Chapter 4

Normal development and anatomy of the fetal abdomen

In this chapter the developmental aspects and normal sonographic appearance of the fetal abdominal wall, gastrointestinal tract, and renal tract are discussed. The last subchapter deals with the role of colour coded Doppler in the visualization of the intraabdominal vasculature.

4.1.1 Developmental aspects of the anterior abdominal wall

The development of the anterior abdominal wall occurs as the embryo folds in cranio-caudal and lateral directions, changing the flat trilaminar embryonic disc into its curvilinear embryonic shape by the third to fourth week²⁶. This folding in both longitudinal and transverse planes is caused by rapid growth of the embryo, particularly of the neural tube. It separates the intraembryonic coelom (peritoneal cavity) from the extraembryonic coelom (chorionic cavity); consequently the amnion fuses with the chorion peripherally and forms the covering of the umbilical cord centrally. Part of the yolk sac becomes thus incorporated into the embryo (midgut) and concurrently the connection of the midgut and yolk sac is reduced to a narrow yolk stalk with relative constriction at the umbilicus. Ultimately, closure depends on the fusion of the four ectomesodermic folds (cephalic, caudal, two laterals). Coalescence of the body stalk with the yolk stalk forms the primitive umbilical cord at 5-6 weeks gestation. As the abdominal cavity is temporarily too small for the rapidly developing intestinal loops, some of these are pushed into the extracoelomic space in the umbilical cord. These extruding intestinal loops form the so-called physiological umbilical hernia (see 4.1.2), which should have spontaneously resolved by 12 weeks of gestation.

4.1.2 Normal sonographic appearance of the fetal anterior abdominal wall

The anterior abdominal wall is formed by skin, subcutaneous fat and muscles. The muscles are visible as hypoechogenic structures. The skin line of the fetal chest and abdomen should be continuous and should have no abrupt angles or disruptions. To adequately judge the integrity of the anterior abdominal wall by ultrasound, the insertion

of the umbilical cord into the fetal abdomen should always be visualized in sagital view and in transverse cross section through the fetal abdomen, because scrutiny of this area will reveal the most common congenital anterior abdominal wall defects (omphalocele, gastroschisis, body stalk anomaly). No signs of protruding abdominal content or a mass adjacent to the anterior ventral wall should be encountered. The so-called physiological umbilical hernia should have resolved by 12 weeks of gestation¹².

Measurements of the fetal abdominal circumference⁹, or area⁴⁰, or abdominal diameters are of great importance in the detection and evaluation of small-for-gestational age and growth-retarded fetuses. These measurements should be determined at the level of the junction of the umbilical vein and portal sinus. The landmarks are the portal vein, the stomach, and the spine⁴⁰.

The role of colour Doppler in normal development of the anterior abdominal wall will be addressed in chapter 4.4.

4.2.1 Developmental aspects of the gastrointestinal tract

The primitive gut forms during the 4th week of gestation as the head, tail, and lateral folds incorporate the dorsal part of the yolk sac into the embryo. The endoderm of the primitive gut gives rise to most of the epithelium and glands of the digestive tract; the epithelium at the cranial and caudal end is derived from ectoderm of the stomodeum (primitive mouth) and proctodeum (anal pit), respectively. The muscular and fibrous elements, and the visceral peritoneum are derived from splanchnic mesenchyme surrounding the endodermal lining of the primitive gut.

The primitive gut is divided into three parts: the foregut, midgut and hindgut, which are supplied by three branches of the dorsal aorta: the celiac, superior mesenteric and inferior mesenteric artery²⁷.

The foregut eventually differentiates into the pharynx, lower respiratory tract, oesophagus, stomach, duodenum until the entrance of the common bile duct, pancreas, and liver and biliary apparatus.

The tracheoesophageal septum separates the oesophagus from the laryngotracheal diverticulum. The oesophagus subsequently elongates rapidly, mainly as a result from cranial body growth, and reaches its final relative length towards the end of the 7th week. The endoderm of the oesophagus proliferates and almost obliterates the lumen; recanalization occurs by the end of the embryonic period.

The stomach first appears as a fusiform dilatation of the caudal part of the foregut, which broadens ventrodorsally. Because the dorsal border grows faster than the ventral border, the greater curvature is formed during the 6th week. As the stomach acquires its adult shape, it rotates 90 degrees in a clockwise direction around its longitudinal axis.

The duodenum develops from the most caudal part of the foregut and the most cranial part of the midgut. These parts grow rapidly and form a C-shaped loop that projects ventrally; the junction of the foregut and midgut is at the apex of this embryonic duodenal loop. During the 5th and 6th week, the lumen of the duodenum becomes reduced and may be obliterated by epithelial cells, but it recanalizes through vacuolization by the end of the embryonic period²⁷.

The midgut gives rise to the caudal part of the duodenum, the small intestines, the caecum and appendix, and the ascending and proximal transverse colon. The initial wide connection between midgut and yolk sac becomes reduced to a narrow stalk or vitelline duct. At first, the midgut is suspended from the dorsal abdominal wall by a short mesentery. Both dorsal mesentery and midgut elongate rapidly. Because of a discrepancy between this elongation and that of the body of the embryo, a series of intestinal movements occur which are usually divided into three stages. (i) As the midgut elongates at the beginning of the 6th week, it forms a ventral U-shaped loop which herniates into the umbilical cord (physiological umbilical hernia) because of shortage of space intraabdominally, mainly caused by the relatively massive liver and kidneys. The yolk sac is attached to the apex of the U-shaped loop. The proximal limb grows rapidly and forms intestinal coils. Except for development of the cecal diverticulum, the caudal limb hardly changes. Within the umbilical cord the midgut rotates 90 degrees counterclockwise around the axis of the superior mesenteric artery. (ii) During the 10th week, probably as a result of the decrease in relative size of mesonephric kidneys and liver, and enlargement of the abdominal cavity, the intestines return rapidly to the intraabdominal cavity. The proximal limb (small intestines) return first and undergo a further 180-degree counterclockwise rotation. The cecum and appendix are now close to the right lobe of the liver. (iii) The third phase is characterized by lengthening of the colon which causes the cecum to descend into the right iliac fossa. As the intestines assume their final positions, their mesenteries are pressed against the posterior abdominal wall. As a result of fusion of the parietal peritoneum and mesentery of the ascending colon, the latter and the main distal part of the duodenum become situated retroperitoneally²⁷.

The hindgut, which extends from the midgut to the cloacal membrane (i.e., endoderm from the cloaca and ectoderm of proctodeum), develops into the distal part of the colon, rectum and upper portion of the anal canal, and part of the urogenital system (bladder and urethra). As a result of fusion of the peritoneum and mesentery of the descending colon, the latter will also be situated retroperitoneally. In the 6th week, the cloaca is divided by a wedge of mesenchyme, the urorectal septum, into the urogenital sinus ventrally and the rectum and upper anal canal dorsally. The area of fusion of the ororectal septum and cloacal membrane becomes the perineal body. The distal third of the anal canal develops from the anal pit (proctodeum)²⁷.

4.2.2 Normal sonographic appearance of the fetal gastrointestinal tract

In the normal situation, the oesophagus is empty, collapsed and situated behind the fetal trachea and heart, and therefore usually only occasionally visualized with ultrasound²¹. Recently, the normal sonographic appearance of the normal oesophagus has been described as a tubular echogenic structure³. Although the thoracic part of the fetal oesophagus could be identified in the majority of 155 uncomplicated pregnancies, the cervical and abdominal segments could only be visualized in a minority of cases³.

With transvaginal ultrasound the embryonic stomach can be detected from 8 weeks onwards while this was the case by 11 weeks of gestation in all fetuses⁵. Early fetal stomach filling probably results from fluid production from the intestinal epithelium because swallowing movements only start from 11 weeks onwards⁵. Also transabdominally, fetal stomach filling should be demonstrated in the left upper abdominal quadrant from 11 weeks in nearly all normal fetuses³³. Although nomograms for fetal stomach size are available¹⁴, interpretation of fetal stomach size should occur with caution since there is a large physiological variety in size and overlap between physiologically and pathologically dilated stomach. In a study on serial measurements of fetal stomach dimensions, Zimmer et al.(1992) also addressed the limited value of the diagnostic use of stomach measurements owing to their dynamically changing nature⁴³. Peristalsis of the fetal stomach has been noted as early as 14 weeks of gestation⁷. Also, absent fetal stomach filling can be a normal variant. The relevance of this phenomenon will be highlighted in chapter 5.2.

The small bowel is normally seen situated centrally in the fetal abdomen, while the large bowel appears as a tubular structure in the periphery. Distinguishing small bowel

from large bowel usually is possible from 20 weeks^{29,42}. Early in the second trimester, the meconium-filled small bowel is often observed as somewhat echogenic and may appear as a pseudomass^{13,25}. Unlike the colon, the small bowel undergoes continuous, active peristalsis^{29,31} which may be visible as early as 18 weeks of gestation²⁰. The internal diameter should not exceed 7 mm^{29,31}.

The large bowel can normally be seen as a long continuous tubular structure located in the flanks and upper abdomen and is filled with meconium. By 30 weeks' gestation haustrations can be clearly seen within the fetal colon^{29,31}. The meconial content appears hypoechoic in comparison to the fetal liver and bowel wall³¹. Peristalsis is less prominent than in the small bowel⁴². The colonic diameter may vary from 3 mm at 20 weeks to up to 20 mm or more near term^{15,31}. However, as with fetal stomach, there is a considerable overlap between physiological and pathological distension.

4.3.1 Developmental aspects of the renal tract

The fetal urinary tract arises from the intermediate mesoderm (kidneys and ureters) and cloaca (urinary bladder and urethra). The development of the renal tract has been described in detail by Moore (1988)²⁸ and by Reuss (1989) from our own centre³⁵. In this chapter only the main features of urinary tract development and growth will be highlighted.

In human embryos three successive sets of excretory organs develop. The first two sets, the pronephros ("forekidney") and mesonephros ("midkidney") degenerate before the third eventual set, the metanephros ("hindkidney") or permanent kidney, appears by the early 5th week of gestation. The metanephros develops from the simultaneous differentiation of the metanephric diverticulum (ureteral bud), which appears near the lower end of each mesonephric (Wolffian) duct and near its entry into the cloaca, and the metanephric mass of mesoderm (metanephrogenic blastema) into which it grows. As the ureteral bud extends dorsocranially, it thus grows into the metanephrogenic blastema which forms a cap over the ureteral bud. Soon after its appearance, the terminal end of the bud (ampulla) quickly starts with repeated dichotomous divisions. Eventually the stalk of the bud forms the ureter while the expanded cranial end of the ampulla becomes the renal pelvis, the calyces and papillae. The metanephrogenic blastema ultimately differentiates into renal parenchyma.

In the ampullar development four stages are recognized. The first stage (8th -14th weeks of gestation) is mainly characterized by repeated dichotomous ampullar divisions ultimately leading to successive generations of branches. The first three to five generations eventually expand to produce the renal pelvis. The next three to five generations form the calyces and papillae whereas the succeeding seven to eight produce the collecting tubules. Each newly formed ampulla induces formation of nephrons. The rate of ampullary division decreases towards the 14th week of gestation. The second stage of ampullar development (14th - 22nd weeks of gestation) is predominantly characterized by nephron formation with only occasional ampullar division. Each nephron attaches to the ampulla by which it was induced. When the next nephron is induced by the same ampulla, the connecting piece of the first nephron shifts. Ultimately, thus only the youngest nephron is directly attached to the ampulla. These arcades consist of four to six nephrons. In the third stage (22nd - 36th weeks) ampullar division has ceased and only further nephron formation occurs. By the end of pregnancy half of the about 1.10° nephrons are directly attached to the collecting tubules. The fourth phase extends into adulthood and is characterized by interstitial growth of tubules and an increase in blood vessels and connective tissue, eventually resulting in the renal corpuscle (glomerulus and Bowman's capsule) and associated tubules (proximal and distal convoluted tubules and Henle's loop). Once nephron induction is brought to halt, it is never resumed. No nephrons develop after birth, except in premature infants, but existing ones complete their differentiation and continue to increase in size until adulthood. The increase in renal size after birth thus results from hypertrophy and not from an increase in number of nephrons.

Initially, the kidneys are in the fetal pelvis, but gradually come to lie in their lumbar fossae as mainly caused by rapid growth of the caudal portion of the embryo. Initially the hilum of the kidney faces ventrally, but as the kidney ascends it rotates 90 degrees so that its hilum is directed medially.

During the 6th week the endodermal cloaca, which until then has been in communication with the mesonephric duct, will become divided into two parts by the caudally growing urorectal septum which reaches the cloacal membrane. The dorsal region forms part of the hindgut (posterior anal membrane which perforates at 7-8 weeks), the ventral part is primitive urogenital sinus (urogenital membrane). The bladder is mainly derived from the endodermal vesico-urethral canal and the lower end of the mesonephric duct. The upper part of the bladder is derived from a minor part of the allantois. The major part of the allantois regresses and becomes converted into the

urachus. During a complicated growth process the ureters come to open into the definitive bladder while the mesonephric ducts open lower down into the pelvic part of the definitive urogenital sinus. The fetal ureters open into the bladder with disappearance of the membrane at the junction at 9 weeks.

4.3.2 Normal sonographic appearance of the fetal renal tract

The fetal kidneys can be visualized reliably as early as 10 weeks of gestation, both by transabdominal¹⁸ and transvaginal ultrasound⁶. Depending on fetal positioning, transvaginal sonography clearly defines at least 90 per cent of fetal kidneys and urinary bladder by approximately 13 menstrual weeks³⁷ while the same can be achieved transabdominally by 20 weeks²⁴. In a transverse section through the fetal abdomen the kidneys can be visualized as two circular structures on either side of the fetal spine. In the longitudinal section they appear as two oval structures in a typical paraspinal location. Normal lobulation of the fetal kidneys is often quite apparent on the sonogram³². The sonographic appearance of the kidneys changes throughout pregnancy. In the early first trimester renal tissue is hyperechoic and gradually becoming less echogenic until after the 11th week, probably due to fluid production within the renal parenchyma. Fetal urine production starts around the 9th week. In the first and early second trimester, the kidneys are usually generally homogeneous in appearance, except for the central collecting structures. An echolucent, circular pelvis can often be seen surrounded by a focal area of echogenicity. As the kidney matures, the pyelo-calyceal system appears as an hypoechoic structure. The renal capsule becomes more visible and the distinction between the relative hypoechoic pyramids and the cortex becomes apparent during the third trimester. Identification of the characteristic configuration of the pyramids in anterior and posterior rows allows positive identification of the kidney. The renal arteries can be visualized with colour coded Doppler ultrasound as is described in detail in Chapter 4.4.

Nondilated fetal ureters are not routinely visualized.

The fetal bladder generally cannot be seen until the 10th week of gestation, but by 12 weeks the bladder can be identified in 50 per cent of cases and occupies an anterior midline position in the fetal pelvis¹⁸. The internal iliac arteries course around the lateral margins of the urinary bladder on their path toward the umbilicus and may assist in definitive identification of the bladder. Changes in bladder size may be frequently observed during a sonographic examination, especially since the fetus empties its bladder

every 30 to 45 minutes^{8,34,41}. Nonvisualization of the bladder with an otherwise normal sonogram and amniotic fluid compartment probably is not clinically significant and requires no follow-up¹⁰. A technique for calculation of fetal bladder volume and hourly fetal urinary production rate (HFUPR) was introduced by Campbell et al.(1973)⁸. The bladder wall is normally thin and virtually invisible, but may hypertrophy in the presence of low-level obstructive uropathy.

The nondilated urethra is difficult to detect in females, and in males it is imaged at a time when the penis is flaccid but appears as an echogenic line extending the length of an erect penis.

Finally, the relation between renal function and amniotic fluid volume should be addressed. During the first trimester the fetal kidneys contribute little if any to the amount of amniotic fluid, as during this period amniotic fluid is considered primarily a dialysate of fetal and maternal serum through the amniotic membrane. In contrast, after 16 weeks gestation fetal urine becomes the primary source of amniotic fluid^{1,16}.

4.3.3 Fetal renal biometry

Several authors have investigated different parameters resulting in normal value curves relative to gestational age. These parameters include kidney length³⁶, width and thickness^{4,18,36}, volume²², kidney perimeter to abdominal perimeter ratio²², kidney area to abdominal area ratio³⁸, and kidney circumference to abdominal circumference ratio ¹⁷. The first four parameters increase throughout gestation, whereas kidney size relative to the fetal abdomen remains more or less constant.

More recently, also more data on renal pelvic dimensions have been published^{2,11,19,30,39}. However, so far no uniform cut-off values have been defined. Also, dilatation of the urinary tract does not always signify obstruction. Conversely, a fetus can have obstructive uropathy in the absence of urinary tract dilatation (see 5.3.3 Obstructive uropathy).

4.4 Colour coded Doppler imaging of the normal (intra)abdominal vasculature

Data presented on colour Doppler imaging are part of a report on the clinical significance of colour coded Doppler ultrasound in normal and abnormal fetal development which was prepared for the "Ontwikkelingsgeneeskunde" Committee of the Dutch Sickness Benefit Council (Ziekenfondsraad) during the period 1990-1993 (OG 89-023).

Colour coded Doppler ultrasound is a method for noninvasively imaging blood flow by displaying characteristics of blood flow (direction, velocity and size) onto the two-dimensional echographic image by means of colour encoding of the Dopplergenerated flow signal.

Doppler systems emit ultrasound that is reflected off the moving red blood cells and then returned at a different frequency. Pulsed-wave Doppler uses a transducer which alternately transmits and receives ultrasound information. The frequency shift detected from blood cells approaching the transducer will be higher than the transmitted frequency thus causing a positive Doppler shift. Alternatively, the frequency shift detected from blood cells moving away from the transducer will be lower than the transmitted frequency and is termed a negative Doppler shift.

Colour Doppler systems add a separate processor that creates the colour flow image based upon the returning data and then integrates it with the two-dimensional anatomic ultrasonic image. All colour Doppler flow imaging systems encode the direction of flow into two primary colours, usually red and blue. Most colour flow systems now display the direction of flow toward the transducer in red (positive Doppler shift) and away from the transducer in blue (negative Doppler shift). There is also relative flow velocity information in the colour hues; the brighter the colour, the higher the velocity detected²³.

The question was addressed whether colour coded Doppler ultrasound provides additional information on (i) normal (intra)abdominal vessel distribution, and (ii) structural and functional information in cases of normal fetal anatomy, when compared with conventional real-time ultrasound. In chapter 5.4 the additional value of colour coded Doppler with respect to structural and functional information in cases of (intra)abdominal pathology will be addressed.

4.4.1 Materials and methods

A total of 218 pregnancies were included in the study, 166 of which presented with normal fetal anatomy and 62 with one or more fetal structural anomalies. Firstly, normal fetal anatomy and fetal biometry (biparietal diameter, head and upper abdominal circumference, femur length) were determined using conventional two-dimensional real-time ultrasound. This was followed by colour coded Doppler examination of the various fetal vessel structures as well as flow direction. The same procedure was adopted in the presence of fetal anomalies. The patient cohort which produced the data presented in this chapter consisted of two groups:

- Patients with an increased risk of an anomaly in their offspring (n=90). Pregnancy duration varied between 18 and 21 weeks of gestation. Reasons for referral were: (i) a previously affected infant; (ii) the patient or her partner were affected; (iii) insulin dependent diabetes; (iv) use of antiepileptic drugs; other drugs which are associated with an increased risk of fetal anomalies (eg. lithium). Data from this subset of patients merely represent normal intra-abdominal vasculature.
- Patients with a suspected fetal anomaly in the present pregnancy (n=76). In this group an abnormal finding elsewhere (uterus large or small for gestational age, oligohydramnios, polyhydramnios, premature labour), resulted in an ultrasound scan which raised suspicion of a fetal anomaly. Data from this subset of patients represent both normal and abnormal intraabdominal vasculature in the presence of fetal structural anomalies.

Scans were performed using transabdominal two-dimensional real-time and colour Doppler equipment (Toshiba SSA 270; carrier frequency 3.75 MHz), which was purchased for the present colour coded Doppler study. Spatial peak temporal average energy output was kept below 100 mW/cm² according to manufacturer specifications.

4.4.2 Normal intra-abdominal vasculature

A total of 166 pregnancies was studied, of which 120 between 14 and 25 weeks and 46 between 26 and 37 weeks of gestation. Emphasis was put on the visualization of the descending aorta, inferior vena cava, renal artery and vein (right and left), common iliac artery and vein (right and left), umbilical artery and vein, portal vein and ductus venosus (Table 1).

Table 1 Visualization of normal intra-abdominal vasculature; comparison between conventional real-time and colour coded Doppler ultrasound

		Conventional real-time ultrasound		Colour coded Doppler ultrasound	
		14-25 weeks $(n = 120)$	26-37 weeks $(n = 46)$	14-25 weeks $(n = 120)$	26-37 wks $(n = 46)$
AA		75%	78%	77%	80%
IVC		80%	82%	86%	83%
RA	Right	24%	32%	49%	50%
	Left	25%	30%	47%	46%
RV	Right	5 %	7 %	38%	41%
	Left	5 %	8 %	39%	44%
CIA	Right	68%	71%	73%	72%
	Left	70%	70%	80%	72%
CIV	Right	18%	21%	52%	76%
	Left	16%	19%	50%	76%
UA		100%	100%	100%	100%
UV		100%	100%	100%	100%
PV		70%	66%	78%	67%
DV		11%	14%	59%	61%

AA=abdominal aorta; IVC=inferior vena cava; RA=renal artery; RV=renal vein; CIA=common iliac artery; CIV=common iliac vein; UA=umbilical artery; UV=umbilical vein; PV=portal vein; DV=ductus venosus.

When compared with conventional 2D real-time ultrasound, colour coded Doppler ultrasound provided additional information regarding the visualization of the following vessels: renal artery (right and left) at 24-25 weeks (student T-test; p < 0.001), renal vein (left and right)(p < 0.001), common iliac vein (right and left)(p < 0.001) and ductus venosus (p < 0.001). Observations regarding the renal vein, common iliac artery and ductus venosus involve both the early and late pregnancy period. It was concluded that colour coded Doppler contributes to the visualization of the intra-abdominal vasculature, in particular the renal circulation, common iliac vein and ductus venosus. An example of colour coded Doppler imaging of the renal circulation is shown on page 73.

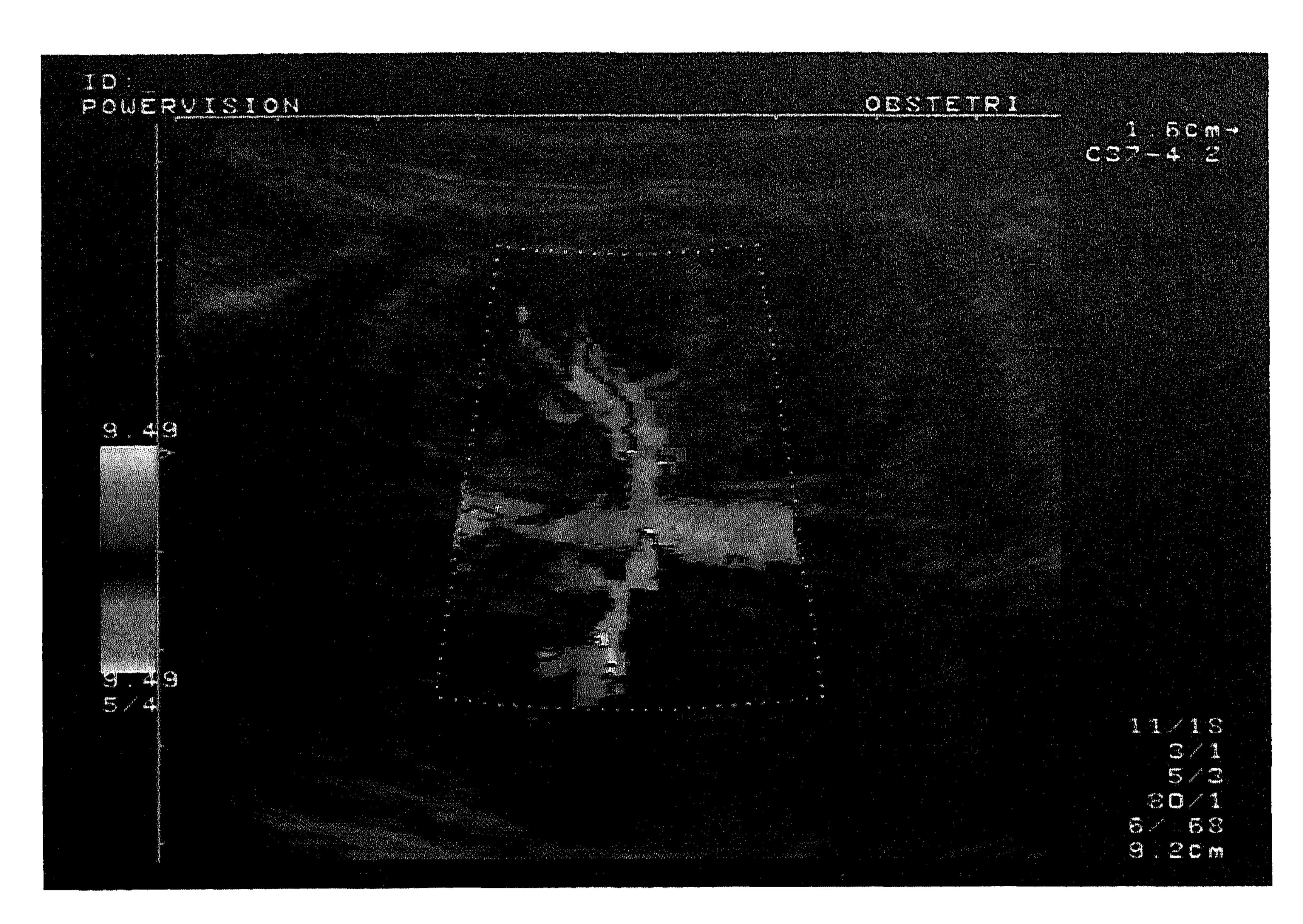


Figure 1 Colour Doppler imaging of the renal circulation (Toshiba SSA-380)



Figure 2 Colour angiography of the peripheral pulmonary vasculature (Toshiba SSH-140)

4.5 References

- Abramovich, D.R. (1978). The volume of amniotic fluid and its regulating factors. In: Fairweather, D.V.I., Eskes, T.K.A. (Eds.): Amniotic Fluid Research and Clinical Application, 2nd ed., Amsterdam, Excerpta Medica, 31-49
- Anderson, N., Clautice-Engle, T., Allan, R., Abbott, G., Wells, J.E. (1995). Detection of obstructive uropathy in the fetus: predictive value of sonographic measurements of renal pelvic diameter at various gestational ages, Am.J.Roentgenol., 164, 719-723.
- Avni, E.F., Rypens, F., Milaire, J. (1994). Fetal esophagus: normal sonographic appearance, J. Ultrasound Med., 13, 175-180.
- 4. Bertagnoli, L., Lalatta, F., Gallicchio, R., Fantuzzi, M., Rusca, M., Zorzoli, A., Deter, R.L. (1983). Quantative characterization of the growth of the fetal kidney, J.Clin.Ultrasound, 11, 349-356.
- Blaas, H.G., Eik-Nes, S.H., Kiserud, T., Hellevik, L.R. (1995). Early development of the abdominal wall, stomach and heart from 7 to 12 weeks of gestation: a longitudinal ultrasound study, Ultrasound Obstet.Gynecol., 6, 240-249.
- Bronshtein, M., Kushnir, O., Ben-Rafael, Z., Shalev, E., Nebel, L., Mashiach, S., Shalev, J. (1990). Transvaginal sonographic measurement of fetal kidneys in the first trimester, J.Clin.Ultrasound, 18, 299-301.
- Bronshtein, M., Yoffe, N., Zimmer, E.Z. (1992). Transvaginal sonography at 5 to 14 weeks' gestation: fetal stomach, abnormal cord insertion, and yolk sac, Am.J.Perinatol., 9, 344-347.
- 8. Campbell, S., Wladimiroff, J.W., Dewhurst, C.J. (1973). The antenatal measurement of fetal urine production, J.Obstet.Gynaecol.Br.Commonw., 80, 680-686.
- Campbell, S., Wilkin, D. (1975). Ultrasonic measurement of fetal abdomen circumference in the estimation of fetal weight, Br.J.Obstet.Gynaecol., 82, 689-697.
- Clautice-Engle, T, Pretorius, D,H., Budorick, N.E. (1991). Significance of nonvisualization of the fetal urinary bladder, J.Ultrasound Med., 10, 615-618.
- 11. Corteville, J.E., Gray, D.L., Crane, J.P. (1991). Congenital hydronephrosis: correlation of fetal ultrasonographic findings with infant outcome, Am.J.Obstet.Gynecol., 165, 384-388.

- Cyr, D.R., Mack, L.A., Schoenecker, S.A., Patten, R.M., Shepard, T.H., Shuman, W.P., Moss, A.A. (1986). Bowel migration in the normal fetus: US detection, Radiology, 161, 119-121.
- Fakhry, J., Reiser, M., Shapiro, L.R., Schechter, A., Pait, L.P., Glennon, A. (1986). Increased echogenicity in the lower fetal abdomen: a common normal variant in the second trimester, J.Ultrasound Med., 5, 489-492.
- Goldstein, I, Reece, E.A., Yarkoni, S., Wan, M., Green, J.L.J., Hobbins, J.C. (1987a). Growth of the fetal stomach in normal pregnancies, Obstet.Gynecol., 70, 641-644.
- Goldstein, I., Lockwood, C., Hobbins, J.C. (1987b). Ultrasound assessment of fetal intestinal development in the evaluation of gestational age, Obstet.Gynecol., 70, 682-686.
- Graham, D., Sanders, R.C. (1982). Amniotic fluid, Semin.Roentgenol., 17, 210-218.
- 17. Grannum, P., Bracken, M., Silverman, R., Hobbins, J.C. (1980). Assessment of fetal kidney size in normal gestation by comparison of ratio of kidney circumference to abdominal circumference, Am.J.Obstet.Gynecol., 136, 249-254.
- Green, J.J., Hobbins, J.C. (1988). Abdominal ultrasound examination of the first-trimester fetus, Am.J.Obstet.Gynecol., 159, 165-175.
- 19. Grignon, A., Filion, R., Filiatrault, D., Robitaille, P., Homsy, Y., Boutin, H., Leblond, R. (1986). Urinary tract dilatation in utero: classification and clinical applications, Radiology, 160, 645-647.
- Hertzberg, B.S., Bowie, J.D. (1990). Fetal gastrointestinal abnormalities, Radiol.Clin.North Am., 28, 101-114.
- Hertzberg, B.S. (1994). Sonography of the fetal gastrointestinal tract: anatomic variants, diagnositic pitfalls, and abnormalities, Am.J.Roentgenol., 162, 1175-1182.
- Jeanty, P, Dramaix-Wilmet, M., Elkhazen, N., Hubinont, C., van Regemorter, N. (1982).

 Measurement of fetal kidney growth on ultrasound, Radiology, 144, 159-162.
- Kisslo, J., Adams, D.B., Belkin, R.N. (1988). In: Doppler Color Flow Imaging, Churchill Livingstone (Eds), New York, 3-28.
- Lawson, T.L., Foley, W.D., Berland, L.L., Clark, K.E. (1981). Ultrasonic evaluation of fetal kidneys, Radiology, 138, 153-156.

- Manco, L.G., Nunan, F.A., Jr, Sohnen, H., Jacobs, E.J. (1986). Fetal small bowel simulating an abdominal mass at sonography, J.Clin.Ultrasound, 14, 404-407.
- Moore, K.L. (1988a). The embryonic period. In: The developing human: Clinically oriented embryology, 4th ed., Philadelphia, W.B.Saunders.
- Moore, K.L. (1988b). The digestive system. In: The developing human: Clinically oriented embryology, 4th ed., Philadelphia, W.B. Saunders.
- Moore, K.L. (1988c). The urogenital system. In: The developing human: Clinically oriented embryology, 4th ed., Philadelphia, W.B.Saunders.
- Nyberg, D.A., Mack, L.A., Patten, R.M., Cyr, D.R. (1987). Fetal bowel: normal sonographic findings, J.Ultrasound Med., 6, 3-6.
- Ouzounian, J.G., Castro, M.A., Fresquez, M., Al-Sulyman, O.M., Kovacs, B.W. (1996). Prognostic significance of antenatally detected fetal pyelectasis, Ultrasound Obstet.Gynecol., 7, 424-428.
- Parulekar, S.G. (1991). Sonography of normal fetal bowel, J. Ultrasound Med., 10, 211-220.
- Patriquin, H., Lefaivre, J-F., Lafortune, M., Russo, P., Boisvert, J. (1990). Fetal lobulation: an anatomo-ultrasonographic correlation, J. Ultrasound Med., 9, 191-197.
- Pretorius, D.H., Gosink, B.B., Clautice-Engle, T., Leopold, G.R., Minnick, C.M. (1988). Sonographic evaluation of the fetal stomach: significance of non-visualization, Am.J.Roentgenol., 151, 987-989.
- Rabinowitz, R., Peters, M.T., Vyas, S., Campbell, S., Nicolaides, K.H. (1989). Measurement of fetal urine production in normal pregnancy by real-time ultrasonography, Am.J.Obstet.Gynecol., 161, 1264-1266.
- Reuss, A. (1989). Renal tract anomalies in the human fetus. Prenatal ultrasound and genetic aspects, Thesis, Rotterdam.
- Romero, R., Pilu, G., Jeanty, P., Ghidini, A. (1988). In: Prenatal diagnosis of congenital anomalies, Appleton & Lange, Connecticut San Mateo, California, 256-259.
- Rosati, P., Guariglia, L. (1996). Transvaginal sonographic assessment of the fetal urinary tract in early pregnancy, Ultrasound Obstet. Gynecol., 7, 95-100.

- Sato, A., Yamaguchi, Y., Liou, S.M., Sato, M., Suzuki, M. (1985). Growth of the fetal kidney assessed by real-time ultrasound, Gynecol. Obstet. Invest., 20, 1-5.
- Scott, J.E., Wright, B., Wilson, G., Pearson, I.A., Matthews, J.N.S., Rose, P.G. (1995). Measuring the fetal kidney with ultrasonography, Br.J.Urol., 76, 769-774.
- 40. Stoutenbeek, P. (1982). Echo-abdominometrie en foetale groei, Thesis, Utrecht.
- Wladimiroff, J.W., Campbell, S. (1974). Fetal urine-production rates in normal and complicated pregnancy, Lancet, i, 151-154.
- 42. Zilanti, M., Fernandez, S. (1983). Correlation of ultrasonic images of fetal intestine with gestational age and fetal maturity, Obstet.Gynecol., 62, 569-573.
- Zimmer, E.Z., Chao, C.R., Abramovich, G., Timor-Tritsch, I.E. (1992). Fetal stomach measurements: not reproducible by the same observer, J.Ultrasound Med., 11, 663-665.