is dangerous because the adult achondroplast's spinal cord frequently fills the spinal canal. However, Walts et al., while noting technical difficulty in performing subarachnoid or epidural anesthesia in eight achondroplastic dwarfs, did not have neurologic sequelae. However, they considered general anesthesia preferable because of the neurologic problems associated with achondroplasia. Although the safe use of ketamine (2 mg/kg) for anesthetic induction in cesarean section has been established, clinical experience with ketamine in AIP is limited. Rizk et al. describe a patient with AIP who underwent induction of anesthesia with ketamine (4 mg/kg) twice within 1 week without neuropsychiatric sequelae. Experimental data are conflicting. Although ALA synthetase activity significantly increased in 17-day-old chick embryos injected with ketamine (5–15 μg), studies in rats failed to demonstrate a change in activity of this enzyme after ketamine 20 mg/kg was administered ip.

The use of barbiturates, narcotics, and possibly regional techniques were contraindicated in this patient. The sparse documentation of clinical experience with various anesthetics in AIP, ketamine, and N₂O with a succinylcholine drip was a reasonable and ultimately safe alternative.

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Total Spinal Blockade during Local Anesthesia of the Nasal Passages

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Total spinal anesthesia is a rare complication of properly performed spinal anesthesia. More commonly it is a complication of lumbar epidural or caudal anesthesia. On rare occasions, a total spinal block has occurred as a complication of stellate ganglion block, brachial plexus block, intercostal nerve block, lumbar paravertebral sympathetic nerve block, or retrobulbar block. Recently, we have encountered a case of total spinal block resulting from injection of local anesthesia into the nasal passages.

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REPORT OF A CASE

A 42-year-old woman was admitted with a diagnosis of chronic ethmoiditis. She had a persistent headache of 3 weeks' duration that was located laterally in the right orbit with radiation to the right ear. An intranasal anterior ethmoidectomy was scheduled to be performed under local anesthesia administered by the otolaryngologist.

Medical history revealed no previous systemic neurologic abnormalities. Anesthetic history included an abdominal hysterectomy and a mandibular resection performed under general anesthesia without complications. Current medications were limited to estrogen, which the patient had taken since her hysterectomy. She was noted to have an allergy to ampicillin.

On physical examination, pertinent positive physical findings were limited to examination of the nasal passages and paranasal sinuses. Tenderness to palpation was noted over the right frontal sinus. The right middle turbinate was noted to be compressing the lateral wall of the nose; thus obstructing the middle meatus. Local anesthesia of the middle meatus with topical application of 2% tetracaine and phenylephrine relieved her symptoms.

She was a normally developed woman (175 cm, 63 kg) without evidence of other physical abnormalities. No neurologic abnormalities were noted. Admission laboratory data, including hemogram, coagulation studies, urinalysis, roentgenogram of the chest and paranasal sinuses, and electrocardiogram, were normal.

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Premedication included pentobarbital 150 mg, im, approximately 3¼ h before surgery, and meperidine 50 mg, promethazine 50 mg, and atropine sulfate 0.4 mg given im 1 h before surgery. On arrival in the operating room, the patient was calm and cooperative. An i.v. infusion of 500 ml of D_{2}W was started.

The patient was positioned on the operating table and draped, and administration of topical anesthesia was initiated with the use of cotton pledges saturated with 10% cocaine applied to the middle turbinate and middle meatus. A total of approximately 4 ml 10% cocaine were used. After the application of cocaine, a submucosal injection of 4 ml 2% lidocaine with 1:100,000 epinephrine was made into the nasal septum, the middle meatus, and the middle turbinate. A 22-gauge spinal needle was used for the submucosal injection. About 10 min later, the surgical procedure started. During this time the patient was awake, cooperative, and conversing intermittently with the surgeon. The middle turbinate was fractured and the anterior ethmoidal cells were removed with a sphenoid punch. Approximately 9 min after surgery had begun, the patient complained of pain. Again, a submucosal injection of 2–3 ml 2% lidocaine with epinephrine was made in the region of the anterior ethmoidal air cells. The surgeon inquired whether the painful area still was painful, and she responded saying that it was not. The surgical procedure was then completed without incident. Within approximately 3 min, the patient became unusually quiet and did not respond to questioning. The surgical drapes were removed and the patient was found to be comatose and apneic. She was noted to have a palpable pulse with a rate of 110 beats·min⁻¹.

Ventilation was controlled with oxygen via mask, and subsequently an endotracheal tube was inserted without the need for a muscle relaxant. Positive-pressure ventilation with oxygen was continued. At this time, the heart rate was 110 beats·min⁻¹ and arterial blood pressure was 170/115 mmHg. Analysis of arterial blood gases revealed a pH of 7.50, P_{a}CO_{2} 33 mmHg, P_{a}O_{2} 505 mmHg, and base deficit 3.0 mEq/l. The pupils were fixed and maximally dilated. She was areflexic in the upper extremities and unresponsive to pain in the head, neck, and upper torso. Babinski reflexes were absent. Knee reflexes were noted to be symmetrical but hypoactive. A slight withdrawal reaction was noted in response to deep painful stimulation over the pretilial areas.

During the course of the resuscitation, the heart rate gradually slowed to 54 beats·min⁻¹, and the blood pressure decreased to 100/80 mmHg. Atropine sulfate 0.6 mg was administered i.v., which restored the heart rate to 78 beats·min⁻¹ and the blood pressure to 130/100 mmHg. After 55 minutes of resuscitation, the patient regained consciousness. She opened her eyes, moved all extremities on command, and was noted to have resumption of normal ventilation. The trachea was extubated in the operating room and she was transported to the recovery room. She did not at any time show any central nervous system signs of excitement or seizure activity suggesting cocaine toxicity.

The postoperative course was uneventful. A complete neurologic examination in the recovery room was within normal limits with exception of the pupils, which were noted to be moderately and asymmetrically dilated and nonreactive. Subsequent neurologic examinations were completely normal. A cranial computerized axial tomography (CAT) scan and EEG were normal. The patient was discharged in good condition on the third postoperative day. The discharge diagnosis was total spinal anesthesia resulting from accidental subarachnoid injection of lidocaine.

**DISCUSSION**

Total spinal anesthesia resulting from local anesthesia of the nasal passages has not been reported previously, despite the common use of local anesthetic techniques by otolaryngologists. Yet the potential for such a complication is readily evident. Topically administered cocaine anesthesia of the nasal passages commonly is supplemented by submucosal infiltration with other local anesthetics.

Attempt to block the anterior ethmoidal nerve by submucosal infiltration may result in inadvertent penetration of the cribiform plate and dura mater, resulting in subarachnoid injection as illustrated in figure 1. The cribiform plate which forms the floor of the anterior cranial vault, is a portion of the ethmoid bone, an exceedingly light and spongy structure. The cribiform plate is thin and has multiple perforations that give it a sieve-like appearance. Fractures of the cribiform plate resulting in cerebrospinal fluid rhinorrhea are not uncommon. Penetration of the structure with a needle during submucosal infiltration in the roof of the nose is very possible. The rapid onset of total spinal block after attempt to supplement topical anesthesia in the anterior superior portion of the nose suggests that a direct subarachnoid injection is the probable mechanism of the complication observed in this case.

In addition to the probability of a direct intracranial injection, a submucosal injection of the olfactory mucosa may result in perineural spread into the cranial subarachnoid space. Moore et al. demonstrated that local anesthetics injected into peripheral nerves may spread via the perineural spaces to the spinal subarachnoid space.

The anatomy of the olfactory mucosa and its neural structures make the possibility of perineural spread of
local anesthetics or other injected substances very likely. The nerve fibers originate as processes of the olfactory cells in the olfactory mucosa, which coalesce to form a plexiform network in the mucous membrane. They then are collected into about 20 branches, the fila olfactoria, which transverse the cribiform plate and end in the olfactory bulb. Each branch has a tubular sheath of dura mater and pia arachnoid. The dura is continuous with the periosteum of the nose, and the pia arachnoid forms the perineural membrane of the fila olfactoria. The perineural spaces within the olfactory mucous membrane are continuous with the subarachnoid space above. The extensions of the subarachnoid space around the bundles of the olfactory nerve fibers have been regarded as a possible source for spread of certain meningeval infections into the cranial cavity.\(^2\) Colored solutions injected into the cranial subarachnoid space have been demonstrated to penetrate into the perineural sheaths of the fila olfactoria as well as into the lymph capillaries of the olfactory mucosa.\(^4\) Solutions injected into the olfactory mucosa also may find their way to the cranial subarachnoid space via the perineural or lymph spaces.

In this case, a total of 400 mg cocaine (4 ml of 10% cocaine) was used by topical application with pledgets. This exceeds the generally recommended maximal dose of 200 mg. Despite this, no symptoms of cocaine toxicity (excitement, delirium, nausea, vomiting, or convulsions) were observed.

Although the normally recommended dose of 200 mg cocaine sometimes is exceeded in clinical practice, the incidence of toxic reactions is quite low.\(^5\) The maximal safe dosage of cocaine is determined by the route of administration, the site and method of application, and the rate of metabolism. As a rule, larger doses can be applied more safely to the nasal mucosa than to the tracheobronchial mucosa. Generally greater absorption of cocaine occurs when it is sprayed or painted on the mucosa rather than when it is applied with pledgets, because much of the cocaine may remain in the pledget.\(^6\) Nevertheless, occasionally toxic plasma concentrations have been documented after intranasal application of 5% cocaine.\(^7\) In this case, the lack of toxic symptoms plus consideration of the site and method of application mitigate against cocaine toxicity as a significant contributory factor. Passage of cocaine into the cerebrospinal fluid after topical application to the olfactory mucosa appears to be unlikely because of the rapid onset of total spinal blockade after previous injection of lidocaine. Furthermore, the duration of approximately 40 min is compatible with lidocaine-induced subarachnoid blockade.

Total spinal block is an unusual complication of regional anesthesia. It may occasionally occur as a result of local anesthesia of peripheral nerves, as well as of spinal and epidural anesthesia. We have described such a case resulting from local anesthesia of the nasal cavity. Attempt to administer local anesthesia in the roof of the nose by submucosal infiltration entails significant risk of passage of the anesthetic into the cranial subarachnoid space. Topical application of anesthetics in this portion of the nose clearly is a safer technique.

Death resulting from total spinal block has been attributed mainly to a lack of preparedness for sudden ventilatory failure. With prompt diagnosis and appropriate resuscitation, no patient should die as a result of the accidental administration of a total spinal anesthetic.\(^1\) Because otolaryngologists most often administer local anesthesia of the nasal passages, it is incumbent upon them to be mindful of the possibility of total spinal block.

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