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Measurement of Sympathetic Blockade: Effect of Epidural and Spinal Anesthesia

To the Editor:—We thank Dr. Magnúsdóttir *et al.*¹ for their recent clinical investigation, which appeared in *ANESTHESIOLOGY*. We agree with their findings of incomplete sympathetic blockade after high thoracic epidural anesthesia (TEA). As they pointed out, their results were in opposition to previous work by others² who reported complete sympathetic blockade with the same TEA technique. We also believe that Magnúsdóttir *et al.*¹ have correctly warned against relying solely on indirect measurement techniques to identify sympathetic activity in humans. Magnúsdóttir *et al.*¹ instead used a microneurographic technique, which is a direct measure of sympathetic nerve (peroneal) activity, and pointed out that the conflicting work² used an indirect thermographic measurement technique. It is interesting that these same authors,² referred to by Magnúsdóttir *et al.*¹ had previously warned against our use of heart rate variability (HRV)³⁻⁵ as a measurement technique to follow sympathetic activity after spinal anesthesia. This rejection of HRV was based on their inability to show changes in low-frequency oscillations^{6,7} after TEA, whereas simultaneous thermographic measurements resulted in changes believed to be consistent with complete sympathetic blockade.² At that time, we suggested that the different results of HRV *versus* thermography techniques might be caused by incomplete cardiac sympathetic blockade after TEA,⁵ and we believe the results of Magnúsdóttir *et al.*¹ now support this explanation.

Magnúsdóttir *et al.*¹ suggest that much of the hemodynamic stability during thoracic epidural results from incomplete sympathetic block above and below the epidural segment. Although we agree with this concept, we disagree with the assumption by Magnúsdóttir *et al.*¹ that TEA completely inhibits cardiac sympathetic activity. HRV measurements indicate that cardiac sympathectomy may not be as common as

was once thought.^{4,5} Although HRV is an indirect measurement of sympathetic activity, it is a technique that is cardiac specific because it uses the heart as the end organ or effector. Because of the complexity and overlap of sympathetic afferent and efferent pathways, it is possible that some sympathetic fibers remain untouched by the epidural anesthetic and could maintain innervation to the heart. Sympathetic preganglionic fibers originate in the intermediolateral cell column of the spinal cord and exit *via* the ventral nerve roots at levels from T1 to L2 or L3. Although the epidural sensory blockade of the dermatomes achieved by Magnúsdóttir *et al.*¹ incorporates the spinal segments thought to contribute to cardiac sympathetic fibers, T1-T4, other less direct pathways to and from the heart could still remain. Sympathetic fibers above and below the segment of epidural anesthesia could travel cephalad or caudad within the sympathetic chain and, in addition to maintaining innervation outside the area of segmental block,¹ could continue to innervate the heart, therefore explaining previous HRV data.^{4,5}

Certainly, measurement of actual neural activity (direct technique) is superior to measurement of effector organ function (indirect techniques) when it is performed correctly. However, microneurographic techniques, as used by Magnúsdóttir *et al.*¹ are very difficult and invasive and have limited application in clinical research. Therefore, all the methods of measuring sympathetic nerve activity have their pitfalls. Nevertheless, we believe we are justified in saying that careful use of direct and indirect measurement techniques can provide useful information about sympathetic activity during spinal and epidural anesthesia. Finally, we believe that this article by Magnúsdóttir *et al.*¹ correctly points out that many of the inconsistencies that are found in reports of the sympathetic effects by different anesthetic techniques

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

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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

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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