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The Use of a Nerve Stimulator for Thoracic Paravertebral Block

To the Editor:—I wish to congratulate Dr. Karmakar on his very thorough and informative review on paravertebral anesthesia and analgesia.[1]

[1]Dr. Karmakar has overlooked one technique of paravertebral blockade that, in my opinion, has shown particular merit and is worthy of further investigation—the use of a nerve stimulator. To my knowledge, the use of a nerve stimulator as a guide to the performance of paravertebral blockade was first alluded to by Drs. J. J. Bonica and F. P. Buckley.[2] I have used and refined this technique for more than 5 yr now. As with any technique it has advantages and limitations. Some of its advantages include the following:

1. A nerve stimulator can be used in a supramaximal mode (Braun, 5.0 mamp) to help identify the paravertebral space. This is especially useful when the anatomy is distorted (*e.g.*, ankylosing spondylosis, previous surgery, local pathology), unusually challenging (*e.g.*, morbidly obese), or when the risk of pneumothorax is increased or its potential occurrence particularly undesirable (*e.g.*, severe chronic obstructive pulmonary disease, ambulatory patient). The diligent use of a nerve stimulator may warn of the "impending danger of a pneumothorax."
2. If desired, the motor or sensory end-point can be fine-tuned to allow for successful blockade of a spinal nerve with as little as 1 ml of local anesthetic. For this reason, this technique may be particularly valuable, in conjunction with imaging (*i.e.*, fluoroscopy), when neurolytic procedures are performed.
3. As outlined by Dr. Karmakar in his excellent review article[1], paravertebral blocks have several potential limitations including the unpredictability of the "multisegmental single injection technique." This technique has been popularized presumably because of reluctance to perform a multiple injection technique "that may incur more patient discomfort and risk." The use of a nerve stimulator allows the precise identification of only those nerves that need to be blocked and provides an additional element of safety. This same principle can be applied to the performance of multiple injection paravertebral blockade when there is uncertainty about the identification of the exact levels targeted (poor correlation between surface landmarks and actual anatomic level) or where the actual levels that need to be blocked are not known with certainty (*e.g.*, a rib fracture) as the exact levels can be ascertained by the motor responses elicited.
4. The nerve stimulator is an excellent teaching and research device. It allows precise correlations to be made between anatomy, physiology (motor responses, electrically elicited paresthesiae, and reproduction of pain in the targeted dermatome/s) and clinical effect. There are many more potential advantages but limited space prevents a more extensive discussion.

Potential disadvantages may include the expense of the nerve stimulator and associated insulated needle, and the inability to easily observe a motor response in obese patients. Therefore, it may be prudent to have an assistant that can palpate a motor response. It is always prudent to use all of your senses to guide the needle, as with any technique.

The technique is simple and can be used on either an awake or heavily sedated or anesthetized patient. Any of the approaches described in Dr. Karmakar's review article can be used.[1] I sedate the patient with intravenous ketamine (2.5–5.0 mg), versed (0.5–1 mg), and sufentanil (2.5–5.0 μg), administer oxygen by nasal prongs (2 to 3 l/min) and monitor with a pulse oximeter. The nerve stimulator (Braun) is set to deliver a supramaximal current (5.0 mamp). I use an insulated 5 cm Stimuplex needle and have found that it is the ideal length for virtually all adult patients in the thoracic region (T_2–T_{12}). The patient is warned that they may feel a pulsating "buzz" or feel some movement in their chest or abdomen. They are asked to report these phenomena as soon as they are perceived. The technique that is chosen determines how the needle is advanced. If the transverse process is encountered the needle is redirected either above or below the transverse process and advanced until a motor or sensory response is elicited. If the transverse process is not encountered the needle is advanced slowly until either a sensory or a motor response is elicited in the distribution of the "ventral" ramus of the spinal nerve. It does not seem necessary to refine the motor end-point although it is my practice to do so. I have found that when the needle is positioned in this manner a motor response will be elicited at a current of between 0.2–2.0 mamp. Approximately 30–40% of the patients will report a simultaneous electrically induced paresthesiae. The technique can also be used to facilitate difficult intercostals nerve blocks.

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In Reply:—Thank you for the opportunity to reply to Dr. Scott Lang's letter, in which he discusses the use of the nerve stimulator to perform thoracic paravertebral block (TPVB). I was aware of various authors alluding to the use of a nerve stimulator to facilitate intercostal nerve block and TPVB. A discussion of this method was not included in my review on TPVB[1] because there was a lack of published data on the specifics and safety. However, after the publication of my review, Wheeler reported on the technical details about using a nerve stimulator to perform TPVB[2] and Dr. Lang now provides additional information.

Because tactile localization of the TPVS using loss of resistance is subjective and indefinite[1] there may be potential merits in using a nerve stimulator to perform TPVB, as suggested by Dr. Lang. Locating the spinal nerve in the TPVS using nerve stimulation is an objective method of performing TPVB and may improve the overall success rate and make the technique safer, especially when clinical conditions make TPVB technically more demanding, such as in patients with morbid obesity or with distorted thoracic anatomy. However, before we can recommend it for routine use there is a need to evaluate its safety, success rate, and complications, because, as outlined by Dr. Lang, there are several potential limitations. These include the need to

use relatively high current intensity (1–5 mA) during initial simulation that can induce paresthesia, inability to readily observe a motor response in obese patients, and the need for an assistant to palpate the chest for the motor response. Moreover, local anesthetic injected at one thoracic level can spread to the contiguous levels (depending on the volume injected) where it may either modify or abolish the intercostal motor response to spinal nerve stimulation predisposing to deep needle insertion and possible pleural puncture during a multiple injection TPVB. Despite some of these potential limitations the technique of using nerve stimulation to perform TPVB is definitely worthy of further investigation.

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Useful Information about the pharmacokinetics and Pharmacodynamics of Midazolam and Lorazepam

To the Editor:— Barr *et al.*[1] have provided useful information about the pharmacokinetics and pharmacodynamics of midazolam and lorazepam when administered by infusion to intensive care patients. However, their conclusions may be misleading and are at variance with other published data.

Methodologically, it is notable that Barr *et al.*[1] report a mean duration of lorazepam infusion more than twice that for midazolam (36.94 *vs.* 15.02 h, respectively), and that the SD of lorazepam infusion duration was approximately 31 h. These differences could easily explain the prolonged time to awakening and extubation associated with lorazepam administration. It is also unfortunate that the authors did not better standardize the analgesic regimens between the two groups, or at least report the doses of fentanyl administered. Differences in drug potency may have been related to the success of the associated analgesic regimen.

Swart *et al.* previously compared the pharmacology of midazolam and lorazepam in a randomized, double-blind trial of critically ill patients.[2] Swart studied a larger and much more ill (Apache II score of approximately 26 *vs.* 9 in the Barr study) group of patients, one that may be more representative of the type of patients requiring continuous infusions of sedative drugs. In addition, patients received sedative infusions for a much longer duration of time in the Swart study (141 *vs.* 15 h). Although Swart *et al.*'s pharmacokinetic analysis was not as sophisticated as Barr *et al.*'s, they did report a longer elimination half life for lorazepam than midazolam (13.8 *vs.* 8.9 h, respectively). However, there was much more variability in midazolam's half life, with a range extending from 2.2–35.5 h. In contrast to Barr *et al.*, Swart found that the infusion rate of midazolam required to achieve a comparable level of sedation was approximately 15 times higher than for lorazepam, despite administration of comparable doses of fentanyl. In addition, Swart *et al.* found that patients achieved the desired level of sedation more frequently with lorazepam than midazolam. Finally, the average daily drug cost was approximately 10 times higher with midazolam than lorazepam.

A comparison of the two studies mentioned above reveals the complexity of sedation of critically ill patients. In the spirit of context-sensitive half-life, the pharmacokinetics of a particular drug may vary markedly with the duration of administration, severity of illness, and so on. Despite the evidence of prolonged sedation in association with lorazepam use in the intensive care unit presented by Barr *et al.*, lorazepam may be a more effective and cheaper agent than midazolam "in the long run."

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In Reply:—We appreciate the points raised by Dr. Deem. Our manuscript addresses the differences in infusion duration.[1] To briefly recapitulate: this was a double-blind, randomized study. Occasionally randomization fails to divide patient covariates evenly between groups. The smaller the study, and the more covariates considered, the more likely it is that not all covariates will be evenly divided between groups. Since randomization failed to provide similar durations of infusions, we used a model-based approach to draw clinical inference from the fundamental PK/PD characteristics. Those inferences support our conclusions. The half-lives reported for midazolam (10 h) and lorazepam (16 h) in our manuscript are consistent with the published PK of midazolam[2–7] and lorazepam.[7–12]

As explained in the manuscript, fentanyl was administered by target-controlled infusion, set to 1.5 ng/ml. Because this was a randomized study, the concept of "standardizing analgesic regimens between groups" is irrelevant. There was no difference in the fentanyl administered to the two groups, nor in the "success of the associated analgesic regimen."

Swart *et al.* reported that 5 of 6 patients with delayed midazolam elimination had been treated with erythromycin for more than 2 days.[7] Obviously such patients should be treated with drugs not metabolized by CYP 3A4, such as lorazepam. Absent those patients, the variability in midazolam and lorazepam reported by Swart was similar to what we observed in our patients, none of who received erythromycin. We

cannot explain why Swart *et al*. were unable to achieve the desired level of sedation with midazolam, despite *average* infusions rates of 16 mg/hr. In our experience, adequate sedation, including complete unconsciousness, can be achieved with either drug, and at far lower doses of midazolam than reported by Swart.

As Dr. Deem notes, Swart *et al* report a 15-fold difference in infusion rates between midazolam and lorazepam. This is much higher than in our study or in other published comparisons of midazolam and lorazepam in the intensive care unit.[13,14] Of note, the dosing differences for midazolam and lorazepam reported by Swart *et al*. correspond exactly with the concentration differences of drugs in their syringes: 0.33 mg/ml of lorazepam *versus* 5 mg/ml of midazolam. Swart *et al*. titrated to deep levels of sedation, where drug effect is difficult to assess precisely. In this study design, lack of precise titration to drug effect would be expected to produce a 15-fold potency difference by default. We believe this is the most likely explanation for their anomalous results.

Our manuscript documents that both midazolam and lorazepam are effective drugs for intensive care unit sedation, and provides guidelines in administering them to achieve comparable results. We did not address which drug was cheaper in the long run, which is a complex question involving far more than drug acquisition costs.

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Increased Total Positive End-Expiratory Pressure Does Not Improve Hypoxemia during One-Lung Ventilation

To the Editor:—We were interested to read the article by Slinger *et al.* regarding the effect of positive end-expiratory pressure (PEEP) on arterial oxygenation during one-lung ventilation (OLV).[1] When application of 5 cm H_2O PEEP to the ventilated lung causes total PEEP (plateau end-expiratory pressure) to increase from a low level toward the maximum curvature point (so-called "lower inflection point") on the static inspiratory pressure-volume (PV) curve during OLV, arterial oxygenation is improved. However, we have two comments regarding the conclusions made.

First, the "lower inflection point" on the inspiratory PV curve does not accurately indicate PEEP required to prevent end-expiratory lung collapse in patients with acute respiratory distress syndrome (ARDS), because PEEP is an expiratory phenomenon.[2] Holzapfel *et al*. reported that the inflection point on the expiratory PV curve was greater than the "lower inflection point" on the inspiratory PV curve, and PEEP set at the inflection point on the expiratory PV curve abruptly decreased pulmonary shunt in the early stage of ARDS.[3]

Second, the conclusions of the authors suggest that increases in total PEEP would improve hypoxemia during OLV in many patients with good elastic recoil. However, we believe that increased end-expiratory volume of the ventilated lung would not improve hypoxemia during OLV. We quantified the magnitude of auto-PEEP during OLV, and found that Pa_{O_2} on F_{IO_2} of 1.0 during OLV was extremely variable and levels of auto-PEEP did not correlate with Pa_{O_2} during OLV.[4] In addition, Katz *et al.* demonstrated that application of 10 cm H_2O PEEP to the ventilated lung did not significantly affect pulmonary shunt during OLV.[5] These clinical findings suggest that end-expiratory volume of the ventilated lung may not represent a major determinant of arterial oxygenation during OLV. Pulmonary blood flow has been considered to redistribute from the nondependent to the dependent lung in the lateral decubitus position. However, an animal study has recently demonstrated that pulmonary blood flow does not redistribute with repositioning from the supine to the left lateral decubitus position during two-lung ventilation.[6] Furthermore, Glenny *et al*. most recently demonstrated that the structure of the pulmonary vascular tree, and not gravity, is the primary determinant of the distribution of pulmonary blood flow in pigs.[7] Whether complete lung collapse produces significant reduction in pulmonary blood flow to the nondependent lung has yet to be fully elucidated. However, these recent experimental studies suggest that excessive pulmonary blood flow to the nonventilated lung, as opposed to decreased end-expiratory volume of the ventilated lung, might play a key role in the development of hypoxemia during OLV.

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In Reply:—On behalf of my co-authors I would like to thank Drs. Yokata and Sari for their correspondence and for sharing our interest in the pathophysiology of one-lung ventilation (OLV) as it applies to intraoperative management of patients during thoracic anesthesia. Their letter highlights three issues that arise out of our study of the interrelation of positive end-expiratory pressure (PEEP) and lung compliance with oxygenation during one-lung anesthesia:

(1) Would it be more useful to use the inflection point derived from the expiratory limb of the pressure-volume (PV) curve of the ventilated lung, rather than the inspiratory lower inflection point (LIP), as a surrogate marker for the functional residual capacity (FRC) and the optimal endexpiratory lung volume? This is a possibility. As they mention, the expiratory inflection point has been demonstrated to be a useful guide for ventilatory management of patients with ARDS in the intensive care unit. Unfortunately, our experimental protocol did not allow for measurements on the expiratory portion of the PV curve. Although there are important differences in the respiratory mechanics between ARDS patients and those having intraoperative one-lung anesthesia, this would be a worthwhile question to study.

(2) The level of auto-PEEP does not correlate with Pa_{O_2} during one-lung ventilation (OLV). This is correct. A comparison of the PV curves of two patients with low levels of auto-PEEP in figure 2 and figure 3 of our manuscript demonstrates this point.[1] The patient in figure 2 had an identifiable LIP, the application of PEEP raised the end-expiratory pressure closer to the LIP level and the patient had an improvement of Pa_{O_2} with PEEP. The patient in figure 3 did not have a measurable LIP. Presumably this patient and others with similar PV patterns do not get down to the level of their FRC at end-expiration and are not helped by applied PEEP. So it is not merely the presence of auto-PEEP but also the underlying lung mechanics that determine whether a patient will benefit from PEEP during OLV.

(3) Gravity and position may not be an important determinant of blood flow redistribution during OLV. The authors quote the study of Mure *et al.*,[2] which was performed in closed-chest dogs to back up their point. While it is correct that we now appreciate that anatomic factors have a major contribution to the distribution of pulmonary blood flow, it is not clear how relevant this is to clinical OLV in the open-chest human. In fact, a recent report has confirmed the importance of operative position on oxygenation during OLV. A study by Watanabe *et al.*,[5] demonstrated that there was a significant difference in oxygenation during OLV dependent on the patients position, with the mean Pa_{O_2} in the lateral position exceeding that in the supine position by greater than 100 mmHg. Since the publication of our manuscript another study has been reported which validates our findings. Fujiwara *et al.*[3] applied PEEP to the ventilated lung or continuous positive airway pressure (CPAP) to the nonventilated lung in a series of patients during OLV. They found that PEEP and CPAP were equivalent therapies and that both significantly increased Pa_{O_2} during OLV. This is very different from the previous findings of Capan *et al.*,[4] who showed that CPAP clearly increased mean Pa_{O_2} during OLV while PEEP decreased mean Pa_{O_2}. The difference between these two studies is in the patient populations. Capan studied patients with moderate or severe COPD having thoracotomies for lung cancer surgery. Fujiwara studied patients having OLV for esophageal surgery who are more likely to have normal pulmonary function. This supports our thesis that patients with normal or supra-normal lung elastic recoil (*e.g.* restrictive lung mechanics: pulmonary fibrosis, obesity) are the patients most likely to reach an end-expiratory lung volume below their FRC, and thus are the most likely to benefit from PEEP to the ventilated lung during OLV.

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Another Explanation for Bowel and Bladder Dysfunction after Spinal Bupivacaine

To the Editor:—We wish to comment on the letter by Drs. Mardirosoff and Dumont[1] describing the case of a patient who experienced bowel and bladder dysfunction after receiving bupivacaine spinal anesthesia. Although direct neural toxicity of several local anesthetics has been reported,[2,3] other factors could be involved in producing the symptoms described by the authors, including lumbar puncture with no drug injection.[4] Positioning the patient for surgery, *i.e.*, knee arthroscopy, can be an important factor. The authors did not mention how the patient was positioned on the operating table. It is known that knee arthroscopic procedures have independently been related to transient

neurologic syndrome,[1] probably caused by surgical positioning together with muscle relaxation if a spinal anesthetic has been employed. Knee arthroscopy usually involves positioning the lower limbs into a device designed to secure the limb but permitting some mobilization for surgical manipulation. Because the authors did not refer to the particular device used, the reader cannot know if this influenced the development of the symptoms the patient experienced. In some instances the pressing device is situated excessively high on the limb, or a perineal bumper is used to secure the patient's body. Both can compress the perineal muscles or nerves (*i.e.*, the III–IV sacral roots constituting the pudendal nerve, and the V sacral and I coccygeal roots) and produce transient peripheral neurologic lesions (similar to a low cauda equina syndrome), which improved in a few hours or days. The perineal nerves are involved in sphynctereal control,[5] and the trauma produced must have induced sphynctereal dysfunction as feces and urine incontinence.

In conclusion, if a transient neurologic dysfunction occurs in a patient who has received spinal anesthesia, not only the drugs used can be the cause, provided morphologic diagnostic studies excluded other causes. However, we agree with the authors that the actual cause of symptoms cannot be elucidated completely in most cases.

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In Reply:—We wish to thank Drs. Errando and Peiró for their remarks. The position of the patient on the operating table was supine, the device used to secure the limbs by our surgeon was a lateral external holding device situated in the upper third of the limb. There was no bumper on the perineal region.

Although we agree that the positioning of the patient can account for some neurologic injuries, it seems quite difficult to explain how, in a case like this one, an external compression device (perineal bumper) could harm the perineal nerves with sphynctereal control that lies in the deep structures of the perineum, without inducing any sensory losses in the perineal region.[1] We might also add that other etiologies, such as hysterical conversions, are also possible, although of a lower probability.[2]

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Epidural Analgesia and Labor: Lack of Efficacity of Walking on Labor Duration due to Short Duration of Walking Time

To the Editor:— We read the article by Dr. Vallejo *et al.*[1] with great interest. In their report the authors found that epidural analgesia followed by walking or sitting does not shorten the labor duration from epidural insertion to complete cervical dilatation.

Our opinion is that the lack of efficacy of walking shown in your study may be caused by the short duration of the walking time. Our experience is that walking for a minimum of 1 h results in shortening of the first stage of labor. It would be interesting to know if in your study a correlation between walking duration and total labor duration was found.

The incidence of low back pain after epidural analgesia does not seem to have been taken into account, and we were very surprised by this. The hypothesis of unnatural position (lack of mobility) following sensory block is frequently evoked as one of important etiology of new onset postpartum back pain after epidural analgesia.[2–5]

The use of ambulation after epidural analgesia could improve and decrease the incidence of low back pain after childbirth. We would welcome your point of view on this point.

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Anesthesiology 2002; 97:526

In Reply:—We appreciate with interest the concerns Dr. Sandefo *et al*. have regarding the lack of efficacy of walking with ambulatory epidural analgesia (AEA). Although our study did not have sufficient power to show statistical significance (if any) in the duration of stage II labor or total labor duration, we did show AEA with walking or sitting did not shorten labor duration from the time of epidural insertion to complete cervical dilatation.[1]

It is the opinion and experience (nonpublished) of Sandefo *et al*. that parturients who ambulate for a minimum of 1 h have shorter labors. The upright position and ambulation are reported to shorten labor.[1] In our study, ambulatory patients walked for 25.0 ± 23.3 min and sat upright in a chair for 40.3 ± 29.7 min which, when combined, adds up to over 1 h.[1] Interestingly, there are at least three other groups who have shown no significant difference in the duration of labor when allowed to ambulate with regional anesthesia.[2–4] In addition, as in our study (P = NS), Asselineau found slightly higher labor duration with ambulation.[1,3]

Unfortunately, the incidence of low back pain after ambulatory analgesia was not one of our measured outcomes, but we did show no differences in Visual Analogue Scale (VAS) scores before and after epidural insertion, at complete cervical dilatation, and at the start of stage II labor.[1]

Regardless of the effect AEA has on labor duration, the biggest advantage of AEA is that it spares motor function, allowing for mobility during labor, and does not impede the ability to push during delivery.

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Potential Choking Hazard when Using a Scented Pen

To the Editor:—We report a potential choking hazard with medical equipment in a 3-yr-old boy scheduled for diagnostic bronchoscopy. The backshell of a transparent facemask was swiped with a scented pen (T 5000 Mask-Ease pens, Trident Medical International, Indianapolis, IN) to facilitate the acceptance of the mask by the child. Inhalational induction was uneventfully performed with sevoflurane in oxygen/nitrous oxide.

During mask ventilation, a floating foreign body was observed by the attending staff anesthesiologist within the transparent facemask (Laerdal Infant Mask, Laerdal Medical, Stavanger, Norway), although it was undetectable for the ventilating anesthesiologist (fig. 1, top).

Mask ventilation was interrupted for a short time for inspection of the backshell of the mask, from which the foreign body was removed (fig. 1, middle). Further inspection showed it to be a broken tip from the Mask-Ease pen (fig. 1, bottom). Two additional cases of broken tips had previously occurred in our department but the tip had been detected before the facemask was used.

The choking potential of foreign bodies in children has not only been reported in connection with common foods and toys, but also with medical equipment such as syringe caps, the caps of inflatable face masks, and nasal cannulas.[1–3] To our knowledge, there has been no publication until now about the choking hazard when using scented pens. The application of scented substances, such as Cherry or Bubble-gum, on the backshell of the facemask is an upcoming facility to make inhalational induction or preoxygenation in children more comfortable. However, as demonstrated in our case, the use of such pens carries the risk of accidentally placing a foreign body in the airway equipment and thus represents a potential choking hazard to the patient.

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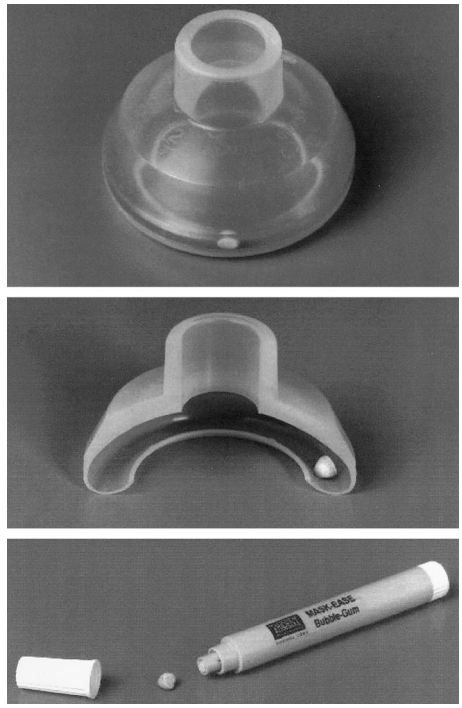

Fig. 1. Transparent facemask with the broken tip of the Mask-Ease pen within the facemask section (*top*). Cross-section of the facemask with the broken tip of the scented pen (*middle*). Mask-Ease pen with the broken tip after swiping the tip across the backshell of the mask (*bottom*).

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Anesthesiology 2002; 97:

In Reply:—The T5000 Mask-Ease scented pens were purchased from an outside vendor of Trident Medicals and sold to a few specific customers in the European Market only. After receiving the initial complaint from the University Children's Hospital in Zurich, Switzerland, a thorough investigation into the root cause of the problem was performed by the Trident Medical quality assurance department.

The conclusions drawn from this investigation and analysis led to the immediate recall of all Mask-Ease scented pens from the market and a discontinuation of any and all future sales of the Mask-Ease scented pens by Trident Medical International.

Bob Richmond, President Trident Medical International, Inc., Indianapolis, IN. brichmond@parholdings.com

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Anesthesiology 2002; 97:527–8

Vulpian and Not Claude Bernard first Proposed the Hypothesis of the Motor End-Plate as the Site of Action of Curare

To the Editor:—Within the anesthesia community, it is commonly believed that Professor Claude Bernard, M.D. (1813–1878), was the first to postulate the motor end-plate as the point of action of curare.

It is certain that he demonstrated experimentally first on a whole curarized frog, that muscle retained its activity when directly stimulated, and second, in the frog model of Galvani in which one leg was protected from the curare by a vascular ligature, that sensory nerves were not affected by curare. However, his position regarding the effect of curare on motor nerve is not so clear. After these experiments he concluded that the motor nerve itself was paralyzed.[1] However, in an experiment with a nerve-muscle preparation where the nerve and then the muscle were successively bathed in a curare solution, he noted that the nerve could be immersed in the curare without loosing its function.[2] He proposed to the Academy of Sciences that the nerve was affected right next to the muscle during its final *ramuscules* (ramifications).[3] He was therefore very close to the correct interpretation, and indeed, the term motor end-plate would later appear in his writing.[4] Nevertheless at the same time, he continued to defend the idea that nerve was destroyed by curare.[5] In addition, he later arrived at an another strange conclusion. He based his view on one observation (whose validity was questioned by Professor Alfred Vulpian, M.D.) where, in a lightly curarized frog, electrical stimulation of the medullar roots leading to the motor nerves was without effect while stimulation of the nerve still produced a response. He concluded that curare uncoupled the nerve from the cord, *i.e.*, the reflex arc was interrupted at the level of the spinal cord and that this was the point of action of curare. Using his own term, curare somehow unhooks (*décroche*) the nerve from the cord. These theories were set out in his "Leçons" of 1865[4] and were again asserted in his last published text about curare where he talked of an unhooking and paralysis coming from the central and not peripheral nervous system.[6] At the end of his life, he still planned new experiments. In his personal notebook, edited long after his death, there are 21 references to curare. For example, he wrote,

Presented at the Satellite Meeting of History of Anesthesia Section Programme, World Congress, McGill University, Montreal, Canada, June 10, 2000.

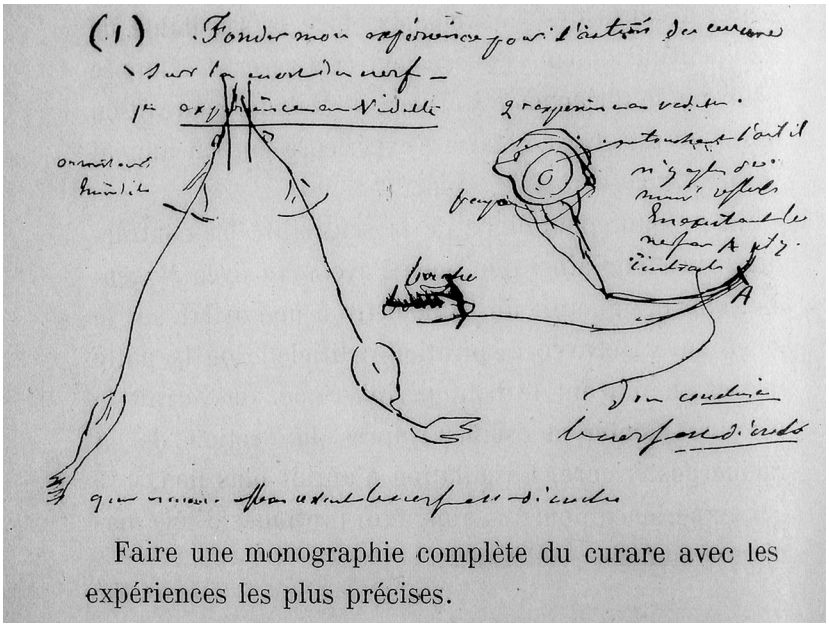

Fig. 1. Notes illustrating a research project (from reference 7, p. 182)[7]: "Fonder mon expérience pour l'action du curare sur la mort du nerf (. . .); en touchant l'artère, il n'y a plus de mouvements réflexes. En excitant le nerf en A, il y a contraction, donc conclure: le nerf est décroché. Quand les . . . (?) reflexes cessent, le nerf est décroché." "Basing my experiment about the action of curare on nerve death (. . .); When touching the artery (*i.e. stimulation of the reflex arc*) there is no longer any reflex movement. When stimulating the nerve at point A there is a contraction. Therefore conclusion: the nerve is unhooked. When the . . .(?) reflexes cease, the nerve is unhooked."

"One could do a neat experiment with curare and strychnine, with the aim of showing that the motor nerve becomes separated from the cord."[7] He illustrated his ideas with diagrams, one of which is shown in figure 1. Had he more time to live and to perform these experiments, perhaps Claude Bernard would have been aware of his error.

It was Alfred Vulpian (1826–1887) who refuted Claude Bernard's hypothesis. Drawing on the publications of contemporary physiologists (especially Wilhelm Kühne, a Berliner physiologist, and Dr. Charles Rouget, Professor of Physiology at Montpellier Faculty) and his own research and reasoning, it is evident from his notes that he had concluded the action of curare to be on the motor end-plate, a concept that was clearly stated in 1875.[8] The fact that Vulpian's work is not widely known outside of France may have led to a mistaken view of the role of Claude Bernard in correctly identifying the true nature of the neuromuscular junction.

The author thanks David J. Baker, M.Phil., D.M., F.R.C.A, (attaché-Consultant, Hôpital Necker Enfants- Malades, Paris, France) for his informed translation.

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Anesthesiology 2002; 97:528–9

Manual Occlusion of the Internal Jugular Vein during Subclavian Vein Catheterization: A Maneuver to Prevent Misplacement of Catheter into Internal Jugular Vein

To the Editor:— Recently our group published a simple method for detecting the misplacement of a subclavian vein catheter into the ipsilateral internal jugular vein (IJV).[1] This technique involved the manual compression of the ipsilateral IJV while transducing the catheter. A clear increase in pressure was noted when the catheter tip was in the IJV. We have now performed a follow-up study to see if a variation on this technique could be used to prevent entry of the subclavian vein catheters into the ipsilateral IJV.

The institute's ethics committee for human studies approved the study. Two hundred adult surgical patients scheduled for central venous cannulation *via* the subclavian approach were included. Patients with chest or neck deformities were excluded. Informed written consent was obtained from all patients. The patients were randomly assigned to one of two groups. All patients were awake, lying supine with head turned to their left. After aseptic preparation and lidocaine infiltration the right subclavian vein was located with the 22-guage hypodermic needle on a syringe, using a standard infraclavicular approach. An 18-guage introducer needle was then inserted and free flow of venous blood was confirmed. The "J-tip" guidewire was threaded through the introducer needle into the subclavian vein. During passage of the guidewire, the ipsilateral IJV was manually compressed in the supraclavicular area in patients belonging to the study group. In the control group the guidewire was inserted without external compression. The IJV was compressed with the index finger of the hand holding the introducer needle while the guidewire was inserted with the other hand of the operator (fig. 1). After placement of the guidewire, the needle was withdrawn and the catheter was passed over the wire (typically 10–12 cm). During placement of the guidewire and the catheter, patients were asked if they felt giddiness, pain, or any unusual sensation in the ear or throat. Difficulty experienced by the operator during insertion was also noted. On conclusion of the procedure, a chest x-ray was performed and the position of the catheter was identified. The incidence of subclavian vein catheter misplacements and untoward effects were noted. Demographic data were analyzed using the Student *t* test.

Support was provided by Sanjay Gandhi Postgraduate Institute of Medical Sciences, Lucknow, India.

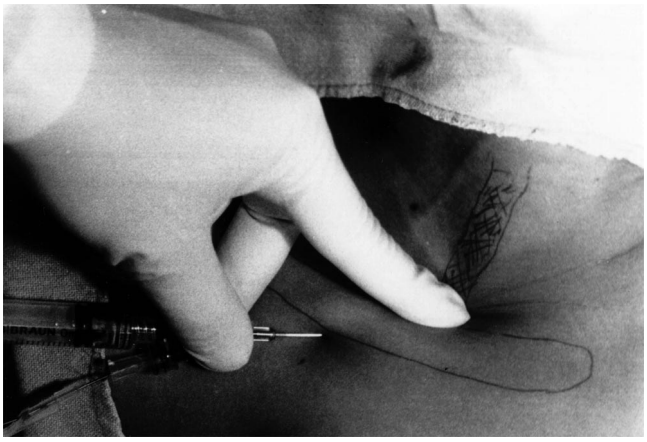

Fig. 1. The introducer needle is held *in situ* after puncture of the subclavian vein. The ipsilateral internal jugular vein (lying beneath the shaded area) is compressed externally in the supraclavicular area with the index finger of the left hand during the introduction of the "J-tip" guidewire with the right hand.

The characteristics of the patients in both groups were comparable (table-1). Ninety-eight patients in the control group and 97 patients in the study group had successful cannulation of the subclavian vein with the introducer needle. In the control group there were seven (7.14%) misplaced catheters detected with chest x-ray; six (6.12%) patients had misplacement of catheter into the ipsilateral IJV, and one (1.02%) into the contralateral subclavian vein. In the study group there were two (2.06%) misplaced catheters and both were in the contralateral subclavian vein. Difficulty was experienced during guidewire insertion with 4 patients of the control group and with 9 patients of the study group. Two patients in each group had mild pneumothoracies, which appeared on the chest x-ray. None of the study group patients complained of any untoward effects. Three patients in the control group complained of pain in the right ear and one patient experienced trickling sensations in the throat during the placement of the guidewire. No difficulties were encountered during the insertion of the

Table 1. Demographic Data and Incidence of Catheter Misplacements in Two Groups

	Control Group (n = 100)	Study Group (n = 100)
Age (Mean ± SD)	42 ± 13	45 ± 12*
Gender (Male:Female)	67:33	62:38*
Body mass index (Mean ± SD)	23.0 ± 2.5	22.5 ± 2.0*
Successful cannulation of SV	98	97*
Total number of catheter misplacements (%)	7 (7.14%)	2 (2.06%)
Misplacement of catheter in IJV (%)	6 (6.12%)	0 (0%)
Pain in ear or trickling throat	4	0 (0%)

* $P > 0.05$ = not significant.

SV = subclavian vein; IJV = internal jugular vein.

catheter. Chest x-ray of these 4 patients revealed misplacement of the catheter into the ipsilateral IJV.

The correct placement of the central venous catheter is essential for accurate monitoring of CVP and long-term use of the catheter. Misplacement of the tip may enhance the risk of clot formation, chemical or bacterial thrombophlebitis, and catheter erosion, in addition to impairing the CVP measurement.[2–4] The most common misplacement of the subclavian vein catheter is into the IJV. This does not vary with the side of insertion nor does it depend on whether the head is turned toward or away from the selected side.[5] The misplacement is typically cephalad into the ipsilateral IJV, although the catheter tip may also be placed in the contralateral IJV or the brachiocephalic vein.[1–3] No reliable method is available to prevent the misplacement of the subclavian vein catheter into the IJV. The incidence of malposition of catheters reported in the literature varies from 4–6%.[1,5] In the current study the incidence of malposition in the control group of patients was 7.14% and most of the misplacements were in ipsilateral IJV (6.12%). The operator encountered difficulty passing the guidewire with 4 patients without IJV occlusion and with 9 patients with IJV occlusion. The occlusion of ipsilateral IJV in the supraclavicular area effectively prevented the cephalad insertion of the guidewire and therefore the subclavian vein catheter into the IJV.

There have been a number of reports of ear pain in patients with subclavian vein catheters misplaced into the IJV.[6–9] König and Roscoe postulated that it occurs secondary to irritation of jugular bulb or cephalad end of ipsilateral IJV, which is innervated by the vagus nerve.[8] It is yet to be explained why all patients who experienced a catheter misplacement into the IJV did not report the same sensation. Also, it is difficult to explain why ear pain or trickling throat occurred, although it is evident that the patients who complained of ear pain or trickling throat had the catheter in the IJV. Therefore, occurrence of ear pain or trickling throat during catheterization of the subclavian vein could be a sign of misplaced catheter into the IJV.

We conclude that manual occlusion of the IJV during subclavian vein catheterization successfully prevents the misplacement of the catheter into the IJV. Our maneuver is simple to perform and requires no extra equipment or expenses.

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