The principal problems posed for the anesthetist by these airway abnormalities are 1) inability to obtain a reasonable mask fit, 2) difficulty in visualization of the larynx,[3,4] 3) difficulty in passing an endotracheal tube due to stenosis of the glottic opening,[2] and 4) postintubation respiratory obstruction.[4]

Preanesthetic evaluation of the acromegalic patient should include obtaining a detailed history, with specific inquiries about the presence or absence of nocturnal dyspnea, loud snoring, and exertional dyspnea or stridor. Physical examination should emphasize a thorough evaluation of the airway. Kitahata[3] recommends radiologic studies of the neck and/or indirect laryngoscopy. When indirect examination is not possible, fiberoptic or direct laryngoscopy during local anesthesia and sedation should be considered. Pulmonary function tests and arterial blood-gas determinations are useful in the evaluation of patients complaining of dyspnea.

On the basis of the preanesthetic evaluation, it is possible to divide acromegalic patients into several categories: 1) those without significant airway involvement, 2) those with hypertrophy of nasal and pharyngeal mucosa but normal vocal cords and glottis, 3) those with glottic abnormalities, including glottic stenosis or vocal-cord paresis, and 4) those with both glottic and soft-tissue abnormalities. Patients in the third and fourth categories should probably undergo tracheostomy either preoperatively or prior to removal of the endotracheal tube. It is in the second category of patients that indications for tracheostomy are the least clear-cut. We feel that the patient whose larynx can be visualized relatively easily can be man-

aged with close observation in the recovery room and intensive care unit. It is suggested, however, that when intubation is unusually difficult, elective tracheostomy be undertaken either preoperatively or prior to extubation.

In this case, massive hypertrophy of the soft tissue obliterated all views of the glottis and the endotracheal tube was passed blindly into the larynx after several attempts. Additional trauma of intubation added an element of edema to the already hypertrophied mucosal folds in the upper airway. It was feared that extubation would be followed by complete supraglottic obstruction. Elective tracheostomy avoided a potential catastrophic airway problem, did nothing to prolong postoperative morbidity, and appears to be a therapeutic modality to be considered in such situations.

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Laryngeal Competence after Tracheal Extubation

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Aspiration of gastric contents is a recognized hazard to postoperative patients whose laryngeal reflexes are inhibited by sedatives or residual effects of general anesthetic agents.[1,2] However, relatively few data have accumulated regarding laryngeal function in postoperative patients who are awake and alert. Gardner[3]

reported aspiration of radiopaque dye given orally by ten of 94 alert, ambulatory patients, two to four days postoperatively. Tomlin et al.[4] challenged awake patients with dye two hours or more after anesthesia of approximately 60 min duration and found aspiration in nine of 41 patients whose tracheas had been intubated. Using a similar dye test 15 min after

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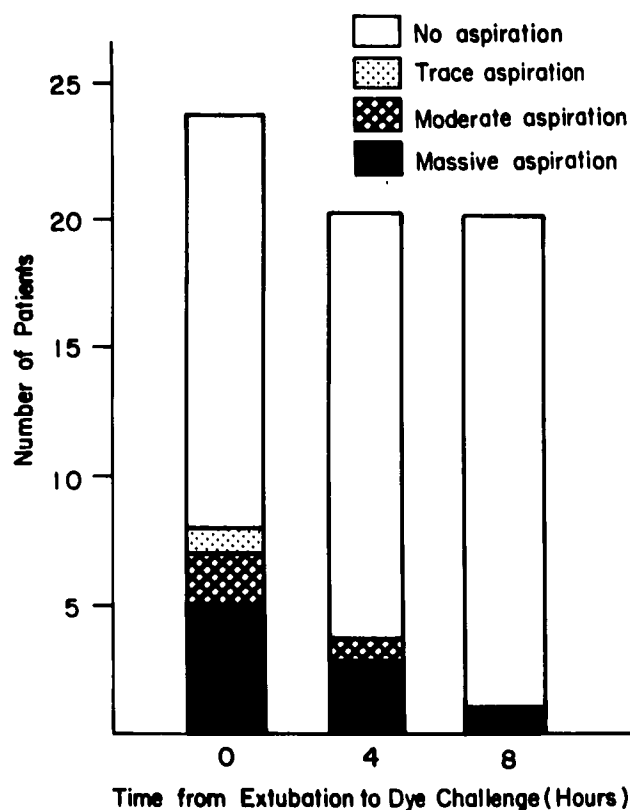
Fig. 1. Numbers of patients who aspirated radiopaque dye given orally immediately after extubation (0 hours), four hours after extubation, and eight hours after extubation. Aspiration in the eight-hour group was significantly less than that in the immediately challenged group ($P < 0.05$).

tracheal extubation, Davis and Cullen‡ found that aspiration of dye occurred in nine of 26 alert, postoperative patients whose tracheas had been intubated for 12–18 hours; however, in their study, clinical evidence of aspiration did not materialize in the three alert patients who had follow-up studies. In this study of alert, awake patients whose tracheas had been intubated for eight to 28 hours during and after cardiac surgical procedures, we sought to determine the capability of the larynx to prevent aspiration and the duration of any detrimental effect of endotracheal intubation.

MATERIALS AND METHODS

Sixty-four patients were studied within the first 24 hours after completion of elective coronary artery-bypass procedures. No patient had a history of chronic

‡ Davis FG, Cullen DJ: Postextubation aspiration following prolonged intubation. Presented at the Annual Meeting of the American Society of Anesthesiologists, October 15, 1974, Washington, D. C.

aspiration, esophageal abnormality, neuromuscular disease, or allergy to iodine or radiopaque dye. Anesthesia for the procedures consisted of induction with diazepam, 5–10 mg, fentanyl, 0.05 mg, and thiopental, 3 mg/kg, iv, followed by inhalational maintenance with enflurane, nitrous oxide, and oxygen. Relaxation for endotracheal intubation and maintenance was accomplished with pancuronium (0.1–0.2 mg/kg). Sterile, disposable, low-pressure–high-volume, cuffed endotracheal tubes were used (8.0 mm ID for women and 9.0 mm ID for men). All patients had nasogastric tubes inserted intraoperatively after anesthetic induction.

Cardiopulmonary bypass was done by a technique previously described.[5] Postoperatively, each patient was mechanically ventilated ($F_{IO_2} = 0.40$) at a rate of 10/min with a tidal volume of 12 ml/kg with an intermittent mandatory ventilation (IMV) circuit incorporated. When the patient was responsive to questions and commands and was able to lift his head from the bed without difficulty, he was evaluated for possible weaning from mechanical ventilation. When arterial blood-gas values were satisfactory, vital capacity was more than 10 ml/kg, and the inspiratory force was greater than -20 cm H_2O, the IMV rate was reduced from 10 min to 0 over a one- to six-hour period as tolerated during monitoring of blood gases and respiratory rate. When vital signs and blood-gas values were stable after the weaning period, tracheal extubation was performed. Patients were not weaned and were not included in the study in the event a vasopressor was necessary in the intensive care unit, or there had been any major intraoperative complication, or blood loss was greater than 150 ml/hour, or urinary output was less than 25 ml/hour.

After tracheal extubation, a 10-ml dose of sterile, radiopaque propyliodine oil suspension (Dionosil Oily, Glaxo Labs., Ltd., Middlesex, England) was given by mouth once, and patients were instructed to swallow the dye.[6] Group I was given the dye immediately after tracheal extubation; Group II, four hours after extubation; Group III, eight hours after tracheal extubation. A roentgenogram of the chest, obtained within 30 min after dye administration, was examined for evidence of aspiration of dye. Aspiration was graded according to the method of Davis and Cullen‡: negative, when no dye could be seen in the trachea or lungs; trace, when a minute amount was visible; moderate, when the trachea or mainstem bronchi were clearly stained; massive, when the peripheral bronchi were seen.

In addition, roentgenograms of the chest of 57 of the 64 patients challenged with dye were examined retrospectively. The results were tabulated without

knowledge of the results of the dye test previously performed. Because almost all of the postoperative roentgenograms had been read as abnormal, the films were considered to show significant pulmonary changes only when new atelectasis could be seen on a roentgenogram obtained three days after tracheal extubation as compared with the roentgenogram obtained immediately after operation.

For each patient, the following data were collected: age, anesthetic agent, duration of anesthesia, time from end of anesthesia to extubation of the trachea, and occurrence of cough upon swallowing the dye. The numbers of patients who aspirated dye in each group were compared by chi-square evaluation, and ages, durations of anesthesia, and times elapsed from end of anesthesia to extubation were compared by the Student t test for unpaired data. Results are expressed as mean ± SE.

RESULTS

Group I

Twenty-four of the 64 patients were challenged with dye immediately after tracheal extubation (fig. 1). The mean age was 57 ± 2 years, the mean duration of anesthesia, 6.1 ± 0.3 hours, and the mean time elapsed from end of anesthesia to tracheal extubation, 12.6 ± 0.8 hours. Eight (33 per cent) of these patients aspirated dye. Of these eight, five showed evidence of massive aspiration; two, moderate aspiration; one, trace aspiration. None of the eight coughed upon swallowing the dye.

Group II

Twenty patients were challenged with dye four hours after tracheal extubation. The mean age was 58 ± 2 years, the mean duration of anesthesia, 5.9 ± 0.3 hours, and the mean time elapsed from end of anesthesia to tracheal extubation, 11.8 ± 1.2 hours (not significantly different from Group I). Four (20 per cent) of these patients aspirated dye (not significantly different from Group I). Three showed massive aspiration and one, moderate aspiration. None of the four coughed upon swallowing the dye.

Group III

Twenty patients were challenged with dye eight hours after tracheal extubation. The mean age was 55 ± 2 years, the mean duration of anesthesia 5.7 ± 0.3 hours, and mean time elapsed from end of anesthesia to tracheal extubation, 11.0 ± 1.2 hours (not significantly different from Group I). One (5 per cent)

TABLE 1. Aspiration Relative to Time Elapsed from End of Anesthesia to Extubation

Time Elapsed (Hours)	Group I Challenged Immediately Following Extubation		Group II Challenged 4 Hours Following Extubation		Group III Challenged 8 Hours Following Extubation	
	Aspiration	No Aspiration	Aspiration	No Aspiration	Aspiration	No Aspiration
4-8	2	2	0	7	0	8
8-12	1	4	2	1	1	4
12-16	4	7	1	4	0	3
16-20	1	2	1	3	0	3
20-24	0	1	0	1	0	1
TOTAL	8	16	4	16	1	19

TABLE 2. Aspiration Relative to Duration of Intubation

Duration of Intubation* (Hours)	Group I Challenged Immediately Following Extubation		Group II Challenged 4 Hours Following Extubation		Group III Challenged 8 Hours Following Extubation	
	Aspiration	No Aspiration	Aspiration	No Aspiration	Aspiration	No Aspiration
8-12	0	1	0	4	0	3
12-16	3	3	1	3	0	8
16-20	1	5	1	2	1	2
20-24	4	5	2	3	0	4
24-28	0	2	0	4	0	2
TOTAL	8	16	4	16	1	19

* Total of anesthetic and postoperative periods.

aspirated dye ($P < 0.05$, compared with Group I). The one patient who aspirated dye showed massive aspiration. No patient coughed upon swallowing the dye.

No relationship was found between the incidence of aspiration as related to postoperative duration of tracheal intubation (table 1) or as related to total duration of tracheal intubation (table 2).

Retrospective examination of roentgenograms of the chest showed that of the 12 patients who aspirated dye, four (33 per cent) had new detectable roentgenographic changes; of the 45 patients who did not aspirate dye, two (4 per cent) had significant new roentgenographic changes three days after extubation. Chi-square analysis of these data showed a significant difference ($P < 0.01$) between the occurrences of roentgenographic changes in those patients who aspirated dye and those who did not. Of the nine patients who aspirated dye massively, two had roentgenographic changes; of the three patients with moderate dye aspiration, two had roentgenographic changes.

DISCUSSION

The potential for aspiration upon attempted swallowing immediately after anesthesia has been documented by others.[4,7] Siedlecki *et al.*[7] found aspiration of radiopaque dye in 27 per cent of responsive patients immediately upon tracheal extubation at the completion of anesthesia; Tomlin *et al.*[4] found aspiration in 22 per cent of patients two hours postoperatively. In their studies the incompetence of the larynx may have been due to laryngeal depression by residual inhalational anesthetic rather than to the recent presence of an endotracheal tube. The patients studied by Davis and Cullen‡ had their tracheas extubated 12–18 hours after anesthesia, and clinically had recovered completely from any effect of the inhalational agent. They had sought to determine whether an endotracheal tube has a detrimental effect on laryngeal function. They reported aspiration of dye in 35 per cent of alert patients when challenged within 15 min of tracheal extubation. Of the 26 awake patients studied, six showed trace aspiration of dye, and three showed moderate or massive aspiration of dye. Follow-up study of the three patients who aspirated moderate or massive amounts of dye revealed no postoperative pneumonia or respiratory complication as determined by roentgenograms of the chest and clinical evaluation.

In our study, all patients had clinically recovered from the effects of the inhalational anesthetic, which had been discontinued a mean of 11.8 hours before tracheal extubation. Aspiration decreased as the time elapsed after tracheal extubation increased, and after eight hours, the decrease was significant. Within each group increased aspiration did not occur with a longer period of tracheal intubation. This result may have been due to examination over too short a period of time, to inadequate numbers of patients at either extreme of times of tracheal intubation, or to the possibility that the detrimental effect of tracheal intubation on the larynx occurs in susceptible individuals during the first few hours of tracheal intubation and thereafter becomes no worse with time. The mechanism of laryngeal incompetence after tracheal extubation has been postulated to be either a sensory or a mechanical impairment of the larynx. The absence of any cough in all of the patients who aspirated dye is evidence that the sensory ability of the larynx was inhibited. A mechanical factor cannot completely be ruled out, however. Definitive conclusions from the retrospective examination of roentgenographic changes alone are not possible, since the times of food intake after tracheal extubation were not controlled, and the effect of the dye is not known.

In addition to tracheal intubation, each patient was exposed to a variety of other factors that have the potential to cause aspiration: residual anesthetic, post-cardiopulmonary-bypass neurologic dysfunction, residual muscle relaxants, and, in some patients, a nasogastric tube. If the gradual diminution of residual effects of anesthetic drugs explained the declining incidence of aspiration with time after tracheal extubation, the incidence of aspiration should have decreased as the time elapsed from the end of anesthesia to tracheal extubation increased within each group; however, such was not the case (table 1). All of our patients were neurologically intact and alert at the time of tracheal extubation, and none showed signs of cerebral dysfunction or somnolence. Intraoperatively, fentanyl, diazepam, and muscle relaxants were given in similar doses to all patients. It was not feasible to include a similar group of cardiac surgical patients who had not been subjected to tracheal intubation.

Although aspiration occurs more frequently in patients with nasogastric tubes in place because of passive regurgitation of stomach contents around the indwelling tube,[2] it is questionable in our series whether a nasogastric tube exerted any detrimental effect directly on laryngeal function. Although all patients had nasogastric tubes in place when challenged immediately after tracheal extubation, none had nasogastric tubes in place during the four- and eight-hour dye challenges. Therefore, the aspiration that occurred in the four- and eight-hour groups cannot be attributed to the presence of a nasogastric tube. In the patients challenged immediately after tracheal extubation, the nasogastric tube had been in place, with intermittent suction, for a mean of six hours before dye challenge, and intermittent suction was continued during the dye challenge; we do not believe the differences between the groups can be attributed solely to the presence of a nasogastric tube.

We conclude that laryngeal ability to prevent aspiration is adversely affected after tracheal extubation in alert postoperative cardiac surgical patients, that the detrimental effect is significantly decreased within eight hours after extubation, and that the mechanism of this effect is, at least partially, the inability of the larynx to sense foreign material. These results cannot be extrapolated to other situations.

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An Electroencephalographic Filter–Processor as an Indicator of Cerebral Ischemia during Carotid Endarterectomy

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A one-channel electroencephalographic filter-processor (EFP)[1]‡ was compared with simultaneous 16-channel EEG recordings and regional cerebral blood flow (rCBF) measurements in 70 patients undergoing carotid endarterectomy to evaluate the efficacy of the EFP in detecting hemispheric cerebral ischemia during carotid artery occlusion.

METHODS

In 15 patients, the two EFP leads were applied symmetrically over the left and right parietal regions as specified in the operating manual. In 55 patients, central and parietal leads were applied unilaterally over the affected hemisphere.

Baseline awake tracings were obtained before the induction of anesthesia, and continuous monitoring was maintained until after tracheal extubation. Particular attention was directed to the EFP and the EEG at the time of carotid occlusion. The EEG was evaluated by an electroencephalographer (FWS) and the EFP by an anesthesiologist (RFC). The criteria for a change indicative of ischemia in the EEG included a reduction in amplitude of the higher-frequency components and the appearance of high-voltage rhythmic slow waves, later replaced by lower-voltage arrhythmic slow waves. The EFP was considered to change only when an obvious decrease in baseline appeared. EEG and EFP tracings at the time of carotid occlusion were correlated with simultaneous arterial blood-gas, temperature, and rCBF measurements. After intracarotid injection of ^{133}Xe the washout curve from the affected hemisphere was analyzed by a tabletop computer to yield rCBF within 1–2 min.[2]

Anesthetic management typically included induction with thiopental, tracheal intubation following an appropriate dose of pancuronium, light levels of inhalational anesthesia with halothane or enflurane, and maintenance of normocarbia by the addition of CO_2 to the inspired gases. Arterial pressure was continuously measured via a cannula in the radial or dorsalis pedis artery, and maintained at preoperative levels. A V_5 electrocardiogram lead was continuously monitored.

Transient cerebral ischemia occurs frequently during the period of carotid occlusion for carotid endarterectomy. Clinical monitoring techniques to help identify the patients in whom such ischemia occurs include neurologic evaluation of the patient during regional anesthesia, measurement of internal carotid artery stump pressure, measurement of regional cerebral blood flow (rCBF), and the use of the electroencephalogram (EEG). This study compares the use of a simplified EFP system in monitoring such patients for the occurrence of cerebral ischemia with our usual techniques of monitoring the standard EEG and regional cerebral blood flow.

RESULTS

Bilateral Leads

The use of a recording electrode over each hemisphere as specified by the manufacturer was relatively ineffective in detecting hemispheric ischemia. Transient cerebral ischemia at carotid clamping was evident by EEG and rCBF (<18 ml/100 g/min) in four