var induction, before surgical stimulation. Only the third case is consistent with hypertension which might have been caused by fentanyl-induced epinephrine release. Another explanation might be that droperidol, like alpha-adrenergic blocking agents,[6] inhibits tissue reuptake of norepinephrine and epinephrine. Corssen and Chodoff[7] showed that droperidol blocks the alpha action of epinephrine more than that of norepinephrine, and in some cases may even potentiate the norepinephrine response. However, Dobkin[8] administered increasing doses of norepinephrine to dogs during Innovar–N_2O anesthesia and found no appreciable increase in blood pressure or pulse rate.

Another possible explanation is that hypertension resulted from the synergistic action of Innovar and other medication. Except for premedication, the three patients had not taken any drugs prior to hospitalization or surgery. In the third case, however, the nasal mucosa was swabbed with 15 per cent cocaine and infiltrated with lidocaine containing epinephrine. Cocaine is known to sensitize the cardiovascular system to epinephrine, presumably by blocking the tissue reuptake of catecholamine.[6] If droperidol also blocks the tissue reuptake, there is reason to believe that these actions would be synergistic or additive and would enhance a catecholamine-induced tachycardia and hypertension.

At this time we only can guess at the underlying mechanism involved in these three unusual cases of sustained hypertension during Innovar and Innovar–N_2O anesthesia. However, it would seem prudent to be on the alert for such responses in patients who might suffer as a result of further elevations in blood pressure: patients with severe hypertension, cerebral aneurysm or pheochromocytoma. If hypertension does occur, one effective treatment is to lower the blood pressure with a ganglionic blocking agent such as Arfonad.

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The Value of Gastric Aspiration in a Comatose Child

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Coma in a previously-healthy child demands stabilization of respiration and circulation as the first step of treatment, but rapid establishment of a diagnosis is necessary for rational specific therapy. Gastric aspiration is often omitted in treating children; yet this one simple procedure may facilitate a prompt diagnosis.

Case Report

A 23-month-old boy was brought to the emergency room of the Bronx Municipal Hospital Center at 7:45 AM, comatose and unresponsive to deep pain. He was areflexic, had small fixed pupils, unobtainable blood pressure, a faint pulse and shallow agonal-type respirations. Endotracheal intubation was performed immediately with a #12

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Cole tube, but the pulse was no longer perceptible. Closed cardiac compression was initiated, controlled ventilation with 100 per cent oxygen started, and 20 mEq/l of sodium bicarbonate infused slowly through a scalp vein. Within one to two minutes, a faint but regular femoral pulse was obtained. However, a norepinephrine drip was required to correct and maintain the blood pressure at close to a 70 mm Hg systolic level. The child's ventilation was then controlled by a Bird respirator. The temperature was 99.0 F rectally, and ear, nose, throat and chest examinations disclosed no abnormalities.

Pertinent history: The mother said the child had been "feverish" the preceding night. She had given him half of a child's aspirin tablet. At approximately 5 AM, the boy entered the parents' bedroom, alert and without complaints, and was sent back to his room. Upon arising at 7 AM, the mother found him on his bedroom floor with shallow breathing and unresponsive to stimuli.

The child had been born by cesarean section after a full-term pregnancy. The mildly diabetic mother was maintained on oral hypoglycemics. Birth weight was 4,325 gm, and both one- and five-minute Apgar scores were good. Blood sugar levels in the immediate neonatal period and subsequently for the next 23 months were normal, as were motor and intellectual development. A week prior to admission, the boy had fallen from his bed and sustained two one-cm lacerations of the right temple. Both lesions were healing well. Also a week before, a 3-year-old sibling had been admitted to the hospital for treatment of chronic lead poisoning. However, the patient himself had no history of pica, lethargy, or irritability, and neurologic examination showed only a flaccid child with no localizing signs.

Diagnostic work-up: Foley catheter specimens of urine were free of sugar and acetone. Dextrose infusion failed to improve the child's status, thus ruling out hypoglycemia due to ingestion of the mother's hypoglycemic tablets. Blood electrolytes, calcium, phosphorus, and sugar were within normal limits. Blood gases obtained following resuscitation showed *p*H 7.42, $Paco_2$ 28 torr, Pao_2 90 torr. Lumbar puncture was performed; opening pressure was 120 cm H_2O, closing pressure, 110 cm H_2O; the cerebrospinal fluid contained few leukocytes, but protein was elevated. Skull radiograms and echogram disclosed no abnormalities. The EEG disclosed a diffusely flat line for 6–8 seconds followed by minimal activity, diffusely, for the same period.

Course: At 11 AM, before the diagnosis had been ascertained, an anesthesiology consultation was called to correct a "leak" around the endotracheal tube. The anesthesiologist noted a marked dilation of the stomach and decided not to reintubate the child with a larger endotracheal tube until the stomach had been decompressed. A nasogastric tube was passed and, along with a considerable amount of air, the aspirate contained about 200 ml of fluid with a strong odor of alcohol. Investigation by the parents produced an empty Isopropyl alcohol bottle from under the child's bed. Subsequent blood studies confirmed the diagnosis.

Following reintubation with a #20 endotracheal tube, the child's respiration and circulation were maintained in the usual manner. He was comatose for two days and then slowly emerged. On the fourth day, the endotracheal tube was removed and oral feedings begun. The EEG reverted to a normal pattern, and the child recovered without further complications. Blood and urinary lead levels were slightly elevated, and therapy for chronic lead intoxication was instituted prior to the child's release from the hospital.

Discussion

The proper initial management of the comatose patient is the establishment and maintenance of an airway, respiration and circulation.[1,2] In accomplishing this sequence, the use, in a 2-year-old child, of an endotracheal tube designed for a neonate may be criticized. Although this was the only tube immediately available, subsequent insertion of a larger tube should have taken precedence over the various diagnostic tests, for an undersized tube causes part of the gas volume delivered by a pressure ventilator to leak back and enter the esophagus as the path of least resistance. Since pediatric endotracheal tubes are uncuffed, and since gastric dilation increases the probability of regurgitation, an undersized endotracheal tube promotes the danger of pulmonary aspiration of gastric contents.

The etiology of the child's coma was difficult to ascertain because of the multitude of possibilities. Infection was considered in view of the mother's observation of feverishness; intracranial bleeding because of the fall a week before; lead encephalopathy due to the elevated cerebrospinal fluid protein content; hypoglycemia since oral hypoglycemic tablets were present in the home. However, once these possibilities were ruled out, ingestion of a toxic agent was the most likely diagnosis, particularly since the small fixed pupils were not characteristic of any of the other etiologies being considered, but rather pointed to ingestion of toxic material.[3] Nearly half the poisonings in children occur in those less than 5 years old; the 2-year-old child is the highest risk, the 1-year-old next. However, only about

3 per cent of these children are in sudden unexplained coma when first seen by the physician.[3,4] Isopropyl alcohol poisoning has been reported following oral intake and following tepid sponging for fever.[5,6] Its toxicity is twice that of ethanol, although the clinical manifestations are similar.[5]

Gastric aspiration, a simple procedure, is one of the most valuable tests in the assessment of coma. Inspection of the aspirate may permit recognition of pills or capsules, while the odor may disclose the nature of other toxic agents. Chemical analysis of the aspirate is considered more informative than that of blood, urine or feces. Finally, removing part of the poison may significantly decrease the duration of toxicity.[7]

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Nasopharyngeal Stenosis

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Nasopharyngeal stenosis as a complication of adenotonsillectomy results from fusion of the tonsillar pillars and soft palate to the posterior pharyngeal wall and leads eventually to obliteration of the normal channel between the nasopharynx and oropharynx. While nasopharyngeal stenosis is not common (approximately 300 cases are cited in the literature)[1] the difficulties that may be encountered in the anesthetic management of patients with this disorder during reconstructive surgery require special alertness on the part of the anesthesiologist and surgeon. The histories of two patients treated within the past year at our institution are summarized.

CASE REPORTS

Patient 1. A 5-year-old girl was admitted for evaluation and treatment of nasopharyngeal stenosis. A week after tonsillectomy and adenoidectomy she developed an acute throat infection, which was treated with antibiotics. Her parents noted that she was mouth-breathing and that her speech was becoming increasingly nasal; otherwise she was well. Examination of the pharynx revealed that the posterior and anterior tonsillar pillars were fused to the posterior pharyngeal wall and a mass of scar tissue which included most of the soft palate had formed. An orifice about 5 mm in diameter in the midline between the nasopharynx and oropharynx was the only visible airway (fig. 1). The epiglottis was not distorted.

Plastic reconstruction of the palate was planned. Following premedication with secobarbital, 50 mg, morphine, 4 mg, and atropine, 0.25 mg, anesthesia was induced with open-hose cyclopropane and maintained with halothane, 1 per cent, nitrous oxide, 60 per cent, and the balance oxygen, via assisted mask ventilation. An oropharyngeal airway was inserted prematurely and laryngospasm occurred. Succinylcholine, 40 mg, was administered, and the chest was easily expanded. Several attempts to visualize the larynx were unsuccessful. The stenotic lumen was too small to admit the laryngoscope blade and the anatomy was too distorted to permit atraumatic introduction of a tube blindly through the nose. A tracheostomy was performed without difficulty and the child was taken to the recovery room in good condition.

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