In Reply:

We appreciate the opportunity to respond to the issue raised by Ramachandran regarding our article[1] on perioperative outcomes in patients with modified metabolic syndrome (mMetS) who undergo noncardiac surgery. We thank the author for his comment that "[our] findings may indeed change the way physicians … look at obese patients in the future." In his letter, the author raises the issue about whether the current study proves the increased risk of mMetS or simply proves "that the preoperative presence of two independent risk factors (and one protective factor) is more significant than having one protective factor?"

Our study was designed to better understand the obesity paradox, the apparent protective effect of obesity on surgical mortality,[2] by distinguishing patients who were obese but "metabolically healthy" from patients with MetS.[3] The major new findings of our study were that patients with mMetS undergoing noncardiac surgery were at higher risk for cardiac, pulmonary, renal, and central nervous system complications.[1] Unlike the obesity paradox observed for mortality,[4] our study did not detect any evidence of a "protective effect" of obesity for these complications. Our analysis does not indicate whether the increased risk associated with the mMetS is due simply to the additive effects of diabetes, hypertension, and obesity, which together make up the modified metabolic syndrome (mMetS). In other words, we have not answered the question of whether the whole is greater than the sum of the parts. However, whether our findings represent, in a statistical sense, an additive effect or an interaction effect is less important than the simple recognition that mMetS is associated with a significantly higher risk of major postoperative complications. In particular, patients with mMetS have a 2- to 3-fold increased risk of cardiac complications and a 3- to 7-fold increased risk of renal complications. These findings are especially striking in light of the previous literature demonstrating an apparently protective effect of obesity on mortality during the perioperative period. From the standpoint of regression modeling, the increased risk associated with mMetS may boil down to the question of "simple math or aberrant physiology." However, in clinical practice, the recognition that patients with mMetS are at a much higher risk of cardiac, pulmonary, and renal complications has both biologic plausibility and important implications for the management of these patients.

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Supported by grant RO1 HS 16737 from the Agency for Healthcare and Quality Research and the Department of Anesthesiology, University of Rochester. The sponsors of this study had no role in the conduct of the study; in the analysis or the interpretation of the data; or in the preparation, review, or approval of the article. The views presented herein are those of the authors and may not reflect those of the Agency for Healthcare and Quality Research.

References

1. Glance LG, Wissler R, Mukamel DB, Li Y, Diachun CA, Salloum R, Fleming FJ, Dick AW: Perioperative outcomes among patients with the modified metabolic syndrome who are undergoing noncardiac surgery. ANESTHESIOLOGY 2010; 113:859–72
2. Dindo D, Muller MK, Weber M, Clavien PA: Obesity in general elective surgery. Lancet 2003; 361:2032–5
3. Neligan PJ, Fleisher LA: Obesity and diabetes: Evidence of increased perioperative risk? ANESTHESIOLOGY 2006; 104:398–400
4. Mullen JT, Moorman DW, Davenport DL: The obesity paradox: Body mass index and outcomes in patients undergoing nonbariatric general surgery. Ann Surg 2009; 250:166–72

(Accepted for publication January 25, 2011.)

Intraoperative Use of an Automated Chest Compression Device

To the Editor:

Cave *et al.*[1] recently reviewed the many techniques available to support circulation during cardiopulmonary resuscitation (CPR): active compression-decompression CPR; phased thoracic-abdominal compression-decompression CPR with a handheld device, impedance threshold device, or mechanical piston device; and load-distributing band CPR or vest CPR. Among all the commercially available devices globally, two devices are currently available in France: LUCAS (Lund University Cardiopulmonary Assist System; Jolife AB, Lund, Sweden), which is a gas- or electric-powered piston device that produces a consistent chest compression rate and depth,[2] and the automated LifeBand® (AutoPulse; ZOLL Medical Corporation, Chelmsford, MA), which is a load-distributing, broad compression band that is applied across the entire anterior chest.[3] These devices can be placed rapidly and used easily. In addition, they do not require extra staff to perform resuscitation.[4] However, the only randomized study published concerning the use of a chest-compression device showed decreased survival.[3] Furthermore, a recent meta-analysis concluded that there is insufficient evidence from human randomized controlled trials to conclude that mechanical chest compression during CPR for cardiac arrest is associated with either benefit or harm.[5] Current American Heart Association[6] guidelines for CPR do not recommend its immediate application in the case of cardiac arrest. Equivalent French guidelines recommend use of this device only in the case of refractory cardiac arrest.[7] These devices are only intended to be used by properly trained personnel[1]—not by a witness of a cardiac arrest, either in or out of the hospital setting, contrary to recommendations for the use of semiautomatic defibrillators. We report here the intraoperative use of a device providing consistent external mechanical cardiac compression for a patient with hypovolemic cardiac arrest.

Support was provided solely from institutional and/or departmental sources.

The decision to apply this device was the result of limited human resources.

A 66-yr-old man with a history of paroxystic atrial fibrillation treated by β-blocker and flecainide acetate was admitted to the emergency department for thoracic pain associated with bowel disturbance for 2 days. He had refused to undergo a colectomy on the left side for colic diverticulosis 1 month before.

On arrival, the patient's heart rate was 83 beats/min; blood pressure, 77/61 mmHg; respiratory rate, 18 breaths/min; hemoglobin, 7.6 g/L^{-1}. He promptly developed respiratory failure, with 80% oxygen saturation. He was treated with continuous intravenous epinephrine, volume loading, and tracheal intubation. A femoral artery catheter was inserted to monitor blood pressure.

Abdominal computed tomography scanning was conducted to identify the source of abdominal bleeding. Computed tomography revealed a hemoperitoneum that was the result of splenic rupture.

Cardiac arrest occurred during patient transfer to the operating room. External manual compression was started immediately. The chest compression device was quickly placed and activated, allowing hospital staff to pursue resuscitation and initiate surgery. Automated chest compressions lasted 40 min. During this time, the patient was in asystole when the device was periodically stopped for a few seconds. The no-flow period (*i.e.*, duration without chest compression) was 0 min; the low-flow period (*i.e.*, duration with chest compression but without spontaneous cardiac activity) was 40 min. During this time, end-tidal carbon dioxide stayed near 15 mmHg. Systolic arterial blood pressure was 70–90 mmHg. Bispectral index monitoring was not possible concurrent with the use of the chest compression device; the quality of signal being low likely as a result of interference.

The patient received 3 L cell-saving recuperation, six packs of red blood cells, four fresh frozen plasma units, 2 g $CaCl_2$, and 250 ml HCO_{3-}, 8.4%, as well as several bolus doses of epinephrine added to continuous infusion. The surgeon performed a splenectomy by a bilateral subcostal incision while the chest compression device was in use. After 40 min, the patient recovered a spontaneous rhythm and the chest compression device was stopped. The epinephrine infusion dose was progressively decreased until it was discontinued after 35 min, when arterial blood pressure reached 110/80 mmHg. Bispectral index was 0 when the chest compression device was stopped; however, this measure increased to 28 within 15 min. Anesthesia was then started with sufentanil and sevoflurane.

The patient was then transferred to the intensive care unit. He was cooled to 34°C for the first 24 h. When sedative drugs were stopped, the patient woke with no neurologic deficit. He was extubated the next day. Pathology results showed a spontaneous spleen rupture from lymphomatic proliferation related to acute lymphoplasmacytic splenic lymphoma. The patient was discharged on postoperative day 16 with a Glasgow Outcome Scale score of 1 (*i.e.*, no neurologic deficit).

The current report describes a case of successful intraoperative resuscitation using an automated external cardiac compression device. Two studies have reported that CPR quality during out-of-hospital[8] or in-hospital[9] cardiac arrest did not meet guideline recommendations. Wik *et al.*[8] reported that adequate CPR was not delivered half of the time. Abella *et al.*[9] calculated that no-flow occurred during 24% of the first 5 min of CPR. One of the explanations for this poor performance is provider fatigue after manual chest compressions lasting a few minutes.[10] Another element that may explain the poor performance of manual CPR is limited staffing resources, as was true in our case (*i.e.*, the incident occurred outside working hours). One anesthesiologist (V.D-N.) and one nurse anesthetist were present during the incident described. The automated chest compression device allowed these two hospital staff members to conduct blood verification, set up the cell-saver device, take blood samples, and manage anesthesia and vasoactive drugs. Despite body movement, the surgeon could perform the surgery without difficulty, as has already been described[11] when the device is used in the catheterization laboratory—particularly during percutaneous coronary intervention, reducing the risk of injury by an instrument.

In conclusion, in some settings (*e.g.*, limited human resources), an automated device for mechanical chest compression can represent an extra and particularly useful "pair of hands," without interfering with surgical procedures.

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References

1. Cave DM, Gazmuri RJ, Otto CW, Nadkarni VM, Cheng A, Brooks SC, Daya M, Sutton RM, Branson R, Hazinski MF: Part 7: CPR techniques and devices: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Circulation 2010; 122: S720–8
2. Wirth S, Körner M, Treitl M, Linsenmaier U, Leidel BA, Jaschkowitz T, Reiser MF, Kanz KG: Computed tomography during cardiopulmonary resuscitation using automated chest compression devices—an initial study. Eur Radiol 2009; 19: 1857–66
3. Hallstrom A, Rea TD, Sayre MR, Christenson J, Anton AR, Mosesso VN Jr, Van Ottingham L, Olsufka M, Pennington S, White LJ, Yahn S, Husar J, Morris MF, Cobb LA: Manual chest compression *vs* use of an automated chest compression device during resuscitation following out-of-hospital cardiac arrest: A randomized trial. JAMA 2006; 295:2620–8
4. Lapostolle F, Agostinucci JM, Bertrand P, Catineau J, Chassery C, Kessler M, Galinski M, Adnet F: Use of an automated device for external chest compressions by first-aid workers unfamiliar with the device: A step toward public access? Acad Emerg Med 2009; 16:1374–7
5. Brooks SC, Bigham BL, Morrison LJ: Mechanical *versus* manual chest compressions for cardiac arrest. Cochrane Database Syst Rev 2011; 1:CD007260
6. Field JM, Hazinski MF, Sayre MR, Chameides L, Schexnayder SM, Hemphill R, Samson RA, Kattwinkel J, Berg RA, Bhanji F,

Cave DM, Jauch EC, Kudenchuk PJ, Neumar RW, Peberdy MA, Perlman JM, Sinz E, Travers AH, Berg MD, Billi JE, Eigel B, Hickey RW, Kleinman ME, Link MS, Morrison LJ, O'Connor RE, Shuster M, Callaway CW, Cucchiara B, Ferguson JD, Rea TD, Vanden Hoek TL: Part 1: Executive summary: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Circulation 2010; 122:S640–56

7. Conseil français de réanimation cardiopulmonaire, Société française d'anesthésie et de réanimation, Société française de cardiologie, Société française de chirurgie thoracique et cardiovasculaire, Société française de médecine d'urgence, Société française de pédiatrie, Groupe francophone de réanimation et d'urgence pédiatriques, Société française de perfusion, Société de réanimation de langue française. Guidelines for indications for the use of extracorporeal life support in refractory cardiac arrest. French Ministry of Health. Ann Fr Anesth Reanim 2009; 28:182–90
8. Wik L, Kramer-Johansen J, Myklebust H, Sørebø H, Svensson L, Fellows B, Steen PA: Quality of cardiopulmonary resuscitation during out-of-hospital cardiac arrest. JAMA 2005; 293: 299–304
9. Abella BS, Alvarado JP, Myklebust H, Edelson DP, Barry A, O'Hearn N, Vanden Hoek TL, Becker LB: Quality of cardiopulmonary resuscitation during in-hospital cardiac arrest. JAMA 2005; 293:305–10
10. Hightower D, Thomas SH, Stone CK, Dunn K, March JA: Decay in quality of closed-chest compressions over time. Ann Emerg Med 1995; 26:300–3
11. Wagner H, Terkelsen CJ, Friberg H, Harnek J, Kern K, Lassen JF, Olivecrona GK: Cardiac arrest in the catheterisation laboratory: A 5-year experience of using mechanical chest compressions to facilitate PCI during prolonged resuscitation efforts. Resuscitation 2010; 81:383–7

(Accepted for publication February 9, 2011.)