7·4 | 7·7 | |
| CHS[155] | | | |
| definite | | | |
| 65–69 | 22 | 12 | |
| 70–74 | 19 | 15 | |
| 75–79 | 32 | 24 | |
| 80–84 | 32 | 25 | |
| 85+ | 29 | 22 | |
| 65+ | 24·4 (18·0–30·8) | 16·6 (12·0–21·2) | 20·0 (16·2–23·8) |
| possible | | | |
| 65+ | 6·2 (3·2–9·2) | 5·1 (2·6–7·6) | 5·6 (3·6–7·6) |

*Framingham data pertain to the 1980s.
#Age adjusted to relevant natural population.
( )95% confidence interval, calculated as 95% CI=(p) ± 1·96 × $\sqrt{((p(1-p))/n)}$ (p=prevalence, n=number of persons in denominator).

in over 70% of cases[82]. Community-based studies other than Framingham have not found hypertension to be such a common cause of heart failure[51,62,72]. This may be partly due to the entirely non-invasive assessment of the aetiology of heart failure in the Framingham Study: many individuals with heart failure due to coronary artery disease (but without a clinical history of myocardial infarction or angina pectoris) may have been misclassified in terms of aetiology because of the frequent coexistence of hypertension with the coronary artery disease. In any case, the importance of hypertension and valvular heart disease as causes of heart failure has declined steadily in the Framingham cohort since the 1950s, concomitant with an increase in the importance of coronary artery disease and diabetes mellitus[75]. There is limited evidence that deaths from heart failure

*Table 5 Prevalence of heart failure, European studies*

| Study | Prevalence (per 1000 population) | | |
|---|---|---|---|
| | Men | Women | Total |
| Europe | | | |
| Sheffield[156] | | | 29·4 (14·2–44·6) |
| Göteborg 1963–80[69] | | | |
| 50 years | 21 (11·4–30·7) | | |
| 67 years | 130 (104·4–156·4) | | |
| Göteborg 1971–7[157] | | | |
| 70 years | 110 (77–143) | 80 (54–106) | 93 (72–115) |
| 75 years | 170 (130–210) | 110 (80–140) | 136 (112–150) |
| London[62] | | | |
| <65 years | | | 0·6 |
| >65 years | | | 28·0 |
| Total population | | | 3·8 (3·2–4·4) |
| Transition,[18] Netherlands | | | |
| 45–64 | 4 | 3 | 4 |
| 65–74 | 33 | 29 | 31 |
| 75+ | 93 | 83 | 87 |
| all (incl. <45) | 10 (8·6–11·4) | 12 (10·6–13·4) | 11 (10·1–11·9) |
| Nijmegen[68] | | | |
| 45–64 | 8 | 3 | 5 |
| 65–74 | 49 | 32 | 40 |
| 75+ | 159 | 162 | 161 |
| all (incl. <45) | 10·4 (9·3–11·5) | 12·6 (11·4–13·8) | 11·6 (10·8–12·4) |
| Italy,[158,159] Castel Franco | | | |
| <65 years | | | 20 (16·6–23·4 |
| >64 years | | | 83 (71·0–95·0) |
| Denmark[160] | | | |
| 40–59 years | | | 1·5 (0–3·9) |
| 60–69 years | | | 38 (26–50) |
| >69 years | | | 190 (165–215) |
| Nottinghamshire[63] | | | |
| 30–39 | | | 0·1 |
| 50–59 | | | 5·5 |
| 70–79 | | | 42 |
| all | | | 13 (10–16) |
| Gen Practice | | | |
| U.K. (1991/2) | | | 0·1 |
| 25–44 | | | 4·2 |
| 45–64 | | | 27·3 |
| 65–74 | | | 74·1 |
| 75–84 | | | 140·3 |
| 85+ | | | |
| all | | | 8·9 |

( )95% confidence interval, calculated as 95% CI=(p) ± 1·96 × $\sqrt{((p(1-p))/n)}$ (p=prevalence, n=number of persons in denominator),

amongst younger individuals (aged 45–54) in the United States have decreased, in parallel with a reduction in incidence of heart failure in that age group in the Framingham Study over the last 4 decades[83]. It is suggested this may be due to hypertension detection and treatment programmes[84]. An overview of trials of pharmacological treatment of hypertension estimated that effective intervention in individuals with hypertension may reduce the age standardized incidence of heart failure by as much as 50%[85]. Whether such an effect will be observed outside the context of carefully conducted clinical trials is as yet unknown.

## Risk factors

Risk factors for the development of heart failure in the general population have been examined in the Framingham Heart Study (Table 8) and the Study of Men Born in 1913[69,71,75]. The presence of cardiovascular disease is, not surprisingly, associated with a greatly increased risk of heart failure. In the Framingham Study almost 20% of those suffering a myocardial infarction developed heart failure within 5–6 years[86]. In hospital series the proportion may be even higher with progressive dilatation of the left ventricle within 4 weeks of

*Table 6 Incidence of heart failure*

| Study | Incidence (per 1000 population) per annum | | |
|---|---|---|---|
| | Men | Women | Total |
| United States | | | |
| Vermont[64] | | | |
| All ages | | | 5·0 (2·8–7·3) |
| North Carolina[64] | | | |
| All ages | | | 3·5 (0·9–6·0) |
| Rochester 1981/2[73] | | | |
| 45–49 | 1 | 0 | |
| 50–54 | 1 | 0 | |
| 55–59 | 3 | 1 | |
| 60–64 | 6 | 2 | |
| 65–69 | 16 | 5 | |
| 70–74 | 9 | 10 | |
| 0–74 | 1·6 (1·0–2·2)# | 0·7 (0·4–1·0)# | 1·1# |
| Framingham*[92] | | | |
| 50–59 | 3 | 2 | |
| 80–89 | 27 | 22 | |
| >=45 | 7·2 | 4·7 | |
| all ages | 2·3# | 1·4# | |
| Europe | | | |
| Göteborg[69] | | | |
| 50–54 years | 1·5 | | |
| 61–67 years | 10·2 | | |
| Eastern Finland[72] | | | |
| (Boston criteria) | | | |
| 45–54 | 1·9 | 0 | |
| 55–64 | 3·1 | 1·5 | |
| 65–74 | 8·2 | 2·0 | |
| 45–74 | 4·0 (2·7–5·3)# | 1·0 (0·5–1·5)# | |
| (Framingham criteria) | | | |
| 45–54 | 2·2 | 0·2 | |
| 55–64 | 3·3 | 2·2 | |
| 65–74 | 7·7 | 2·9 | |
| 45–74 | 4·1 (2·8–5·4)# | 1·6 (0·9–2·3)# | |
| Transition, Netherlands[18] | | | |
| 45–64 | 3·1 | 2·5 | 2·8 |
| 65–74 | 20·1 | 16·2 | 18·0 |
| 75+ | 50·3 | 39 6 | 43·5 |
| Nijmegen[68] | | | |
| 45–64 | 2 | 2 | 2 |
| 65–74 | 17 | 11 | 13 |
| 75+ | 45 | 38 | 40 |
| all ages | 3·1 (2·5–3·7) | 3·4 (2·8–4·0) | 3·3 (2·9–3·7) |
| Gen Practice[67] | | | |
| U.K. (1991/2) | 1·4 | 1·0 | 1·0 |
| 45 64 | 9·3 | 7·4 | 8·3 |
| 65–74 | 22·7 | 16·2 | 18·6 |
| 75–84 | 29·1 | 32·9 | 32·0 |
| 85+ | | | |
| all ages | | | 2·3 |

*Framingham data pertain to the 1980's.
#Age adjusted to relevant natural population.
( )approximate 95% confidence interval, calculated as 95% CI=(I) ± 1·96 × $\sqrt{n/PY}$ (I=incidence, n=number of cases, PY=person years of observation).

infarction greatly increasing the risk[87–89]. Hypertensive cardiovascular disease with electrocardiographic evidence of left ventricular hypertrophy confers a more than 15-fold increased risk of developing heart failure[75]. Echocardiography provides a more sensitive and reliable indicator of increase in left ventricular mass, which is associated with higher rates of cardiovascular disease and death[90] but no population-based studies relating echocardiographically assessed left ventricular mass to occurrence of congestive heart failure have been

*Table 7 Aetiology of heart failure*

| Aetiology | Teerlink *et al.**[80] (%) | Framingham Heart Study**[75] (%) | |
|---|---|---|---|
| | | Men | Women |
| Ischaemic | 50·3 | 59 | 48 |
| Non-ischaemic | 49·7 | 41 | 52 |
| Hypertensive | 3·8 | 70 | 78 |
| Idiopathic | 18·2 | | |
| Valvar | 4·0 | 22 | 31 |
| Other*** | 10·3 | 7 | 7 |
| No aetiology provided | 13·3 | | |

*Based on 31 reports on heart failure published from July 1989 to June 1990.
**Framingham Heart Study, 32 year follow-up. Percentages added up to over 100% as hypertension and coronary artery disease were not regarded as mutually exclusive causes in the Framingham Heart Study.
***Other: viral, ethanol, amyloidosis, postpartum etc.

*Table 8 Relative risks for the development of heart failure (Framingham Heart Study; 36 year follow-up)[75]*

| | Men (age; years) | | Women (age; years) | |
|---|---|---|---|---|
| | 35–64 | 65–94 | 35–64 | 65–94 |
| Serum cholesterol* | 1·2 | 0·9 | 0·7 | 0·8 |
| Hypertension** | 4·0 | 1·9 | 3·0 | 1·9 |
| Glucose intolerance | 4·4 | 2·0 | 7·7 | 3·6 |
| Electrocardiographic LVH*** | 15·0 | 4·9 | 12·8 | 5·4 |

*Serum cholesterol >6·3 mmol . $l^{-1}$.
**Hypertension: blood pressure >160/95 mmHg or on antihypertensive treatment.
***Voltage criteria for left ventricular hypertrophy on resting electrocardiogram.
LVH=left ventricular hypertrophy.

published. There are many other pathophysiological markers associated with an increased risk of heart failure, reflecting the changes associated with increasing cardiac damage: increased heart volume on chest radiograph, T wave abnormalities on the electrocardiogram, and a reduced peak expiratory flow rate amongst many others[9,69,91].

Diabetes mellitus appears to be a more powerful risk factor in women than men[92] and only part of this increased risk can be attributed to concomitant hypertension, obesity and dyslipidaemia. Diabetes alone may induce important structural and functional changes in the myocardium that increase the risk of heart failure. Body weight is also an independent risk factor for heart failure[69,92], but interestingly, total cholesterol is not. A high total cholesterol to high density cholesterol ratio is, however, powerfully associated with an increased risk of heart failure presumably due to coronary heart disease. Cigarette smoking increases the risk of heart failure, possibly through the same mechanism, but the relationship becomes weaker with increasing age[69].

The importance of the above risk factors and markers lies in the ability to identify those individuals at especially high risk of heart failure, and early modification of such risk factors may prevent, or at least postpone, the onset of heart failure.

## Prognosis

The prognosis of untreated heart failure is unknown. All studies providing information on survival and prognosis date from the era in which diuretics have been in use. Information on the prognosis of heart failure can be derived from population-based studies, hospital series and the placebo arm of heart failure trials. The latter source, however, reflects the highly selected group of patients that enter clinical trials. Whatever the source of data, heart failure is associated with a marked reduction in life expectancy at any age.

### *Population based studies*

The Framingham Heart Study reported a mortality rate higher than that found in the trials of therapy in mild to moderate heart failure. Once heart failure had developed only 25% of men and 38% of women were alive at 5 years[70] with a median survival of only 1·66 years in men, and 3·17 years in women. This reflects a mortality rate 6 to 7 times that of the general population of the same age. Heart failure in the event of acute myocardial infarction carries an especially poor prognosis[93]. If death within the first 90 days of development of heart failure is excluded from the data (e.g. deaths from heart failure soon after myocardial infarction) as is the case in most clinical trials, the median survival in Framingham Heart Study subjects increases to 3·2 years in men, and 5·4 years in women[70]. The poor prognosis of heart failure was confirmed in the study in Rochester, Minnesota, with only 66% alive 1 year after the diagnosis of heart failure[73]. Mortality 5 years following the onset of heart failure was 26% in men with mild to moderate heart failure in the Study of Men Born in 1916[94]. NHANES (National Health and Nutrition

Examination) published a 10 year mortality of 43% in men and women aged 25 to 74 years with self reported heart failure[42]. The mortality rate of patients with heart failure may increase with age: the Framingham Study reported a 27% increase in mortality rate per decade of life in men, and a 61% gradient in women[92]. This has not been confirmed in other studies[95,96]. Framingham data also suggest that women not only develop heart failure less frequently than men, but may also benefit from a lower mortality rate when they develop heart failure[92].

In the Framingham Heart Study approximately 50% of all deaths in those who developed heart failure were 'sudden' (death within 1 hour of the onset of symptoms), the incidence of such a mode of death being five times that of the general population[97]. Despite lack of agreement on the definition of sudden death[98] recent trials of pharmacological therapy in heart failure have also reported that approximately half of all deaths are sudden[99], the remainder, with few exceptions, being cardiovascular, with the majority being due to progressive pump failure. The precise mechanisms implicated in sudden death in patients with heart failure remain to be elucidated, but antiarrhythmic therapy (with the possible exception of amiodarone in severe heart failure[100]) has not been shown to prevent such deaths[101,102]. The prognostic benefit of angiotensin converting enzyme (ACE) inhibitors in recent heart failure trials is largely due to a reduction in the number of deaths from progressive pump failure, with little or no effect on the number of sudden deaths[103].

## *Hospital-based studies*

Hospital series tend to reflect the patient population of specialist referral centres and thus often include only the more severe cases of heart failure[104]. Franciosa reported a mortality rate of 34% at 1 year, 59% at 2 years, and 76% at 3 years in a series of 182 men with chronic heart failure refractory to standard medical therapy at that time[95]. Wilson documented another series of patients with severe heart failure referred for consideration of vasodilator therapy[96]. All of these patients experienced symptoms at rest or on minimal exertion. Mortality was 48% at 1 year and 68% at 2 years. However, Andersson and Waagstein[105] reported a much lower mortality rate (50% at 5 years) in a retrospective study of all patients aged less than 65 years discharged from hospitals in Western Sweden with a coded diagnosis of heart failure. The authors checked all coded patients' hospital records to confirm the diagnosis of heart failure, but the criteria for inclusion in this study were less stringent than those used in the Framingham Heart Study. The authors would have presumably missed cases of heart failure that were not coded as such on discharge from hospital, but such cases may be expected to be less severe, if anything, than those correctly coded. A recent hospital-based study from Denmark reported a 1 year mortality of 21% in 190 patients under age 76 years who had heart failure by clinical criteria[106]. Patients with myocardial infarction and malignant disease were excluded. A similar study from Montreal, Canada of 153 consecutive patients presenting to the emergency room with decompensated heart failure reported a 1 year mortality of 33%[107].

## *Pharmacological trials*

Further data on the prognosis of heart failure is available from the placebo arms of the trials of pharmacological agents in heart failure[108–111]. As these trials were composed of a highly selected group of patients the generalizability of such results is not clear. The first CONSENSUS Study enrolled only patients with severe heart failure (symptoms at rest) and reported a mortality rate of 52% at 1 year in the placebo group[109]. Two later trials included patients with a wider spectrum of severity and reported a correspondingly lower mortality of between 15 and 20% at 1 year in the placebo group[110,111]. The Acute Infarction Ramipril Efficacy (AIRE) Study reported a 1 year mortality of 14·0% in the placebo arm in patients with symptomatic heart failure in the first few days following myocardial infarction[108]. This study excluded patients with severe symptoms (NYHA Class IV) and the heart failure needed only to be transitory for entry into this study.

## *Predictors of prognosis*

There is conflicting evidence on whether patients with underlying coronary artery disease do better or worse than those with other (or unknown) aetiologies of heart failure. Franciosa reported a significantly lower survival in patients with severe heart failure and electrocardiographic or coronary arteriographic evidence of coronary artery disease as compared with idiopathic dilated cardiomyopathy (54% vs 77% at one year, $P<0·01$) in a hospital-based series[95]. Similar differences in survival were found in the first Veterans Administration heart failure study[110]. Wilson *et al.* did not observe a difference in survival between heart failure patients having coronary artery disease or primary cardiomyopathy[96]. This is in marked contrast to community data from Framingham which suggest that, if anything, men who develop heart failure due to coronary artery disease do better than those with other aetiologies of heart failure[92]. Such a discrepancy between studies remains unexplained although many biases may exist in selected hospital series. Differences in establishing the diagnosis of coronary artery disease may also play a role: angiographic evidence of coronary artery disease is routinely available in population-based studies.

There has been much interest in the clinical and investigative features predictive of outcome in patients with heart failure[112]. Of the numerous variables that have been studied[113] maximal oxygen uptake and duration of exercise testing, right and left ventricular

ejection fractions, pulmonary capillary wedge pressure, ventricular arrhythmias, levels of catecholamines and atrial peptides have been shown to predict prognosis in patients with heart failure[36,114–122]. In the SOLVD-Registry functional class as assessed by the New York Heart Association Classification[38], the result of the 6 minute walk test (a simple symptom-limited exercise test), and left ventricular systolic function as measured by the ejection fraction were the strongest independent predictors of the 1 year mortality rate and hospitalization rate[36]. Not all series have found that such variables do predict survival: functional class appears to be the most consistent — the more severe the functional impairment, the worse the prognosis. Arrhythmias appear to provide little prognostic information regarding the occurrence of sudden death[101].

### *Secular trends in prognosis*

Information on secular trends can be obtained from The Framingham study because of the long follow-up period and the uniform case definition throughout the study. No improvement in survival over 4 decades has been noticed[92], but the effect of the relatively recent introduction of ACE inhibitor therapy on survival has not been examined as yet. A recent meta-analysis demonstrated an impressive 23% reduction in mortality in trials of ACE inhibitors in heart failure[123] but the gain in life expectancy is measured in months rather than years. Furthermore, it appears that a substantial proportion of heart failure patients outside the context of clinical trials are not receiving such optimal treatment[124,125].

## Mortality

In the United States, the number of deaths ascribed to congestive heart failure as the underlying or contributing cause of death rose from 130 000 in 1970 to 267 000 in 1988[75,84]. Part of this increase may be explained by the increasing proportion of the elderly within the population, but age-adjusted death rates for heart failure as the underlying cause of death (per 100 000 population) increased from 7·2 to 8·8 for white men, and from 4·6 to 6·3 in white women from 1979 to 1988. For blacks these figures were 11·4 and 13·2 for men and 8·6 and 10·1 for women respectively. After 1988 age standardized rates started to decline; to 7·2 and 5·3 for white men and women in 1990 and 11·0 and 8·2 in blacks respectively[126]. However, as the U.S. standard certificate of death was revised in 1989 this decline may to some extent be artificial, and depends on the accuracy of the death certificates.

At all ages, the death rate from cardiac failure is greater in black than white patients in the United States[127,128]. This may be related to the increased prevalence of hypertension[129,130] and early coronary artery disease in the black population[131], although poorer access to health care may also play a part[132], as may any difference in the accuracy of death certificates in blacks compared with whites.

In Canada, despite an increase in the absolute number of deaths with a primary diagnosis of heart failure, age-adjusted death rates for heart failure have declined in the period between 1980 and 1990[133]. It has been suggested this is due to the improved survival of patients with heart failure but it is difficult to exclude a spurious decline because of temporal changes in coding practice.

Mortality data from other countries is limited. In the United Kingdom the death certificate explicitly forbids heart failure to be entered as the primary cause of death, and instead the underlying pathological process is specified, e.g. coronary heart disease. Attempts to estimate mortality figures for deaths from heart failure from death certification in the UK are unlikely to be valid[134].

## Hospital morbidity

Hospital morbidity data is readily obtainable but relates only to those individuals who have required hospital (usually inpatient) treatment and therefore do not necessarily reflect the incidence or prevalence of the condition within the community. Any change in the number of patients admitted or discharged from hospital over time may relate more to changes in the perceived usefulness of inpatient assessment and treatment, and changes in awareness of the condition, than to any real change in incidence or prevalence. The accuracy of available data may also vary within and between countries and over time[58]. Such data, however, do demonstrate the increasing demands that heart failure is placing on hospital resources. European data suggest that approximately 1% of national health care budgets is spent on individuals with heart failure[135–138], the bulk of the costs being due to the costs of hospital admission.

The number of hospital discharges with heart failure coded as the primary diagnosis in Scotland rose by 60% between 1980 and 1990 to 210 per 100 000 population per annum[139]. A similar increase has also been recorded in Sweden for the years 1970–1986: counting only one admission per year for any individual there was an 80% increase in discharges for heart failure in men, and a 130% increase in women[94], with an even more marked increase in those aged over 75 years. Data from Holland suggest an increase of the same magnitude[140].

In the United States in 1991 congestive heart failure was the primary discharge diagnosis in approximately 790 000 hospitalizations and constituted the leading diagnostic related group among hospitalized patients aged over 65 years of age[141]: more than double the number observed in 1978, and more than five times the number in 1970[142,143]. This reflects a year-on-year age-adjusted increase in hospitalizations from 82 per 100 000 population in 1970 to 281 per 100 000 in 1990.

The method of reimbursement for medical expenses throughout that period of time changed with the introduction of diagnostic related groups in 1983 and this may have affected the absolute numbers to some degree, but is unlikely to explain such a massive and steady increase. Among black patients in the US, rates of hospitalization are even higher than white patients, probably reflecting a greater incidence and prevalence of heart failure in the black population[142,144].

A high readmission rate is characteristic of patients with heart failure[145,146]. A survey of seven hospitals (two university and five general) in the Netherlands in 1991 and 1992 indicated that 16% of patients were readmitted with heart failure within 6 months of their first admission[140]. Given this frequent rehospitalization of patients with heart failure, the number of hospital discharges rather than the number of individual patients discharged may exaggerate the size of the problem. The Swedish study[94], however, demonstrated an age-adjusted increase in the number of heart failure admissions, even after exclusion of readmissions. The increase particularly concerned persons older than 75 years, as was the case in Scotland and the Netherlands[139,140].

The hospital case-fatality rate for heart failure is reported to have fallen in the U.S.A.[144], the Netherlands[140], and Scotland[139] over the last decade, as has the average duration of hospitalization. The latter may reflect changing patterns of care as much as any improvement in therapy.

## Conclusions and directions for future research

Heart failure is an important and growing public health problem: it is the cause of substantial morbidity and mortality, and consumes a significant proportion of the health care budget in most developed countries. Notwithstanding the lack of a clear definition and differences in methodology of the studies discussed in this paper, some general conclusions can be drawn. The incidence and prevalence of heart failure increase markedly with age and the most common aetiology of heart failure is coronary artery disease. There is no evidence to date that the prognosis of heart failure in the community has improved despite the advances in therapy over the last 4 decades. However, the effect of widespread use of drugs that, in clinical trials, have been shown to prolong life in patients with heart failure may not have become evident at the population level as yet and it is of concern that many patients do not receive optimal treatment[124,125].

The number of hospital admissions due to heart failure has been steadily increasing in developed countries for some time, and this rise is only partially explained by changes in the proportion of the elderly within these populations. This rise may appear paradoxical in view of the declining cardiovascular (and coronary heart disease) mortality rates and improvements in hypertension treatment[147,148]. It is conceivable, however, that more individuals are surviving the initial cardiac damage produced by e.g. myocardial infarction, only to develop heart failure at a later date when the heart can no longer compensate. The Framingham study tends to confirm this impression: over the last 40 years the average age of onset of heart failure has been steadily rising[70]. It has also been suggested that the treatment of hypertension merely postpones the onset of heart failure to an older age, rather than preventing it[84]. The improved treatment options may explain the declining hospital case fatality rates, and at the same time the increasing number of patients at increased risk of readmission.

What does the future hold for the burden of heart failure within our communities? A recently developed simulation model predicts a transition from acute to chronic cardiovascular disease in the near future in the Netherlands, resulting in an age-adjusted increase in the number of patients with heart failure[4]. This increase will be markedly accentuated by ageing of the population, resulting in a large increase in the number of elderly heart failure patients, in whom presentations of symptoms may be atypical[149,150] and readmissions are frequent[145,146]. It is likely that all industrialized countries will go through a similar transition.

We need to develop and validate diagnostic criteria for heart failure which utilize modern cardiological investigations, and which can be applied in both clinical and epidemiological research. A clinical case definition is necessary in population surveys which aim to measure more than just the frequency of this syndrome, as classifying individuals correctly is essential in relation to aetiology, cardiac anatomy and function, morbidity and mortality.

Future studies of heart failure should be population based if the aim is to reliably measure the incidence and prognosis of unselected cases of heart failure. Hospital-based series cannot provide this information. Such an approach will permit an evaluation of the comparative importance of the various aetiological factors, the development of more powerful prognostic indices, and sound comparisons between populations, within populations and over time: vital statistics are too unreliable to allow meaningful comparisons. The burden of heart failure within our communities can also be monitored by this approach, and by systematically following-up incident cases given current best treatment any change in the clinical course of this syndrome will be detected. Whilst clinical trials of new treatment should ideally be conducted in the same patient populations inevitably there is case selection and so it is incumbent on those randomizing patients to define, through registers, the populations from which these patients come. It will then be possible to describe what proportion of heart failure patients are likely to benefit from such new treatments in the future. Finally, we need to ensure current scientific evidence on treatment is translated into clinical practice to ensure maximum benefit to the population.

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# Appendix

## Description of six scores for the classification of heart failure (Also see Table 2)

### *Framingham Heart Study*[43]

(1) Developed for assessment of heart failure in a population-based study.
(2) Heart failure present when two major (X) or one major (X) and two minor (x) criteria are fulfilled.

| | |
|---|---|
| Paroxysmal nocturnal dyspnoea (x) | |
| Neck vein distension (x) | |
| Rales (x) | |
| Cardiomegaly (X) | |
| Acute pulmonary oedema (X) | |
| S3 gallop (X) | |
| Weight loss >=4·5 kg in 5 days in response to treatment* (X) | |
| Hepatomegaly (x) | |
| Peripheral oedema (x) | |
| Nocturnal cough (x) | |
| Dyspnoea on ordinary exertion (x) | |
| Pleural effusion (x) | |
| Decrease in vital capacity by 1/3 from maximum*** (x) | |
| Tachycardia** (x) | |

*Weight loss >=4·5 kg in 5 days in response to treatment. Major criterion if weight loss occurred during therapy for heart failure, otherwise minor.
**Increased jugular venous pressure: >16 cm of water.
***Tachycardia: heart rate >=120 $min^{-1}$.
***Vital capacity down 1/3 from maximum.

### *Study of men born in 1913*[44]

(1) Developed for assessment of heart failure in a population-based study.
(2) No, latent or manifest heart failure if stage is 0, 1, 2/3 respectively.

| | |
|---|---|
| No history, no signs and no treatment for heart failure | Stage 0 |
| Cardiac score >0 | Stage 1 |
| Cardiac score >0 and dyspnoea or treatment with digitalis or loop diuretic | Stage 2 |
| Cardiac score >0, dyspnoea and treatment with digitalis or loop diuretic | Stage 3 |

| *Cardiac score* | |
|---|---|
| History of heart disease (@) | 1–2* |
| Angina pectoris | 1–2* |
| Swollen legs at the end of the day | 1 |
| Dyspnoea at night | 1 |
| Pulmonary rales | 1 |
| Atrial fibrillation | 1 |

*1=in the past, 2=during the last year. (@) Angina pectoris or myocardial infarction. A maximum of 2 points was given for those reporting both a history of heart disease and angina.

### *Walma*[45]

(1) Developed for assessment of heart failure in patients on diuretic therapy in general practice.
(2) Heart failure (arbitrarily) considered present if score >=3.

| | |
|---|---|
| Paroxysmal nocturnal dyspnoea | 3 |
| Dyspnoea on exertion | 2 |
| Increased venous pressure | 2 |
| All other items | 1 |
| (Tachycardia present if heart rate >100 $min^{-1}$) | |

### *Boston score*[47]

(1) Developed for assessment of heart failure in 150 general medical outpatients who were on long-term digitalis therapy.
(2) No, possible or definite heart failure if score equals 0–4, 5–7, 8–12 points respectively.

| *History* | |
|---|---|
| Rest dyspnoea | 4 |
| Orthopnoea | 4 |
| Paroxysmal nocturnal dyspnoea | 3 |
| Dyspnoea on walking on level | 2 |
| Dyspnoea on climbing | 1 |

| *Physical examination* | |
|---|---|
| Heart rate 91–110 min, 1; >110 min, 2 points | 1/2 |
| Elevated jugular venous pressure >6 $cmH_2O$ 2 points; >6 $cmH_2O$, plus hepatomegaly or oedema 3 points | 2/3 |
| Rales basilar 1; >basilar 2 points | 1/2 |
| Wheezing | 3 |
| S3 gallop | 3 |

| *Chest X-ray* | |
|---|---|
| Alveolar pulmonary oedema | 4 |
| Interstitial pulmonary oedema | 3 |
| Bilateral pleural effusion | 3 |
| Cardiothoracic ratio >=0·5 | 3 |
| Upper-zone flow redistribution | 2 |

No more than four points allowed from each of the three categories

### *NHANES score*[42]

(1) Developed for assessment of heart failure in a national health survey.
(2) Heart failure present if score >=3.

| *History* | |
|---|---|
| Short of breath when hurrying on the level or up slight hill? | 1 |
| Short of breath when walking at ordinary pace on the level? | 1 |
| Do you stop for breath when walking at own pace? | 2 |
| Do you stop for breath after 100 yards on the level? | 2 |

| *Physical examination* | |
|---|---|
| Heart rate 91–110 min, 1 point; >110 min, 2 points | 1/2 |
| Rales basal 1 point; >basal 2 points | 1/2 |
| Neck vein distension | 1 |
| Neck vein distension and oedema/hepatomegaly | 2 |

| *Chest X-ray* | |
|---|---|
| Cephalization of pulmonary veins | 1 |
| Interstitial oedema | 2 |
| Alveolar fluid and pleural fluid | 3 |
| Interstitial oedema and pleural fluid | 3 |

*Gheorghiade*[46]

(1) Developed to assess severity of heart failure in patients on digoxin with 'documented congestive heart failure based on the presence of clinical or radiographic evidence of heart failure or both'.

(2) Each item contributes one point. Heart failure (arbitrarily) considered present if score >=3. ¶Cardiomegaly on chest X-ray if cardio-thoracic ratio >0·5.