Transient swelling of the parotid glands following general anesthesia—
“anesthesia mumps”

BARBARA JACOBSON, CRNA
Scranton, Pennsylvania

This article focuses on the transient swelling of salivary glands during and following general anesthesia. A case report is presented as an example. To apprise both the anesthetist and patient of what to expect, the author delves into the possible causes of this condition.

Among the usual causes of acute and chronic swelling of the salivary glands are: epidemic parotitis (mumps), thyrotoxicosis, postoperative parotitis, amyloidosis, tuberculosis, systemic lupus erythematosi s, Wegener's granulomatosis, idiiosyncrasy to inorganic or organic iodides, radiation parotitis, chronic sialadenitis with calculi and Sjogren's syndrome.

Asymptomatic, noninflammatory parotid-gland enlargement occasionally is seen in association with nutritional deficiency, (lack of vitamin A), endocrine disorders, obesity, liver disease, and impaired glucose tolerance. Parotitis may also appear in patients with uremia, typhus, and relapsing fever.

The first report of swelling of the salivary glands in association with anesthesia was by Attas and associates. In December, 1966, soon after induction of anesthesia and intubation of the trachea in a Negro woman, it was noticed that all the salivary glands were greatly enlarged. During the next 15 minutes, the glands decreased in size; within 30 minutes, they were barely palpable. Since neither published literature nor intramural consultations threw light on the possible causes of this enlargement, the incident was almost forgotten until the same thing occurred in a second patient the next month.

The author observed the following case during April, 1977.

Case report

A 30-year-old, 80 kg Caucasian female was admitted for excision of a breast tumor with frozen section. Her history and physical were essentially normal. She denied any apparent complications from anesthetic experience which included: excision fibrocystic breast tumor, 1974; cystoscopy, 1975; and tubal ligation, 1976.

Preanesthetic sedation consisted of morphine sulfate 10 mg and scopalamine hydrobromide 0.4 mg (IM) given one hour prior to surgery. Anesthesia was induced smoothly with Thiopental 250 mg IV. Maintenance was uneventful with nitrous oxide and oxygen (4L:2L) administered by mask and fentanyl 0.10 mg IV. A Berman oral airway was utilized during the procedure. Total anesthesia time was 45 minutes. Recovery from anesthesia was rapid and smooth.

Two hours and forty-five minutes after the end of anesthesia, a marked swelling of both parotid glands was found. The patient expressed no discomfort or pain. The swelling disappeared within 6 hours, with no treatment.

At the postoperative visit, the pa-
tient admitted that following a previous anesthetic exposure, she had experienced severe swelling of the parotid glands. The swelling occurred after arrival back to her room and lasted for a much longer period of time. The swelling at that time disappeared without treatment; hence, the reason she did not realize it was an apparent complication.

Discussion

It is of particular interest to note that only three articles have been published in anesthesiology literature concerning surgical patients developing acute swelling of the salivary glands in association with general anesthesia. Attas described seven cases of glandular swelling associated with general anesthesia. Reilly reported three such cases on emergence from anesthesia. Neither author could relate the glandular swelling to age, sex, race, type of surgery, premedications, anesthetics, or past medical history. Yet, six of their reported patients were Negroes, comprising 60% of the total. The combination of atropine followed by the depolarizing neuromuscular-blocking agent, succinylcholine, was present in seven of their patients (70%). Valsalva maneuver, coughing, and straining were present in approximately 60% of the total cases.

Many conditions causing parotid swelling are associated with, or even are secondary to, an increase or decrease in the rate of salivary secretion. To determine if a correlation existed between volume of salivary secretion and salivary-gland enlargement, Attas collected secretions. The volume of secretion collected was correlated with the degree of salivary-gland enlargement according to an arbitrarily graded scale from 1+ to 4+. No correlation was found.

Matsuki, et al, reported five cases of transient swelling of salivary glands during and following endotracheal anesthesia. Tubocurarine was given to their five patients for muscle relaxation but no definite association with the onset of glandular swelling could be found. Matsuki was impressed that the violence of straining and coughing produced by intraoral manipulation for intubation is the most likely cause of the swellings. This is observed following peroral endoscopy in nonanesthetized patients as described by Maruyama, et al; Palmer; and Slaughter and Boyce.

Preoperative sedation consisted of narcotics and sedatives; neither general anesthetics nor muscle relaxants were given to these patients. One cause of the swelling is presumed to be due to air distention of blind branchial cleft remnants. This swelling is not so firm and can easily be squeezed flat. Another possible cause was straining produced by insertion of the endoscope during which Wharton’s duct or Stenson’s duct was distended or displaced transiently. This type is relatively firm and cannot be deflated by pressure.

Yet, another related cause of the swelling of the salivary glands may develop because of mechanical stimulation associated with the insertion of the endoscope which results in an increase in secretion of saliva and in distention or displacement of Wharton’s and/or Stenson’s duct. Matsuki deducted this same mechanism may be used to explain the cause of the swelling in association with endotracheal anesthesia.

Of interest to note is the fact that one of the patients reported on by Attas developed swelling after premedication and prior to induction of anesthesia. One of the cases reported by Reilly, as well as the case presented in this article, both consisted of smooth anesthesia courses administered via mask.

Anticholinergic drugs (belladonna alkaloids) are employed in preanesthetic medication to reduce the undesirable effects of excessive parasympathetic stimulation. All secretory glands have a dual innervation consisting of both parasympathetic fibers and sympathetic fibers. Many types of stimuli may provoke salivation. In the mouth, this may be simple contact with buccal mucosa, grinding of teeth, move-
ment of jaws, or placement of an oral airway. The use of anticholinergic drugs probably would not provide complete protection, since usual doses produce a partial muscarinic-type block.

Summary

The cause of the salivary gland swelling is, at present, unknown. Several factors, such as the administration of belladonna alkaloids and depolarizing neuromuscular blocking agents, straining, coughing and/or the procedure of endotracheal intubation are suggested as possible causes. The most common factor in the reported cases is the administration of a belladonna drug in premedication.

The swelling is usually bilateral but unilateral swelling has been described. It is completely painless but may be very frightening to both the anesthetist and the patient. The onset of the glandular swellings have occurred prior to induction, during maintenance, and following general anesthesia up to 24 hours. The swellings have disappeared between 30 minutes to 72 hours after occurrence. No particular treatment has been rendered.

As to the incidence of the abnormal response: Reilly reported 3 of 1,500 cases (0.2%) and Matsuki reported 5 of 3,000 cases (0.16%). The incidence may increase with more careful observation of surgical patients.

This benign anesthesia complication must be contrasted with other types of parotitis. It is of importance to be aware of the abnormal response in order to prevent unnecessary anxiety.

REFERENCES


AUTHOR

Barbara Jacobson, CRNA, is a 1963 graduate of Community Medical Center School of Nursing in Scranton, Pennsylvania. She is a 1968 graduate of Allentown Hospital School of Anesthesia in Allentown, Pennsylvania. She also attended the University of Scranton. Presently, she is a staff nurse anesthetist at Community Medical Center, Scranton, Pennsylvania.

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