Daily variability in waist circumference

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*Heart* 2010 96: 550
doi: 10.1136/hrt.2009.179242

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To the Editor: A recent issue of Heart contained two articles concerning abdominal obesity.1 2 Both describe waist circumference as a proxy measure of central fat distribution, linked to atherothrombotic inflammatory abnormalities seen in the “metabolic syndrome”, associated with adverse cardiovascular events.

This “vital sign in clinical cardiology”2 needs accurate assessment. Standardised measurement techniques are more complex than one might think. Waist circumference can vary substantially and rapidly during the respiratory cycle (between 91.0 and 94.1 cm in one subject) and through volitional alteration of abdominal distension (between 86.5 and 94.6 cm in another).3 To negate this, the World Health Organization recommends marking the midpoint between the bottom rib and the iliac crest with a pen, placing the patient’s arms relaxed by their sides. The measuring tape should be directly on the skin—snug but not compressing.4

Yet despite this, personal experience suggests that involuntary changes in waist circumference occur throughout the day, due to the effects of food, faeces and flatus. We performed three small studies in hospital inpatients (n=50) who were mobile, eating a “normal diet”, free of malignancy or oedematous states and not clinically dehydrated. In 48 patients, the waist circumference at 08:00 (prebreakfast) was smaller than that measured at 14:00 (postlunch). This difference was significant (paired t test, p<0.001) for absolute values (1.99 cm, 95% confidence interval 1.53 to 2.45 cm) and percentage change (2.16%, 95% confidence interval 1.69% to 2.64%). The maximum absolute change was 5.8 cm and maximum percentage change 6.5%.

These differences are unlikely to be of relevance in epidemiological studies but become important for individuals who may cross the boundary for diagnostic cut-off criteria over the course of a normal day. Both authors comment that there is no scientific rationale for the use of thresholds or “cut-points” for waist circumference—which, like systolic blood pressure, has a continuous positive relationship with risk. Yet such “cut-points” are used in the diagnosis of the metabolic syndrome—being a mandatory criterion in the International Diabetes Federation guidelines.5 Perhaps it is time to stipulate the use of a fasting or preprandial early morning waist circumference in the diagnosis of the metabolic syndrome.

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Competing interests None declared.
Provenance and peer review Not commissioned; not externally peer reviewed.

REFERENCES

Frailty: the great confounder, the great forgotten

To the Editor: Ryan et al1 present the results of SAFE-PACE-2, which are at odds with SAFE-PACE-1.2 The authors discuss that this may be due to greater frailty in SAFE-PACE-2 participants. This point is valid and merits further reflection in relation to recent frailty literature.

Frailty is a well-recognised clinical syndrome, but it remains difficult to define and measure. Central to the concept of frailty is the dysregulation of multiple systems (eg, balance, muscle strength, cognition), all of which independently contribute to the high incidence of falls in frail older people.3 If falls can be a hallmark of frailty, any research on interventions with falls as endpoint should be able to control for this confounder. Many frailty assessment tools have been (and are still being) proposed but are rarely used to help interpret results of trials in geriatric medicine (eg, by using frailty scales as covariate, conducting subgroup analyses based on increasing frailty categories).

The above would help establish which types of patients are most likely to benefit from an intervention. Along these lines, Ryan et al1 argue that the main beneficiaries of pacing may be non-frail patients, who are at low risk of falls from other sources. It is reasonable to presume that frail patients will continue to fall, despite minimising the contributing effect of carotid sinus hypersensitivity.

The issue calls for a frailty-adjusted meta-analysis of all trials of pacing for carotid sinus hypersensitivity before the intervention is regarded as lacking evidence. The point by Alboni et al4 remains valid, that in the treatment for recurrent vasovagal syncope “can be chosen by considering the clinical context, the risk of trauma and possible comorbidities, in addition to utilising the little or controversial knowledge available, as well as common sense”. Elderly people are very heterogeneous. We need frailty measures that serve as common language for comparing studies that, while asking the same questions and using the same methodology, happened to recruit from different populations. This would contribute towards a better evidence base in geriatric medicine.

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Competing interests None declared.
Provenance and peer review Not commissioned; not externally peer reviewed.

REFERENCES

Segmental coronary endothelial dysfunction in patients with minimal atherosclerosis

To the Editor: We read with great interest the paper published by Lavi et al5 and feel that it is worthy of comment. We admire the foresight and technical expertise that has gone into creating this study, to try and prove a worthy concept. However, we do have an issue regarding the measurement of necrotic core in minimal plaques. The authors state that after selecting the appropriate frames, ‘two independent investigators’ measured each frame and the results were calculated. Sections with endothelial dysfunction had larger necrotic core plaques (0.15 (IQR 0.03–0.35) mm² versus 0.0 (IQR 0.0–0.07) mm², p<0.001) and more dense calcification (0.05 (IQR 0.0–0.15) mm² versus 0.0 (IQR 0.0–0.10) mm², p<0.01).

The absolute value of the numbers calculated for constituent plaque area is very small (in keeping with the modest disease). Have the authors performed intraobserver and interobserver variability testing to prove that they are able to reproduce this level of accuracy? Having analysed thousands of intravascular ultrasound-based virtual histology frames and published work on intravascular ultrasound-based virtual histology variability,6 we are aware that minimal discrepancies in manual border detection, between operators, can significantly influence calculated plaque measurements.