Acute Transient Sialadenopathy during Induction of Anesthesia

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In December, 1966, soon after induction of anesthesia and intubation of the trachea had been accomplished smoothly in an obese Negro woman, it was noticed that all the salivary glands were greatly enlarged and of rubbery consistency. 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 (fig. 1) the next month. This abnormal response was seen in five more patients during the following six months (figs. 2 and 3).

FIG. 1. This 45-year-old woman had marked swelling of the right parotid and left submandibular glands immediately after smooth induction of anesthesia and intubation of the trachea. There was moderate swelling of all other glands. Note moderate swelling of left lacrimal gland and marked swelling of the left submandibular gland. The swelling subsided within 30 minutes.

Glandular swelling in these seven patients could not be related to age, sex, race, type of surgery planned or past medical history. All patients except one had received pentobarbital, meperidine and atropine before operation, in doses compatible with age and physical status. One patient had received atropine only, and parotid swelling was observed before induction of anesthesia (fig. 3). Induction of anesthesia had been smooth in three patients and was associated with coughing and straining in the other three. Since the only constant factor in six of the seven patients was administration of atropine followed by succinylcholine, a study was undertaken to determine if sialadenopathy could be produced at will by this combination of drugs.

METHOD

Stenson's and/or Wharton's ducts were cannulated in three patients and secretions were collected from a single parotid gland with a double suction cup. This approach was abandoned when it was found that the volume of secretion from one gland was not related to the volumes secreted by the other glands, that the collecting catheters were easily dislodged during intubation, and that cannulation of the ducts was sufficiently irritating to induce salivation by itself. All secretions were therefore collected from the oropharynx by a large-bore

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catheter with a graduated cylinder interposed in the suction line. Secretions were collected continuously for periods of five minutes each (a) before induction of anesthesia, (b) after the intravenous administration of 200 mg. thiopental, 60 or 80 mg. succinylcholine, and endotracheal intubation, and (c) during paroxysms of coughing purposely induced by endotracheal suction during extubation. The volume of secretion collected was correlated with the degree of salivary gland enlargement. Gland swelling was arbitrarily graded from 1+ to 4+, depending on number of glands involved, degree of enlargement (just palpable to obviously visible), and consistency ("rubbery" to "woody hardness").

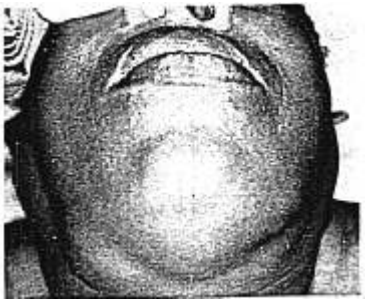

FIG. 2. Swelling of all salivary glands (parotid, submandibular, and submental) in a 42-year-old woman. Induction of anesthesia was stormy and intubation was associated with profuse secretions.

RESULTS

The lack of correlation between volume of secretion collected and salivary gland enlargement is readily apparent in figure 4. Patients 1, 2, 5 and 6 received 0.4 mg. atropine intramuscularly two hours before study and produced copious flows of watery secretions after succinylcholine. Patients 3, 4, and 7 received 0.6 mg. atropine intramuscularly 30–60 minutes before study and produced slight secretions. Although healthy conscious subjects given succinylcholine reported that the drug produced dryness of the mouth,[1] succinylcholine apparently stimulated secretions in these patients. Enlargement of salivary glands was not related to the volume of secretion but did appear in four of seven patients in whom coughing was induced at the end of operation and in the only patient who strained after intubation of the trachea (Patient 6). The swelling persisted only for 3–5 minutes, and in no case was it as dramatic as that observed in the first group of patients.

DISCUSSION

Acute, transient swelling of one or both parotid glands occurring in nonanesthetized patients without obvious cause has been described.[2] The enlargements varied from a slight fullness of the face to swelling so prominent that mumps was diagnosed by mistake. The glands were uniformly smooth and firm, and could not be palpated between attacks. Swelling was frequently associated with allergic symptoms, and plugs containing eosinophils were recovered in the parotid secretions of some patients. Maynard[3] suggested that the rapid onset of swelling and decompression was due to retention of the saliva resulting from a blocking of the ducts by swelling of the lining epithelial cells. Blockage of the salivary ducts could explain the swelling in the patient whose swelling preceded induction of anesthesia. It could not, however, explain the swellings which appeared in the three patients in whom stormy inductions were accompanied by profuse salivary secretions. Glandu-

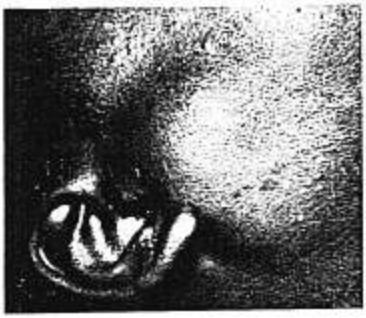

FIG. 3. Right parotid swelling in an 18-year-old youth scheduled for repair of an inguinal hernia. This patient received only 0.6 mg. atropine, intramuscularly, an hour before the photograph was taken. No anesthetic had been given.

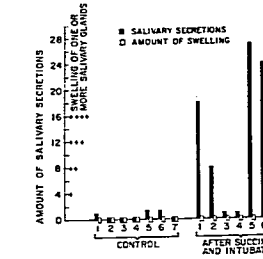

FIG. 4. Histogram showing volumes (ml.) of oropharyngeal secretions collected and grades of salivary gland swelling in the seven patients studied. No relation between volume of secretion and degree of swelling existed. Swelling was associated with straining during intubation in Patient 6 and occurred during extubation in four of the seven patients.

lar swelling of a minor degree appeared to be related to the violence of straining and coughing produced by intratracheal manipulation, possibly secondary to increased intrathoracic pressure and venous engorgement of all the tissues of the head and neck. This swelling was small compared with that observed in the original group of patients; the cause of their glandular swelling remains unknown.

We have been unable to find any reports of others who have observed sialadenopathy during general anesthesia. The only remote association is the observation of Hall *et al.*,[4] who noted brawny edema affecting the soft tissues of the lower jaws in three boars of a single litter who received 100 mg. succinylcholine intravenously. Since this was accompanied by violent convulsions, an atypical response in this species, the edema and convulsions were ascribed to an unusual genetic pattern in the litter.

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An Acoustic Switch for Use in Constant Monitoring

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A dual stethoscope attached to a molded monaural earpiece to monitor both pulse and blood pressure continuously was described in 1955.[1] This system has gained considerable acceptance[2, 3, 4] because it is simple, inexpensive and reliable. It has been recommended that the device "should be a part of the armamentarium of all anesthesiologists."[5]

The cardiac pulse pickup is either a conventional stethoscope chest piece positioned over the precordium or an esophageal pickup. The pulse pickup used is connected to one arm of the Y piece and the blood pressure pickup is connected to the other arm. A monaural earpiece makes the third connection to the Y. When taking the blood pressure, the anesthesiologist either clamps the pulse pickup tubing or uses a simple three-way stopcock as a valve.

We have recently improved this device by designing a simple and inexpensive valve that frees the anesthesiologist's hands. On blood pressure cuff inflation, the valve automatically blocks the acoustic pathway of the cardiac pulse pickup and allows reception of Korotkoff sounds. On cuff deflation, cardiac pulse sounds return automatically (figs. 1 and 2). No valve

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