and symptoms of postspinal headache, aseptic hemogenic meningitis,[11] and disease of the cervical spine. Postspinal headache may consist almost entirely of pain in the back of the neck, caused by traction on structures below the surface of the tentorium cerebelli; it is transmitted chiefly by the ninth and tenth cranial nerves and the upper three cervical nerves, and is referred to the suboccipital region in the back of the neck.[12] The definite increase in the severity of symptoms in the upright posture should lead one to strongly suspect postspinal headache, particularly when associated with vomiting, dizziness, or cranial nerve palsy. Persistent nuchal rigidity in a horizontal posture is suggestive of aseptic meningitis, as also occurred in the second case.

Although no definite cause-and-effect relationship was demonstrated it seems plausible that a decrease in cerebrospinal fluid pressure, as a result of recent lumbar puncture, led to a shift in brain tissue and rupture of small cerebral vessels lying within the meninges. The rarity of such an occurrence is attested to by the relative lack of similar cases reported in the literature. In view of the widespread use of spinal anesthesia in this country, this complication, if indeed related to spinal anesthesia, does not occur with sufficient frequency to constitute a contraindication to it. It does, however, underline the potential hazards of performing spinal anesthesia in patients with known or suspected intracranial tumors or vascular abnormalities. Similarly, the early prophylaxis and treatment of postspinal headache by fluid administration or epidural patching should perhaps be considered very strongly in management of all patients who have headache following lumbar puncture prior to leaving the hospital. The early diagnosis and treatment of postspinal headaches is important to allow differentiation between the effects of lowered cerebrospinal fluid pressure and more potentially serious causes of headache, such as spontaneous hemorrhage.

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Airway Management for a Neonate with Congenital Fusion of the Jaws

DAVID D. ALFERY, M.D.,* C. F. WARD, M.D.,† IVAN R. HARWOOD, M.D.,‡ FRANK L. MANNINO, M.D.‡

Congenital fusion of the jaws is a rare malformation that may prevent oral intubation.[1] We recently encountered a newborn with this anomaly who required general anesthesia. Nasotracheal intubation was carried out under direct vision, using a 3.2-mm fiberoptic bronchoscope with an attached teaching eyepiece. Subanesthetic doses of ketamine were used to provide sedation while maintaining spontaneous ventilation. We know of no previous report of securing an airway in the above-described manner, with or without this oral anomaly present. This technique offers an alternative to tracheostomy in a newborn when oral or blind nasal endotracheal intubation is not feasible.

* Resident III in Anesthesia.
† Assistant Professor of Anesthesiology.
‡ Assistant Professor of Pediatrics.

Received from the Departments of Anesthesiology and Pediatrics, University of California Medical Center, San Diego, 225 Dickinson Street, San Diego, California 92103. Accepted for publication February 20, 1979.

Address reprint requests to Dr. Alfery.

Report of a Case

A 3,760-g term female infant was delivered vaginally from a gravida 4, para 3, mother after a normal gestation. The 1- and

5-minute Apgar scores were 8 and 8, respectively. The pediatric and ear, nose, and throat services were consulted soon after birth because of inability to open the infant's mouth. A decision was made to withhold oral feedings immediately after the infant's birth, and administration of parenteral fluids was started.

Physical examination revealed apparent bilateral fusion of the alveolar ridges and retrognathia. Anteriorly the gums were open a distance of 2.0 cm wide by 0.5 cm high, and they could not be opened further (see fig. 1). Additionally, hypertelorism and abnormalities of the ears were found. The examination disclosed no other abnormality. Computerized axial tomographic (CAT) scan of the skull disclosed no abnormality; x-rays of the mandible revealed hypoplasia with cystic lucencies bilaterally. Mandibular tomograms showed an aplastic right mandibular condyle and an hypoplastic left condyle, with no evidence of bony fusion to temporal bone on either side. Lateral x-rays of the neck were not obtained.

Three days after birth the patient was brought to a heated operating room for an examination under anesthesia and an attempt at mobilization of the mandible. Atropine, 0.07 mg, iv, was administered, and a blood pressure cuff, electrocardiogram, precordial stethoscope, and rectal temperature monitor were applied. Maintenance of the airway was evaluated with a rebreathing bag and mask, and it was felt that there was only a marginal ability to ventilate the patient manually. Phenylephrine spray (0.2 ml, 0.1 per cent) and lidocaine jelly (0.5 ml, 2 per cent) were used to topicalize the nares. A 3.2-mm OD Olympus® fiberoptic bronchoscope (Model #BF, Type 3A) was passed down the left nostril by a pediatric pulmonologist (IH) and the hypopharynx visualized. A warm, softened 3.0-mm endotracheal tube was passed down the right nostril by an anesthesiologist (CW). He was able to visualize the tip of the tube through the teaching eyepiece of the bronchoscope. During the remainder of the procedure, 100 per cent oxygen 2 l/min was passed through the endotracheal tube into the oropharynx. A second anesthesiologist (DA) manipulated the patient's head and monitored respiration, heart rate, and patient movement. Ketamine in 0.5–1-mg increments (total dose 5 mg) was given iv when patient movement or rapid, irregular breathing interfered with attempts to visualize the glottis. After 40 min, with a combination of head extension and elevation, manipulation of the endotracheal tube, and movement of the bronchoscope, the trachea was successfully intubated. During the intubation procedure heart rates ranged from 150 to 208 beats/min, and systolic blood pressure remained stable at 50–60 torr. In addition, otolaryngologists were present and prepared for tracheostomy.

Following endotracheal intubation the infant was anesthetized uneventfully with enflurane in oxygen with spontaneous ventilation. Examination revealed no bony or fibrous fusion; rather, the alveolar ridges were wedged together as a result of temporomandibular joint hypoplasia. No other abnormality of the oropharynx or larynx was found. Forceful mechanical separation of the alveolar ridges allowed the mouth to be opened to a distance of 1 cm. The patient was returned to the special care nursery, where the trachea was extubed three hours later. Daily forceful dilatation to a distance of 1.5 cm allowed the infant to nipple-feed. She was sent home after several days, to be followed as an outpatient with progressive oral dilatation.

Discussion

Congenital fusion of the gums is a rare malformation of oral development.[1] It may be partial or complete; most reported cases have had associated severe congenital anomalies such as aglossia or facial hemiatrophy.[2] At least one case of fusion of the gums at birth in a patient with no associated oral abnormality has been reported; this patient was managed with a tracheostomy followed by surgical excision of fibrous adhesions of the mandible.[3]

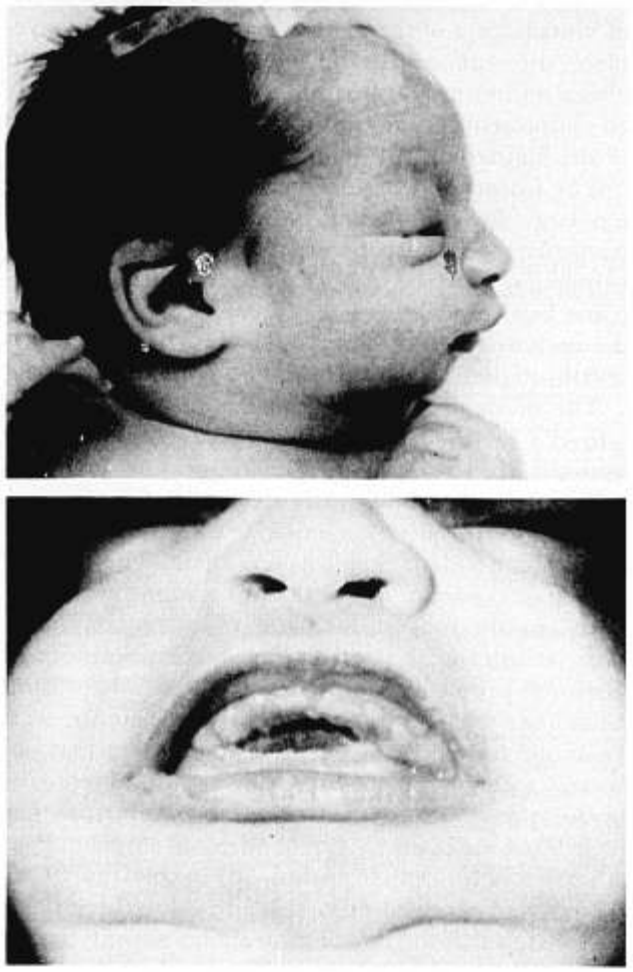

Fig. 1. Upper, lateral view demonstrates retrognathia; lower, oral opening measures 2.0 cm × 0.5 cm.

The inability to open the mouth of a newborn presents an uncommon challenge to the anesthesiologist. The infant described here required a secure airway and general anesthesia for full oral examination and attempted correction. With possible blood loss into the oral cavity, we did not consider an insufflation technique appropriate. Blind nasal intubation was also avoided, due to potential problems of trauma and resultant laryngospasm, and the obvious technical difficulties with so small an infant. Tracheostomy was reserved as a last resort, because of the increased morbidity in neonates who undergo this procedure.[4,5] We chose the use of the 3.2-mm fiberoptic bronchoscope because it could offer us the only possible means

of visualization of the glottis. Using the teaching eyepiece, the endoscopist and the anesthesiologist could obtain optimum coordination. We were unable to pass an endotracheal tube over the bronchoscope because of size limitations; a 4-mm-ID tube is the smallest that can be introduced by this method, and this size was too large for this patient. Other limitations of this particular bronchoscope are the lack of suction and ventilation capabilities, but these problems were overcome by the introduction of a suction catheter into the oral cavity as necessary and by insufflation of oxygen through the endotracheal tube in the right nostril.

The method of endotracheal intubation used required a relatively quiet field, whereas we were presented with a vigorous, active infant. Our goal was to limit motion while providing minimal respiratory depression; we chose ketamine, to be administered in small subanesthetic doses, as our agent. There is disagreement concerning the use of ketamine as the primary anesthetic agent for patients undergoing intraoral or intranasal procedures.[6-8] We acknowledge potential problems of laryngeal reflexes when using ketamine.[9-12] However, laryngeal competence with ketamine has been shown to be superior to that observed with diazepam, especially in the absence of opiate premedication.[13] In addition, ketamine has been used successfully for blind nasal intubation in adults[14] and for rigid bronchoscopy in children.[15] Our patient had received no oral feeding for three days, and we felt that the risk of aspiration was outweighed by the need for spontaneous and relatively undepressed ventilation and immobility of the patient.

We emphasize the need for a third person to be present to manipulate the head and monitor the general condition of the patient. In addition, equipment and personnel for emergency tracheostomy should be present in this situation. Direct visualization with a fiberoptic bronchoscope for nasal intubation and sedation with low incremental doses of ketamine offer an alternative to tracheostomy in securing the airway of a newborn who has a compromised oral passageway.

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