The Stomach and Esophageal Atresia Repair

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Introduction

Esophageal atresia (EA) with or without a tracheoesophageal fistula (TEF) is an abnormality of embryologic differentiation. There are a range of phenotypes, and EA-TEF is associated with developmental anomalies in other organ systems. The gastric cavity is intimately related to the esophagus anatomically and functionally. In clinical practice gastric morphological anomalies are not reported in patients with EA, and there is little discussion on gastric function. In this chapter we examine clinical and experimental data that includes the stomach in patients with EA. Gastric neuromuscular development during embryogenesis, electrophysiology, and gastric function after EA repair are reviewed in relation to EA-TEF and EA without TEF. The effects of EA repair surgery and fundoplication on gastric function are examined and whether there is an impact of this on long-term outcome of patients with EA.

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Embryology

The relationship between the esophagus and stomach is an example of the coordinated function that characterizes the alimentary tract. The smooth sequential movement of the upper intestinal tract is made possible by their common origins. The embryology of the foregut is discussed elsewhere in this book. In brief the primitive foregut gives rise to the pharynx, respiratory tract, esophagus, stomach, and proximal duodenum. Differentiation of the stomach commences around 7 weeks of gestation. The gastric cavity and esophagus therefore develop in concert during embryologic differentiation of the foregut. While the precise event or sequence of events that lead to the various EA defects is not defined, there is data that shows anomalies of the sonic hedgehog and related signaling pathways are involved [1]. The same pathways are involved anatomically and functionally in the normal foregut [2]. Furthermore atretic malformation of segments of the alimentary tract distal to the stomach is associated with the EA-TEF spectrum [3].

Gastric Anatomy at Birth

During fetal growth functional maturity of the alimentary tract involves the flow of amniotic fluid, and in utero foregut obstruction leads to abnormalities of fluid volume [4]. The most

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common form of EA comprises a blind-ending upper esophageal pouch and a TEF to the lower esophageal end so that fluid can flow via the tracheal fistula into the distal esophagus and therefore the remainder of the alimentary tract. In EA without a TEF to the distal esophageal pouch, amniotic fluid cannot flow through to the lower esophagus and stomach, and therefore development of the stomach can potentially be affected in such cases. Indeed diminished or absence of air in the stomach is a radiological feature for the diagnosis of pure EA [5].

Sase et al. examined fetal gastric volume using ultrasound [6]. Women with normal singleton pregnancies between 18 and 39 weeks of gestation were included in their study. Gastric measurements were also performed in 13 fetuses with digestive tract obstruction. While the cases of EA were not defined in terms of the presence or absence of a fistula, the gastric area ratio was below the 95 % confidence interval for the predicted value in all five fetuses with EA and greater than the 95 % confidence interval for the predicted value in 7 of 8 with duodenal atresia or distal intestinal tract obstruction. While it is reasonably assumed that amniotic fluid has a role to play in the development of the gut, there has been no systematic study on the development of the stomach and gastric anatomy in patients with EA. Indirect evidence comes from an investigation of the neurohistopathology of the lower esophagus, gastroesophageal junction, and gastric cardia associated with EA. Nakazato et al. used a microdissection technique to study the upper gastrointestinal tract of five patients with EA-TEF prior to surgical repair [7]. A looser than normal Auerbach's plexus configuration was present in the distal esophagus, and the nerve plexus was abnormal in the gastric fundus of all the patients. The authors concluded that the findings suggest the existence of congenital functional impairment of the upper gastrointestinal tract in patients with EA-TEF, due to abnormal development of the myenteric plexus. In contrast to this, a report in which the authors utilized a manometric approach to examine the lower esophageal sphincter (LES) prior to EA repair showed that the relaxation of the LES was normal in response to contractions in the upper esophageal pouch suggesting that the neurophysiology of the gastroesophageal junction is normal in these patients [8, 9]. Our series of patients comprises the largest group of EA patients without a distal TEF undergoing primary EA repair [10]. We have also noted that in cases of pure EA where there is almost no discernible length to the distal esophageal pouch (Fig. 44.1), the LES can still be visualized as a distinct structure (Fig. 44.2). Furthermore we have assessed the stomach visually and with contrast to define gastric anatomy and noted that the dimensions of the gastric cavity have not been compromised in typical cases of EA without a distal TEF (Fig. 44.3) [10]. Conversely a report on nine babies with pure EA, albeit over a decade ago, noted that after initiation of gastrostomy feeds, seven (78 %) developed gastric complications, including two posterior gastric perforations (one fatal). The authors proposed that the high complication rate was due to a small, abnormal stomach that was vulnerable to damage by operative trauma and the effects of handling large volumes of feed. They hypothesized that the stomach is abnormal because it has not been exposed to the maturing effects of amniotic fluid in utero [11].

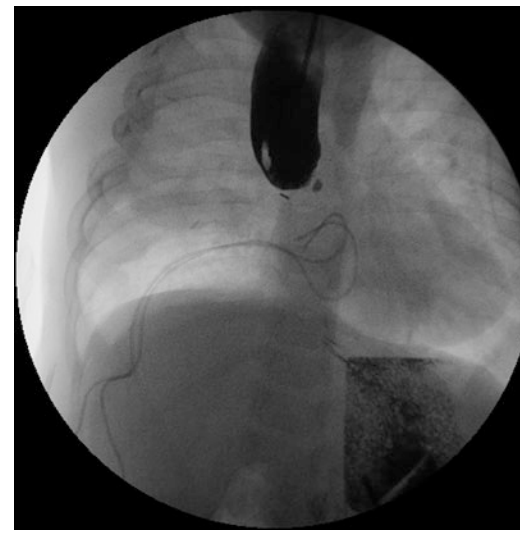

Fig. 44.1 An extremely short lower esophageal remnant that cannot be easily seen even when a probe is used to distend the area of the stomach in a case of pure esophageal atresia. The gastric volume is not diminished

Gastric Electrophysiology

The electrical activity of the stomach was first defined almost a century ago. There is a consistent pattern whether recorded from the serosal or mucosal surface of the stomach or the skin surface [12]. The characteristic electrical activity comprises slow waves of three cycles per minute (cpm) or 0.05 Hz. This is driven by pacemaker cells high on the greater curvature and is an inherent property of the smooth muscle of the stomach and related to cell membrane permeability changes and movement of sodium – interstitial cells of Cajal have been defined as the primary pace setting cells of the gastrointestinal tract [13]. Spikes waves correspond to action potentials of muscular contractions. A maturation pattern of the gastric electrical activity has been demonstrated dependent on the gestational age; a normal electrical rhythm can be detected during early gestation [14, 15]. Chen and McCallum reported that normal slow-wave frequency in the EGG was related to normal gastric motility and that abnormal slow-wave frequencies were associated with motility disorders [16]. Dysrhythmias have been reported in patients with pseudo-obstructive disorders with associated gastric dysfunction suggesting that disordered gastric electrical activity is a sign of intrinsic neuropathic disorders of the gut [3]. Surface skin recording or electrogastrography (EGG) and its uses in clinical practice are discussed in detail elsewhere in this book. Patients with EA have been studied using this technique. In a study of 16 EA patients in comparison to controls, the authors found a wide frequency distribution with two individuals showing tachygastria (frequency ≥5 cpm) and another two demonstrating bradygastria (frequency <2 cpm) [12]. The authors postulated that the wide range suggests that there is an electromechanical dissociation that results in abnormal gastric contractions. In a similar study by Yagi et al., 13 children with a history of EA repair underwent EGG, 5 of whom demonstrated abnormalities [17]. The dysrhythmias continued in the postprandial period and were persistent in 3 of 5. The authors concluded from this that there must be a congenital neuronal defect associated with these findings in patients with EA. In support of this hypothesis, there is evidence to show that the smooth muscle cells of the stomach can exhibit an abnormal slow-wave frequency after inhibition of cholinergic activity [18]. Gastric dysrhythmias are suggested to cause antral dysmotility by inhibiting the strength of

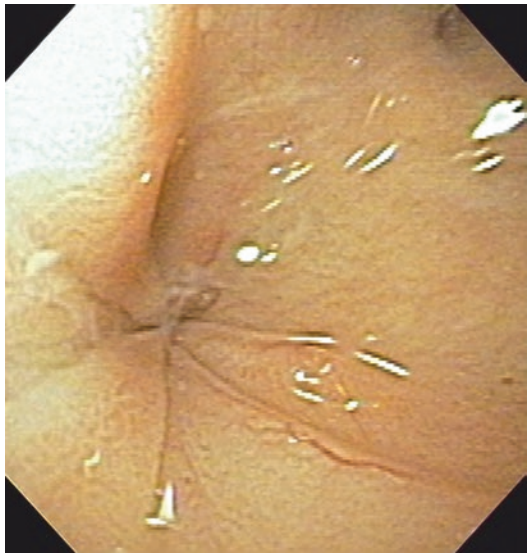

Fig. 44.2 The appearance of the lower esophageal sphincter in the case from Fig. 44.1

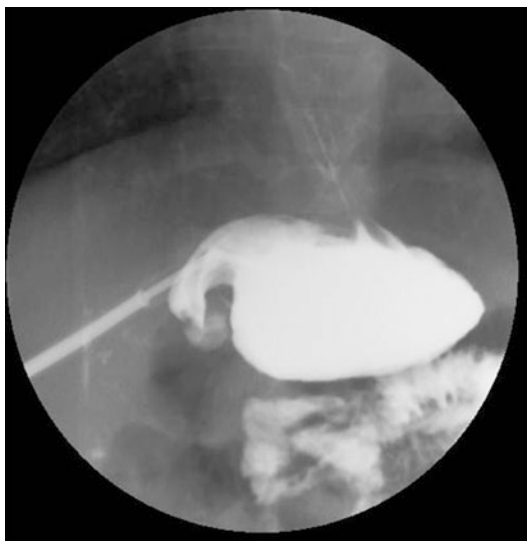

Fig. 44.3 Contrast study after esophageal atresia repair and fundoplication in a case of pure esophageal atresia; the gastric volume does not appear to be diminished

contractions or reducing aboral propagation of contractions [19]. In a study by Bokay et al., there was a significant increase in bradygastria and decrease in tachygastria in the postprandial from the preprandial period in patients with a history of EA repair in comparison to controls [20]. Abnormal EEG patterns were present in 11 of 15 of the EA patients, while in 12 of 15 some clinical evidence of gastroesophageal reflux (GER) had been noted, and 60 % of the EA patients showed reflux on esophageal pH monitoring. There was no difference in the distribution of gastric myoelectrical activity between those with and without esophageal reflux on pH monitoring either before or after a meal. The authors of this study hypothesized that the significant increase in bradygastria and decrease in tachygastria in the postprandial period indicate that the myoelectrical response to ingested meals is sluggish [20]. The lack of a relationship between symptoms and abnormal myoelectrical activity evident in this study was a feature of the studies of Cheng et al. and Yagi et al. In particular there is no clear association between the presence of GER and abnormalities of gastric slow waves from these studies.

Gastric Function

There are limited data on the mechanical function of the stomach after EA repair. Investigators have focused on the stomach mainly to try and explain upper digestive symptoms that are reported in patients after EA repair in children and adults [21]. Gastric emptying by scintigraphy is the gold standard for the study of gastric function [21, 22]. It was first described by Griffith et al. in 1966 and is now used routinely in adult and pediatric patients to assess gastric emptying of solids and liquids [22]. Jolly et al. used scintigraphy to assess liquid-phase gastric emptying as well as GER in children after EA repair [3]. The authors noted that gastric emptying delay and GER were related to the use of tension on the esophageal ends in achieving primary EA repair. In a study by Montgomery et al. using a mixed meal comprising of pancakes in older children gastric emptying was abnormal in two of ten children with GER [23]. Romeo et al. examined gastric emptying in patients who had EA repair in childhood, 60 % of whom had symptoms of dyspepsia and dysphagia [24]. They found longer gastric emptying times in EA patients compared to controls with overt gastric delay in 4 of 11 patients using solid-phase emptying. The authors concluded that delayed gastric emptying is common and may be responsible for GER in EA patients. Our own data (unpublished) has been based on long-gap EA patients [10]. We examined liquid-phase gastric emptying in nine infants with only one showing delay (gastric emptying half-life – T ½ of >90 min); all our patients had undergone a fundoplication. In the most recent publication on the subject by Caldaro et al., 12 of 39 patients with EA exhibited delayed gastric emptying [25]. The possibility of gastric emptying delay as a cause of significant GER though elegant is not clear from the pediatric data. In a study by Aktas et al., gastric emptying times did correlate with the degree of scintigraphically assessed GER in infants [26]. In children with severe neurological injury from birth (cerebral palsy), gastric emptying times were found to be the same in patients with pathological GER diagnosed with pH monitoring and control patients [27]. Gastric emptying, foregut dysmotility, and GER often coexist. This has been noted in specific patient groups such as the abovementioned children with neurological injury [28], as part of morphological syndromes and foregut anatomical disorders. Manometric study of gastric motility in EA patients was conducted by Romeo et al. in the study of EA patients discussed above [24]. The investigators were able to recognize the features of the migrating motor complex in the interdigestive phases: phase I, a quiescent period; phase II consisted of irregular motor activity; and phase III a period of coordinated contraction. Three peristaltic wave types (I, II, and III) cycle were also identified. In 5 of 11 patients, the duration of the third phase and the frequency and amplitude of the peristaltic waves were abnormal. The authors found that antral hypomotility was due to increased duration of the third fasting phase and to reduced amplitude of type III

peristaltic waves. The significance of the findings was less clear in that two patients with GER were symptomatic and had delayed gastric emptying and abnormal gastric manometry, two others with delayed gastric emptying and abnormal manometry had no symptoms, and one patient had manometric abnormality but without major symptoms or gastric emptying problems. The authors noted that the alterations observed had some similarities with those observed in patients with primary or secondary dyspeptic syndromes. As noted above antral dysmotility may be a feature abnormal slow-wave activity [20, 29]. It could be postulated that foregut dysmotility gives rise to gastric emptying delay which in turn results in GER. In addition primary repair of EA could potentially involve disruption of the vagus nerve further complicating the etiological relationship between electrophysiology, manometry, and symptomatology in these patients [30].

Fundoplication

Gastroesophageal reflux is a common sequel to congenital disorders of the foregut. In one study almost all patients treated for EA developed GER [31]. Not surprisingly therefore most case series of EA patients show that a proportion of children undergo fundoplication [32]. Fundoplication in children with preexisting upper gastrointestinal dysmotility may however be problematic. In a series of children evaluated for symptoms of upper gastrointestinal motor dysfunction having undergone fundoplication for severe GER symptoms were unchanged or worsened after fundoplication [33]. The outcome of fundoplication may be specifically related to gastric motility. Loots et al. examined dysphagia after fundoplication and found that children who developed postoperative dysphagia were those with preoperative gastric emptying delay as compared to children without gastric emptying problems [34]. Conversely mean gastric emptying time was shown to be reduced in patients after undergoing Nissen fundoplication [35]. The acceleration in gastric emptying after fundoplication was elaborated to represent a shift toward normal gastric emptying times in the vast majority of patients in one study [36]. This would imply that while post-fundoplication symptoms may develop in patients with preoperative gastric emptying delay, ultimately gastric emptying may be improved in this group. The findings in relation to post-fundoplication gastric emptying are however not consistent. In a study of children undergoing Nissen fundoplication for GER, the investigators were able to demonstrate reduction in gastric compliance, an increase in minimal gastric distending pressure, exacerbation of the sensation discomfort with gastric distension, and yet no effect on gastric emptying [37].

Patients with EA are likely to need a fundoplication if tension is necessarily applied to the esophageal ends to achieve primary anastomosis [19]. Wheatley et al. described wrap disruption and recurrent reflux in 33 % of a pediatric population treated for EA [38]. The authors theorized that upward tension on the wrap owing to the presence of a shortened esophagus probably predisposed these patients to an increased frequency of fundoplication failure. Snyder et al. also concluded that the same factors responsible for the development of reflux in children with EA (poor acid clearance, altered motility, esophageal shortening) may contribute to the higher failure rate [39]. The authors showed that a complete or Nissen fundoplication failed twice as commonly as partial wrap fundoplication.

Apart from failure of the fundoplication, there are other consequences to a Nissen fundoplication in patients after EA repair. In the study by Curci et al. of 14 of 31 patients who underwent a Nissen fundoplication, dysphagia requiring supplemental gastrostomy feeding became an issue in 5 [32]. Of those five patients, four underwent postoperative manometry and extended pH monitoring, which revealed normal LES pressure, normal pH results, and marked esophageal dysmotility. The authors postulated that the fundoplication created a mechanical obstruction for those patients with a dyskinetic esophagus that cannot generate the pressure to open the LES. Other investigators have also concluded that particularly in children with EA, fundoplication cannot be considered a procedure

without complications and that problems resulting from disturbed gastric and esophageal motility should not be underestimated [40]. As discussed above fundoplication is inevitable in cases of long-gap EA where significant tension is necessary for primary repair, and, as noted by Esposito et al., dysphagia occurs independent of the anti-reflux mechanism adopted and that dysphagia especially when associated with respiratory symptoms may be a consequence of the primary dysmotility of the esophagus that typifies EA [41]. In our true long-gap EA patients, fundoplication was always performed after EA repair, and we have shown that the children are able to develop normal feeding milestones [42].

Long-Term Outcome

The most commonly reported long-term problem after EA repair is GER [43–48]. An Australian long-term follow-up study showed that older symptomatic patients had a high risk of complications: 63 % had reflux symptoms and 19 % had severe symptoms [43]. Another long-term study of 227 EA patients, spanning more than two decades, found GER in 127 patients (58 %); 56 patients (44 %) required an anti-reflux procedure [49]. Similarly in a series of 31 EA patients treated for GER 14 patients (45 %) required a Nissen fundoplication [32]. A study of adults found that GER was a common problem that impaired quality of life in 30 % of patients [44]. Tovar et al. showed symptoms were related to reduced acid clearance as a result of ineffective peristalsis in the distal esophagus in relation to GER [45], and as discussed above disordered esophageal motility is known to be a constant feature of the repaired esophagus in EA and therefore poor esophageal clearance [43–50]. Apart from low LES pressure and lack of propagating motility, esophageal contractions are simultaneous and weak, especially in the lower esophagus [20, 50, 51]. Low LES pressure in such patients has been associated with more severe reflux and aspiration pneumonia [52]. However, no correlation was found between pulmonary problems and the presence of GER, esophagitis, or esophageal dysfunction [49, 53].

Summary

The available data on gastric pathophysiology in the setting of EA is limited but does allow some conclusions to be drawn. It can be reasonably argued that the foregut neurophysiology may not be normal at birth at least in a proportion of EA patients; however, the genetic data which is largely based on animal models does not allow us to differentiate between the known human phenotypes. At birth the morphology of the stomach is not greatly altered in EA-TEF, and there is insufficient data to suggest gastric volume is reduced in cases of EA without TEF. Gastric emptying delay can be a problem in patients and may contribute to GER. The etiology of gastric delay may involve antral hypomotility related to congenital neuronal disruption in some patients. The effect of EA surgery and possible injury to the vagus nerve can be considered as contributing to gastric emptying delay. Tension applied to the esophagus affects the gastric cardia, and fundoplication is known to have an effect on gastric physiology though how this affects patients with EA is less clear. Our own findings based on long-gap EA patients along with other data indicate that indeed a small proportion of children consistently demonstrate abnormalities of gastric function; however, this does not account for the proportion of patients with a history of EA-TEF repair that expresses upper gastrointestinal symptoms. Apart from GER there is evidence to show that the upper digestive and pulmonic symptomatology is related to poor clearance from the esophagus and may be the major factor to consider in adults who present with esophagopulmonic symptoms.

References

1. Arsic D, Cameron V, Ellmers L, Quan QB, Keenan J, Beasley S. Adriamycin disruption of the Shh-Gli pathway is associated with abnormalities of foregut development. J Pediatr Surg. 2004;39(12):1747–53.
2. van den Brink GR. Hedgehog signaling in development and homeostasis of the gastrointestinal tract. Physiol Rev. 2007;87(4):1343–75.
3. Jolley SG, Johnson DG, Roberts CC, et al. Patterns of gastroesophageal reflux in children following repair of esophageal atresia and distal tracheoesophageal fistula. J Pediatr Surg. 1980;15(6):857–62.

4. Gross H, Filly A. Potential for a normal stomach to simulate the sonographic 'double bubble' sign. J Can Assoc Radiol. 1982;33:39–40.
5. Andrassy RJ, Mahour H. Gastrointestinal anomalies associated with esophageal atresia or tracheoesophageal fistula. Arch Surg. 1979;114:1125–8.
6. Sase M, Asada H, Okuda M, Kato H. Fetal gastric size in normal and abnormal pregnancies. Ultrasound Obstet Gynecol. 2002;19(5):467–70.
7. Nakazato Y, Landing BH, Wells TR. Abnormal Auerbach plexus in the esophagus and stomach of patients with esophageal atresia and tracheoesophageal fistula. J Pediatr Surg. 1986;21:831–7.
8. Shono T, Suita S. Motility studies of the esophagus in a case of esophageal atresia before primary anastomosis and in experimental models. Eur J Pediatr Surg. 1997;7:138–42.
9. Shono T, Suita S, Arima T, et al. Motility function of the esophagus before primary anastomosis in esophageal atresia. J Pediatr Surg. 1993;28:673–6.
10. Foker JE, Kendall-Krosch TC, Katton K, Munroe F, Khan KM. Long-gap esophageal atresia treated by growth induction: the biological potential and early follow-up results. Semin Pediatr Surg. 2009; 18(1):23–9.
11. Kimble RM, Harding JE, Kolbe A. The vulnerable stomach in babies born with pure oesophageal atresia. Pediatr Surg Int. 1999;15(7):467–9.
12. Cheng W, Spitz L, Milla P. Surface electrogastrography in children with esophageal atresia. Pediatr Surg Int. 1997;12(8):552–5.
13. Hashitani H, Garcia-Londoño AP, Hirst GD, Edwards FR. Atypical slow waves generated in gastric corpus provide dominant pacemaker activity in guinea pig stomach. J Physiol. 2005;569(Pt 2):459–65.
14. Cucchiara S, Salvia G, Scarcella A, et al. Gestational maturation of electrical activity of the stomach. Dig Dis Sci. 1999;44:2008–13.
15. Riezzo G, Indrio F, Montagna O, et al. Gastric electrical activity and gastric emptying in term and preterm newborns. Neurogastroenterol Motil. 2000; 12:223–9.
16. Chen J, McCallum RW. Electrogastrogram: measurement, analysis and prospective applications. Med Biol Eng Comput. 1991;29:339–50.
17. Yagi M, Homma S, Iwafuchi M, Uchiyama M, Matsuda Y, Maruta T. Electrogastrography after operative repair of esophageal atresia. Pediatr Surg Int. 1997;12(5–6):340–3.
18. Sarna SK, Daniel EE. Threshold curves and refractoriness properties of gastric relaxation oscillators. Am J Physiol. 1974;226(4):749–55.
19. Dubois A. Gastric dysrhythmias: pathophysiologic and etiologic factors. Mayo Clin Proc. 1989;64(2):246–50. Review.
20. Bókay J, Kis E, Verebély T. Myoelectrical activity of the stomach after surgical correction of esophageal atresia. J Pediatr Surg. 2005;40:1732–6.
21. Orringer MB, Kirsh MM, Sloan H. Long term esophageal function following repair of esophageal atresia. Ann Surg. 1977;186:436–43.
22. Urbain J-LC, Charkes ND. Recent advances in gastric emptying scintigraphy. Semin Nucl Med. 1995;25: 318–25.
23. Montgomery M, Escobar-Billing R, Hellström PM, Karlsson KA, Frenckner B. Impaired gastric emptying in children with repaired esophageal atresia: a controlled study. J Pediatr Surg. 1998;33(3):476–80.
24. Romeo C, Bonanno N, Baldari S, et al. Gastric motility disorders in patients operated on for esophageal atresia and tracheoesophageal fistula: long-term evaluation. J Pediatr Surg. 2000;35(5):740–4.
25. Caldaro T, Garganese MC, Torroni F, et al. Delayed gastric emptying and typical scintigraphic gastric curves in children with gastroesophageal reflux disease: could pyloromyotomy improve this condition? J Pediatr Surg. 2011;46(5):863–9.
26. Aktas A, Ciftci I, Caner B. The relation between the degree of gastro-esophageal reflux and the rate of gastric emptying. Nucl Med Commun. 1999;20:907–10.
27. Spiroglou K, Xinias I, Karatzas N, Karatza E, Arsos G, Panteliadis C. Gastric emptying in children with cerebral palsy and gastroesophageal reflux. Pediatr Neurol. 2004;31(3):177–82.
28. Werlin SL. Antroduodenal motility in neurologically handicapped children with feeding intolerance. BMC Gastroenterol. 2004;4:19.
29. Haight C, Towsley HH. Congenital atresia of the esophagus with tracheoesophageal fistula. Extra pleural ligation of the fistula and end to end anastomosis of esophageal segments. Surg Gynecol Obstet. 1943;76:672–88.
30. Davies MR. Anatomy of the extrinsic motor nerve supply to mobilized segments of the oesophagus disrupted by dissection during repair of oesophageal atresia with distal fistula. Br J Surg. 1996;83(9):1268–70.
31. Ottolenghi A, Camoglio FS, Valletta E, Giacomello L, Pasquini A. Primary and secondary gastro-esophageal reflux in pediatric age. Minerva Pediatr. 2004;56(1):91–6.
32. Curci MR, Dibbins AW. Problems associated with a Nissen fundoplication following tracheoesophageal fistula and esophageal atresia repair. Arch Surg. 1988;123(5):618–20.
33. Di Lorenzo C, Flores A, Hyman PE. Intestinal motility in symptomatic children with fundoplication. J Pediatr Gastroenterol Nutr. 1991;12(2):169–73.
34. Loots C, van Herwaarden MY, Benninga MA, Vanderzee DC, van Wijk MP, Omari TI. Gastroesophageal reflux, esophageal function, gastric emptying, and the relationship to dysphagia before and after antireflux surgery in children. J Pediatr. 2012. doi:10.1016/j.jpeds.2012.08.045. doi:pii: S0022-3476(12)01003-7, [Epub ahead of print].
35. Pacilli M, Pierro A, Lindley KJ, Curry JI, Eaton S. Gastric emptying is accelerated following laparoscopic Nissen fundoplication. Eur J Pediatr Surg. 2008;18(6):395–7.
36. Estevão-Costa J, Fragoso AC, Prata MJ, et al. Gastric emptying and antireflux surgery. Pediatr Surg Int. 2011;27(4):367–71.

37. Mousa H, Caniano DA, Alhajj M, Gibson L, Di Lorenzo C, Binkowitz L. Effect of Nissen fundoplication on gastric motor and sensory functions. J Pediatr Gastroenterol Nutr. 2006;43(2):185–9.
38. Wheatley MJ, Coran AG, Wesley JR. Efficacy of the Nissen fundoplication in the management of gastroesophageal reflux following esophageal atresia repair. J Pediatr Surg. 1993;28:53–5.
39. Snyder CL, Ramachandran V, Kennedy AP, Gittes GK, Ashcraft KW, Holder TM. Efficacy of partial wrap fundoplication for gastroesophageal reflux after repair of esophageal atresia. J Pediatr Surg. 1997;32(7):1089–91.
40. Holschneider P, Dübbers M, Engelskirchen R, Trompelt J, Holschneider AM. Results of the operative treatment of gastroesophageal reflux in childhood with particular focus on patients with esophageal atresia. Eur J Pediatr Surg. 2007;17(3):163–75.
41. Esposito C, Langer JC, Schaarschmidt K, et al. Laparoscopic antireflux procedures in the management of gastroesophageal reflux following esophageal atresia repair. J Pediatr Gastroenterol Nutr. 2005;40(3):349–51.
42. Khan KM, Kendall Krosch TC, Eickhoff JC, Sabati AA, Brudney J, Rivard AL, Foker JE. Development of feeding milestones after primary repair of long-gap esophageal Atresia. Early Hum Dev. 2009;85(6):387–92.
43. Taylor AC, Breen KJ, Auldist A, et al. Reflux and related pathology in adults who were born with esophageal atresia: a long-term follow-up study. Clin Gastroenterol Hepatol. 2007;5(6):702–6.
44. Somppi E, Tammela O, Ruuska T, et al. Outcome of patients operated on for esophageal atresia: 30 years experience. J Pediatr Surg. 1998;33:1341–6.
45. Tovar JA, Diez-Pardo JA, Murcia J, et al. Ambulatory 24-hour manometric and pH metric evidence of permanent impairment of clearance capacity in patients with esophageal atresia. J Pediatr Surg. 1995;30:1224–31.
46. Biller JA, Allen JL, Schuster SR, et al. Long-term evaluation of esophageal and pulmonary function in patients with repaired esophageal atresia and tracheoesophageal fistula. Dig Dis Sci. 1987;32:985–90.
47. Krug E, Bergmeijer JH, Dees J, de Krijger R, Mooi WJ, Hazebroek FW. Gastroesophageal reflux and Barrett's esophagus in adults born with esophageal atresia. Am J Gastroenterol. 1999;94(10):2825–8.
48. Deurloo JA, Ekkelkamp S, Taminiau JA, et al. Esophagitis and Barrett esophagus after correction of esophageal atresia. J Pediatr Surg. 2005;40(8):1227–31.
49. Engum SA, Grosfeld JL, West KW, Rescorla FJ, Scherer 3rd LR. Analysis of morbidity and mortality in 227 cases of esophageal atresia and/or tracheoesophageal fistula over two decades. Arch Surg. 1995;130(5):502–8.
50. Werlin SL, Dodds WJ, Hogan WJ, et al. Esophageal function in esophageal atresia. Dig Dis Sci. 1981;26:796–800.
51. Dutta HK, Grover VP, Dwivedi SN, Bhatnagar V. Manometric evaluation of postoperative patients of esophageal atresia and tracheo-esophageal fistula. Eur J Pediatr Surg. 2001;11(6):371–6.
52. LeSouef PN, Myers NA, Landau LI. Etiologic factors in long term respiratory function abnormalities following esophageal atresia repair. J Pediatr Surg. 1987;22:918–22.
53. Lopes MF, Botelho MF. Midterm follow-up of esophageal anastomosis for esophageal atresia repair: long-gap versus non-long-gap. Dis Esophagus. 2007;20(5):428–35.