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Letters to the editor

OVERVIEW

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

Sarcopenic obesity: under recognised and over treated?

Editor – Cruz-Jentoft and Landi's fine review of the growing importance of sarcopenia (*Clin Med* April 2014 pp 183–6) omitted to discuss the increasingly recognised condition of sarcopenic obesity. At an individual level the classification of overweight and obesity by body mass index (BMI) as a measure of (excess) fat and lean tissue mass is increasingly recognised as flawed,[1] and many older people with apparently 'healthy' BMIs may in fact be sarcopenic.[2] Meta-analyses consistently show that mortality and morbidity associated with overweight and obesity only increase at a BMI above 30 kg/m^2 in the elderly. In addition, the incidence of cardiovascular disease, mortality and all-cause mortality is higher in those with sarcopenic obesity than those who are 'simply' obese. Sarcopenia probably lies at the heart of the so-called obesity paradox – the finding that modest overweight is beneficial. Thus in the elderly, weight loss interventions are best offered to patients who are obese rather than overweight (by BMI definition) and who have functional impairments, metabolic complications or obesity-related diseases that can benefit from weight loss. Physical activity and exercise should form part of any weight loss therapy, but are of particular importance in the elderly. ■

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References

1 Finer N. Better measures of fat mass – beyond BMI. *Clinical Obesity* 2012;2:65.
2 Mathus-Vliegen EM, Obesity Management Task Force of the European Association for the Study of Obesity. Prevalence, pathophysiology, health consequences and treatment options of obesity in the elderly: a guideline. *Obes Facts* 2012;5:460–83.
3 Atkins JL, Whincup PH, Morris RW *et al.* Sarcopenic obesity and risk of cardiovascular disease and mortality: a population-based cohort study of older men. *J Am Geriatr Soc* 2014;62:253–60.

Delirium: a synthesis of current knowledge

Editor – van Munster and de Rooij are two highly experienced delirium investigators, but I am concerned about four points in their article on delirium (*Clin Med* April 2014 pp 192–5).

1 A key element in their article is the change from the fourth edition of the *Diagnostic Statistical Manual* (DSM-IV-R) to the fifth (DSM-V). However, the 33 references do not include the source on DSM-IV-R or DSM-V. I consulted both the American Psychiatric Association website (www.psych.org) for DSM criteria on delirium and the hard copies of DSM-IV-R and DSM-V. Neither DSM-IV-R nor DSM-V criteria define 'acute onset' as 1 day. DSM-V suggests 'several days', but in practice most investigators, except me, ignore this and do not report speed of onset.
2 The authors are concerned about underdiagnosis of delirium, which is common outside geriatric medicine or old age psychiatry. However, the opposite process – overdiagnosis – is prevalent.[1] Labelling acute behavioural change in dementia as a delirium instead of behavioural and psychological symptoms of dementia (BPSD) is the leading reason for this. There are many reasons for overdiagnosis; in a country with a national health service, general practitioners (GPs) experience difficulty in convincing hospitals to admit patients with BPSD, whereas labelling it 'delirium' is the instant ticket to hospital admission. Diagnosis related group (DRG) funding in some hospitals favours delirium over dementia.
3 Although the authors label their article 'a synthesis of current knowledge', they have completely ignored dissenting views in medical journals that publish the greatest number of delirium articles. This is a logical fallacy known as suppressed evidence. We demonstrated that confusion assessment method (CAM) positive delirium in 647 acute geriatric admissions had no effect on survival in hospital or at 30, 90, 180 or 365 days post admission.[2] Subsequent articles on the Central Coast Australia Delirium Intervention Study (CADIS; ClinicalTrials.gov NCT01650896) showed that a 25% decline in attention, executive function or memory in 24 hours produced a more robust phenotype than the CAM with respect to eliminating false positives, such as BPSD and Parkinson's disease psychosis, and generating high reversibility.[3,4] The phenotype of delirium is to asthma what dementia is to the chronic obstructive pulmonary disease (COPD) phenotype.
4 Every delirium research proposal, investigator guideline and methods section in articles must describe how the investigators tested hearing before any cognitive tests and corrected hearing with portable amplifiers, which are as essential as the stethoscope in cognitive research for older people.[6] ■

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