comprehensive account of cardiac amyloidosis, and describe two cases of AL-amyloidosis. As a haematologist with some experience of systemic AL-amyloidosis, I would like to add further relevant comments with regard to diagnosis and treatment.

Firstly, with respect to diagnosis, measurement of serum free light-chains is a very important recent development not mentioned in the article. The monoclonal gammopathy in AL-amyloid is often very subtle, below the level of detection by the standard test of immunoelectrophoresis; this serum free light-chain assay is approximately 500 times more sensitive,[1] much more convenient than collecting a quantitative 24-hour urinary collection, and a useful monitor of response to treatment. Its use is recommended in the British Committee for Standards in Haematology (BCSH) 'Guidelines on the diagnosis and management of AL amyloidosis'.[2]

The serum amyloid-P scan is mentioned as being useful only in the preoperative assessment for cardiac transplant. Although poor at assessing cardiac amyloid, it still has an important role in the initial analysis of the patient's overall amyloid load and number of other organs involved,[3] which has an impact on overall prognosis and bone-marrow transplant-related mortality rate.

Secondly, there are two main problems with chemotherapy treatment in this condition. The first is the time taken to achieve a response (often longer than predicted survival). The treatment aims to reduce the monoclonal light-chains, hence shifting the balance from deposition to mobilisation of amyloid, known to be a dynamic process, but occurring particularly slowly in cardiac tissue. The second is the inability to tolerate the agents used, eg fluid retention caused by steroids. In addition to standard use of melphalan, a report by Sanchorawala et al[4] of continuous use of low-dose melphalan in patients with significant cardiac involvement concluded that this well-tolerated regime was effective in patients receiving total doses of >300 mg. Novel agents are increasingly used in the management of myeloma, eg proteasome inhibitors, which may also be helpful in AL-amyloidosis. We recently reported successful treatment of a patient with cyclophosphamide, thalidomide and dexamethasone.[5]

Finally, patients with amyloidosis have significant bleeding risk from vasculopathy due to amyloid deposition in vessels and various coagulation defects. Patients requiring anticoagulation therefore need particularly close monitoring.

BETHAN MYERS
Consultant Haematologist
Queen's Medical Centre, University Hospital,
Derby

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In response

We are grateful for Dr Myers' comments, which highlight a number of issues surrounding the use of chemotherapeutic agents, steroids and anticoagulation in this condition. We agree that the measurement of serum free light-chains will simplify both diagnosis and follow-up of patients with cardiac amyloid and have recently begun using it in our own practice.

Dr Myers' comments about the difficulties of chemotherapy in cardiac amyloid emphasise the need for close cooperation between cardiologists and haematologists in the management of these complex patients. Fluid retention can be a major problem and there is often a very fine balance between symptomatic oedema and symptomatic intravascular volume depletion.

NEIL MAREDIA
Specialist Registrar in Cardiology
James Cook University Hospital, Middlesbrough

SIMON RAY
Consultant Cardiologist
Wythenshawe Hospital, Manchester

Conundrum of BMI measurements

Editor – 'What is wrong with the Body Mass Index?' in Conversations with Charles (*Clin Med* May/June 2005 pp 301–2) was especially interesting and entertaining. Indeed, it was a welcome note highlighting the myths surrounding the accuracy of Body Mass Index (BMI) measurements as a sole measure of the population's health. This topic has received much public attention not just since the Radio 4 debate on BMI and health but also since the release of the much acclaimed book *Super Size Me* by the Amercian author Morgan Spurlock.

Charles is right to point out that BMI (weight/height^2) is more closely associated with the observed risk compared with the 'ponderal index' (weight/height^3), which is closely associated with body fat – the latter measurement providing a more reliable comparitive measure. In addition, he is also right to recognise that increasing abdominal obesity is critical when assessing risk. Hence, in the proposed 'Charles index' or 'health index' he incorporates waist measurement – weight/(waist^2 × height). However, one cannot help but feel this proposed health index may be too simplistic and that it requires a combination of factors to be considered.

There is a growing body of evidence to suggest that other factors apart from BMI alone may be important in assessing health. For example, increasing BMI is associated with higher death rates for all cancers (stomach and prostate in men; breast, uterine and ovarian in women).[1] Furthermore, waist/hip ratio has been shown to be a better marker of abdominal fat.[2] This has been shown also to be an independent marker of high blood pressure[2] and risk for developing ovarian cancer.[3] In the European Prospective Investigation into Cancer and Nutrition (EPIC) study,[4] hip measurement alone correlated with the risk of breast cancer in

pre-menopausal women. Even the amount of physical activity undertaken may have an impact on conditions modulated by inflammation.[5]

Thus, it is apparent that assessment of a population's health is multifactorial and we should consider incorporating measurement of waist/hip ratios and perhaps even physical activity (using a short questionnaire as in the EPIC study) in addition to BMI. Using BMI alone to assess health status in the population may explain the discrepency seen between BMI measurements of a 'weedy' individual with a protuberant belly (normal BMI) compared with a 'large fit chap with a small tummy' (high BMI).

RP ARASARADNAM
Teaching and Research Fellow
Human Nutrition Research Centre
University of Newcastle upon Tyne, Newcastle

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In response

Thank you for giving us the opportunity to comment on Dr RP Arasaradnam's letter, and to Dr Arasaradnam himself for his kind and largely supportive comments. The conversation was necessarily edited and shortened. In it we agreed that waist/hip ratio is a better measure of risk than BMI, although the curve must be J-shaped because of the Belsen effect. Charles did consider incorpo-

rating hip circumference in his index. As this is beneficial it would have to be in the numerator and another length measurement must be introduced into the denominator to maintain an index, making it more complicated. If we scale by height^2, then the index becomes (weight $\times$ hip)/(waist^2 $\times$ height^2). The alternative is to use two existing measures and divide ponderal index by waist/hip ratio. The ratio has no dimensions but incorporates waist rather than waist^2. However, as waist^2, as a measure of abdominal cross-section (abdominal height being predetermined), is the natural power, this might underestimate the effect of abdominal fat. Only observation in large populations could sort out which is most appropriate. Unless and until that is done, Charles would say 'Keep it simple!' He would suggest that in the meantime we should ban BMI and insist on a health index. He would use hip and waist circumference, but present the ratio as hip/waist, thus making it the greater the better.

The question of the difference between fat and muscle density also came up. The error arising from ignoring the difference is relatively small and largely irrelevant to the argument, as individuals of the same proportions will necessarily have the same proportion of fat. Nevertheless, where one is comparing the muscular with the obese, BMI will underestimate the volume of fat as opposed to muscle. If the conjectures in the conversation are correct, this might suggest that volume-for-volume strong abdominal muscle is even more deleterious than abdominal fat. 'An intriguing thought!' to quote Charles.

COEMGENUS

Post-script

The waist-to-hip ratio wins! A recent publication in the *Lancet* has demonstrated its clear superiority over teh Body Mass Index as a predictor of myocardial infarction risk world wide.[1] Well done, both Charles and Dr Arasaradnam.

EDITOR

Reference

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Clinical & Scientific letters

Letters not directly related to articles published in *Clinical Medicine* and presenting unpublished original data should be submitted for publication in this section. Clinical and scientific letters should not exceed 500 words and may include one table and up to five references.

Prescribing patterns in acutely admitted medical patients: changes over 10 years

Advances in treating chronic illnesses inevitably lead to polypharmacy and the associated risks of adverse drug reactions and interactions.[1–3] There are surprisingly few data from secondary care on the absolute numbers of drugs that patients are taking. We studied the prescription lists of emergency patients admitted to the department of medicine of a UK district hospital, at two points 10 years apart.

Methods

All emergency adult admissions to the medical unit at Wycombe Hospital during the months of April 1994 and April 2004 were identified. The first 100 medical admissions for each study month were analysed. Reason for admission was categorised as cardiac, respiratory, gastrointestinal/hepatic, neurological or miscellaneous (endocrine, musculoskeletal, falls, thromboembolic disease, miscellaneous infections, etc). Patient records were scrutinised for the numbers and types of prescribed drugs on admission and on discharge during the two study periods.

Based on the frequency of administration, the total number of daily drug doses was also calculated. Drugs administered 'as required', eg nitrolingual spray, were arbitrarily counted as one dose per day, for the frequency of drug dose. Simple analgesics and non-prescription medicines were not included. Drug types were classified by indication as listed in the chapters of the