

## CORRESPONDENCE

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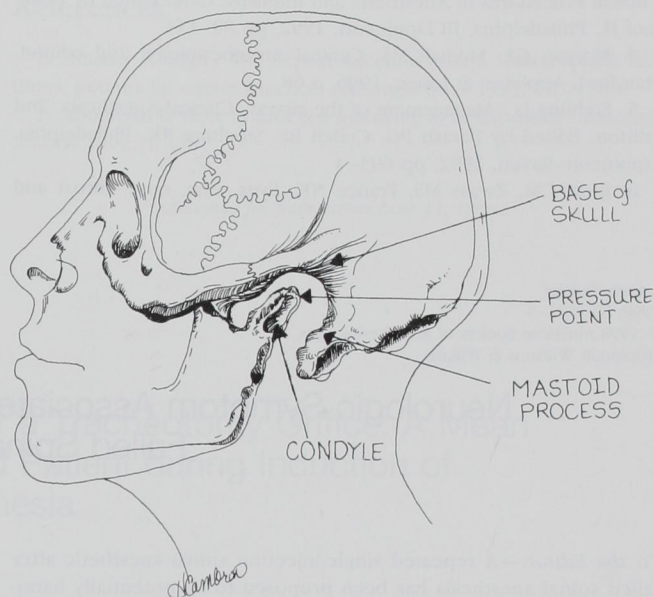
## Laryngospasm—The Best Treatment

*To the Editor:*—When considering the treatment of laryngospasm, standard textbooks of anesthesiology suggest virtually the same sequence: namely jaw thrust at the angle of the mandible while applying positive-pressure ventilation with oxygen, 100%, by bag and mask and, if that fails, administering succinylcholine, the recommended dose varying from 0.25 to 1 mg/kg intravenously or 4 mg/kg intramuscularly.<sup>1-8</sup> In addition, some texts recommend suctioning foreign material from the oropharynx, administering lidocaine, 1 to 1.5 mg/kg, and removing or avoiding any painful stimulus. One author recommended digital elevation of the tongue by inserting an index finger deep into the pharynx, a treatment of substantial risk to the therapist.<sup>9</sup>

Almost 40 years ago, Dr. N. P. Guadagni showed me a technique for prompt termination of laryngospasm, which I have used countless times with complete success. Because I have used the technique so often myself and have taught it to hundreds of residents and nurses, I presumed that it was common knowledge and well documented in the literature. However, a thorough literature search has not revealed any mention of it. The technique involves placing the middle finger of each hand in what I term the *laryngospasm notch*. This notch is behind the lobule of the pinna of each ear. It is bounded anteriorly by the ascending ramus of the mandible adjacent to the condyle, posteriorly by the mastoid process of the temporal bone, and cephalad by the base of the skull (fig. 1). The therapist presses *very firmly* inward toward the base of the skull with both fingers, while at the same time lifting the mandible at a right angle to the plane of the body (*i.e.*, forward displacement of the mandible or "jaw thrust"). Properly performed, it will convert laryngospasm within one or two breaths to laryngeal stridor and in another few breaths to unobstructed respirations.

The most common mistake made by those learning the technique is to place the fingers lower on the ramus of the mandible or at the angle of the jaw. Pressure and forward displacement of the mandible at these locations will elevate the tongue from the posterior pharyngeal wall but will not correct laryngospasm. To be effective for laryngospasm, the pressure must be firm and must be applied at the most cephalad portion of the laryngospasm notch. It is desirable to administer oxygen while performing the technique. This is easily performed by holding a mask over the patient's face with the thumb and index fingers of each hand while using the middle fingers for applying pressure and forward displacement. The technique is effective in infants, children, and adults. Because the patient is making respiratory efforts at the time of treatment, there is no need to apply positive pressure on the reservoir bag of the anesthetic machine, although an assistant can do so if the anesthesiologist wishes. I believe this technique for treatment of laryngospasm is far superior to those recommended herein because it is absolutely reliable, it resolves the spasm more rapidly than positive pressure ventilation, and it is much quicker and safer than administering succinylcholine or lidocaine. The technique also may be used to maintain a patent airway during mask anesthesia.

The obvious question is, Why does it work? Unfortunately, a sound, scientific answer cannot be provided. It works in part because forward displacement of the mandible corrects airway obstruction caused by the



**Fig. 1.** Schematic illustration of laryngospasm notch bounded anteriorly by the condyle of the mandible, posteriorly by the mastoid process, and superiorly by the base of the skull. Digital pressure is applied firmly inwardly and anteriorly on each side of the head at the apex of the notch (see pressure point arrow), which is slightly cephalad to the plane of the earlobes (not shown).

tongue falling back against the posterior pharyngeal wall. However, contrary to the recommendation that painful stimulation be avoided, an essential component of the treatment is the severe pain that the patient experiences because of the firm pressure that is applied to the ramus of the mandible, the facial nerve, and perhaps the deep lobe of the parotid gland. The parotid gland is innervated in part by the glossopharyngeal nerve, which in turn has connections with the vagus nerve and the superior cervical sympathetic ganglion by way of the petrosal ganglion.<sup>10</sup> The interconnections of the nerves at this location are complex and specific functions are not completely understood. It is likely that the painful stimulus relaxes the vocal folds and vocal cords by way of either the parasympathetic or sympathetic nervous systems.

The thanks Helen Cambron, R.N., for the illustration.

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## Neurologic Symptom Associated with a Repeated Injection after Failed Spinal Anesthesia

*To the Editor:*—A repeated single-injection spinal anesthetic after failed spinal anesthesia has been proposed to be potentially harmful.<sup>1</sup> We present a patient in whom neurologic symptoms developed associated with repeated single injection after failed spinal anesthesia. In this patient, dibucaine was repeatedly injected into the subarachnoid space. Although this is an agent virtually never used in the United States, the clinical course of the patient may provide important issues from a theoretical point of view when clinicians deal with failed spinal anesthesia. A 33-yr-old woman with no medical history was scheduled to undergo conization. The patient was placed in the right lateral position on a horizontal operating table. A 25-gauge Quincke needle was introduced into the subarachnoid space at the L<sub>3</sub>-L<sub>4</sub> interspace on the first attempt, and clear cerebrospinal fluid (CSF) flowed freely. Hyperbaric dibucaine, 7.5 mg, was injected, commercially prepared as Percamin S (Teikoku Kagaku, Tokyo, Japan; 0.3% dibucaine in 5% sodium chloride solution, specific gravity 1.037). Clear CSF was aspirated immediately before and after the injection. There were no signs of pain or paresthesia during insertion of the needle or during injection. The patient then was turned to the supine position on a horizontal operating table. Because she could flex fully both knees and feet 15 min after the spinal injection, we decided to repeat the lumbar puncture. At this time, we failed to test for a block by an evaluation of the sacral dermatomes. The second dural puncture was performed on the first attempt with a 25-gauge Quincke needle at the L<sub>3</sub>-L<sub>4</sub> interspace, with the patient in the right lateral position on a horizontal operating table. The CSF that flowed out of the needle still was clear. Six milligrams of the same anesthetic was injected into the subarachnoid space. Clear CSF was aspirated immediately before and after the injection. Neither pain nor paresthesia was elicited during placement of the needle or drug injection. The

patient was turned to the supine position on a horizontal operating table, and sensory analgesia to pin-prick was reached at S<sub>1</sub> 10 min after the subsequent spinal injection. After the patient was placed in the lithotomy position, the gynecologic procedure was uneventful and lasted 25 min. When the operation was terminated, a pin-prick test revealed the sensory analgesia to be L<sub>1</sub>, and a Foley urinary catheter was inserted into the bladder. On the morning of the first postoperative day, the patient first noted the loss of sensation in the buttocks and was unable to void. A urinary catheter was used before noon. At this time, the patient started to complain of numbness in bilateral S<sub>2</sub>-S<sub>3</sub> dermatomes. She needed an indwelling urinary catheter until the seventh postoperative day, and thereafter she could urinate in an interrupted stream with the help of considerable straining. There was no bowel dysfunction or motor weakness. Magnetic resonance imaging performed at the twentieth postoperative day showed no abnormality in the lumbosacral spine. Urinary difficulties completely resolved within 4 weeks. The numbness in the buttocks gradually subsided but continued for 6 weeks.

The neurologic symptom observed in this patient may not be associated with trauma because there were no signs of pain or paresthesia during insertion of the needle or during injection. Clear CSF was aspirated before and after the injection, both in the initial and the subsequent punctures. Consequently, the combined dose of hyperbaric local anesthetic most likely was delivered into the subarachnoid space. In the current patient, the total dose of dibucaine from the two injections exceeded that recommended for single-injection spinal anesthesia. In addition, we repeated lumbar puncture at the L<sub>3</sub>-L<sub>4</sub> interspace at which the initial puncture was performed. The restricted sacral distribution indicates that the combined dose was not diluted by CSF, resulting in the regional concentration to be neurotoxic. Maldistribution of hyperbaric local