a "walking epidural" (40 μg epidural sufentanil after a lidocaine-epinephrine test dose) to a CSE technique (10 μg intrathecal sufentanil). In our study, there was no significant difference in cervical dilation at time of subsequent dose (5.6 ± 1.6 in the epidural group *vs.* 5.5 ± 1.8 in the CSE group), nor was there a difference in the time from analgesic administration to full cervical dilation (295 ± 160 min in the epidural group *vs.* 297 ± 155 min in the CSE group). This was specifically for patients who received epidural analgesia in the latent phase of labor.

Perhaps it is not the CSE technique that is associated with a more rapid cervical dilation; rather, it may be that administering high (0.25%) concentrations of local anesthetic (0.25% bupivacaine) to nulliparous patients is associated with slower cervical dilation.

For the past 10 yr, we have rarely administered any labor epidural with 0.25% bupivacaine; our most common "local anesthetic" epidural is 0.1% bupivacaine with 3 μg/ml fentanyl. Perhaps the results of Tsen *et al.*[1] would have been different if they had used a lower concentration of epidural bupivacaine or if their epidural technique had consisted of opioid without bupivacaine.

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In Reply:—We appreciate the interest in our work and comments expressed by Drs. Rosman and Connelly *et al.* with regard to our recent article.[1] Rosman expresses concern that the collective effects of subtle, nonsignificant differences between the combined spinal-epidural (CSE) and epidural groups could lead to an observation bias in the rate of cervical dilation. The influence of the factors he cites (earlier rupture of membranes and more aggressive use of oxytocin) is controversial but has been found to be unimportant in a large randomized trial from our institution.[2] Nonetheless, a multivariate analysis of initial cervical dilation rate *versus* analgesic group, controlling for use of oxytocin before analgesia, artificial or spontaneous rupture of membranes, and rupture of membranes before analgesia, with or without first-order interactions, still found group assignment to be a highly significant determinant (main effects model, $P = 0.0024$). Moreover, the difference between groups in examination frequency was not significant clinically (approximately 20 min) or statistically. The time after analgesia to the next examination was also not significant (CSE group, 1.6 ± 1.3 h, *vs.* epidural group, 1.6 ± 1.3 h, $P = 0.92$). Therefore, we do not believe the examination frequency could have significantly altered the observed rates of dilation. Rosman's concern regarding the length of ruptured membranes before analgesia is also unlikely to have influenced our observations. The inclusion of only those patients in whom ruptures occurred before analgesia (as opposed to all patients, even if ruptures occurred after analgesia, as reported in the original article) shows that in the CSE group ruptures occurred closer to the time of analgesia initiation than in the epidural group (5.2 ± 4.2 h *vs.* 6.8 ± 2.6 h, $P = 0.04$). In summary, although it is not practical to standardize every aspect of labor management given the uncertainties of nulliparous labor, we believe that there were no important differences that could have materially altered our results.

In addition, because a dural puncture is part of the CSE technique, we assume that Rosman is referring to the potential for headaches as a complication after regional techniques; of interest, use of the CSE technique has been suggested to prevent dural puncture with the larger epidural needle because it allows for confirmation of the dural space with a smaller needle. Although we cannot make any strong conclusions about potential complications of these two techniques because our study was not designed for that purpose and remains underpowered to make robust conclusions of that nature, we did not observe any differences in postdural puncture headache, fetal bradycardia, maternal hypotension, nausea, pruritus, or excessive blockade.

Connelly *et al.* suggest that our findings could be the result of epidural analgesia slowing the progress of labor, rather than CSE analgesia enhancing it. Although this is a possibility, we do not believe it to be likely nor supported by their previous work.[3] Our epidural group experienced a mean cervical dilation rate of 1.3 ± 0.7 cm/h for the first stage of labor in nulliparous women, a rate considered to be normal by Friedman,[4] the American College of Obstetricians and Gynecologists,[5] and major, recent obstetric texts.[6] Of note, although the timing of cervical dilation relative to analgesia was neither specified nor standardized in the work by Dunn *et al.*,[3] it appears that the mean cervical dilation in both their intrathecal and epidural groups was even slower than in our epidural group.

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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 others[2] 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 work[2] 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