PATIENTS who are anesthe-
tized or heavily sedated 
surrender their ability to convey 
signs and symptoms of low blood 
pressure, particularly those associ-
ated with cerebral hypoperfusion 
(i.e., light headedness, mental 
status changes, or syncope). Con-
sequently, physicians have come 
to rely on empiric definitions of 
what constitutes the lowest toler-
able blood pressure during surgery 
or, stated differently, the defini-
tion of intraoperative hypotension.

There remains debate, although, 
on what value of blood pressure 
in relation to preoperative baseline 
should be considered as hypoten-
sion with much variability in defi-
nitions between investigations.1,2 
The need for a precise definition of 
intraoperative hypotension is 
supported by observational stud-
ies in adults that have linked low 
blood pressure with adverse patient 
outcomes after cardiac and noncar-
diac surgery, including 30-day and 
1-yr mortality.3–8 In this issue of 
ANESThESIOLOGY, Walsh et al.9 con-
firm 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 col-
lected 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 
creatine kinase-MB ≥28.8 ng/ml). Of 
ote, patients with chronic kidney 
disease and those who underwent 
urologic surgery, nephrectomy, or 
renal transplantation were excluded 
because they did not have postop-
erative creatinine measurements. 
Serum myocardial injury biomark-
ers were selectively measured only 
in high-risk patients and those 
with clinical evidence of myocar-
dial ischemia. Patients without 
myocardial injury biomarker data 
were assumed not to have suffered 
an MI. Blood pressure was mea-
sured noninvasively every 2–5 min 
in most patients, but 44.5% of 
patients had invasive arterial pres-
sure 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 oth-
ers 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.1–8 
As questioned in the title of the article by Walsh et al.9 
are physicians now able to derive an empiric definition of 

Illustration: A. Johnson.

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◆ This Editorial View accompanies the following article: Walsh M, Devereaux PJ, Garg AX, Kurz A, Turan A, Rodseth RN, Cywinski J, Thabane L, Sessler DI: Relationship between in-
traoperative mean arterial pressure and clinical outcomes after 
noncardiac surgery: Toward an empirical definition of hypoten-
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. 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. 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. 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. 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. 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. 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 autoregulation 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., 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. 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. 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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EDITORIAL VIEWS


