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Intraoperative Hypotension and Patient Outcome:

Does "One Size Fit All?"

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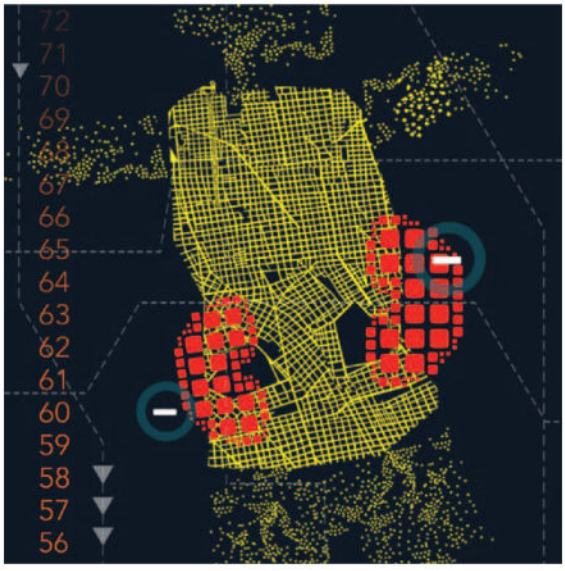
Patients who are anesthetized or heavily sedated surrender their ability to convey signs and symptoms of low blood pressure, particularly those associated with cerebral hypoperfusion (*i.e.*, light headedness, mental status changes, or syncope). Consequently, physicians have come to rely on empiric definitions of what constitutes the lowest tolerable blood pressure during surgery or, stated differently, the definition of intraoperative hypotension. There remains debate, although, on what value of blood pressure in relation to preoperative baseline should be considered as hypotension with much variability in definitions between investigations.[1,2] The need for a precise definition of intraoperative hypotension is supported by observational studies in adults that have linked low blood pressure with adverse patient outcomes after cardiac and noncardiac surgery, including 30-day and 1-yr mortality.[3–8] In this issue of Anesthesiology, Walsh *et al.*[9] confirm and extend these growing data when they report that mean arterial pressure (MAP) less than 55 mmHg during noncardiac surgery is associated with risk for postoperative acute kidney injury (AKI) or myocardial infarction (MI).



In their study, Walsh *et al.*[9] analyzed prospectively collected data obtained from the electronic medical records of 33,330 patients who underwent noncardiac surgery at the

Cleveland Clinic (Cleveland, Ohio). They have assessed the association between MAP less than 55–75 mmHg and post-operative AKI (defined as increases in serum creatinine of greater than 1.5-fold or 0.3 mg/dl from baseline) or MI (defined as serum troponin T ≥0.04 μg/l or creatinine kinase-MB ≥8.8 ng/ml). Of note, patients with chronic kidney disease and those who underwent urologic surgery, nephrectomy, or renal transplantation were excluded because they did not have postoperative creatinine measurements. Serum myocardial injury biomarkers were selectively measured only in high-risk patients and those with clinical evidence of myocardial ischemia. Patients without myocardial injury biomarker data were assumed not to have suffered an MI. Blood pressure was measured noninvasively every 2–5 min in most patients, but 44.5% of patients had invasive arterial pressure monitoring every 1–2 min. A MAP threshold of less than 55 mmHg was found to be associated with risk for AKI and MI, events that occurred in 7.4 and 2.3% of patients, respectively. They further report an incremental exposure–risk relationship whereby increased duration of MAP less than 55 mmHg (1–5, 6–10, 11–20, and >20 min) increased the risk for AKI and MI. Moreover, 30-day mortality was significantly associated with more than 20 min of MAP of less than 55 mmHg.

The current study by Walsh *et al.*[9] and data from others draw important attention to the fact that blood pressure management during surgery might be a factor that can be modified as a means for improving patient outcomes.[3–8] As questioned in the title of the article by Walsh *et al.*,[9] are physicians now able to derive an empiric definition of intraoperative hypotension as a MAP less than 55 mmHg for adult patients undergoing noncardiac surgery? The study has many strengths, including the large number of patients, which allows for careful risk adjustments. As with any such analysis, however, it is difficult to account for all variables or residual confounders that might affect the results. The authors acknowledge and attempt to address many of these factors, including potential bias by their exclusion of patients without postoperative serum creatinine data and MI biomarker data. One source of bias that was not directly addressed was whether patients who had surgery of longer duration might have had more blood pressure measurements and a higher risk for hypotension than those whose surgery was of shorter duration. Additionally, bias might occur for patients who received direct arterial blood pressure measurement because they had more blood pressure measurements than did those whose blood pressure was measured noninvasively. Patients in whom direct arterial blood pressure monitoring was performed likely had higher comorbidity and/or more complex surgery. Furthermore, rather than being the proximate cause of AKI and MI, might intraoperative hypotension be a marker for some unmeasured characteristic of patients who are also prone to AKI and MI?

An important consideration in interpreting the results reported by Walsh *et al.*[9] is that adverse cerebral outcomes were not evaluated. Classically, it is believed that cerebral perfusion is more dependent on MAP, whereas cardiac perfusion is more dependent on diastolic blood pressure, and renal perfusion is dependent on both MAP and cardiac output. That is, the kidney can be hypoperfused at normal MAP if cardiac output is compromised, even while cerebral and cardiac perfusion is maintained.[10,11] Therefore, the historic rationale of choosing 50 mmHg as a goal for MAP has been to preserve cerebral perfusion, specifically citing the autoregulatory limit of 50 mmHg published by Lassen in 1959.[12] Although 50 mmHg is descriptive of the lower limit of cerebrovascular pressure

autoregulation in a large number of patients, the applicability of such a limit to *all* patients is frequently questioned.[13] Indeed, our work in patients undergoing cardiac surgery with cardiopulmonary bypass has revealed the startling finding that the lower limit of cerebral blood flow autoregulation varies widely between individuals and ranges from 40 to 90 mmHg.[14] These limits are difficult to predict based on clinical variables, including preoperative blood pressure. Importantly, we have found that regional cerebral oxygen saturation derived from noninvasive near-infrared spectroscopy serves as a suitable surrogate for cerebral blood flow autoregulation monitoring. [15,16] This method involves monitoring of the correlation coefficient between cerebral oxygen saturation and MAP in the low frequencies associated with autoregulation vasoreactivity and provides a continuous measure of auto-regulation at the bedside. Although much work is required before the use of such monitoring can become widespread, these methods will enable physicians to individualize blood pressure of patients to maintain MAP in the autoregulation range. Of relevance to the study by Walsh *et al.*,[9] we have found that the magnitude and duration of blood pressure below the limits of cerebral blood flow autoregulation measured with cerebral oximetry independently predict AKI.[17] One is tempted to conclude from this that a MAP threshold that allows for cerebrovascular autoregulation will also allow for renovascular perfusion. However, we have seen in animal models that decrements in cardiac output can ablate renovascular reactivity and result in large decreases of renal blood flow, *even at normal arterial pressure, when cerebral blood flow is uncompromised.*[10] On the basis of these findings, one would predict that the lower limit of cerebrovascular autoregulation is specific for compromise of renal perfusion but is not sensitive in low-output states. Notably, during cardiopulmonary bypass, systemic flow is controlled such that MAP is an important variable for ensuring organ perfusion.

The combined data to date suggest that hypotension during surgery may be associated with poor patient outcome even up to 1 yr after surgery. Hence, careful management of blood pressure may lead to improved patient outcomes. However, it remains unknown whether it is untreated intraoperative hypotension or the treatment of such hypotension with IV fluids, vasoconstrictive drugs, or inotropes that contributes to the observed adverse outcomes in these studies. We are currently conducting a randomized clinical trial to compare neurologic outcomes of patients whose MAP targets during cardiopulmonary bypass are based on real-time autoregulation monitoring to outcomes of patients who receive standard of care (trial registration www.clinicaltrials.gov: NCT00981474). Such studies in noncardiac surgical patients are needed to determine whether early treatment or prevention of adverse intraoperative events leads to improved patient outcomes. Regardless, we believe that the combined data suggest that a single blood pressure target derived from group summary data cannot be extrapolated to be optimal for all patients, or, “one size does not fit all.”

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