

that this technique may be preferable in cases of suspected or known cervical spine injury.⁸ This has been our experience also, and in fact we frequently use the lightwand at our institution for the intubation of patients with cervical spine injuries.

The major disadvantage of the lightwand technique is the need for an extra piece of equipment with the ever-present risk of equipment failure, which contrasts with the blind nasal technique and its lack of required apparatus. The need to dim the room lights may also be viewed as a disadvantage. However, we believe that the benefits of faster intubation, less trauma, and lack of a need for a spontaneously ventilating patient far outweigh the disadvantages. It should be remembered that other techniques for managing the difficult airway are either dependent on sophisticated equipment requiring frequent practice to maintain skills (*i.e.*, the fiberoptic laryngoscope) or more invasive methods such as the retrograde wire technique¹ or cricothyroidotomy.⁴

In summary, lightwand-guided endotracheal intubation has recently been compared with direct-vision laryngoscopic intubation of the trachea.⁸ The lightwand technique was found to be similar to the direct laryngoscopic technique in efficacy, safety, and incidence of complications. We chose to compare the lightwand with blind nasal endotracheal intubation because the blind nasal technique is a frequently used alternative method, particularly when the patient has a known or suspected difficult airway. We found lightwand-guided endotracheal intubation to be

superior to the blind nasal approach in terms of greater speed, fewer attempts, and reduced incidence of complications.

We recommend the lightwand technique as an easily learned, highly efficacious method for endotracheal intubation of the awake patient, as well as for management of the difficult airway.

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Cervical Epidural Steroids in Reflex Sympathetic Dystrophy

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Reflex sympathetic dystrophy, or algodystrophy, is the result of dysfunction of the spinal cord and neuraxis,^{1,2} and/or dysfunction of the peripheral nerves.³ Early signs and symptoms of reflex sympathetic dystrophy are burn-

ing pain, hyperpathia, allodynia, vasomotor, and sudomotor changes.^{3,4} Weakness, atrophy, and trophic changes occur at a later stage of the disease. The three grades are: 1) clinical symptoms of classical causalgia, *i.e.*, severe pain, with severe vasomotor and sudomotor changes; 2) diffuse pain with mild vasomotor and sudomotor changes; and 3) borderline between normal response to trauma and the previous grades.³ Without treatment, the condition passes through three stages^{3,4} and is downgraded.

Stage I is characterized by pain, hyperesthesia, hyperalgesia, localized edema, muscle spasm, and tenderness. In this stage, regardless of grade, the syndrome is still considered as accessible to treatment. Therefore, it is imperative to recognize the syndrome in an early condition. Treatment consists of either blocking the sympathetic nerve supply by injecting the sympathetic chain and gan-

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glia with a local anesthetic³ or intravenous injection of guanethidine during occlusion of the circulation.⁵ Also, physical therapy is considered a useful adjuvant.

Stage II develops in approximately 3–6 months in untreated patients. Pain decreases gradually, edema spreads, and trophic changes become apparent. Radiograms will show spotty osteoporosis. In Stage III there are mostly irreversible and marked trophic changes. Interosseous muscle atrophy appears, and the limb is useless. There is diffuse bone atrophy.

The occurrence of reflex sympathetic dystrophy in cortex motor area damage,⁶ the ability of oral corticosteroids to improve clinical features of reflex sympathetic dystrophy,^{6–10} and the presence of specific target neurons for 3H-corticosterone and glucocorticoid receptor immunoreactive nerve cells in laminae 1, 2, 3 and 7 in the rat spinal cord^{11,12} made us consider the epidural administration of corticosteroids in a patient with reflex sympathetic dystrophy resistant to conventional therapy.

REPORT OF A CASE

A 50-yr-old woman presented, 1 month after surgical resection of the first rib and scalenotomy, with continuous burning pain in the left hand and part of the forearm. The pain was aggravated by slight touch. On examination the left hand was pale and cold. The skin was glossy and the nails were brittle. The triceps tendon reflex was absent on the left side; also, a slight paresis of the triceps, pronator teres, and supinator muscles was found. Slight touch was extremely painful in the first two thoracic dermatomes (allodynia). A lower left brachial plexus lesion and reflex sympathetic dystrophy, Grade 3–2, Stage I, of the left upper limb was diagnosed.

Initially, bupivacaine 0.25% 10 ml was injected twice a week in a 6-week period in close proximity of the left stellate ganglion (stellate ganglion block by anterior [paratracheal] approach¹³). The effectiveness of the block was assessed by thermography. However, this treatment, together with physical therapy, failed to institute significant improvement, as indicated by pain self-rating and physical examination. Therefore, iv injection of guanethidine (10 mg diluted in 25 ml of prilocaine 0.25% with 5 mg of heparin) during occlusion of the circulation of the affected arm⁵ was tried twice a week for a period of 6 weeks. Again, an observable but incomplete response (short-lasting vasodilatation) occurred, while the disease progressed to Stage III.

After cervical epidural puncture with a 16-gauge Tuohy needle, 60 mg of methylprednisolone acetate in 3 ml of NaCl 0.9% was injected. A noticeable, significant increase in the left hand temperature occurred within 1 h after injection. The patient reported improved functioning and a marked reduction of pain during the first week after the injection. Subsequent cervical epidural steroid injections, once a week for 4 weeks, were associated with further improvement in that marked pain relief, improved motor control, and reduced muscular contracture and trophic changes occurred. An observed side effect in the course of treatment was the occurrence of spontaneous contractions of muscles in the neck.

DISCUSSION

The functional state of the nervous system can be considered in terms of the input–output relationships of the sensorimotor reflex arc. As an input, the action potentials are conducted along the primary afferents of the spinal

cord. Their nerve endings are found in the deeper layers of the spinal cord (for example, laminae 3 and 4)^{14–17} at and rostral to their level of entry. The output at the spinal level is the secondary local motor and sympathetic activity, as well as the generation of action potentials along ascending tracts to supraspinally located structures. Some of these are part of the feedback loops that are known to participate functionally in the “pain control system” by means of descending inhibitory tracts.

The initial therapy (*i.e.*, blockade of the stellate ganglion, intravenous guanethidine during occlusion of the circulation of the arm, and physical therapy) modified the input–output relationship through the peripheral part of the sensorimotor reflex arc. Corticosteroids are considered to have central effects. In the spinal cord of rats—within the substantia gelatinosa, laminae 3 and 7, and especially in the spinal motor area—neurons can be labeled with corticosterone.^{10,12} In animal experiments motor neurons are influenced by corticosteroids.¹⁸ Motor neurons in the cortex⁶ and at the spinal level,³ the latter incorporated in a sensorimotor reflex arc, play a role in reflex sympathetic dystrophy, although the exact function of these neurons in this syndrome is not known. However, corticosteroids may have a beneficial influence at the spinal level by modifying the functional state of neurons, which constitute the neuronal pool between input and output and which are involved in the sensorimotor reflex arc with supraspinal control in reflex sympathetic dystrophy.

In analogy to the perispinal route of administration of opioids, the main reason and intention to use the perispinal route for the administration of the corticosteroids is to limit the presence of the drug to the considered target of its effect.

Due to additives, intrathecal injections of solutions with steroids can cause serious side effects.^{19–22} Epidural treatment with steroids is not associated with such side effects,²³ although there is experimental evidence of lasting depression of the pituitary–adrenal axis.^{24,25}

Indications of a modification in input–output relationships are the occurrence of spontaneous contractions of the muscles in the neck in the course of epidural steroid administration, increase of skin temperature, reduction of trophic changes and contractures, and improved functioning. Further research, both experimentally and clinically, is needed to establish the actual relationships at the spinal level and their consequences for the therapy of reflex sympathetic dystrophy.

Even though the pathogenesis of the reflex sympathetic dystrophy syndrome remains to be elucidated, central mechanisms involving a dysregulation of spinal input–output relationships should be considered. Possibly, epidurally injected steroids may restore such relationships and improve the clinical condition of such patients.

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No Prophylactic Effect of Early Sympathetic Blockade on Postherpetic Neuralgia

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We propose to determine whether sympathetic blockade given during an acute herpes zoster infection could

prevent the development of postherpetic neuralgia. There is no reliable treatment available for postherpetic neuralgia; however, various treatments^{1,2} have been used

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