# letters to the editor

Please submit letters for the editor's consideration within three weeks of receipt of *Clinical Medicine*. Letters should ideally be limited to 350 words, and sent by email to: clinicalmedicine@rcplondon.ac.uk

## Conversations with Charles

Editor – I wish to thank you for the kind remarks in your editorial (*Clin Med* December 2010, pp 645–6). Shortly after my last manuscript was submitted I realised that contrary to Charles's usual approach I had not gone back to basic principles in my penultimate article and so missed the obvious solution to the problem of establishing an absolute proxy for body bulk.

If one considers the body as a cylinder then bulk is reflected in circumference, and so is essentially two-dimensional. Hence body mass index (BMI) (mass/height$^2$) is a good proxy for relative bulk. To correct for the height of the cylinder one has to scale for height by dividing BMI by actual height/median height of the reference population, say 1.7. This gives an absolute proxy for bulk. This can be simplified to Ponderal index (mass/height$^3$) multiplied by median height. This standardised BMI would have the great advantage of maintaining the familiar 25 as ideal. Its main benefit would be to avoid underestimate of malnutrition in thin tall people, though in the bulky, where body composition is more relevant, standardised waist measurement might remain as good if not better.

CK CONNOLLY

## Acute decompensated heart failure secondary to thiamine deficiency: often a missed diagnosis

Editor – We read with great interest Saunders *et al*'s excellent educational paper (*Clin Med* December 2010 pp 624–7) on malnutrition. Thiamine deficiency leads to dysfunction of cardiovascular system, commonly known as wet beriberi. This tends to present as acute decompensated heart failure and also with signs of hyperdynamic circulation.[1,2] Cardiac beriberi is usually missed in clinical practice because of the absence of classically described symptoms, such as pedal oedema/anasarca. It has been reported that these patients have ongoing myocardial damage with troponin rise.[3] Patients in the high risk group of thiamine deficiency are likely to be suffering from chronic alcoholism, social isolation or poor dietary intake, including elderly.[4] As the body storage of thiamine is often small, with a high turnover rate and a half life of 10 to 18 days, high risk patients can enter into thiamine deficiency rapidly. Coexisting hypomagnesaemia, a likely result of chronic alcohol abuse, further aggravates the myocardial damaging effect of thiamine deficiency.[5]

These patients are likely to be diagnosed with idiopathic dilated cardiomyopathy as coronary angiography rules out ischaemic cardiomyopathy. From our experience, erythrocyte transketolase levels are under requested for patients presenting with heart failure.

Treatment of such patients includes usual heart failure management and aggressive replenishment of thiamine. This has been shown to have dramatic response to haemodynamic state in a few hours.[1,2]

We suggest that thiamine deficiency should be excluded in malnourished patient's presenting with acute decompensated heart failure.

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### References

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### *In response*

Wet beriberi is fortunately a rare condition in the western world, even allowing for a degree of underreporting and missed diagnosis. Instead, interest focuses on patients with moderate thiamine deficiency and the potential role in chronic heart failure. The incidence of thiamine deficiency in patients with heart failure is reported to range from 13–98% and is exacerbated by malnutrition and loop-diuretics, which promote thiamine excretion.[1–2] There have been mixed results from trials assessing the role of thiamine replacement but one small trial did show some promise with improvements in left ventricular function.[3]

While it is important to obtain a diagnosis if wet beriberi is clinically suspected, given the relative safety of thiamine replacement, it should not be withheld pending laboratory results. Thiamine testing is relatively expensive, has a long laboratory turn around time and blood analysis in acutely unwell patients may not represent tissue availability.

It is also worth remembering that all characteristic deficiency states are the end result of progressive utilisation of micronutrient stores in an attempt to compensate for the shortfall. Identification of one