unnecessary use of mobile phones should be avoided by health promotion activities, such as group discussion, public presentations and through electronic and print media sources. In addition, further large sized studies, along with detailed clinical examinations, are needed to study the long-term effects of mobile phone use and hearing and vision disorders.

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References

1. Hyland GJ. Physics and biology of mobile telephony. ***Lancet*** 2000; 356 : 1833-1836.
2. Kellenyi L, Thuroczy G, Faludy B, Lenard L. Effects of mobile GSM radiotelephone exposure on the auditory brainstem response (ABR). ***Neurobiology*** 1999; 7: 79-81.
3. Oftedal G, Wilen J, Sandstrom M, Mild KH. Symptoms experienced in connection with mobile phone use. ***Occup Med*** 2000; 50: 237-245.
4. Sandstrom M, Wilen J, Oftedal G, Mild KH. Mobile phone use and subjective symptoms. Comparison of symptoms experienced by users of analogue and digital mobile phones. ***Occup Med*** 2001; 51: 25-35.
5. Mobile phone blindness- 2 cases thought to be caused by mobile phones [cited Dec 14, 2003] Available from: www.greenhealthwatch.com http://www.ehn.clara.net/mobilephones.htm#newsafetylimits

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Duodeno-jejunal anastomosis with trans anastomotic nasojejunal tube for congenital duodenal obstruction

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Duodenal atresia, severe duodenal stenosis, or both, necessitate an urgent reestablishment of the duodenal continuity, by duodeno-duodenostomy or duodeno-jejunostomy. Calder[1] reported the first description of duodenal atresia in 1773, but Vidal[2] who performed a gastro-jejunostomy records the first successfully treated case in 1905 in France. The first successful duodeno-jejunostomy was in Denmark performed by Ernst[3] in 1914. Duodeno-duodenal or duodeno-jejunal anastomosis may not always function correctly due to some type of a dysmotility of the duodenum as a result of it being so thick and dilated. Neonatal intensive care and total parenteral nutrition have significantly improved the outcome of these patients. Although some studies have reported that the operation of duodeno-jejunostomy with trans-anastomotic stenting is to be reserved for a second time repair of this congenital anomaly,[4] we stressed in this study our satisfaction with the results of this procedure.

From November 1993 to February 2004, 21 neonates with congenital duodenal obstruction underwent a trans meso-colic duodeno-jejunal anastomosis stented by a 6 French (Fr) trans-anastomotic naso-jejunal feeding tube (TNJT) at Al-Hada Military Hospital, Attaif and King Faisal Specialist Hospital and Research Centre, Jeddah, Kingdom of Saudi Arabia. Anastomosis was always chosen to be at the distal lower aspect of the dilated duodenum. The TNJT is kept in place for 2 weeks. A second decompressive nasogastric tube size 8 Fr is also inserted during the procedure and removed before starting oral feeding on day 4, when simple abdominal radiograph showed the normal intestinal containing air distribution. Epidemiological, clinical, radiological and therapeutic data as well as their postoperative course were abstracted from their files. The complications and the outcome of surgery were also registered. The types of anomalies were defined during surgery. Extrinsic obstruction secondary to malrotation, intra luminal valve and, short stenosis was excluded from this study.

First degree of consanguinity was found in 16 cases. Prenatal diagnosis was suspicious in 10 of the 19 cases that underwent a prenatal ultrasound, 10 were associated with polyhydramnios, and prematurity as noted in 11 cases out of 21 (**Table 1**). The associated congenital anomalies were found in 12 cases, the most common was cardiac in 7/21, Down's syndrome in 7/21, renal anomalies requiring a urology follow up in 2/21, malrotation with atresia in 2/21, musculo-skeletal in 2, and cloacae in one. None of the patients presented with other intestinal atresia or exteriorized biliary tract anomalies. In 19 cases, the postnatal diagnosis was confirmed within the first 48 hours by the presence of the classical radiological double bubble shape. Abdominal radiograph was requested to confirm the prenatal ultrasound finding or as a work up for recurrent vomiting. In the 2 cases of annular pancreas with simple stenosis, the diagnosis was carried out on the fourth and fifth day by contrast meal for feeding intolerance (gastric content copious vomiting occurring shortly after each meal), the simple abdominal radiograph was not conclusive. In 18 cases, laparotomy was performed within 48-hour post diagnosis, the others were postponed for preoperative cardiac evaluation, or infections secondary to aspiration pneumonia.

Laparotomy confirmed the type of the obstruction, atresia with a duodenal gap was the most common cause in 11, annular pancreas in 8, and cordonal atresia in 2 cases. None had the classic apple-peel syndrome. In 18, the short-term postoperative course was uneventful, oral feeding started on the fourth day, including one patient referred with 2 days history of drained anastomotic leak that occurred on the second day of his duodeno-duodenal anastomosis. Leak was confirmed by ultrasound for abdominal distension, showing the presence of significant intra-abdominal fluid. This patient recovered with the normal delay from immediate re-exploration, resection of the duodenojejunal loop and, duodeno jejunal latero-terminal anastomosis with TNJT and protective gastrostomy. In 3 cases, the TNJT fell down at 48 hours, 4 days and 9 days post op, the first was discharged against medical advice on day 12 before tolerating oral feeding, the others had an initial resurge in the symptoms of duodenal obstruction, to be resolved later, on day 16 in the second patient and day 13 in the third patient, TNJT in place was removed 4 days later. We report that one mortality occurred after cardiac surgery was carried out at the age of 23 days: case of great vessels transposition, duodenal atresia and, cloacae malformation. His duodenal atresia repair carried out at the age of 3 days was uneventful. Long term follow up was uneventful, except in one patient who had an episode of adhesive intestinal obstruction treated conservatively, and another 2-year-old girl still under regular follow up for single gallstone of 10 mm diameters, diagnosed at the age of 3 months.

Failure of canalization of the duodenum between the eighth and tenth gestational week can lead to its atresia or stenosis. On the contrary, there are 2 major hypotheses concerning the development of annular pancreas, adhesion of the right ventral anlage to the duodenal wall (Lecco's theory), and persistence of the left ventral anlage (Baldwin's theory).[5] Atresia or complete obstruction may be seen with duodenal muscular continuity -cord type- or with a gap usually filled in with pancreatic tissue. An intact membrane in the shape of a "wind-sock" web is a well-known anomaly and is almost always intimately involved with the entry of bile ducts, which call for extra vigilance during the operation. The overall incidence of intestinal atresia was estimated worldwide to be one per 2500 live births, making this anomaly approximately twice as common as esophageal atresia or congenital diaphragmatic hernia and almost 3 times more common than Hirschsprung disease. It seems to be more frequent in our region by the high percentage rate of consanguinity. The sex distribution is almost equal, and familial occurrence is rare and complex (for example: Feingold syndrome, an autosomal dominant condition, including tracheo-esophageal atresia, microcephaly, hand and foot anomalies, fascial dysmorphism, and developmental delay).[6] Associated anomalies are frequent and variable.[7] Polyhydramnios is very high in 30-78% of cases, prematurity is noted in more than one third of cases, and almost 50% are associated with other congenital abnormalities represented by cardiac anomalies which could dictate the final outcome more than the urinary tract, digestive or musculo-skeletal anomalies. Up to 40% have trisomy 21. The distal biliary tree anomalies are common, in 85% of cases it opens in the proximal duodenal segment. Association with chyloascites has not been reported previously. The prenatal diagnosis is frequently performed by the prenatal ultrasound showing the dilated stomach and proximal duodenum; after the 25th gestational week, the discovery of polyhydramnios should incite the research of an upper intestinal obstruction. Post-natal diagnosis should not be delayed until the occurrence of pulmonary complications. Bile stained gastric fluid, feeding intolerance or recurrence of clear or bile stained vomiting starting within hours of birth require an abdominal radiograph to confirm the diagnosis by the classic finding of double bubble.

Table 1 - Presentation pictures.

Birth weight	Gender	Type of anomaly	Dilated stomach on prenatal ultrasound	Polyhydramnios
1000g	1 male	Atresia, 1 with Down's syndrome	Negative	0
1550 - 2500g	4 male 5 female	Atresia- 5 Annular pancreas - 4 Down's syndrome - 4	3 of 8 were negative	3: 2 noticed by ultrasound
More than 2500g	6 Male 5 Female	Atresia - 7, Annular pancreas 4: 2 with simple stenosis and 2 with Down's syndrome	2 of 10 were negative	7: 5 noticed by ultrasound

The presence of intestinal gas beyond the duodenum indicates incomplete obstruction, in this case a mid gut volvulus cannot be excluded and even in absence of its evidence, urgent exploration is recommended as soon as the patient is stable. Intestinal perforations are very rare complications. In cases of feeding difficulty or recurrent vomiting with unclear double bubble shape on the abdominal radiograph, other radiological modalities play an important role in the work up of more than direct diagnosis. Some cases of an incomplete obstruction are not recognized until adult life, usually diagnosed during the work up of peptic ulcer.[4] A thorough clinical examination to rule out other congenital anomalies, resuscitation, and gastric decompression, should precede the systematic and methodic surgical exploration. The type of the anomaly could orient on the etiology of congenital duodenal obstruction: malrotation, anterior portal vein. Associated biliary and intestinal anomalies must be considered before abdominal closure. We do not dissect the biliary tract unless an evident anomaly is seen. Simple malrotation without atresia is treated by the Ladd procedure, and the simple web or short stenosis needs plasty or resection, for all other cases we performed a trans mesocolic duodenojejunal anastomosis stented by a trans anastomotic feeding tube size 6 Fr for 2 weeks. This method provided full satisfaction due to its simplicity, early oral feeding tolerance as early as 4 days post op and early discharge with a small feeding tube shortened to the paranasal area and fixed by a simple adhesive tape. Whatever the surgical technique, the slow anastomotic function is a common problem in duodenoduodenal anastomosis, which is not always feasible and may require more extensive dissection to approximate the duodenal ends.[8] Duodenal tapering runs a higher risk of fistula and injury to the ampulla of Vater.[8] Currently, the laparoscopic approach is recommended,[9] yet whatever the surgical technique employed, trans anastomotic stent provides early oral feeding without adjunct complication. There were no complications related to the stent in our series. Endoscopic excision is reserved for partial web, fiber optic endoscopy identifies the obstruction and endoscopic retrograde cholangio-pancreatography has been able to document the abnormalities of the bile and pancreatic ducts system. Post operative complication had been reported in 70%, with 18% surgical redo surgery, anastomotic leak, and delay in feeding tolerance from 6-45 days. Long-term complication includes alkaline reflux and peptic ulceration, duodenal stasis with blind loop syndrome, recurrent abdominal pain or diarrhea. Gallstone has been also reported following duodenal atresia repair. Generally, the survival in infants with duodenal anomalies is more than 95%. Mortalities are the result of severe cardiac anomalies. Growth retardation and development delay are also very rare out of major associated anomalies.

In conclusion, congenital duodenal obstruction is a frequent anomaly; total parenteral nutrition as well as the great progress in the neonatal intensive care improved the outcome greatly. Trans mesocolic duodeno-jejunal anastomosis with TNJT provides early oral feeding and has no inherent specific complications.

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References

1. Calder E. Two examples of children born with preternatural conformation of the guts. ***Med Essays*** (Edinburgh) 1733; 1: 203.
2. Vidal E. 18e Congres de chirurgie, Paris: Proces verbaux memoires et discussion. ***Ass Fr Chir*** 1905; 18: 739.
3. Ernst NP. A case of congenital atresia of the duodenum treated successfully by operation. ***BMJ*** 1916; 1: 1644.
4. Cilley RE, Coran AG. Duodenoduodenostomy. In: Spitz L, Coran AG, editors. Pediatric Surgery. 5th ed. London (UK): Chapman & Hall; 1995.
5. Kamisawa T, Yuyang T, Egawa N, Ishiwata J, Okamoto A. A new embryologic hypothesis of annular pancreas. ***Hepatogastroenterology*** 2001; 48: 277-278.
6. Holder-Espinasse M, Ahmad Z, Hamill J, Pahari A, Misra D, Drake D. Familial syndromic duodenal atresia: Feingold syndrome. ***Eur J Pediatr Surg*** 2004; 14: 112-116.
7. Tashjian DB, Moriarty KP. Duodenal atresia with an anomalous common bile duct masquerading as a midgut volvulus. ***J Pediatr Surg*** 2000; 36: 956-957.
8. Alexander F, Difiore J, Stallion A. Triangular tapered duodenoplasty for the treatment of congenital duodenal obstruction. ***J Pediatr Surg*** 2002; 37: 862-864.
9. Seyaert H, Valla JS, Van Hoord E. Diaphragmatic duodenal atresia: lapatoscopic repair. ***Eur J Pediatr Surg*** 2003; 13: 414-416.

The prevalence of *Candida dubliniensis* among germ tube positive candida samples isolated from the respiratory tract

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Candida dubliniensis (*C.dubliniensis*) is one of the germ tube and chlamydospore forming Candida species, which was first recognized in 1995. It is difficult to differentiate from *Candida albicans* (*C.albicans*) with the standard diagnostic laboratory methods due to their similar phenotypic characteristics. However, *C.dubliniensis* can be