CORRESPONDENCE

are a result of the method of measuring sympathetic activity and its interpretation.

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References

1. Magnúsdóttir H, Kirno K, Ricksten S-E, Elam M: High thoracic epidural anesthesia does not inhibit sympathetic nerve activity in the lower extremities. ANESTHESIOLOGY 1999; 91:1299-304

2. Hopf H-B, Weibbach B, Peters J: High thoracic segmental epidural

Anesthesiology 2000; 93:302-3 © 2000 American Society of Anesthesiologists, Inc. Lippincott Williams & Wilkins, Inc.

In Reply:—We appreciate the interest and comments expressed by Drs. Introna, Blair, Martin, and Yodlowski. We fully agree that indirect methods of measuring sympathetic nerve activity can provide useful, qualitative information. Quantification of sympathetic nerve activity, however, is difficult with these techniques.

The main issue raised by Introna *et al.* is whether a high thoracic epidural anesthesia (TEA) completely inhibits cardiac sympathetic activity. Based on studies measuring heart rate variability, these authors are convinced that TEA with T1-T5 sensory blockade does not result in complete cardiac sympathetic blockade. However, a comparative study of heart rate variability, cardiac norepinephrine spillover, and muscle sympathetic nerve activity in humans by Kingwell *et al.*¹ invites some caution because it showed heart rate variability to be dependent on multiple factors in addition to cardiac sympathetic nerve activity and not directly related to cardiac norepinephrine spillover.

The degree of thoracic sympathetic blockade was not specifically addressed in our recent study,² which was primarily aimed at evaluating sympathetic function caudal to the TEA-induced sensory blockade and showed no sign of sympathetic blockade. However, previous microneurographic studies of lumbar epidural and spinal anesthesia have shown a fairly close relation between the extent of sensory and sympathetic blockade.^{3,4} Because the nerves to internal organs are not accessible to microneurographic recording in humans, we previously used biochemical measurements of nerve transmitter release to quantify cardiac sympathetic nerve activity.⁵ We used an isotope dilution technique with radiolabeled norepinephrine to demonstrate that TEA prevented the sympathetically mediated surgical stress response during coronary artery bypass surgery. Although this finding could be explained by an afferent nociceptive blockade or an efferent blockade

anesthesia diminishes sympathetic outflow to the legs, despite restriction of sensory blockade to the upper thorax. ANESTHESIOLOGY 1990; 73:882-9

3. Introna R, Yodlowski E, Pruett J, Montano N, Porta A, Crumrine R: Sympathovagal effects of spinal anesthesia assessed by heart rate variability analysis. Anesth Analg 1995; 80:315-21

4. Hopf H-B, Skyschally A, Heusch G, Peters J: Low-frequency spectral power of heart rate variability is not a specific marker of cardiac sympathetic modulation. ANESTHESIOLOGY 1995; 82:609–19

5. Introna RPS, Montano N, Yodlowski EH, Martin DC, Pruett JK, Crumrine RS: Low-frequency component of heart rate variability. AN-ESTHESIOLOGY 1995; 83:884-7

6. Malliani A, Pagani M, Lombardi F, Cerruti S: Cardiovascular neural regulation explored in the frequency domain. Circulation 1991; 84: 482-92

7. Montano N, Ruscone TG, Porta A, Lombardi F, Pegani M, Malliani A: Power spectrum analysis of heart rate variability to assess the changes in sympathovagal balance during orthostatic tilt. Circulation 1994; 90:1826-31

(Accepted for publication March 21, 2000.)

of cardiac sympathetic nerve fibers, supportive evidence for the existence of a cardiac sympathetic blockade after TEA has been provided by Taniguchi *et al.*,⁶ who directly measured efferent cardiac sympathetic nerve activity after TEA in an experimental study on cats. Our recent finding² that vasomotor and sudomotor reflexes were abolished in the hands but remained in the feet after TEA also suggests a thoracic sympathetic blockade. Therefore, although we agree with Introna *et al.* that "sympathetic fibers above and below the segment of epidural anesthesia could travel cephalad or caudad within the sympathetic chain" and "continue to innervate the heart," we remain convinced that TEA can abolish sympathetic reflexes within thoracic segments.

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References

1. Kingwell BA, Thompson JM, Kaye DM, McPherson GA, Jennings GL, Esler MD: Heart rate spectral analysis, cardiac norepinephrine spillover, and muscle sympathetic nerve activity during human sympathetic nervous activation and failure. Circulation 1994; 90:234-40

2. Magnúsdóttir H, Kirnö K, Ricksten S-E, Elam M: High thoracic epidural anesthesia does not inhibit sympathetic nerve activity in the lower extremities: ANESTHESIOLOGY 1999; 91:1299-304

3. Lundin S, Kirnö K, Wallin BG, Elam M: Effects of epidural anaesthesia on sympathetic outflow to the skin. Acta Anaesthesiol Scand 1990; 34:492-7 4. Kirnö K, Lundin S, Elam M: Effects of intrathecal morphine and spinal anesthesia on sympathetic nerve activity in humans. Acta Anaesthesiol Scand 1993; 37:54-9

5. Kirnö K, Friberg P, Grzegorczyk A, Milocco I, Ricksten S-E, Lundin S: Thoracic epidural anesthesia during coronary artery bypass surgery: Effects on cardiac sympathetic activity, myocardial blood flow and metabolism, and central hemodynamics. Anesth Analg 1994; 79: 1075-81

6. Taniguchi M, Kasaba T, Takasaki M: Epidural anesthesia enhances sympathetic nerve activity in the unanesthetized segments in cats. Anesth Analg 1997; 84:391-7

(Accepted for publication March 21, 2000.)

Anesthesiology 2000; 93:303 © 2000 American Society of Anesthesiologists, Inc. Lippincott Williams & Wilkins, Inc.

OR Scheduling Algorithms

To the Editor:—In a recent article¹ describing their computer analysis of various operating room (OR) scheduling algorithms, Dexter *et al.* provided important additional insights into this vexing problem. I have questions regarding some of their assumptions and methods.

The duration of each simulated add-on case was generated by sampling from a log-normal probability distribution that fit the historical data for each OR suite. Each case thus selected was not then subjected to a second simulation designed to cause duration to vary around the result of the first selection according to the known probability distribution of the durations of individual add-on cases. This adjustment is necessary if the simulation is to mirror the true occurrence of individual variance because that variance is not revealed to the Monte Carlo simulation by the statistic describing the overall add-on case duration, as described in the Methods section. Omitting the case-duration variance as applied to the simulations could have an important impact on the results. The authors provide no data to justify the omission.

This impact may have been mitigated by the method used to gather data needed to generate the probability distribution of the "open time available for add-on cases in each OR," although this is not made clear in the article. Were the open times taken from daily, projected nextday schedules available at the official cutoff time on the scheduling day? Or were they taken from the actual recorded open time available for each OR after the scheduled operations were completed and before handling of known add-ons was commenced on the day scheduled? If the latter, intuitively, the simulation results would be significantly more dependent on the probability distribution of the individual add-on case durations. Even in the former case, the effect on the results may be large. The authors touch on this issue only briefly in their discussion when, in another context, they state correctly, "The mean time to complete a series of consecutive cases approximately equals the sum of the mean times to complete each of the consecutive cases." In either case, it would have been suitable and not complex to include the effect of variation in case duration as part of the simulation.

In calculating use, did the authors account for overtime caused by the use of "fuzzy constraints" by including additional time in the denominator? If so, was this time overweighted to effectively penalize the algorithm for extending the schedule past 3 PM?

Long turnover times were truncated at 1 h. Did the authors test the effect of a different maximum? It could be important in assessing the results. Some long OR turnover times result directly from unplanned adverse effects that the scheduling methods can have on the behaviors of the personnel. Analyzing the causes of long OR delays was beyond the scope of the study and, therefore, properly omitted. But longer intervals between cases will have a direct bearing on the results of the analysis, and it would be helpful to know their magnitude.

Finally, the pressing question is whether any of the algorithms were tested in those same OR suites to provide the needed confirmation of theory. The authors have not provided evidence that the "variablesized bin packing" model, notwithstanding that the tested algorithms have been extensively evaluated for that model by management scientists, behaves sufficiently like the OR scheduling problem to impart confidence that their results are applicable to real problems in the absence of real world testing.

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Reference

1. Dexter F, Macario A, Traub RD: Which algorithm for scheduling add-on elective cases maximizes operating room utilization? ANESTHESI-OLOGY 1999; 91:1491-500

(Accepted for publication March 23, 2000.)

Dennis M. Fisher, M.D., was acting Editor-in-Chief for this correspondence.