

## Paresthesia but No Motor Response: What's Going On?

*To the Editor:*—The article by Urmey and Stanton<sup>1</sup> tested the hypothesis that if a needle should touch a nerve eliciting a paresthesia, an electrical current passing through that needle should also elicit a motor response. Since paresthesia was successfully elicited in 100% of cases but only 30% of patients exhibited any motor response to electrical stimulation up to 1 mA (200- $\mu$ s pulse width), Drs. Urmey and Stanton seem to suggest that eliciting a motor response is unreliable, unnecessary, and may fail to signify proximity of the needle tip to the nerve. However, this conclusion is based on several assumptions, which deserve further comment.

The mechanism by which paresthesiae are elicited still remains the object of passionate debate. Paresthesia is a subjective symptom, which in many cases is difficult to differentiate from pain, and can be elicited by a variety of stimuli, including pressure rather than direct nerve contact. Although, we agree that eliciting a paresthesia suggests that the needle is producing some kind of mechanical stimulation of the nerve, the ability to elicit paresthesia in every patient does not constitute irrefutable evidence of direct nerve contact, and does not provide any insight on which part of the needle was involved in generating the paresthesia itself. This is a crucial point, because, on the contrary, nerve stimulation is mainly produced by the needle's tip. Rather than postulating a selective contact between the needle tip and a geographically isolated sensory component of the nerve (intriguing hypothesis but quite speculative), it seems equally plausible that the shaft of the needle, rather than the tip, was actually producing the mechanical stimulation. This hypothesis is also supported by the observation that associated motor responses occurred more frequently with noninsulated needles (in which the electrical field is also extended along the shaft). The observation that, once elicited, the motor responses were not related to the site of paresthesia further suggests that the shaft of the needle probably touched one nerve, while the needle's tip stimulated another nerve. However, it must also be considered that, though subtle, the withdrawal reaction upon eliciting a paresthesia may displace the needle tip from the position in which the paresthesia is obtained, and this may further affect the experimental model used by Drs. Urmey and Stanton.

The authors also suggest the high success rate of nerve block as an indirect evidence of the specificity of the paresthesia technique for nerve location. However, this result can be reasonably explained by the very large dose of local anesthetic administered (50 ml/750 mg mepivacaine). The relationship between the injected volume and success rate is well known.<sup>2</sup> Excellent success rates with interscalene brachial plexus block have been reported with doses of local anesthetic ranging from 40 to 60% of those used by Urmey *et al.*<sup>3–5</sup> Furthermore, the use of a low-current nerve stimulation ( $\geq 0.2$  mA) technique as the primary method to localize the interscalene brachial plexus, results in a 95% success rate with only 35–40 ml of local anesthetic, even if no patient reports paresthesia in the brachial plexus distribution before or during motor stimulation.<sup>6</sup>

Finally, although the authors carefully verified the output of the nerve stimulator before each use, the current intensity actually delivered to the patient was not measured. This is especially important when considering that the needles and nerve stimulator came from

different manufacturers. The lack of an appropriate documentation of the actual current delivered to the patients may further affect the validity of the experimental model. Such caution is even reinforced when assuming, as stated by the authors, “the needle tip was in direct contact with a sensory nerve following the elicitation of a mechanical paresthesia.” Indeed, in this condition, the activation of the nerve stimulator should also result in an “electrical paresthesia” in the same distribution as the original “mechanical paresthesia,” especially considering the relatively high electrical energy applied (1-mA intensity with a 200- $\mu$ s pulse width).<sup>7</sup> However, this was likely not the case, as it was not reported. Using a low-power nerve stimulator, Smith and Allison<sup>8</sup> reported that despite an often protracted search for paresthesiae, they were elicited in only 39% of cases, whereas the electrical paresthesia using nerve stimulation was obtained in all patients, and resulted in much higher success rates. Therefore, it is again extremely difficult to assume that indeed the needle inducing the paresthesia was directly contacting the nerve with its tip, raising one more time the question related to what paresthesia is.

In conclusion, while acknowledging the contribution of Drs. Urmey and Stanton in the still ongoing debate between the use of paresthesia or electrical stimulation for nerve location, we need data from studies using a similar design, but paying more attention to the definition of paresthesia, using lower volumes of local anesthetic and a well defined technique for nerve stimulation, as well as verifying that mechanical paresthesia would also result in electrically induced paresthesiae.

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## Motor Response following Paresthesia during Interscalene Block: Methodological Problems May Lead to Inappropriate Conclusions

*To the Editor:*—We read with great interest the article by Urmeý and Stanton.<sup>1</sup> It would appear, however, that the validity of the results must be viewed with some caution and the authors' conclusions, in particular, call for several comments.

First, use of fresh batteries for a nerve stimulator is not sufficient to prove its adequacy. A peripheral nerve stimulator in use for several months might not perform as well as a new one.<sup>2</sup> For use of this importance, the stimulator output should have been tested with a calibrated oscilloscope. A stimulator that underestimates current output can mislead the operator. Moreover, a stimulator with clearly marked polarity would have been preferable. If polarity is inversed inadvertently, more current is needed.

Nerves are not fixed in the surrounding tissue. Despite the needle presumably being near the nerve and immobilized when paresthesia are reported, imperceptible patient movement or breathing can dislodge the needle. There is no doubt that for patient comfort and security, as well as ethical reasons, the solution was injected after applying the stimulator only long enough to achieve paresthesia and not while maintaining paresthesia. To date, how close a needle must be to a sensory fascicle to produce mechanical paresthesia has not been established.<sup>3</sup> Is the needle situated inside the nerve in contact with the sensory fascicle of the reported paresthesia, just touching the nerve, or somewhere in the vicinity of the nerve? Shoulder paresthesia can be provoked by deep palpation of the interscalene groove in thin patients.

Why varied responses are observed after mechanical or electrical stimulation of the same root or trunk can be explained by fine details of anatomy. With the interscalene technique, the plexus is located at the level of the trunk and/or anterior branch of the spinal nerve. As a result, the response elicited is clearly metameric. The upper trunk and C5-C6 contain sensory fascicles that distribute to the upper lateral brachial cutaneous nerve (axillary nerve; C5), the lower lateral brachial cutaneous nerve (radial nerve; C5-C6), the lateral antebrachial cutaneous nerve (musculocutaneous nerve; C6), and the palmar cutaneous branch of the median nerve (C6). Consequently, paresthesia extending from the shoulder to the hand is not surprising with the interscalene approach. Similarly, with a nerve stimulator, the contractions commonly mediated by the cephalad roots (C5-C6) or upper trunk are those of the supraspinatus, infraspinatus (C4-C6), deltoid (C4-C6), biceps brachii (C5-C6), brachioradialis (C5-C6), extensor carpi radialis longus (C6-C7), pronator teres (C6-C7), and flexor carpi radialis (C6-C8) muscles. A distal paresthesia or motor response does not necessarily imply that the needle has been inserted too deeply, contrary to what had been suggested elsewhere.<sup>4</sup> Lastly, satisfactory results require taking care not to regard as positive the shoulder responses that are due to stimulation of a collateral branch either directly or with the shaft of an uninsulated needle.

In addition, electrophysiologic aspects of nerve stimulation and its clinical application are important to consider. Nerves coursing from the plexus are mixed, consisting of different nerve fascicles, including groups of axons that determine a common and specific motor or sensory innervation. With the minimum current able to produce a stimulus, large A  $\alpha$  motor fibers are stimulated, while small A  $\delta$  and C fibers are not. In such cases, a motor response is obtained without pain or patient discomfort. The shoulder is innervated by 30% of the fibers of the brachial plexus, 28.4% for motor innervation, and only 1.6% for sensory innervation.<sup>5</sup> The assumption by Urmeý and Stanton that each paresthesia constituted evidence of contact with a sensory fascicle alone was probably unfounded. Sensory and motor fascicles do not appear to be very distant from each other in the brachial plexus.

Eliciting a motor response rather than a sensory response should occur frequently.

However, when pure sensory fibers were stimulated, confirmation of the needle tip being in contact with the nerve is obtained by eliciting paresthesia with each pulsation.<sup>2</sup> For successful peripheral nerve blockade, electrical paresthesia at 0.4–0.6 mA (100–200  $\mu$ s) have been used as endpoint without evidence of mechanical paresthesia or nerve damage.<sup>6,7</sup> If contact persisted with a sensory component of the nerve root, how was it possible for none of the patients to report electrical paresthesia to the two observers in spite of the maximum attained current of 1.0 mA, when they clearly indicated having the corresponding mechanical paresthesia several seconds earlier? As a very high stimulus is needed once the tip is some distance away from the nerve according to Coulomb's law, the only credible explanation is that, in 70% of the patients studied, the needle was sufficiently remote from the nerve after the initial paresthesia.

On the contrary, a very low current was sufficient to produce the response in the other 30% of cases. An uninsulated needle would have required more current to stimulate the nerve. Achievement of a response with such minimal intensity indicates that the needle remained in very close contact or was touching the nerve.<sup>8</sup> Urmeý and Stanton are to be congratulated because they provided good evidence that the needle has to be quite close to the nerve when paresthesia is elicited. A motor response at 0.1–0.2 mA for 200  $\mu$ s was obtained in several patients after the paresthesia disappeared. When responses are observed for currents this low, injection should be avoided, notably in unconscious subjects.<sup>9</sup> When performing nerve stimulation with an insulated needle, the proper endpoint is the minimal stimulating current. The needle is then released and if the response persists, the needle-nerve relationship should remain unchanged at low current (0.2–0.5 mA for 100  $\mu$ s). The operator should verify that the motor response disappears by applying decreased current once again before injection. The response should be abolished instantaneously with a painless and easily injected 1 ml-test dose.

Paresthesia occurs and serves as a warning with all techniques including use of nerve stimulators. Paresthesia is usually difficult to elicit with a short-bevelled, insulated needle.<sup>3</sup> The observed 30% increase in ability to obtain nerve stimulation with a long-bevelled needle is most likely due to a tendency of short-bevelled needles to press or push the nerve away.<sup>10</sup> Long-bevelled needles are sharper, may penetrate the nerve easier, and potentially increase the risk of postoperative dysesthesia. In contrast, despite frequent unintentional paresthesia during block placement, the withdrawal and redirection of stimulating short-bevelled needles is not associated with an increased incidence of neurologic complication, even when using a multiple injection technique.<sup>11</sup> Urmeý and Stanton would probably have consistently observed a motor response before mechanical or electrical paresthesia had they chosen to begin with an adequate procedure of nerve stimulation at a recommended higher current.

Currently, no compelling evidence exists to endorse a single technique or needle as superior with respect to success rate or incidence of complications. There are no prospective randomized controlled studies that compare the relative risks of regional anesthesia performed on anesthetized or conscious patients. Nevertheless isolated case reports,<sup>12</sup> and medicolegal reports provide a background of suspicion, which is difficult to refute with a lack of data either for or against the practice. The incidence of dramatic complications appears to be higher following interscalene approaches to the brachial plexus.<sup>12</sup> In the absence of firm data to the contrary, the weight of clinical practice suggests that the majority of peripheral nerve blocks, with the possible

exception of the interscalene approach to the brachial plexus, can be performed under sedation. The anesthesiologist should carefully consider whether the benefits of regional anesthesia performed on an anesthetized patient are greater than the risk of a catastrophic outcome. It is important that authors not draw conclusions erroneously based on slight imperfections in a study design, especially conclusions leading to recommendations or contraindications with significant medicolegal connotations.

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## What Happened to the Paresthesia?

*To the Editor:*—We read with great interest and enthusiasm the investigation by Urmev and Stanton<sup>1</sup> of the correlation (or lack of) between paresthesia and motor response during interscalene block. Unfortunately, a fundamental concept of their work was not addressed, discussed or, at worst, may be faulty. As a result, we are unsure how to apply their study and results to our clinical practices.

In the discussion, the authors state "the ability to elicit paresthesia in every patient in this study constituted evidence that nerve contact was made by the tip of the needle in every case." They present no data or evidence to support this statement. We dispute whether paresthesia necessarily indicates needle contact with a nerve. As an anesthesia resident, I witnessed an open placement of an axillary catheter under local anesthesia. Each time the surgeon applied traction to a connective tissue septum within the neurovascular "sheath," the patient experienced a mild median nerve paresthesia. Each time the paresthesia was produced, there were no instruments touching any nerves. Curiously, when the median nerve was retracted to allow passage of the plexus catheter, no paresthesia resulted. Finally, when the catheter was advanced within the neurovascular compartment, multiple median nerve paresthesias resulted. Clearly, paresthesia does not necessarily equal needle contact with nerve. In the absence of a clear understanding of the cause, or causes, of paresthesias, we find it difficult to interpret their results.

In our experience, most paresthesias are mild and immediately resolve in spite of the needle being immobile. The fact that the paresthesia resolves implies that "the event" that produced the paresthesia no longer exists in spite of the needle being "immobilized." Taken further, many practitioners I know are reluctant to administer local anesthetic or choose to give a very small "test dose" in the setting

of a persistent paresthesia for fear of an intraneural injection. Using this chain of logic, by the time Urmev and Stanton stimulated the needle, the conditions that produced the paresthesia no longer existed. In other words, the position of the needle relative to the nerve has changed. This alternative explanation is also consistent with their data showing that noninsulated needles produced motor response more frequently than insulated.

Finally, we tried to apply their study results to our own practice except we approached the problem from the other direction. We performed 19 consecutive interscalene blocks using a 22-gauge, insulated, short bevel needle and a nerve stimulator (Stimuplex Dig RC, B Braun, Bethlehem, PA). None of the patients received premedication. The stimulator current was set at 0.50 mA. None of the patients (0 of 19) experienced a paresthesia before a motor response was observed. All 19 blocks were successful (loss of deltoid/biceps and surgery performed without need for general anesthesia). We have a hard time understanding and explaining these data in light of the results and speculations by Urmev and Stanton.

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*In Reply:*—We appreciate the interest expressed in our recent publication. Whereas it was not our intention to incite such passionate and emotional responses from our respected peers, we certainly welcomed and carefully considered the many comments regarding the interpretation

of our data and implications of our findings. In our article, we simply described a phenomenon, *i.e.*, that the deliberate elicitation of a paresthesia by advancing a needle to a nerve root during interscalene block is not always associated with the ability to elicit a motor re-



sponse to electrical stimulation. Conversely, we have also observed what Carter and Sandberg reported,<sup>1</sup> that is, that a motor response to needle advancement is not always associated with a paresthesia. Our observations are reproducible and similar observations have been made at other institutions during axillary block.<sup>2</sup> We have videotaped the phenomenon. We do not completely understand nor pretend to be able to definitively explain the easily observed pronounced dissociation between sensory and motor nerve stimulation that occurs in many patients.

Upon review of these letters, there clearly exists some misunderstanding with regard to our intentions. It was not our intention to endorse or condemn any single technique or needle type. At our institution, we routinely use paresthesia techniques, peripheral nerve stimulation techniques, long-beveled needles, and short-beveled needles. With proper technique, we are convinced that all of these are very useful and extremely safe. Use of peripheral nerve stimulation to locate nerves for local anesthetic blockade was a major advance in regional anesthetic practice.

It appears that the questions raised by the authors of all three letters relate either to our methodology or our conclusions.

Regarding our methodology, our reported results cannot simply be dismissed as being secondary to a faulty nerve stimulator that: 1) was brand new, 2) had new batteries, the voltage output of which was checked by digital voltmeter immediately prior to each use, 3) was functionally checked by the ability to transcutaneously stimulate the facial nerve of the first author at 2 mA immediately prior to each use, 4) was equipped with a digital meter indicating the delivered current amplitude in mA. Further, this phenomenon has been observed with more than one manufactured brand of nerve stimulator.

Regarding our conclusions, we believe that our data interpretation was sound and responsible and that we kept an open mind when interpreting new data rather than clinging to preconceived notions that lack scientific support. To this end, we were puzzled by the inconsistencies, double standards, and biased conclusions included in the letter by Choquet *et al.*<sup>3</sup> In their letter, these authors cite the need for a "calibrated oscilloscope" but drew significant conclusions based upon several referenced studies that lacked this same rigorous standard. They state that "confirmation of the needle tip being in contact with the nerve is obtained by eliciting a paresthesia with each pulsation," based upon reference to a review article published in 1985 that neither specifically studied this nor provided supporting experimental data for this statement.<sup>4</sup> It appears to be anecdotal. They state "when responses are observed for currents this low (motor response at 0.1–0.2 mA for 200  $\mu$ s), injection should be avoided..." but later contradict themselves by advocating injection following motor response "at low current (0.2–0.5 mA for 100  $\mu$ s)," again, without data. The fact is that there are presently no compelling data that link injection at low current to nerve injury. Injection at low minimal current has never been prospectively compared to injection at higher minimal currents. Indeed, all three catastrophic complications reported by Benumof<sup>5</sup> that involved the use of a peripheral nerve stimulator, reported injection with current levels in the 0.81 mA to 1.0 mA range. Choquet *et al.* include editorial commentary on the presumed advantages of short-beveled needles compared to long-beveled needles without statistically significant data from prospective controlled studies to support their views. The fact is, as Neal *et al.* recently published, "there are no randomized clinical trials to support or refute the ability of various needle types and bevel configurations to impale human nerves."<sup>6</sup> Choquet *et al.* omit relevant data that short-beveled needles caused more severe nerve lesions in animal studies and that the lesions took longer to heal.<sup>7</sup> The "tendency of short-beveled needles to press or push the nerve away" is a myth. With minimal pressure, under direct vision, a short-beveled needle will travel into the substance of an isolated nerve<sup>7,8</sup> or through an anesthetized animal nerve (William F. Urme, M.D., unpublished observations; 22-gauge, short-bevel needle was easily passed through brachial plexus fascia and nerves in an anesthetized dog, Harvard School of Public Health, Boston, MA, October

1987). In fact, three of the four catastrophic cases reported by Benumof<sup>5</sup> documented the use of a short-beveled needle that clearly entered the neural tissue itself. Finally, Choquet *et al.* claim "paresthesia(s) are usually difficult to elicit with a short beveled insulated needle," but contradict this a few lines later by stating "frequent unintentional paresthesia" occur during block placement by withdrawal and stimulation of short-beveled needles.

On the other hand, we thank Casati *et al.*<sup>9</sup> and Carter and Sandberg<sup>1</sup> for their constructive critiques and agree that an "ability to elicit a paresthesia in every patient does not constitute irrefutable evidence of direct nerve contact." There is no obvious way to ethically prove this. Perhaps two-dimensional ultrasonographic examination of nerves during paresthesia can eventually help to answer this question. (Joseph Kay, M.D., Assistant Professor of Anesthesiology, University of Toronto, Sunnybrook Hospital, Toronto, Canada, personal communication, July 2002). Nevertheless, it is extremely unlikely and represents very wishful thinking to believe that a needle deliberately advanced in the direction of superficial large diameter nerve roots causes paresthesia without ever making nerve contact. Our contention that nerve contact was the event that caused paresthesia is supported by the depth at which paresthesia was reported. By contrast, we agree that with use of a peripheral nerve stimulator, it is clear that a motor response may be elicited without contact with the needle's tip, at a distance from the nerve. Nevertheless, in practice, we have frequently encountered further evidence that the needle tip may be in the nerve when motor response is elicited with peripheral nerve stimulator. Often, motor responses occur at very low amperage, disappear with further advance of the needle, only to reappear when the needle is slightly retracted. This constitutes credible evidence that the shielded needle tip may travel through the nerve during our searching. It is illogical to believe that the needle's shaft rather than its point caused the mechanical paresthesias observed in our study; as the needles were all advanced axially, initial contact would be with the needle's point. This does not mean that the needle tip was intrafascicular or intraneuronal. This we believe is exceedingly rare based upon the extremely low incidence of permanent neuropraxias that occur in association with interscalene block.

In summary, there is an old saying, "In life, you will be forgiven your lies, but heaven help you if you attempt to tell the truth." In our opinion, it is denial on our part as anesthesiologists to perpetuate and cling to folklore that we can deliberately aim and advance needles at superficial, large nerves, gently sneaking up on them while never making nerve contact. Although it is tempting to believe in such a never proven concept of "immaculate conduction," there is compelling scientific evidence and unfortunate complications of intraneural damage that argue the contrary. That is, there is every indication and reason to believe that nerve contact occurs during peripheral nerve or plexus blockade. Maybe we are asking the wrong questions. Perhaps, when considering peripheral nerve location by needle exploration, the burden of proof should not lie with having to demonstrate that paresthesia occurs in response to needle tip to nerve contact. Instead, we might begin to ask ourselves how we can prove that no nerve contact occurs when we elicit sensory or motor responses during peripheral nerve blocks. Despite this theoretical routine nerve contact, with responsible technique, peripheral nerve blocks are extremely safe.<sup>10</sup> Use of either accepted paresthesia or electrical nerve stimulation techniques are very safe, if performed carefully on patients who are not overly sedated or anesthetized. Our publication as well as that of Choyce *et al.*<sup>2</sup> represent an initiative in determining the relationship between paresthesia and stimulator techniques used for nerve location. Additional, more sophisticated studies are needed. Forthcoming data should be analyzed openly rather than defensively.

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## 2-Octyl Cyanoacrylate Glue for the Fixation of Continuous Peripheral Nerve Catheters

*To the Editor:*—Continuous peripheral nerve blocks have been associated with sustained effective postoperative analgesia,<sup>1-3</sup> opioid sparing,<sup>4,5</sup> improved rehabilitation,<sup>6</sup> and improved patient well being. However, the catheters used to deliver local anesthetic are frequently difficult to secure and maintain in the correct position. Several methods to fasten catheters have been advocated, such as suturing, retrograde subcutaneous tunneling,<sup>7</sup> cutaneous sutures,<sup>8</sup> and different methods of taping. Nevertheless, accidental dislodgement is still a frequent problem particularly with freely mobile sites, such as the neck and the axilla or lumbar region where perspiration is prominent. Having a simple, noninvasive, reliable method for catheter fixation would be beneficial. We report cases of three patients undergoing outpatient continuous nerve catheters secured with 2-octyl cyanoacrylate glue (Dermabond® Topical Skin Adhesive, Ethicon, Somerville, NJ).

The first patient was scheduled for left knee manipulation followed by use of a continuous passive range of motion machine. As part of the anesthetic plan a lumbar plexus block<sup>9</sup> was performed. The patient's back was prepped using Betadine® (Purdue Fredrick, Stamford, CT) and isopropyl alcohol. The block was placed using an 18-gauge 150-mm insulated Tuohy needle (B. Braun Medical, Bethlehem, PA) and a 20-gauge standard polyamide epidural catheter. After placement, the catheter was pulled taught against the skin. Using a sterile applicator the 2-octyl cyanoacrylate adhesive (Dermabond® Topical Skin Adhesive) was applied from the puncture site along 5 cm of the catheter's length. After approximately 4 min, the adhesive formed a clear coating that was not tacky to touch. The puncture site was then covered with a sterile transparent dressing. The patient was discharged home the next day with a disposable infusion pump delivering local anesthetic. On return to the ambulatory center 56 h later, the lumbar plexus transparent dressing was firmly adherent but easily peeled off. With moderate force, the catheter was then pulled away from the skin, from its distal attachment toward the puncture site. The catheter was then easily removed. No abrasion or irritation of the skin was noted along the previous location of the catheter.

The second patient was scheduled for a rotator cuff repair and placement of an interscalene catheter. Using a 3.8-cm insulated Tuohy needle and the same technique, a continuous interscalene block was performed.<sup>10</sup> After the catheter was placed, it was also secured using the 2-octyl cyanoacrylate adhesive as described with the first patient. This patient spent the first postoperative night in the recovery care center and was discharged home with a continuous interscalene infusion. On return to the ambulatory center 59 h after the initial block, there was no leaking at the insertion site. This catheter was also firmly

adherent but easily removed. A transparent strip of adhesive approximately 1 cm wide was still firmly attached to the catheter. No abrasion or irritation of the skin was noted along the previous location of the catheter.

A third patient was scheduled for left knee arthroscopy. The surgeon anticipated an extensive repair with expected intense postoperative pain. A continuous lumbar plexus block was performed without difficulty. After placing the catheter, it was secured using the 2-octyl cyanoacrylate adhesive. The arthroscopy revealed less damage than the initial physical examination and imaging studies and no formal operative procedure was done. The patient was expected to have minimal postoperative pain and be discharged the same day. In the post anesthesia care unit, despite its recent placement, the catheter was easily removed. A 5-cm abrasion was noted along the previous location of the catheter. The patient reported no pain with removal.

## Discussion

The bonding agent used in these three cases was 2-octyl cyanoacrylate (Dermabond® Topical Skin Adhesive). The adhesive comes as a sterile liquid in a monomeric formulation along with the colorant D & C violet #2. It is supplied in a single-use sterile blister pouch. The applicator is composed of a crushable glass ampoule contained within a plastic vial with attached applicator tip. Once applied to the skin, the liquid adhesive is slightly more viscous than water and polymerizes within 1-3 min. This forms a flexible coating that is permanently bound to the keratin in the epidermis and cannot be removed with water-based products. During the bonding process, a mild exothermic reaction takes place that may be perceived as heat by the patient. The bond may be weakened with acetone or petroleum jelly. Prior to administration, it is necessary to remove iodine-based antiseptic solutions because these may inhibit polymerization. After a typical application, the coating lasts approximately 5-10 days and sloughs off naturally with the epithelium. The product is approved by the Food and Drug Administration in place of topical sutures or staples and is marketed for its improved cosmetic results.

In these three cases, the glue formed a transparent shiny layer over the insertion site and appeared to completely seal the needle puncture site as well as the catheter. No break in the layer could be detected. However, as a precaution against contamination, a transparent dressing was also placed over the entry site. Because of the cyanoacrylate, the dressing adheres tighter than it normally does to skin. We hypothesize that placing the dressing over the glued layer may be superfluous but clinical data are necessary to support this.

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An obvious disadvantage of an adhesive that binds so permanently to the skin is seen when a catheter needs to be removed soon after administration. During this time, the epidermis may not have had time to slough off, making removal difficult. In the third patient presented here, this situation occurred. Despite the tight bond, we were able to peel the catheter from the skin. This maneuver created a thin (diameter of the 20-gauge catheter) 4-cm-long abrasion that was barely visualized, where the keratin of the epidermis was removed with the catheter. We suspect that if the catheter had been glued with a greater surface area (coiled on the skin) or below a flat piece of plastic it would have been harder or more traumatic to remove.

In summary, this technique offers a simple, alternative method to secure a catheter for a prolonged period of time. Because of the permanent bond, this method may be advantageous in highly mobile locations as well as areas subject to perspiration. The technique may have applicability for other uses, such as epidural catheters, invasive lines, and surgical drains. Given the results in these patients, further study comparing this technique with more traditional methods seems warranted.

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## A Simple Arm Positioning Aid for Fracture Table Cases

*To the Editor:*—When positioning a patient on the fracture table for open reduction/internal fixation of a femoral neck fracture, it is commonly necessary to secure the arms over the chest in a crossed fashion to avoid contact with the fluoroscope. Most hip fracture patients are elderly, and are at risk for skin abrasions if tape is used for this purpose. Wrapping sheets around the patient to secure the arms can prevent access to the peripheral intravenous site, and may not adequately restrain the arms during manipulation of the lower extremities by the surgical team.

We have successfully been using a soft foam donut-style headrest to gently but securely restrain the patient's arms across the chest. This method is well tolerated by a conscious patient with a regional block. It allows unobstructed access to hands and arms for peripheral venous or arterial line manipulation, and permits the use of a standard safety belt to hold the patient on the table.

To use a foam doughnut headrest for this purpose, first insert the patient's left arm into the hole and advance the headrest above the elbow until it is around the distal third of the humerus. Position that arm across the chest with the left hand lying over the right biceps area. Then cross the right forearm over the left forearm so the left wrist rests in the antecubital area of the right arm, and insert the right hand and wrist into the hole. The circular insert from the foam headrest may then be placed between the arms to pad one from the other. Of course, right and left arm positions may be switched as dictated by line placement or convenience. The final arrangement is shown in figure 1.



**Fig. 1.** Arms secured by foam donut headrest with circular foam insert between wrists.

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