

was confirmed before induction of anesthesia, the inability to determine BP intraoperatively should prompt clinicians to rapidly employ a different modality that ensures reliable BP determination so that large gaps devoid of BP readings do not occur during an anesthetic. Finally, given the frequent difficulty reported by Mathis *et al.* in determining BP intraoperatively in the majority of anesthetics, perhaps unrecognized and untreated hypotension could have also contributed to the primary outcome of acute kidney injury.

Competing Interests

The authors declare no competing interests.

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In Reply:

We thank Drs. Barbara and Freundlich *et al.* for their thoughtful responses to our recent article.¹ In their responses, they highlight critical points regarding the management of patients with left ventricular assist devices (LVADs) presenting for noncardiac surgery. These points include: (1) LVAD patients are at high risk for perioperative complications; (2) frequency of invasive arterial line monitoring continues to decrease, despite high rates of intraoperative monitoring gaps; (3) potential hemodynamic instability in the setting of inadequate blood pressure monitoring may lead to increased incidence of complications (including acute kidney injury); and, as such, (4) alternatives to automated noninvasive cuff measurements for blood pressure monitoring must be more aggressively pursued.

We agree with Drs. Barbara and Freundlich that the LVAD population is, by definition, high risk and that a decreasing frequency of arterial line monitoring observed over our study period is not justified by the high rate of

blood pressure monitoring gaps also observed. We support arterial line placement for major procedures requiring general anesthesia in this population; however, we highlight alternatives to routine arterial line use for minor procedures with sedation, as alluded to in Dr. Barbara's and Dr. Freundlich's responses.

With the increased prevalence of LVAD patients presenting for noncardiac procedures, rapidly growing demands are placed on limited anesthesiology department resources. Arterial line placement can occasionally be a technically challenging, time-consuming task in the LVAD patient, often requiring ultrasound guidance in the setting of nearly nonpulsatile blood flow. While we do not discourage such attempts, we *strongly* encourage anesthesiologists to seek access to—and develop a familiarity with—other means of blood pressure monitoring, most notably a Doppler cuff. In the LVAD population, Doppler measurements demonstrate success rates of 91 to 100%, a vast improvement upon automated cuff measurements (50 to 63%).^{2–4} As a result of these findings, we have developed a staff education program at our institution to improve departmental awareness and access to Doppler devices for the specific purpose of LVAD patient monitoring; we support efforts to do the same among institutions caring for LVAD patients.

With regards to the context of arterial line usage and monitoring gaps observed, we acknowledge limitations of the retrospective nature of our study. Anesthesiologist justification for arterial line use, whether planned or unplanned, was unavailable for study. In most instances of gaps in monitoring, gaps occurred after induction of anesthesia; in such cases, we can speculate that the monitoring gap may have been associated with an automated cuff failure in the setting of decreased preload or afterload and diminished pulsatility. Beyond seeking a means of improved blood pressure monitoring, it has been our experience in caring for LVAD patients that efforts to maintain pulsatility—including judicious fluid boluses and vasopressor administration concurrent with gentle induction of anesthesia—can often successfully maintain automated cuff monitoring capability and prevent unrecognized hypotension.

In addition to a familiarity with blood pressure monitoring in the LVAD population, we encourage *all* anesthesiologists to become familiar with basic settings for continuous-flow LVADs, including pump flow, speed, power, and pulsatility index.⁵ Although we describe an association between intraoperative hypotension and acute kidney injury in our study, a correlation between LVAD pump flows and outcomes remains understudied. Pump flow generated by a specific pump speed may be a sensitive indicator of the balance between preload and afterload and may be a useful aid in patient management. Finally, no hemodynamic parameters monitored should serve to replace an understanding of the pathophysiology of the preload-dependent, afterload-sensitive LVAD patient; such an understanding remains equally important in clinical decision making.

In conclusion, we thank Drs. Barbara and Freundlich *et al.* for their valuable feedback regarding our study. Although

the optimal means for hemodynamic monitoring in the LVAD patient is yet to be fully elucidated, it is clear that this population poses a challenge to American Society of Anesthesiologists Standards for Basic Anesthetic Monitoring.⁶ Given the potential for increased postoperative complications with inadequate monitoring, our study represents a call to action for consensus guidelines addressing anesthetic monitoring specific to this increasingly common, high-risk population.

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Competing Interests

The authors declare no competing interests. The University of Michigan performs contract research with St. Jude, Pleasanton, California, and HeartWare, Framingham, Massachusetts.

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A Deeper Look at Anesthesia Depth

To the Editor:

The editorial by Garcia and Sleight¹ provided an outstanding discussion of ketamine's complexities. Their conclusion, that we use a flawed concept of anesthesia depth, was insightful and provides a reason as well as an opportunity to suggest something more meaningful.

Anesthetic “depth” is one of our profession's oldest and most used metaphors. As a metaphor, greater depth has long held connotations of an increased anesthetic dose and a traditionally strong connection to our observations of deep sleep.

In the past, this did not pose a particular problem, but now it does. Connecting greater depth to deeper sleep tends to push our thinking toward a unitary concept of anesthetic action, even though the unitary concept has been discredited. In this way, our most common metaphor actually hampers our using more appropriate concepts of anesthetic actions and interactions.

But shifting the depth connection away from sleep and toward anatomy can resolve this problem. The key to this is that increased depth is synonymous with higher minimum alveolar concentration (MAC) values. When these MAC values are aligned with the relevant neuroanatomy, a connection between depth and anatomy arises that is far more functional than the connection between depth and sleep.

To see this, consider some specific anesthetic effects associated with specific MAC values. Loss of movement results from the equipotent dose of the γ -aminobutyric acid-enhancing anesthetics known as 1 MAC. Loss of consciousness occurs at approximately 0.3 MAC (MAC-Awake)² and 1.3 MAC produces suppression of the sympathetic nervous system (MAC-BAR).³

These three functions—consciousness, movement, and sympathetic suppression—can be attributed to three more or less distinct regions of the nervous system. Consciousness is associated with the cerebral cortex, movement goes with the spinal cord, and a significant component of sympathetic suppression occurs outward from the spinal cord.^{4,5} When you match these locations to the appropriate MAC values, you find that an increasing anesthetic dose, or increasing depth, produces effects first in the cortex (0.3 MAC), then down the spinal cord (1 MAC), and then finally further out toward the periphery (1.3 MAC).

This gives the metaphor of depth an actual, if coincidental, anatomic correlation. Increased doses of anesthetic produce effects first in the “uppermost” region of the nervous system, then further “down,” and finally further “out.” In other words, “depth” is a descent down the nervous system as anesthetic dosage increases, and an ascent back up as the dosage level is reversed.

This makes increasing anesthetic depth a metaphor for anesthetic affect on an increasingly larger number of neural

This letter was sent to the author of the original article referenced above, who declined to respond.—Evan D. Kharasch, M.D., Ph.D., Editor-in-Chief.