We welcome letters to the Editor concerning articles which have recently been published. Such letters will be subject to the usual stages of selection and editing; where appropriate the authors of the original article will be offered the opportunity to reply.

Letters should normally be under 300 words in length, double-spaced throughout, signed by all authors and fully referenced. The edited version will be returned for approval before publication.

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Obesity in total hip replacement

Sir,

We read with interest the article by Andrew et al in the April 2008 issue entitled, “Obesity in total hip replacement”, and wish to bring to attention two issues.

Firstly, the authors mentioned in the Introduction that there was a conflicting background of evidence regarding the effect of obesity on clinical outcome in total joint replacement. It is important to note that the studies which suggested that obese patients have no increased risk of complications following joint replacement were studies with small samples (130 to 341 subjects). In contrast, the studies that supported a positive correlation between obesity and surgical complications were significantly larger in size (1211 to 3309 subjects). Indeed, among studies throughout the literature that involve samples of 1000 or more, the consistent finding is that obesity confers a higher risk of complication after total joint replacement. As such, it is likely that the lack of difference arising in studies with small samples most likely reflects a lack of power.

Secondly, the authors appear to have made an error in the analyses of their data regarding the crucial outcome of deep infection after joint replacement. If, as indicated, two of the 1071 (0.2%) non-obese subjects developed deep infection after joint replacement compared with five of the 332 obese subjects (1.5%), then application of a chi-squared test to this difference yields a two-sided p-value of 0.011, not 0.115 as reported. The interpretation should be that the incidence of deep infection in obese patients was 7.5 times higher than in non-obese patients, and statistically significantly so. This result concurs with the current literature, which includes a large study from our institution which found obesity to be an independent risk factor for prosthetic infection following primary total hip replacement.

It is time to acknowledge that obesity is a risk factor for total joint replacement. The intent is not to deny patients surgery, but rather to identify high-risk patients so that their treatment can be tailored to ensure the best possible outcomes.

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Author’s reply:

Sir,

We thank Professor Choong et al for their letter and comments in response to our paper.

With regard to the first point about conflicting evidence, we agree in part with their sensible comments regarding effect size. Indeed, many of the studies have small sample sizes, however, most are still large enough to be able to detect clinical effects of a certain magnitude. Together, they would identify any obvious or dangerous compromise requiring immediate attention. One could also argue that if the effect size is so small (requiring only large sample sizes of 1000+) then the clinical relevance of such findings is questionable. Hence, in our eyes at least, the evidence does remain conflicting.

With regard to the second issue over infection, we have rechecked our data and analysis in great detail and have found that the data and analysis for the paper is correct. However, Professor Choong is quite right that a single printed p-value for infection comparison (only) in the text is erroneous and should have read < 0.05. Similarly to his own findings, the obese group had a significantly (statistically but not clinically) greater infection rate.

Whilst this error should be documented for completeness we would suggest that it does little to change the message that obese patients are not compromised compared with non-obese patients. This is for the following reasons:

1. The incidence in all groups is very small (worst case is 1.4%). We agree with Professor Choong that whilst it can be expressed somewhat sensationalistically as a ‘sevenfold’ difference, such comparisons need the appropriate context. A sevenfold increase in a very small value remains a small value. Again, we would suggest that the clinical relevance of a difference of 1% is debatable. The 1.5% is still well within the published infection rates seen in many other studies (0.2% to 3.9%).

2. Interestingly, the morbidly obese group had no deep infection at all. Should obesity and infection be strongly linked then we
would have expected some incidence of deep infection in the morbidly obese group.

3. In all other outcome measures there was no difference between the groups. This has two implications. First, a small elevation in infection rate (in obese patients) appears not to affect outcome. Irrespective of their higher risk of infection, they still gain the same benefit of surgery as non-obese patients. Secondly, the evidence from arguably more meaningful outcome data (OHS, revision, dislocation, etc) shows that, in general, obese patients do equally well in terms of outcome as non-obese patients.

In the paper we are not condoning obesity, we are merely highlighting that in this current economic climate, such patients may not be offered surgery for fear of complications. It seems that this risk is very small to non-existent.

We would like to express our thanks to our colleagues in Melbourne for their keen interest and highly appropriate observations relating to this work.

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