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Anesthesiology 1998; 88:291 - 2 © 1998 American Society of Anesthesiologists, Inc. Lippincott-Raven Publishers

Using the Skin Vasomotor Reflex to Access Autonomic Reactivity to Laryngoscopy and Intubation

DISCOVERING a measure of the "depth of anesthesia" has been a holy grail for many in anesthesiology. "Depth of anesthesia" means different things to different people, but one goal of this search has been to find methods for predicting the hemodynamic responses to anesthetic drugs and to intraoperative stimuli. A practically applicable method of evaluating sympathetic tone or responsiveness would be of considerable clinical utility.

In this issue of ANESTHESIOLOGY, Shimoda *et al.*¹ report on the skin vasomotor reflex (SVmR) as a method to assess autonomic reactivity during a sevoflurane anesthetic. They used a laser Doppler flowmeter to make repeated measures of the transient decrease in skin blood flow to a finger, which occurs after brief, painful, percutaneous electrical (tetanic) stimulation of the ipsilateral ulnar nerve. At a constant end-tidal concentration of sevoflurane, the magnitude of the cutaneous flow reduction after each stimulus was reasonably consistent. In all patients, the concentration of sevoflurane was gradually increased while the SVmR was assessed repeatedly.

In the "control group," laryngoscopy (a clinically relevant stimulus) was performed at the discretion of the anesthesiologist without reference to the SVmR, and the subsequent changes in heart rate and blood pressure were noted. In the "monitored group," intubation was performed only after the amplitude of the evoked reduction in skin blood flow had decreased to <10% of that seen just after thiopental induction. This occurred when the end-tidal sevoflurane concentration was $\approx 2.7\%$. In the control group, systolic blood pressure increased from 107 to 145 mmHg with endotracheal

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This Editorial View accompanies the following article: Shimoda O, Ikuta Y, Sakamoto M, Terasaki H: Skin vasomotor reflex predicts circulatory responses to laryngoscopy and intubation. Anesthesiology 1998; 88:297-304.

Accepted for publication October 31, 1997.

Key words: Anesthesia; blood flow; monitor; sevoflurane; vasomotor reflex.

intubation, whereas heart rate increased from 87 to 105 beat/min. By contrast, in the monitored group, blood pressure did not change (although heart rate increased). Further evaluation showed that the pressor response to intubation was correlated with the SVmR amplitude; *i.e.*, the lower the elicited SVmR amplitude, the smaller the blood pressure increase. The authors conclude that the SVmR is a useful measure of autonomic responsivity and that the hemodynamic response to laryngoscopy and intubation can be prevented if sevoflurane concentrations are increased until the SVmR is minimized.

What does the SVmR really represent? First, it may only reflect a local neural discharge (i.e., direct stimulation of nerves fibers to the hand). However, the response is probably more general than this. Experience in my laboratory has shown that in awake volunteers, painful electrical stimulation of C fibers in the median nerve of one arm will elicit cutaneous vasoconstriction in the contralateral arm. If stimulus intensity is decreased to non-painful levels, the contralateral response is minimal, suggesting that nociception is an important component. Inspection of the figures in Shimoda's article shows transient, though small, pressor responses after each stimulus. It is therefore likely that the ipsilateral SVmR is simply one component of a widespread response; simultaneous bilateral measurements of SVmR (during the application of a unilateral stimulus) would have been extremely interesting.

Shimoda *et al.* should be congratulated for their efforts in evaluating an easily applied technique to assess autonomic responses during anesthesia. This technique potentially represents a standardizable, repeatable nociceptive stimulus that elicits a quantifiable vascular response to stimulation of sympathetic C fibers. It may be extremely useful for continuous monitoring of the influence of anesthetics on sympathetic tone and reactivity. Unfortunately, the response of some clinicians to a first reading of this article may be "Interesting, but so what?" After all, large pressor responses to intubation are common and, although the subject of great discussion, are not obviously deleterious, at least in most situations. They can also be reliably attenuated by simply increasing volatile agent concentrations or by

supplementing the anesthetic with opioids, barbiturates, or lidocaine. Finally, who needs another monitor in the operating room? As will be discussed, it could be also be useful on other levels.

No one yet knows the answer to these questions, nor the practical value of the described method. For example, it is not yet known how well the measurements described by Shimoda et al. will work with other anesthetics. It is also not known how the method will work in high-risk patient groups in whom uncontrolled hemodynamic events are most common and most likely to be most detrimental. For example, even mildly hypertensive humans have elevated sympathetic neural tone² and are thought to have exaggerated responses to sympathoexcitatory stimuli. Can patients at risk for exaggerated responses be identified by their SVmR? Conversely, the method might be used to identify patients with autonomic dysfunction. For example, diabetic patients³ have reduced autonomic tone and maximal response to noxious stimuli. Diabetics with disturbed autonomic function are also more prone to hypotension after anesthetic induction.⁴ Does decreased basal tone (i.e., a reduced baseline SVmR amplitude) predict reduced responses to noxious intraoperative stimuli, or might it predict greater degrees of postinduction hypotension? Can assessment of SVmR permit the detection of such individuals? At present, no one knows.

The technique in Shimoda *et al.* may parallel and supplement the pioneering work of Dr. Thomas Ebert *et al.*, who have used a more invasive and sophisticated technique (microneurography) for evaluating autonomic responses to anesthetics. They have shown that propofol-induced hypotension is a result of sympathoin-hibition.⁵ Further, again using microneurography, they have reported that desflurane causes a 250% increase

in sympathetic nerve activity. Dr. Ebert's work shows clearly the value of assessing autonomic effects of anesthetics. Thus, although Shimoda *et al.* may provide important clinical information, their introduction of a technique that could provide information on how anesthetics affect autonomic responses to intraoperative stimuli may be of even greater value. There is little doubt that the application of this and related methods in anesthesia and in critical care medicine will improve our understanding of basic anesthetic mechanisms and, hopefully, how best to treat patients.

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References

- 1. Shimoda O, Ikuta Y, Sakamoto M, Terasaki H: Skin vasomotor reflex predicts circulatory response to laryngoscopy and intubation. Anesthesiology 1998; 88:297 304
- 2. Anderson EA, Sinkey CA, Lawton WJ, Mark AL: Elevated sympathetic nerve activity in borderline hypertensive humans. Hypertension 1989; 14:177-83
- 3. Hoffman RP, Sinkey CA, Kienzle MG, Anderson EA: Muscle sympathetic nerve activity is reduced in IDDM before overt autonomic neuropathy. Diabetes 1993; 42:375–80
- 4. Latson TW, Ashmore TH, Reinhart DJ, Klein KW, Giesecke, AH: Autonomic reflex dysfunction in patients presenting for elective surgery is associated with hypotension after anesthesia induction. Anesthesiology 1994; 80:326-37
- 5. Ebert TJ, Muzi M, Berens R, Goff D, Kampine JP: Sympathetic responses to induction of anesthesia in humans with propofol or etomidate. Anesthesiology 1992; 76:725-33
- 6. Ebert TJ, Muzi M: Sympathetic hyperactivity during desflurane anesthesia in healthy volunteers. Anesthesiology 1993; 79:444-53