CRITICAL CARE

COMMENTARY

# Separating wheat from chaff: examining the obesity paradox in the critically ill

Rishi Rattan and Stanley A Nasraway Jr*

See related research by Wacharasint *et al.*, http://ccforum.com/content/17/3/R122

**Abstract**

Obesity is an increasing burden globally. In the general population, the obese have an increased mortality risk. Regarding the critically ill, a growing body of literature supports the obesity paradox, the notion that obesity confers a protective effect in certain disease states. However, the paucity of methodologically sound trials prevents definitive interpretation and may obscure risks.

In the previous issue of *Critical Care*, Wacharasint and colleagues' investigation into obesity in septic shock adds to the contradictory studies on the effects of obesity in critical illness [1]. Since 1980, global obesity has nearly doubled, with over 1.4 billion adults overweight or obese, defined by a body mass index (BMI) ≥25 kg/m$^2$ [2]. Obesity is a major risk factor for cardiovascular disease, diabetes, obstructive sleep apnea, and certain malignancies. Yearly, 2.8 million deaths worldwide are attributable to obesity [2].

Up to 40% of ICU patients are obese [3]. Obese ICU patients have higher rates of ventilator-associated pneumonia, deep vein thrombosis, pulmonary embolism, and cardiovascular complications [4]. Early studies found an increased mortality rate in the critically ill obese, particularly in long-stay, morbidly obese (BMI ≥40 kg/m$^2$) subjects [5,6]. However, more recent literature supports the obesity paradox, the notion that obesity confers a protective effect in certain disease states. Obesity in critical illness appears to exert no effect on mortality, or may even decrease mortality, despite longer ICU stay and time to resolve organ failure [7]. In the past two decades, it seems there has been a shift in care and outcomes of the critically ill obese.

*Correspondence: snasraway@tuftsmedicalcenter.org
Department of Surgery, Tufts Medical Center, 860 Washington St, Boston, MA 02111, USA



In Wacharasint and colleagues' cohort of patients in septic shock, the obese had decreased mortality. Surprisingly, this was seen even in the subcohort of morbidly obese patients [1]. Infection profiles differed, with lower rates of lung and fungal infection in the obese. Like other studies, they found that most intensivists still approach adults of varying weights with uniform doses of fluid and vasopressors. Thus, the obese received a lower weight-based dose. The significance of this is still speculative. Excessive fluid resuscitation may cause harm [8]. Perhaps the lower weight-based dose confoundingly leads to a perceived protective effect of obesity.

Sussing out useful information on the critically ill obese remains difficult. It appears that obese patients may respond differently than nonobese patients in critical illness. The basis for this, though, is unknown. Given the contradictory data on inflammatory markers, it is likely that the appropriate complement of markers for study in critical illness remains unidentified. Despite the known difficulties of ventilating obese patients given decreased chest wall compliance, increased gastric reflux, increased ventilator-associated pneumonia, and Wacharasint and colleagues' finding of inappropriately high tidal volumes during mechanical ventilation when compared to their nonobese cohort, the obese still had lower rates of lung infection [1]. These mortality data contradict earlier findings in the critically ill morbidly obese and the use of low tidal volumes in acute lung injury [5,6,9].

How can we explain the decreased mortality in the obese, particularly the morbidly obese, observed in this and other studies [1,3]? We agree that the obese have a different immune response in critical illness. We acknowledge that the Acute Physiology and Chronic Health Evaluation scoring systems overestimate the severity of illness in the obese by incorporating markers that differ in otherwise healthy obese patients, like creatinine and oxygenation.

There are three possible explanations for how the obese, especially morbidly obese, may experience better outcomes in the ICU today as compared to decades past. First, and most likely, this special population has received heightened attention and the consequence has been

improved outcomes [10,11]. The study of pharmacokinetics in the obese has advanced. ICUs have developed better turning, skin, mobilization, oral, and pulmonary toilet protocols. Newer mattresses and beds attenuate pressure sore formation. It is possible our inadvertent 'under' dosing of obese patients' fluids and vasopressors conferred a protective effect. Indirect calorimetry can accurately estimate energy expenditure in the obese. Use of ultrasound when obtaining vascular access has decreased complications. Imaging tables that support the greater weight of morbidly obese patients have opened up modalities and improved the diagnosis of life-threatening illness. Improvements in glycemic control have benefited the obese, who are likelier to have dysglycemia and diabetes. Improved ventilation and extubation protocols have probably improved outcomes in the obese who have higher rates of atelectasis, hypoxemia, and obstructive sleep apnea.

Second, the observation of an obesity paradox in critical illness typically arises from retrospective or *post hoc* analyses or meta-analyses of the same. These studies pose interesting questions but do not prove the existence of an obesity paradox. Even when matching severity of illness in the obese and non-obese, scoring systems do not do justice when comparing a 70 kg patient with a 270 kg patient. Scoring systems have never been validated in the obese. Moreover, one must discriminate between mild obesity and morbid obesity. It may be that outcomes in the mildly obese are no worse than for normal or overweight subjects.

Third, the finding of an obesity paradox in the critically ill, counter to general population outcomes, may be a mirage, an optical phenomenon that does not actually exist. That morbid obesity is 'protective' in critical illness strongly conflicts with bedside observations in the crucible of real life. In the morbidly obese, emergency airway or vascular access can be difficult to secure, chest compressions are less effective, and simply turning a morbidly obese patient may require up to eight bedside providers. Overall, the protective effect of morbid obesity in critical illness does not pass the 'smell test' for those working bedside. Discrepant findings in suboptimal studies underscore our lack of understanding of obesity, inflammation and critical illness, and the need for further research.

**Abbreviations**
BMI, body mass index.

**Competing interests**
The authors have no competing interests relevant to the content herein.

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