Have we jugulated the epidemic of heart failure?
One swallow does not make a summer . . .

See page 209 for the article to which this Editorial refers

Epidemiology is a useful tool for describing the frequency (incidence and prevalence) of a disease and its demographic and clinical characteristics, in a given population, at a given time. This is what we should call ‘snapshot’ epidemiology. It may also be used to assess the prognosis and outcome in a given cohort of patients during a given follow-up period. Repeating the snapshots at various periods may provide information on changes over time in the frequency and characteristics of the disease. Trends may be generated, which are tempting to analyse as the consequence of changes in patient management throughout the same period. The paper of Stewart et al.\[1\], in this issue, describes trends in hospitalization for heart failure that occurred from 1990 to 1996 in Scotland.

It raises the intriguing hypothesis that the ‘epidemic’ of hospitalization for heart failure in Scotland previously reported between 1980 and 1990, may have peaked around 1993–1994. The authors speculate that this may be a sign of modern-day strategies for the prevention and the treatment of heart failure.

Let us first praise the Information and Statistics Division of the National Health Service in Scotland for its Scottish Morbidity Scheme. Thanks to a single individual identifier, the scheme allows data collection on all hospital discharges, and links the data to information on morbidity and mortality held by the General Register Office of Scotland\[2\]. It is therefore extraordinarily efficient for epidemiological studies. Only a few countries in Europe generate such comprehensive data. Nevertheless, however sophisticated and comprehensive, the data are collected retrospectively and rely heavily on a coding system which relates hospitalization to heart failure as diagnosed by the hospital physician. Given that rapid changes have taken place in awareness, perception and management of cardiovascular disease in general, and of heart failure in particular, the accuracy and reliability of heart failure diagnosis in Scottish hospitals and elsewhere has certainly changed over time. For instance, in the Stewart et al.\[1\] report, only heart failure coded as a primary diagnosis for hospitalization tended to level off. Heart failure coded as a secondary diagnosis, and thus, the total heart failure hospitalizations seemed to continue to rise, even though at a slower rate after 1993–1994. One possible explanation for this discrepant trend is that physicians may now better investigate patients for the primary or precipitating cardiovascular cause of heart failure. This primary cause would then be coded more frequently as the primary diagnosis of hospitalization, instead of heart failure per se. This would result in heart failure being more frequently coded as secondary diagnosis. Indeed, because of a better diagnosis work-up (ergometry, angiography . . .), a patient hospitalized for ischaemic heart failure would be coded as coronary heart disease as a first diagnosis and as heart failure only as a second diagnosis. This is consistent with the authors’ finding that heart failure as a secondary diagnosis (relative to primary diagnosis) was more frequent in cardiology units compared to non-cardiology units.

Thus, drawing trends from data generated using a rapidly changing instrument (diagnosis performance) may be misleading. Relating the observed peaking of hospitalization to better prevention and treatment of the disease is speculative and may be hazardous. A whole range of confounding factors may account for the retrospectively observed trend.

Very few recent reports of changes in rates of heart failure hospitalization are available for reference. In their report on seasonal variations in heart failure hospitalization and mortality in France, Boulay et al.\[3\] have documented the number of heart failure hospitalizations from 1995 to 1997 in this country. The total number of hospitalizations rose on average from 91 610 in 1995 to 113 776 in 1996 and 118 827 in 1997. Although the observation was limited to 3 years (the paper did not include information on heart failure hospitalization before 1995), the trend showed a consistent rise, which does not confirm the findings of Stewart et al.\[1\]. Interestingly, 1-year case fatality decreased in the report by Stewart et al., from 1990 to 1996 in Scotland, while it remained remarkably stable in France, from 1992 to 1996\[3\].
Concomitantly, in both cases the total number of heart failure hospitalizations rose steadily throughout the same period.

Can we infer from the results of Stewart et al[1] that modern-day treatment and management strategies have begun to bear fruit? However tempting, the speculation that the epidemic of heart failure is being juggled cannot be accepted before similar, consistent and persistent trends in different countries and different health systems are reported. Modern-day treatment of hypertension and ischaemic heart disease, the most common causes of heart failure, is effective in preventing heart failure. Controlling high blood pressure in the elderly may decrease heart failure incidence by as much as 40%. Use of angiotensin converting enzyme (ACE) inhibitors in hypertensive patients and in patients with other cardiovascular risk factors may decrease new occurrences of heart failure[6]. Better management of acute myocardial infarction using thrombolytic and anti-thrombotic therapy as well as primary angioplasty do improve patient survival.

Would prolonged survival result in the increased incidence of heart failure? In the various clinical trials in acute myocardial infarction where heart failure was prospectively monitored, it was usually less frequently observed in the active treatment groups. However, it may be argued that, given the average age of patients at inclusion in these trials, which is usually lower than that of patients affected by heart failure, and given the relatively short period of follow-up, which rarely exceeded 3–4 years in all these trials, one cannot rule out that ‘late’ heart failure in myocardial infarction survivors is indeed increasing. With the increasing age of the population and the longer survival of patients with chronic heart disease, the predicted growing prevalence of heart failure has been interpreted as ‘the ironic failure of success’. As medical and surgical advances have lengthened survival rates in chronic heart disease patients, the number who live long enough to develop heart failure may be increasing[5–7]. The combined effects of ageing and of chronic heart disease on the cardiovascular system would inevitably lead to heart failure. As a result, the age of patients with a first ever hospitalization for heart failure may be progressively rising, which is confirmed by the observation of Stewart et al.

The work of Stewart et al[1] is limited to the description of hospitalization for heart failure and does not address the general incidence and prevalence of heart failure. Nearly all available epidemiological descriptions of heart failure are based on retrospective analysis of hospital records of patients admitted to hospital, referred for or presenting to the emergency room with heart failure, and only few observations are population-based[5–8]. Because out-patient management of patients with heart failure has improved, one can expect that patients who in the past would have required hospitalization are now treated in ambulatory conditions and may remain out of hospital for longer periods. On another hand, because patients with heart failure are getting older, they may be more frequently institutionalized and managed within nursing homes and therefore relatively less often referred for hospitalization. Thus, a stabilization of hospitalization related to heart failure cannot be safely interpreted as a stabilization of the incidence or prevalence of heart failure in itself. Moreover, because of large variations in health systems and in clinical practice among different western countries, one should caution against the generalization of data derived only from hospital records.

Epidemiological data are essential for estimating and decision making concerning the utilization and allocation of health care resources. They are also critical for planning prospective studies of therapeutic interventions and care management and particularly for sample size calculation and risk stratification. However important, epidemiological data limited to hospitalized patients do not provide information on the huge hidden part of the iceberg, which consists of mild and asymptomatic patients with heart failure. In epidemiological studies of hospitalized heart failure patients, case ascertainment relies on the diagnosis made by the managing physician. The lack of agreement on a definition of heart failure, as well as the lack of gold-standard diagnostic criteria, may result in a considerable heterogeneity in the diagnosis of heart failure in epidemiological studies. Ideally, the definition of heart failure should combine clinical features with an objective measure of cardiac performance and/or neurohormonal markers. Available epidemiological data in heart failure describe only a fraction of patients with this syndrome and are in no way comprehensive. Epidemiological data describing patients with advanced heart failure[9] are more likely to be comprehensive, because, at this stage of the disease signs and symptoms, ventricular dysfunction and neurohormonal activation do coincide and diastolic failure with no evidence of systolic failure is usually rare.

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References

ST-segment mapping in the diagnosis of acute myocardial infarction: a new role for an old method

See page 218 for the article to which this Editorial refers

ST-segment precordial mapping for the diagnosis of repolarization abnormalities in experimental and clinical acute myocardial infarction was introduced in the 1970s[1,2]. This was followed by use of the much more extensive ‘total thoracic’ ST wave mapping using standard exercise protocols to improve sensitivity and specificity when diagnosing ischaemic heart disease[3,4]. As known from theoretical studies, ‘the strength of the surface maps is the direct association of the areas of positive and negative surface potentials with major wavefronts of the heart[5], and — since electrocardiographic potentials are recorded from large areas of the chest — body surface potential mapping enables the detection of significant physiological and diagnostic information not transmitted to the left precordial zones that are usually studied.

Whereas mapping directly explores the torso, the standard 12-lead electrocardiogram relies on remote-field bipolar and unipolar limb leads to compensate for the limited thoracic sampling. Based on these concepts, body surface potential mapping has been applied successfully in the study of various abnormal clinical conditions, such as chronic myocardial infarction, exercise stress testing and the diagnosis of accessory atrioventricular bypass tracts[6]. However, the real clinical relevance of body surface potential mapping has remained rather limited, since the breakthrough of invasive catheter diagnostics and other imaging methods, such as echocardiography and nuclear imaging, which seemed to overcome the diagnostic problems.

The results of large population, evidence based studies, such as GUSTO-IIb, show that the electrocardiogram is still the most accessible and widely used diagnostic tool in patients arriving at an emergency department with symptoms suggestive of acute myocardial ischaemia[7]. According to the GUSTO-IIb study, the 30-day incidence of death or myocardial reinfarction was 10.5% in a total of 12 142 patients with ST-segment depression. A similar number of adverse events occurred in 12.4% of the patient group with ST-segment elevation and depression. The results of GUSTO-IIb has also strengthened the value of two diagnostic tools available in the emergency department: the ECG and creatine kinase determinations, which enable bedside risk stratification and prediction of cardiac events.

It has been known for many years that the ST segment is not electrically inactive, and ST segment elevation measured on the electrocardiogram has been considered to be a characteristic of acute ischaemia, and thus the subject of numerous studies since the first report of Pardee[8] in 1920 on ECG alterations during acute ischaemia. When the whole thoracic surface is explored, the ST segments of normal subjects are quite uniform, with some minor individual variations, and are characterized by a potential maximum in the left mid-ster nal region and a posterior minimum[6]. This pattern is stable and increases in intensity during repolarization without any significant spatial displacement. The ST segment is, in fact, the early part of repolarization, during which the potential distributions may depend on both the distribution of cellular action potential shapes and the sequence of ventricular activation. In normal