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Facial Paralysis after General Anesthesia

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Damage to peripheral nerves in the anesthetized patient has been attributed to ischemia caused by direct compression or to stretching of the nerve.†‡ Paralysis of the brachial plexus, radial nerve, ulnar nerve, and common peroneal nerve has been frequently reported. In contrast, facial nerve paresis has been rarely described. The following case is reported as an instance of a fortunately rare complication of general anesthesia.

REPORT OF A CASE

A 54-yr-old male physician presented for arthroscopic menisectomy of the knee as an outpatient procedure. Past medical and surgical history was unremarkable and the decision was made to proceed with general anesthesia. After uneventful induction of anesthesia with thiopental 400 mg and fentanyl 75 μg iv, anesthesia was maintained with halothane and nitrous oxide in oxygen. An appropriate face mask was used supported by a Connell harness. Throughout the 75 min of anesthesia, cardiovascular and respiratory variables remained normal, and the only difficulty encountered was in maintaining a clear airway by the usual method of holding the chin forward. Lifting the jaw forward by bilateral digital pressure applied behind the angle of the mandible resulted in an unobstructed airway. Surgery and anesthesia proceeded uneventfully. Recovery was prompt and without immediate complication other than a mention by the patient that the angles of his jaw were sore. The next day a bilateral partial facial nerve paresis developed with striking weakness of the lower facial muscles, poor control of saliva, and bilateral parotid swelling and tenderness. The paresis resolved over 3 weeks, and the parotid swelling receded over 5 weeks to complete recovery.

DISCUSSION

Pressure behind the mandible to relieve pharyngeal respiratory obstruction is employed with such frequency that it is surprising that damage to the facial nerve is not more frequently reported. The only previously reported cases in the English literature were those of Fuller and Thomas[2] in 1956. Their first patient manifested weakness and asymmetry of the face and impaired control of food during chewing following 30 min of pressure behind the angles of the mandible. Full function returned after 3 months. A second patient had minor weakness of one side of the mouth after 10 min of forward digital pressure. Recovery was complete in 3 weeks. Unlike the present case, the degree of pharyngeal obstruction in both patients was severe enough to warrant fairly early endotracheal intubation.

Britt and Gordon[2] cite only the cases of Fuller and Thomas[1] in their extensive review of peripheral nerve injuries associated with anesthesia. Several standard textbooks[3–6] draw attention to the danger of injury from stretching of the nerve caused by forward traction on the jaw and from direct pressure applied to the nerves as they pass over the ramus of the jaw. They cite either no references or the solitary case reports of Fuller and Thomas.[1] A total of six cases of involvement of the facial nerve has been noted in the German and Russian literature.[7,8,§]

After leaving the skull *via* the stylomastoid foramen, the facial nerve becomes superficial to the mandibular ramus and enters the parotid gland where it divides into its branches. While the temporal and zygomatic branches turn upward, the buccal and mandibular branches turn downward to run over or behind the ramus of the mandible where pressure may render them liable to injury, a factor that may be compounded by the stretching force caused by strong forward traction.

Lisitsyn[8] studied 20 facial nerve dissections and found three types of anatomic variation, all or any of which could be pertinent to the present and other cases. First, the trunk may give off its branches at varying levels in relation to the parotid gland. Second, the nerve may lie superficial rather than deep to the parotid gland. This renders the buccal branch liable to pressure injury from a too tightly fitted mask or head strap. My patient noted an inability to whistle, an act requiring a fully functioning orbicularis oris muscle, the lateral part of which is supplied by the buccal branch of the facial nerve. Finally, the mandibular branch usually runs high in relationship to the angle of the mandible and will not be brought to lie on the edge of the angle no matter how far the jaw is pushed forward.

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† Denny-Brown D, Brenner C: Paralysis of nerve induced by direct pressure and by tourniquet. Archives of Neurology and Psychiatry 51: 1–26, 1944.

‡ Denny-Brown D, Doherty M: Effects of transient stretching of peripheral nerve. Archives of Neurology and Psychiatry 54:116, 1945.

§ Polovinkova EP: Paralysis of inferior branch of facial nerve following anaesthesia. Sovetskaia Khirurgii 10:124–126, 1935.

However, in rare cases, it occupies a low position and bends around the edge of the angle of the jaw, where any pressure would readily cause direct compression of nerve against bone. Possibly, the rarity of this complication lies in the rarity of this anatomic variant.

The cautionary moral in the management of this anesthetic complication concerns the "departure from usual" management simply because the patient was an esteemed physician colleague. Obviously, a really difficult or dangerous airway management problem would have been treated in this instance as in any other patient with prompt endotracheal intubation. But airway patency was not difficult to maintain provided the anesthesiologist was prepared to work harder than usual to provide anterior displacement of the jaw. In order to protect our colleague from the potential added morbidity of endotracheal intubation and the effects of a depolarizing muscle relaxant, we persisted with this well-intentioned plan. Physician patients should be treated as patients and not as physicians, and in this case early intubation would have prevented the complication described.

In summary, a patient with partial facial paralysis after general anesthesia is described. Early endotracheal intubation is called for when airway maintenance can only be achieved by strong pressure applied to the lower jaw.

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Patients with Burns Are Resistant to Atracurium

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Patients with thermal burns may demonstrate a different response than unburned patients when given muscle relaxants. Succinylcholine, a depolarizing muscle relaxant, may cause acute hyperkalemia,[1,2] whereas *d*-tubocurarine (DTC) and metocurine (MTC)—nondepolarizing muscle relaxants—have been shown to be less effective in several studies of burned patients.[3–5] As much as three times the dose of MTC used in normal subjects may be required to effect an equivalent degree of relaxation.[5]

Atracurium is a nondepolarizing muscle relaxant with few side effects up to three times its ED_{95} of 0.2 $mg \cdot kg^{-1}$.[6,7] It has a rapid onset and short duration of action, and its elimination is independent of the kidney and liver.[8] As such, it might be a suitable alternative to succinylcholine to facilitate endotracheal intubation and maintain relaxation.

The purpose of this study was to determine the response to an intubating dose of atracurium, 0.5 $mg \cdot kg^{-1}$, in burned patients compared with that in a population of normal unburned control subjects. We also investigated the relationship between the response to atracurium and: 1) the per cent of total body surface area burned (TBSA); and 2) the number of days postburn injury.

Methods

The study was approved by the Human Studies Review Committee of the University of Washington, and oral consent was obtained from the patients. Subjects for the study were 36 (22 men; 14 women) patients scheduled for excision of burn wound or scar revision. They ranged in age from 18 to 65 yr (33 ± 2.6 yr) and in size of burn from 1% to 80% of total body surface area (TBSA). Pa-

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