Chronic heart failure: a multisystem syndrome

See page 1860 for the article to which this Editorial refers

Chronic heart failure is a clinical syndrome, not a single diagnosis. Although initiated by a reduction in left ventricular function, it is characterized by substantial biochemical, hormonal, metabolic, and functional alterations in the periphery[11]. Non-cardiac factors frequently become the major determinants for both symptom generation and limitation of exercise tolerance. The microvasculature, both structurally and functionally, is disordered, leading to underperfusion of vital organs. Even large arterial function is abnormal. Major abnormalities have been described in skeletal muscle structure, function and metabolism, including early depletion of phosphocreatinine, early acidification and accumulation of inorganic phosphate and adenosine diphosphate, and a reduction in the rate of resynthesis of phosphocreatinine[2]. It is easy to see how these changes could produce muscular fatigue! Explaining the common symptom of dyspnoea is more difficult. Putative abnormalities in lung structure and function have proved difficult to isolate convincingly; abnormal ventilatory control system function such as chemoreflex or ergoreflex overactivity may be more important[3,4].
Neuroendocrine overactivity is considered by many to be a hallmark of the chronic heart failure syndrome. Initially activated as a way of compensating for blood and fluid loss or sodium depletion, these systems, which include the renin–angiotensin–aldosterone system, the sympathetic nervous system, the vasopressin system, and a reduction in vagal tone may, when maintained chronically, be harmful, inducing organ hypoperfusion, myocardial toxicity, and increased susceptibility to ventricular arrhythmias.

Are these 'peripheral' abnormalities the end of the story, or are even more complex and apparently unrelated pathologies in fact true components of the chronic heart failure syndrome, playing important roles not only in symptom generation, but in disease progression and prognosis as well? Should we be looking beyond haemodynamics to metabolic targets for the next therapeutic advance in heart failure?

In this issue, Opasich and co-workers have confirmed a high prevalence of the 'sick euthyroid syndrome' in a large group of heart failure patients, in whom thyroid hormone handling in the cells is deranged by an increase in reverse triiodothyronine, and have related the presence of this syndrome to outcome and to other measures of severity. They also documented partial reversion after successful cardiac transplantation. This may be only the tip of the iceberg as far as an endocrine dimension of chronic heart failure is concerned. Plasma insulin levels are increased in heart failure, associated with decreased sensitivity to the glucose transport effects of insulin. In advanced cardiac cachexia alterations in sex hormones, growth factors, and catabolic and anabolic hormones along with a shower of catabolic cytokines and immune dysfunction have been described. Many appear to have prognostic value, and we are only beginning to understand how these changes come about, what they mean when they occur, and how best to manage a patient when this catabolic storm occurs. Hippocrates recognised the cachexia of heart failure; we are only now appreciating how important it might be to our patients.

References


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Postinfarction ventricular septal rupture

See page 1841 for the article to which this Editorial refers

Postinfarction ventricular septal rupture continues to be a major cause of morbidity and mortality in coronary care patients. It is the cause of death in one third of all patients who suffer from myocardial infarction. In this issue, Cox et al[11] report the results of a large retrospective study that was carried out to evaluate the potential benefits of coronary angiography and subsequent coronary artery bypass grafting in 109 consecutive patients who underwent