In Reply.—We appreciate Dr. Youngs' interest in our study of transient neurologic symptoms (TNS) after spinal anesthesia with lidocaine and prilocaine. Her letter raises important issues concerning local anesthetic neurotoxicity and the factors that affect TNS after spinal anesthesia.

As Dr. Youngs notes, rate of injection is a critical factor affecting subarachnoid distribution of catheter-injected local anesthetic. Data from our modeling studies suggest that slow injection through a caudally directed catheter results in maldistribution,2 with relatively high regional anesthetic concentrations, a pattern consistent with limited sensory anesthesia. Repetitive administration can overcome the restricted block, but anesthetic concentrations achieved within the subarachnoid space may be sufficient to induce permanent neurologic injury.2-4

Dr. Youngs suggests that maldistribution likewise may be an important factor for development of TNS after lidocaine spinal anesthesia. She also questions whether small-gauge pencil-point needles may promote maldistribution and whether their current popularity may account for the high incidence of TNS reported in recent studies. These hypotheses have intuitive appeal. However, they fail to recognize important distinctions between catheters and needles and between TNS and neurologic injury.

First, small-gauge needles have far less resistance than microcatheters because of their shorter length and slightly larger diameter. Although Dr. Youngs refers to a 1-ml injection over 10 s as “slow,” this is an injection rate that cannot be physically accomplished with a microcatheter. Second, 0.1 ml/s may be a slow flow rate, but when used with a 25-gauge needle, it induces a high-stream velocity, and this promotes mixing. Additionally, needles are generally positioned near the peak of the lumbar sacral curve, which encourages solution to move cephalad and caudal. The result is generally a reasonably favorable distribution with relatively low subarachnoid concentrations. For example, when 1 ml lidocaine, 5%, was injected over 10 s in our model, the highest concentration measured in the mock cerebrospinal fluid was 0.5%, a concentration only 25 to 50% of that achieved with a sacrally directed 20-gauge catheter.2 Similarly, other investigators subsequently reported that significant maldistribution did not occur with solution administered at 2 ml/10 s, even when injection was made through a 25-gauge Whitacre needle with the side port directed sacrally.3 Unfortunately, these authors stated in the manuscript that “sacral needle direction and slow rate of injection with Whitacre needles may predispose to neurotoxic concentrations,”5 a conclusion often cited without recognizing that “slow” was defined as 0.035 ml/s, an injection rate not routinely used in clinical practice. In our clinical study of TNS, all injections were performed with the side port of the needle directed cranially.3 Our injection rates were not timed. However, we believe they were between 0.1 and 0.2 ml/s.

These theoretical considerations aside, the available data do not identify neurotoxic maldistribution as an important factor for development of TNS. In the three studies referenced by Dr. Youngs,1,6,7 and in previous,3-11 TNS occurred despite well-distributed sensory anesthesia, arguing against maldistribution as a contributing factor. Moreover, in a recent prospective epidemiologic study of 1,865 patients, we found that neither sensory dermatomal level nor adequacy of block affected risk of TNS.14 Perhaps most relevant, we did not observe an effect of needle type or needle size on the incidence of TNS.14

In conclusion, we do not believe TNS has emerged as a problem caused by the introduction of small-gauge needles in clinical practice. Although maldistribution can occur with any device, these needles do not favor a restricted distribution when used with normal technique. Moreover, maldistribution does not appear to be an important factor contributing to TNS, despite an apparent role in recent neurologic deficits associated with spinal anesthesia.3-15 This distinction underscores the uncertainty regarding a commonality of mechanism between transient pain/dysesthesia after spinal anesthesia and anesthetic-induced neurologic injury.

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Shearing of Plastic Coating of Stylet with Double Lumen Tube: Another Incident

To the Editor—Shearing of the plastic sheath of the stylet leading to endotracheal tube obstruction has been reported with smaller tubes.\(^1\)

It has been suggested that this happens because of the tight-fitting stylet with a pliable coating and a firm grasp of the endotracheal tube over the stylet.\(^2\)

Recently, we also experienced a similar incident with a 6.0 mm inner diameter Carlen’s double-lumen tube (DLT) (Portex, Kent, England), which has a stylet provided by the manufacturers. A 38-yr-old man presented for removal of a hydatid cyst of the left lung during general anesthesia. A 6.0 mm Carlen’s DLT was passed into the trachea easily with the aid of stylet provided with the tube. At removal of the stylet, it was noticed that the plastic coating over the distal part of the stylet was missing. The DLT was immediately removed. Fortunately the sheared part of the plastic coating was found stuck in the tube. The patient was ventilated with the face mask and was reintubated with the help of another stylet after lubricating it with sterile water-soluble jelly.

Shearing of the plastic coating of a stylet usually occurs at the point where it is angled to assist in the intubation.\(^3\) This also happened in our patient. Some force is bound to be exerted at this point during removal of stylet because of the contour of the airway and the tube. The more angulation produced, the greater will be this shearing force.

This case shows that shearing can occur even with larger tubes, and one should always inspect the stylet after withdrawal for integrity of the plastic coating.

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