right innominate vein, it is likely that this complication will occur most often as a result of forceful attempts when catheterization via the internal jugular vein is difficult.

REFERENCES

Severe Congenital Subglottic Stenosis in Association with Congenital Duodenal Obstruction

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Congenital subglottic stenosis is infrequent. Holinger and Brown reported 53 cases collected during a period of 30 years in a pediatric hospital and a university endoscopic clinic.1 Fearon and Cotton collected 84 cases during a ten-year period in a similar setting.2 The occurrence of congenital subglottic stenosis in association with congenital duodenal obstruction is very unusual. It does not appear in the listings of anomalies associated with congenital duodenal obstruction reported by Aitken,3 Moore,4 Kiesewetter and Koop,5 or Rickham.6

REPORT OF A CASE

A 1½-day-old white male infant delivered by cesarean section because of previous cesarean section was admitted June 10, 1977, with a history of wheezing and vomiting. An upper gastrointestinal series performed at the referring hospital showed no passage of barium beyond the stomach, and a diagnosis of duodenal atresia was made.

On admission to this hospital, the infant, who weighed 2560 g, appeared normal except for slight to moderate wheezing on inspiration. X-ray of the chest showed no abnormality. Laparotomy with general anesthesia was scheduled. Aspiration of oropharyngeal secretions suggested a possible cause for the wheezing.

Several attempts to achieve awake intubation were made by the anesthesiologist, using endotracheal tubes of 3.0 mm ID and 2.5 mm ID, with and without a stylet, but obstruction was repeatedly encountered below the vocal cords. The anesthesiologist then suggested the possibility of subglottic stenosis. After failing in an attempt to pass a 2.5 mm-ID endotracheal tube, the surgeon proceeded to perform a tracheostomy using local anesthesia, with oxygen delivered under intermittent positive pressure from the anesthesia circuit. Insertion of the tracheostomy tube provided complete relief of wheezing. Laparotomy was postponed until two days later, at which time exploration revealed the duodenal obstruction to be due to an annular pancreas with malrotation. A duodenoduodenostomy and lysis of Ladd’s bands were performed.

The postoperative course was complicated by recurrent obstruction of the tracheostomy tube, left pneumothorax, and bilateral pneumonia. Weight gain was slow, and allergic dermatitis appeared.

Laryngo-tracheoscopy was performed three weeks after tracheostomy and again at seven weeks. At the initial examination circumferential narrowing was encountered 1 cm distal to the vocal cords. The apparent aperture would not admit a 2.5-mm bronchoscope, nor could the tracheostomy tube be visualized distal to the obstruction. A polyethylene catheter 1 mm in diameter was successfully passed through the apparent aperture. On the second examination a 3-Fr polyethylene catheter was inserted through the area of constriction but a 2.5-mm bronchoscope again could not be passed. A tracheogram obtained two days after the second laryngo-tracheoscopy (fig. 1) showed a segment of stenosis 1–2 mm in diameter extending from the glottis inferiorly approximately 1 cm. The patient by this time had resolution of the pulmonary problems and was showing a steady weight gain. When his weight reached 2.880 g he was transferred to a convalescent facility with a 00 Shiley tracheostomy tube in place.

DISCUSSION

Congenital subglottic stenosis is a cause of neonatal asphyxia. Tracheostomy is necessary in 50 per cent

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of cases. Stridor with or without episodes of cyanosis may begin at birth, depending on the severity of the stenosis. In mild cases of stenosis recurring attacks of croup are common and stridor may be manifest only during respiratory infection. Occasionally, stenosis may be detected when the child is being anesthetized for some unrelated surgical procedure, as the anesthetist tries to pass an endotracheal tube considered to be the correct size for the age of the patient and unusual resistance to passage is encountered in the subglottic space. The point of maximal obstruction is usually 2 to 3 mm below the level of true cords.

A congenital laryngeal disorder in the neonate should be suggested by stridor that is maximal on auscultation of the larynx, with or without cyanosis, and a normal chest x-ray. Complete investigation is indicated so that any necessary precautions can be taken to prevent asphyxia.

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