1.1 Introduction

Evaluating the normal anatomy and detecting an abnormality of fetal GI tract is based on sonography: it is an evidence. However, the prenatal US demonstrates true insufficiencies for diagnosing GI tract malformation (Corteville et al. 1996; Haeusler et al. 2002; Phelps et al. 1997). Actually, sonographic imaging is only able to detect a bowel loop dilatation or hyperechogenic bowel. Rarely, etiological diagnosis may be done, such as Down syndrome with duodenal stenosis. Obviously, even where the mechanism of bowel atresia is unknown, the infant will be operated on in the first days of life. Nevertheless, a simple dilatation may refer to a more severe disorder: bowel volvulus, multiple atresia, apple peel syndrome. A volvulus may impair the bowel vitality, and the diagnostic emergency cannot be assessed with US. In the same way, while US may show and measure a normal fetal colon, it is actually unable to affirm a microcolon, which is an extremely interesting etiological marker: small or large bowel atresia, meconium ileus, megacystis-microcolon-intestinal hypoperistalsis syndrome.

These true insufficiencies of US explain the development of fetal bowel MR imaging. This relatively recent examination demonstrates specific and different signals of the normal content of small and large bowel: the normal GI tract anatomy is accurately assessed. These potentialities are evident, especially with severe fetal bowel abnormalities (Benachi et al. 2001; Brugger and Prayer 2006; Daltro et al. 2005; Farhataziz et al. 2005; Hill et al. 2005; Hubbard and Harty 2000; Levine et al. 1996; Mendez et al. 2003; Miyakoshi et al. 2001; Ozkan et al. 2004; Saguintaah et al. 2002; Sasaki et al. 2006; Veyrac et al. 2004).

Thus, the US detection and improving MR diagnosis will deeply change the neonatal management: delivery within a medical center including pediatric
surgery department, radiological and sonographic imaging during first hours of life, early medical or surgical treatment.

1.2
The Normal Fetal GI Tract

While the 1st trimester of gestation is the embryonic phase of anatomical GI tract formation, the 2nd and 3rd trimesters represent the fetal phase of enzymatic and functional development: complete maturation of intestinal motricity, secretion and absorption will be required for normal neonatal intestinal function. These anatomical and physiological data should be known to understand fetal bowel imaging.

1.2.1
Anatomical Development of the Fetal GI Tract

1.2.1.1
Molecular Biology

The anatomical development of the GI tract is well known (Grand et al. 1976) and the literature widely describes the neural crest cells bowel colonization (Burns and Le Douarin 2001), the smooth muscle differentiation (Wallace and Burns 2005) and the early appearance of intestinal villi (Moxey and Trier 1979). During these last years, the advances in molecular biology, based on experimental (Hermiston et al. 1993; Shi and Ischizuya Oka 1996) and human (Walters et al. 1997) studies have shown the reality of a genetic programming. For example, the right/left isomerism is early genetically determined (Gebbia et al. 1997). The responsibility of a gene RET mutation in Hirschsprung disease is well established. Finally, genes HOX mutations are demonstrated in several gastrointestinal abnormalities: abnormal HOX-C4 gene induces esophageal obstruction with abnormal epithelial cell proliferation and muscle development (Boulet and Capecchi 1996); gene HOX D-12 is involved in disorders of the anal muscle wall formation (Kondo et al. 1996). Thus molecular biology is important in understanding the ontogeny of GI tract and the development of congenital abnormalities with the future hope of genetic therapy (Rustgi and Podolski 1997).

1.2.1.2
Embryologic Development of the Fetal GI Tract

The primitive gut appears at the 4th week’s gestation as a hollow tube extending from the buccopharyngeal to the cloacal membrane, initially closed. It is made of three parts (Diagram 1.1): the foregut, midgut and hindgut. The foregut gives origin to the pharynx, thoracic and abdominal esophagus, stomach and upper half of the duodenum; it is supplied by vitelline arteries. The midgut gives origin to the lower half of duodenum, the jejunum, ileum, cecum, appendix, ascending colon and the right 2/3 of the transverse colon; it is supplied by celiac and superior mesenteric arteries. The hindgut gives origin to the left 1/3 of transverse colon, the descending colon, sigmoid colon and rectum; it is supplied by the inferior mesenteric artery.

1.2.1.2.1
The Foregut

With rupture of the buccopharyngeal membrane at the beginning of the 4th week, the GI tract communicates with amniotic cavity. The esophagus, separated from the trachea by a tracheoesophageal septum, rapidly lengthens; this elongation results from the cephalic pole development with pharyngeal ascension, the heart development and head retroflexion. The stomach first appears (5th week) as a fusiform dilatation. At the 6th–8th weeks, it undergoes a 90° rotation. At the 8th week the typical shape is obtained with greater and lesser curvatures, body, fundus, antrum, pylorus. The duodenum originates from both the caudal portion of foregut and cranial portion of midgut. During the stomach rotation, the duodenum runs to the right before becoming fixed in a retroperitoneal position. In parallel, glandular structures develop from endodermal buds of the duodenum. The hepatic and biliary buds appear at the 3rd week and the pancreatic bud at the 4th week.

1.2.1.2.2
The Midgut

The midgut rotation results in the final configuration of small and large bowel. During the 5th week, the gut grows rapidly; there is insufficient room in the abdominal cavity (filled by the liver and kidneys) to contain all its different parts. Consequently, the midgut loop herniates into the extra embryonic coelom where it will remain and develop until the 10th week.