Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

Conflict of Interest

None.

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Bhargava V. Devarakonda, MD, DM
Saravana Babu, MD, DM, FTEE
Rupa Sreedhar, MD, PDCC
G.J. Murukendiran, MD, DM
Markose L. Paret, MD, DM
Subin Sukesan, MD, DM
Division of Cardiothoracic and Vascular Anesthesia, Sree Chitra Tirunal Institute for Medical Sciences and Technology, Trivandrum, India

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Clearing Up the Obesity Paradox in Cardiac Surgery

To the Editor:

We read with interest the recent article by Burgos et al. on the presence of the "obesity paradox" in cardiac surgical patients.[1] The authors found that overweight patients experienced lower unadjusted mortality rates and fewer adverse outcomes after cardiac surgery. However, after adjusting for preoperative variables, body mass index (BMI) did not have an independent effect on mortality, which calls the existence of an "obesity paradox" into question. We commend the authors for their excellent work. We wish to delve deeper into the debate surrounding the "obesity paradox" and discuss how one might reconcile this medical hypothesis with the extensive body of literature on atherosclerosis and cardiovascular disease.

First described in 2003,[2] the obesity paradox, or reverse epidemiology principal, states that there are better health outcomes for obese individuals than for normal weight−matched individuals with certain medical conditions, such as diabetes, end-stage renal disease, hypertension, heart failure, coronary artery disease, and peripheral artery disease. There is even some evidence that a higher BMI confers a positive survival value on sudden cardiac arrest, both out-of-hospital and in-hospital cardiac arrest, with shockable rhythms (ventricular fibrillation and pulseless ventricular tachycardia), suggesting that the obesity paradox applies to the post-arrest population.[3] Since its inception, the obesity paradox has generated hundreds of articles and studies on disparate clinical subpopulations.

The obesity paradox is an example of a statistical phenomenon known as bias resulting from observational studies. The mere association of 2 variables does not in and of itself demonstrate causality as the old adage, "correlation is not causation" states. This is particularly true for retrospective studies. Lack of statistical adjustment for cigarette smoking, for example, may lead to erroneous underestimation of the risk conferred by obesity in some early studies[4] because smokers tend to have lower BMIs. The same logic holds true for nursing home residents or terminally ill or cancer patients. It is necessary to account for individual disease severity and frailty. Use of the term "obesity," or use of BMI alone, is insufficient and may be misleading. Obesity should be subdivided into the following categories: class 1 (BMI of 30-34.9 kg/m^2), class 2 (BMI of 35-39.9 kg/m^2), and class 3 (BMI >40 kg/m^2). Studies that cannot parse obese participants into these categories should not assume subgroup homogeneity. Some studies that took a more rigorous view of this problem still appear to validate the obesity paradox.[3] This may be a result of the varying levels of fitness of study participants, a variable difficult to quantify post hoc. Outcome studies that take a rigorous look at the effect of BMI,[5] on the other hand, often do not support the obesity paradox. Perhaps we're looking at obesity the wrong way. Obesity is not a static variable, but consists of several different subpopulations. Obesity class 1 participants frequently may be otherwise healthy, with varying degrees of cardiovascular fitness, whereas obesity class 3 subjects have higher incidence of cardiovascular disease, metabolic syndrome, and obstructive sleep apnea. Frequently used phenotypic indicators of the latter 2 of these conditions are abdominal circumference and waist-to-hip ratio.[6,7] Central obesity correlates better than BMI as a measure of cardiovascular risk.[6,7]

The debate on the obesity paradox is continuing and moving into the subspecialty realm. As anesthesiologists, we're struck that the wrong message is being given to the general public. The obesity paradox is a misnomer.[8] Obesity is not a benign condition, and obese individuals are not homogenous; they are affected by their personal physical fitness and fragility—items all- too- often not included in the routine assessment tools. Nonetheless, obesity, particularly severe obesity, increases cardiovascular, diabetic, cancer, and stroke risks; shortens life expectancy; and has been shown to be associated with worse coronavirus disease 2019 outcomes.[9-13] We believe that the obesity paradox should be revealed for what it is—a statistical bias of a limited subset that is sometimes correct.

Conflict of Interest

None.

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Jon D. Samuels, MD
Briana Lui, BS
Robert S. White, MD, MS
Department of Anesthesiology, Weill Cornell Medicine, New York, NY

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Choosing Sides: Contralateral Tension Pneumothorax after Thoracotomy

To the Editor:

A 73-year-old man underwent right hemi-clamshell incision for right upper lobectomy and mediastinal lymph node dissection for lung adenocarcinoma. After chest closure, before removal of the left-sided double-lumen endotracheal tube, chest x-ray showed a moderate left basilar pneumothorax, with partial collapse of the nonsurgical lung (Fig 1, A). While image interpretation was pending, the patient was breathing spontaneously on pressure-support ventilation mode. He was coughing, causing transient hypotension, so the endotracheal tube was removed. He developed tachypnea, shallow breaths, and oxygen desaturation. After transient improvement with jaw thrust and positive pressure mask ventilation, he developed worsening hypoxia, tachycardia, and hypotension that persisted despite reintubation with a single-lumen endotracheal tube and vasopressor support. The chest tubes on the surgical side were functioning appropriately. Upon review of the prior chest x-ray, it was suspected that the contralateral pneumothorax had developed tension physiology. A left-sided chest tube was placed emergently, with an audible gush of air and immediate, profound improvement in oxygenation, ventilation, and hemodynamics (Fig 1, B).

Intraoperative tension pneumothorax is uncommon. Although the thoracic surgery population is possibly at increased risk for ipsilateral or contralateral pneumothorax as a result of preexisting lung disease, barotrauma during one-lung ventilation, traumatic airway placement or exchange, thoracic epidural placement, or surgical manipulation, tension pneumothorax in the setting of one-lung ventilation also is considered rare.[1] Presentation of tension pneumothorax in patients receiving assisted ventilation includes hypoxia, hypotension, and cardiac arrest and, although most patients develop severe signs within minutes of presentation,[2] diagnosis and treatment can be dangerously delayed if these signs are incorrectly attributed to complications of one-lung ventilation while the double-lumen endotracheal tube is in place, or to a malfunctioning chest tube on the surgical side without consideration of contralateral tension pneumothorax. Anesthesiologists ought to be alert to the possibility of contralateral tension pneumothorax after thoracotomy. Communication between surgical and anesthesiology services is paramount.

Conflict of Interest: The authors declare no competing interests.