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Gastrointestinal surgery in the neonate

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Summary The surgical treatment of neonates has enjoyed rapid development during the last 50 years. Neonatal surgery has now become an independent branch of general surgery, which requires the expertise of dedicated paediatric surgeons. Much of the development of neonatal surgery is due to the close collaboration between paediatric surgeons, neonatologists, anaesthetists, pathologists, radiologists, biochemists, and nurses. There has been a steady improvement in the outcome of most neonatal diseases requiring surgery. For example at Great Ormond Street Hospital for Children, London, the mortality for the most common congenital intestinal obstructions has declined from 50% in the 1950s and 1960s to 25% in the 1960s and 1970s to a current mortality of less than 5%. The reasons for this improvement can be ascribed to a better understanding of the physiology of surgical neonates, to the advances in fluid management, nutrition, mechanical ventilation, and not least to refinements in surgical techniques. There is still considerable work to be done to improve the survival of neonates with severe conditions such as necrotizing enterocolitis (NEC). Surgery of the gastro-intestinal tract is required for congenital abnormalities and acquired lesions. In this article the authors focus on the most common congenital anomalies of the gastro-intestinal tract requiring surgery. In addition they discuss two acquired conditions requiring surgery in neonates: NEC and hypertrophic pyloric stenosis. It is beyond the scope of this article to discuss anomalies of the abdominal wall, gastro-oesophageal reflux, and anomalies of the pancreato-biliary system.

Congenital anomalies of the gastro-intestinal tract (GIT)

Oesophageal atresia (OA) and tracheoesophageal fistula (TOF)

This complex group of anomalies with an incidence of 1 in 2440–4500 livebirths [1,2] results from failure of correct division of the tracheal primordium from the oesophagus during early embryonic development. The precise aetiology is unknown with a number of embryological theories proposed to explain the different variants of this anomaly.

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There is a high incidence of co-existing abnormalities including the VACTERL syndrome, CHARGE association and isolated cardiovascular anomalies.[3]

Clinical features
OA is commonly associated with maternal polyhydramnios. The diagnosis may be made in the antenatal period particularly if there is no TOF. In the postnatal period symptoms associated with the condition include excessive salivation, feeding difficulties, respiratory distress, and cyanotic episodes. Cases of OA (with the exception of the rare OA with double fistula) can be confirmed by failure of passage of a nasogastric tube into the stomach. Cases of TOF in the absence of OA (i.e. H-type fistula) may present later, usually with recurrent episodes of respiratory distress or pneumonia.

Treatment
The overall aim of surgical correction is early division of any fistula with the respiratory tract to protect the lungs and airway, and restoration and maintenance of oesophageal continuity to allow normal feeding. After diagnosis a Replogle tube is placed in the upper oesophageal pouch which allows suction of secretions and minimizes the risk of pulmonary aspiration. Surgical repair involves ligation and division of any fistula and primary anastomosis of the two ends of the oesophagus where possible. Infants in whom the gap between the two ends of the oesophagus is too wide for primary anastomosis to be achieved pose a problem. These infants are initially fed by gastrostomy and the oesophageal anastomosis is re-attempted after 6 weeks. If recurrent attempts at this remain unsuccessful oesophageal replacement alternatives including colonic interposition and gastric transposition are considered.

Outcome
The majority of patients do well following anastomosis but a number of complications may occur and they require recurrent procedures. Complications include anastomotic leakage, anastomotic stricture, recurrent TOF, gastro-oesophageal reflux and disordered peristalsis.

The stomach

The most common abnormality of the stomach in the neonatal period is hypertrophic pyloric stenosis. Whether this is truly a congenital abnormality or an acquired disorder is questionable. It is further discussed in the section on gastro-intestinal problems of the newborn.

Other congenital conditions affecting the stomach including congenital microgastria, gastric volvulus, and congenital gastric outlet obstruction due to a pyloric web or atresia are all extremely rare and are mentioned only for completeness.

Obstructive lesions of the duodenum, jejunum and ileum

The most common congenital conditions affecting the duodenum, jejunum and ileum all result in partial or complete gastrointestinal obstruction.

Clinical features
Obstructive lesions of the small intestine from the pylorus down to the ileocaecal valve may give rise to polyhydramnios in the antenatal period which is detectable by antenatal ultrasonography.

After birth, the most common and important clinical manifestation of obstructive lesions of the GIT is bile-stained vomiting. Vomiting with truly bilious staining is always abnormal in the neonatal period and always requires investigation. Lesions in the duodenum and jejunum usually result in bilious vomiting within hours. In addition the abdomen may appear empty or even scaphoid and visible gastric peristalsis may be observed. Lesions lower in the ileum result in a distended abdomen if the obstruction is complete and there may be failure to pass meconium. Obstructive lesions may also give rise to intestinal perforation in the neonatal period and occasionally antenatally. In all cases of neonatal intestinal obstruction infants become progressively hypovolaemic and are prone to circulatory and respiratory collapse. They require fluid resuscitation and may require ventilatory support. GIT obstruction should therefore be considered in any infant who is dehydrated especially if there is a history of vomiting.

Stenotic lesions of the small bowel in which the obstruction is incomplete may give rise to increased diagnostic difficulty. Affected infants often present with intermittent vomiting and episodes of partial obstruction. They eventually fail to thrive or develop complete obstruction at which stage they are fully investigated and the diagnosis becomes apparent.

Investigations
The aim of investigating cases of suspected obstruction of the small intestine is twofold: first to identify the nature and anatomical location of the lesion to allow for planning of correct treatment, and second, to identify cases of malrotation in whom there is a risk of midgut volvulus and intestinal ischaemia. These cases require urgent surgical intervention to reduce the risk of potentially catastrophic intestinal necrosis. The history and examination may give clues as to the location of the lesion as described above. An abdominal X-ray may simply confirm the presence of dilated intestinal loops but may also give further clues in some cases. A double bubble appearance on abdominal X-ray with a lack of air in the distal intestine is characteristic of duodenal obstruction. Multiple air-filled loops of proximal bowel often with air–fluid levels along with a paucity or complete absence of gas in the distal bowel is highly suggestive of obstruction of the ileum. Intestinal perforation if present will usually be apparent on abdominal X-ray and in the rare cases of antenatal perforation there may be widespread or localized flecks of calcification representing calcified meconium within the peritoneum.

In cases in which the diagnosis is not clear on abdominal X-ray or in which midgut malrotation or volvulus is suspected a limited upper gastrointestinal contrast study is indicated. The classical finding in cases of malrotation is that the duodenojejunal flexure lies to the right side of the spine instead of in its normal left-sided position. This finding should prompt urgent surgical treatment due to the risk of co-existing midgut volvulus. The contrast study may also