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# Dysphagia in children: a paediatric surgical perspective

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**Abstract**

Dysphagia in childhood encompasses all disorders of swallowing. The patient groups seen frequently in paediatric surgical practice are those with repaired oesophageal atresia, some patients with gastro-oesophageal reflux (GOR) disease and those with neurological impairment.

Oesophageal atresia patients frequently experience dysphagia that is most troublesome in early childhood, fortunately for most the long-term outlook is good. Gastro-oesophageal reflux may give rise to dysphagia as a result of peptic stricture, dysmotility associated with oesophagitis or as a complication of anti-reflux surgery. Children with neurological impairment and swallowing problems are very difficult for their carers to manage. Gastrostomy represents a pragmatic solution to their nutritional needs.


*Keywords:* Dysphagia; Child; Oesophageal atresia; Gastro-oesophageal reflux; Fundoplication; Gastrostomy

## 1. Introduction

Dysphagia in children may be broadly defined so as to include all swallowing problems in this age group, due both to oropharyngeal and oesophageal disorders. The differential diagnosis is wide and many of the individual causes are extremely rare.

As a paediatric surgeon, there are three patient groups who I see fairly frequently with dysphagia.

Firstly, children who have previously undergone repair of oesophageal atresia, second some of those with gastro-oesophageal reflux (GOR) disease and third the group of

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neurologically impaired children with either aspiration or nutritionally inadequate oral intake.

## 2. Oesophageal atresia

Oesophageal atresia occurs approximately once in every 4000 live births. In most neonates, a primary oesophago-oesophageal anatomosis is achieved and, in contemporary series, the survival is now 95% or more [1].

Long-term follow-up of the survivors of oesophageal atresia repair has shown that between 60% and 70% experience problems with dysphagia. This usually first becomes evident at the time of introduction of solids into the diet and may be particularly troublesome in the early years of childhood. The prevalence of dysphagia does not appear to alter significantly with increasing age, but the severity of symptoms reduces, perhaps as children learn strategies to enable them to overcome their swallowing problems. Most adults are able to take a normal diet and have a normal lifestyle. Many modify their eating behaviour, for example taking frequent sips of fluid whilst eating and some will avoid certain troublesome foods. Nutrition is usually good and growth normal [2].

Food impaction is a particular problem in these patients. It occurs in between 13% and 50% of patients on at least one occasion [2,3]. The peak incidence is about age 2 years, when a more adult diet is being taken but appropriate eating behaviour has not yet been learnt. In older patients, food impaction is less common, but it has been reported in as many as 16% of over 15-year-olds [2]. The site of impaction is usually at the level of the oesophageal anastomosis, but in older patients it may be lower down in the distal oesophagus. The impacted food bolus usually requires endoscopic removal. Children who experience food impaction frequently have prior symptoms of dysphagia and evidence of gastro-oesophageal reflux; however, these seem to occur with equal frequency in those who do not experience impaction.

GOR is very common in these children. It can be demonstrated in up to 50% in the first 5 years of life, but only 18% will experience symptoms. Interestingly, adult survivors do not report symptoms of GOR more frequently than the general population [4].

Reflux may be important in the aetiology of dysphagia in these patients as it may be associated with strictures either at the anastomosis or in the distal oesophagus. The development of an anastomotic stricture, particularly one that recurs after initial dilatation should prompt a search for evidence of reflux and aggressive medical or surgical management if it is discovered. Another possible mechanical cause of dysphagia is a coexistent congenital oesophageal stenosis. These are normally in the distal oesophagus and are resistant to dilatation, often containing ectopic tracheal cartilage in the oesophageal wall. Resection is usually required.

In the majority of patients, however, dysphagia is not due to stricture or stenosis, but appears due to impaired oesophageal peristalsis. It has been demonstrated that the vagal supply to the oesophagus is incomplete as part of the anomaly and also that there are abnormalities of the intrinsic nerve plexuses in the oesophageal wall [5]. Further damage to the innervation of the oesophagus may occur if extensive mobilisation of the oesophageal ends is required to achieve anastomosis in a wide gap.

Patients with symptoms of dysphagia or food impaction should be investigated with contrast radiography to exclude a stricture or stenosis. Anastomotic strictures usually respond well to balloon dilatation [6]. Endoscopy or pH studies are indicated to exclude GOR. In those who do not have a stricture or stenosis, then management is expectant as long as a good nutritional intake is maintained, in view of the excellent long-term prognosis.

## 3. Gastro-oesophageal reflux disease

GOR may produce dysphagia as a result of the development of a peptic stricture, sometimes as a result of "uncomplicated" oesophagitis and perhaps, most commonly, as a complication of anti-reflux surgery.

Peptic stricture is rare in childhood, with only few small series reported [7]. Strictures may be managed by dilatation and medical treatment with proton pump inhibitors; however, in my experience, dilatations need to be repeated frequently, usually under general anaesthetic. An anti-reflux procedure combined with intra-operative dilatation usually produces long-lasting resolution of the stricture and postoperative dilatations are rarely necessary.

There has recently been reported an interesting series of children whose presentation with GOR was with food bolus obstruction [8]. In these children, there were often repeated episodes of obstruction, but other symptoms of reflux were minimal. Contrast radiography showed no evidence of stricture but endoscopy and biopsy in all cases showed evidence of reflux oesophagitis. Manometry was abnormal in most of the children, with very high-pressure peristaltic waves in the oesophagus provoked by reflux and also occurring during sleep.

In all cases, treatment of reflux prevented further episodes and led to normalisation of manometry. I have encountered two similar patients, both of whom have remained well on medical treatment, but in one further food impaction occurred when he stopped treatment.

Unfortunately, the most common cause of significant dysphagia in GOR patients in my practice is anti-reflux surgery. Dysphagia as a result of fundoplication is well recognised in adult practice but is less well described in paediatric series. In adults, early dysphagia is reported in between 2% and 44% after open fundoplication. Fortunately, the rate of dysphagia falls over 3–6 months, from 34% to 6% in combined series [9]. Prospective evaluation of patients undergoing fundoplication reveals that 40–50% experience dysphagia pre-op, the majority improving after surgery, but in some dysphagia develops de novo and in others may worsen.

In some cases, this may be due to technical errors or complications, such as over tight closure of the oesophageal hiatus, para-oesophageal herniation or an excessively tight fundal wrap.

In most, however, there is no obvious problem and the cause is unclear. It is known that fundoplication alters the physiology of the gastro-oesophageal junction, increasing the length of the distal oesophageal high pressure zone and raising the resting and nadir pressures of the lower oesophageal sphincter; however, changes do not correlate with the presence or absence of dysphagia [9].

Various technical operative factors have been investigated; laparoscopic as opposed to open fundoplication was associated with an increased risk in one randomised controlled trial, but not in other series. The type of fundoplication does not seem important, dysphagia occurring equally in complete and partial wraps. Division of the short gastric vessels does not alter the incidence of dysphagia with Nissen fundoplication [9].

If dysphagia after fundoplication is very troublesome and not settling after 6 weeks, then oesophageal dilatation is often helpful [10]. In experience with 50 laparoscopic Nissen fundoplications at the Royal Hospital for Sick Children, Edinburgh, there was evidence of early dysphagia in 20%, two patients with near total dysphagia required early re-operation and conversion to a partial wrap, two underwent balloon dilatation and six were treated conservatively. At mean follow-up of 3 years, no patients report persisting problems.

## 4. Neurological impairment

A significant number of children with neurological impairment have problems with oral feeding, either aspiration of feeds into the larynx and trachea or extremely slow and time consuming oral feeding leading to nutritionally inadequate intake. Many of these children also have problems with vomiting, which may be due to GOR or to a more widespread upper gastro-intestinal dysmotility including inappropriate triggering of the emetic reflex. For these children, the pragmatic solution is to either avoid oral feeding or supplement it by tube feeding. This is most conveniently delivered by means of a gastrostomy. The commonest method of gastrostomy insertion is the percutaneous endoscopic gastrostomy (PEG) originally described by Gauderer et al. [11]. This has significantly reduced the morbidity of gastrostomy insertion but is not without the risk of major and minor complications [12]. In some patients, the co-existence of severe GOR may make simultaneous fundoplication and gastrostomy desirable, but fundoplication should be avoided in those in whom vomiting is due to inappropriate triggering of the vomiting reflex [13].

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