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Type D Type E Figure 18.1 Anatom

ical variations in tracheoesophageal /f_i s t u l a w i t h o r w i t h o u t oesophageal atresia. In Type C, the upper pouch ends in the neck or upper chest but occasionally it reaches the /f_i stula where muscle /f_i bres are shared.

/uni25CF /uni25CF /uni25CF /uni2192 /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF Robert L Replogle , 1931–2016, Chicago, the last trainee of Robert E Gross. A /uni00A0 nasogastric tube coiled in the upper oesophageal pouch on a chest radiograph suggests the diagnosis. A nasal or oral sump Replogle tube is placed to drain saliva and prevent aspiration. Positive airway pressure is avoided as air passing through the fistula causes gastric distension, compromised ventilation, and risks perforation (Figure 18.2). If pressure support is needed, perhaps because of RDS, prompt fistula ligation is needed. Types A and B typically have a long gap and may require oesophageal replacement; options include colonic or jeju - nal interposition or gastric transposition some months after a cervical oesophagostomy and a gastrostomy . In many cases the ends can be brought together by progressive traction and yed anastomosis. dela In types C and D, the fistula is divided through a right y or thoracoscopically . If the neonate is stable thoracotom and the gap favourable, an anastomosis is fashioned over a ly feeding. If a primary trans-anastomotic tube, facilitating ear anastomosis is not possible, then options include a delayed anastomosis after a few weeks of growth, or the use of traction sutures and an earlier anastomosis, or a much later interposition. Traction sutures can be internal or external. Nutrition is supported through a

gastrostomy . Complications after a repair include anastomotic leaks, oesophageal strictures and refistulation. Minor leaks often set - tle without intervention, strictures need dilating with a bougie or a balloon, and refistulation needs repair. Type E is an isolated ‘H’-type tracheoesophageal fistula he fistula is usually found in the neck on a without atresia. T rent chest infec - contrast swallow . Type E presents with recur tions or coughing after feeds and is usually repaired in the neck.

infants. Airway Intubation can be challenging as the occiput /f_l exes the neck, the tongue is large and the epiglottis is long, angulated and positioned high and close to the soft palate. A straight blade laryngoscope, an uncuffed tube and a neutral position for the neck Abdomen The liver is large and fragile and the bladder rises out of the pelvis. The abdomen must be entered carefully. The umbilical vein is patent for many days after birth and is ligated before being

divided. Respiratory (respiratory distress syndrome [RDS], chronic lung disease) Preterm delivery, gestational diabetes and birth asphyxia all lower pulmonary surfactant levels, resulting in decreased lung volume and compliance and promoting airway collapse on expiration and atelectasis. Fewer type 1 muscle /f_i bres in the diaphragm and intercostals increases early fatigue. Chronic in /f_l ammatory lung disease with scarring is seen in pr eterm babies from prolonged ventilation, overin /f_l ation, high

pressures and oxygen toxicity. Surfactant, oxygen, continuous positive airway pressure (CPAP) or mechanical ventilation Cardiovascular A fall in pulmonary vascular resistance (PVR) at birth helps establish the postnatal circulation. In the early postnatal period, hypoxia, stress, high P CO or 2 acidosis may raise PVR; if the ductus arteriosus and foramen ovale are open, blood shunts R L causing hypoxaemia An underdeveloped barore /f_l ex means unchecked blood loss leads rapidly to hypotension Fluids and

electrolytes Excess total body water and extracellular /f_l uid are excreted after birth in a physiological diuresis. Insensible losses increase with low birth weight and low gestational age. The immature kidney loses sodium, bicarbonate, glucose, amino acids and phosphates. Low glycogen stores at birth promote hypoglycaemia, particularly in the preterm. Use local neonatal intensive care unit (NICU) protocols. Maintenance /f_l uids need 10% glucose and appropriate electrolytes Watch for

hyperglycaemia with hypernatraemia, which increases the risk of intraventricular haemorrhage in the preterm Replace nasogastric losses or stoma losses (>15 /uni00A0 mL/kg/day) millilitre for millilitre with 0.9% NaCl, 0.15% KCl Nutrition Reserves are de /f_i cient in the premature and postnatal starvation affects neurological development. Start central parenteral nutrition as a matter of urgency Thermoregulation A high surface area to bodyweight ratio increases heat loss; particularly

during exposure for anaesthesia (exacerbated by vasodilation) and surgery, there is an inability to shiver. Low temperatures promote coagulopathy, which is compounded by the acidosis from poor peripheral perfusion and myocardial depression. Warm incubators, limit exposure for procedures, warm theatre, warm /f_l uids Figure 18.2 Tracheoesophageal /f_i stula/oesophageal atresia with gastric perforation in a 28-week gestation, 1000-g baby. Note the endotracheal tube (ET), Replogle

tube in the upper pouch, the umbil

ical venous catheter (UVC), free abdominal air around the liver and either side of the falciform ligament above the UVC and patchy lung /f_i elds of respiratory distress syndrome.

Figure 18.3 Double bubble in duodenal atresia (gastric and /f_i rst part of the duodenum). Note the umbilical cord and clamp in the lower part of the image.

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