OLIGOHYDRAMNIOS: SONOGRAPHIC ASSESSMENT & CLINICAL IMPLICATIONS

INTRODUCTION

A decreased amniotic fluid volume is frequently one of the first clues to an underlying fetal abnormality. The sonographer/sonologist should, therefore, have a basic understanding of the mechanisms responsible for normal amniotic fluid production. Once the derivation of amniotic fluid is understood, the potential mechanisms that can result in oligohydramnios can be better appreciated.

Amniotic fluid has a number of important roles in embryo/fetal development:

1. Permitting fetal movement and the development of the musculoskeletal system.
2. Swallowing of amniotic fluid enhances the growth and development of the gastrointestinal tract.
3. The ingestion of amniotic fluid provides some fetal nutrition and essential nutrients.
4. Amniotic fluid volume maintains amniotic fluid pressure thereby reducing the loss of lung liquid - an essential component to pulmonary development. (Nicolini, 1989).
5. Protects the fetus from external trauma.
6. Protects the umbilical cord from compression.
7. It's constant temperature helps to maintain the embryo's body temperature.
8. It's bacteristatic properties reduces the potential for infection.

The factors involved in regulating amniotic fluid volume are still not completely understood. The 6 proposed pathways (Brace, 1997) for fluid movement into and out of the amniotic cavity include:

PATHWAY	ML/DAY TO THE FETUS	ML/DAY TO AMNIOTIC FLUID
Fetal swallowing	500-1000	-
Oral secretions	-	25
Secretions from the respiratory tract	170	170
Fetal urination	-	800-1200
Intramembranous flow across the placenta, umbilical cord and fetal	200-500	-
Transmembraneous flow from the amniotic cavity into the uterine circulation	-	10

OLIGOHYDRAMNIOS DEFINITION

Brace and Wolf (1989) reviewed the literature and compiled 705 measurements of amniotic fluid between 8 and 43 weeks' gestation. All of the measurements were either by a dye-dilution technique or by direct measurement at hysterotomy. They reported that amniotic fluid volume increased progressively until 33 weeks' gestation and then plateaued. The mean amniotic fluid volume between 22 and 39 weeks' gestation was 777 ml with the 95% confidence interval ranged from 302 ml to 1997 ml. This mathematical modeling of amniotic fluid volume provides a statistical definition of oligohydramnios. Whether this definition is clinically relevant with respect to fetal/neonatal outcome has not been determined.

SONOGRAPHIC ASSESSMENT

The sonographic assessment of amniotic fluid volume is semiquantitative. Historically, amniotic fluid volume was simply evaluated visually and graded as decreased, normal, or increased (Figure 1).

The appearance of fetal crowding and an obvious lack of amniotic fluid were used to define oligohydramnios. Goldstein and Filly (1988) have reported good intra-observer and inter-observer agreement between subjective assessment and the single largest pocket determination of amniotic fluid volume. One disadvantage of the subjective assessment of amniotic fluid volume is an inability to compare results from serial examinations as the fetal or maternal condition changes.

Manning and Platt (1980) measured the single deepest pocket of amniotic fluid free of fetal extremities and umbilical cord to assess amniotic fluid volume (Figure 2).

Fig 1. Visually normal amniotic fluid volume at 18 weeks' gestation. Click for larger image.

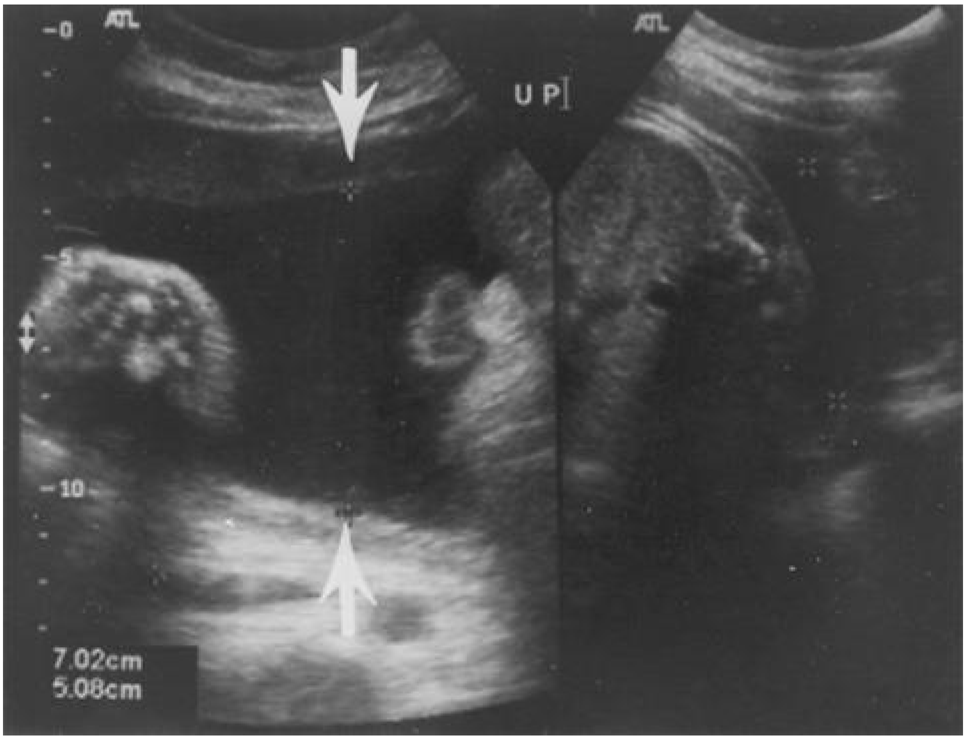

Fig 2. 30 week gestation. A single deepest pocket of amniotic fluid (7 cm), indicating a normal amniotic fluid volume. Click for larger image.

Oligohydramnios was defined as the absence of a single pocket of amniotic fluid with a depth < 1.0 cm (Figure 3). This definition was found to be too restrictive. Manning redefined normal amniotic fluid volume as one pocket of amniotic fluid that measures at least 2.0 cm in two perpendicular planes (Manning 1995)(Figure 4).

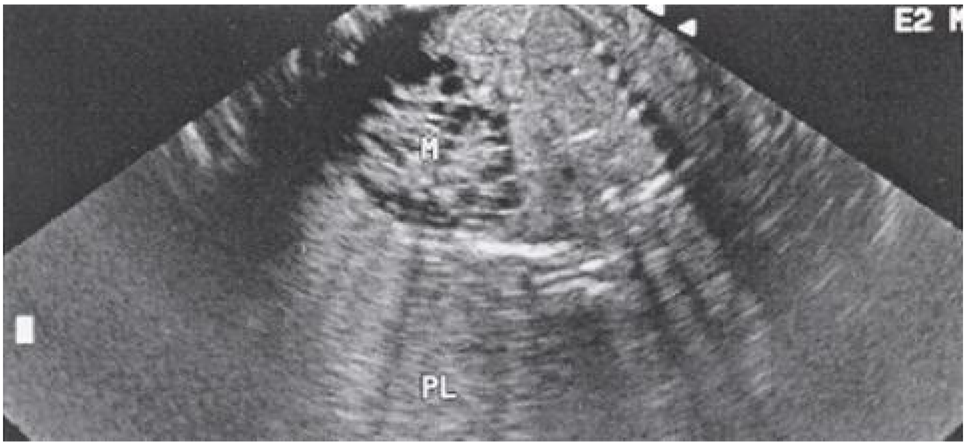

Fig 3. Subjective assessment of amniotic fluid volume. 20 week fetus with a unilateral multicystic kidney (m) and congenital absence of the other kidney, resulting in anhydramnios (PL = placenta). Click for larger image.

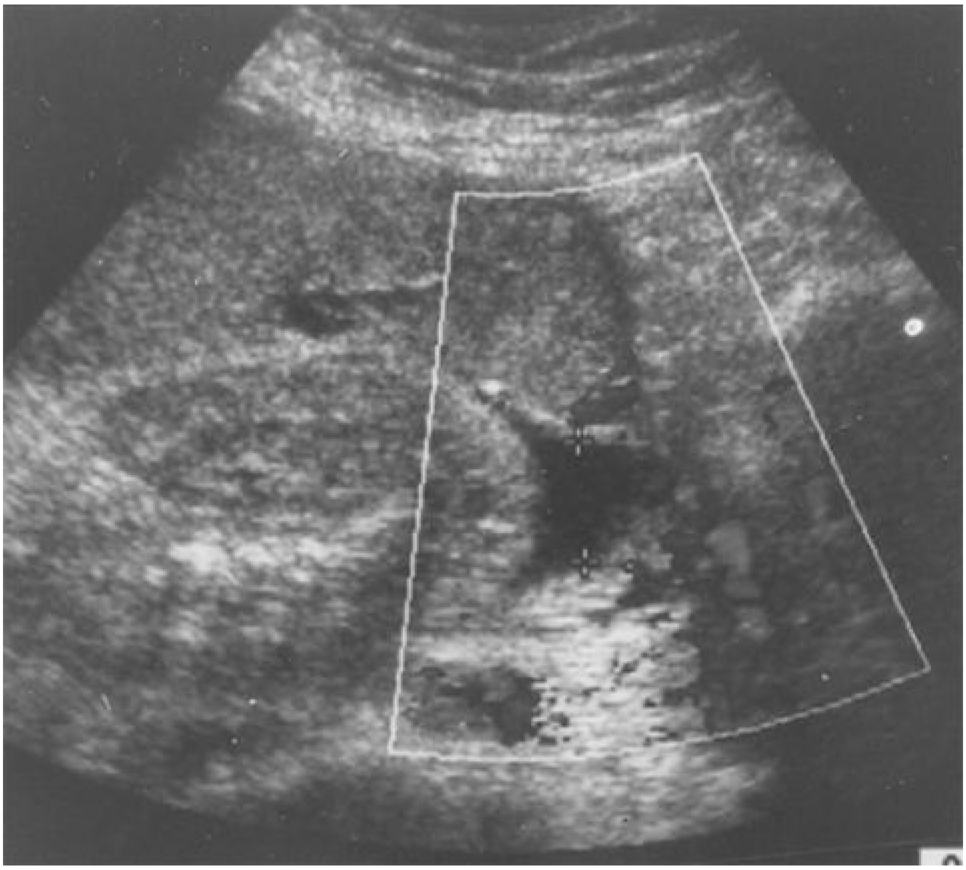

Fig 4. 1.8 cm pocket of amniotic fluid indicating oligohydramnios. The color box confirms that umbilical cord is not present in the pockets of amniotic fluid. Click for larger image.

The amniotic fluid index (AFI) was proposed as a way to more fully assess the amount of amniotic fluid throughout the uterine cavity (Phelan, 1987a). This method summed the maximum vertical pocket of amniotic fluid in each quadrant of the uterus. Oligohydramnios was defined as an amniotic fluid index < 5.0 cm (Phelan, 1987a & b) (Figure 5A and B and Figure 6A and B)

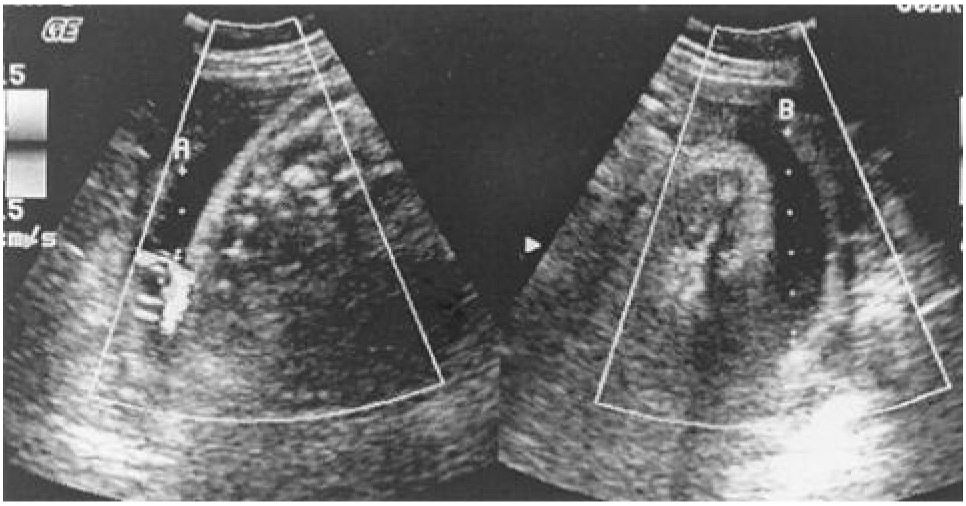

Fig 5a. Upper quadrant. A normal amniotic fluid index of 19.6. The color box is used to ensure an absence of intervening umbilical cord. Click for larger image.

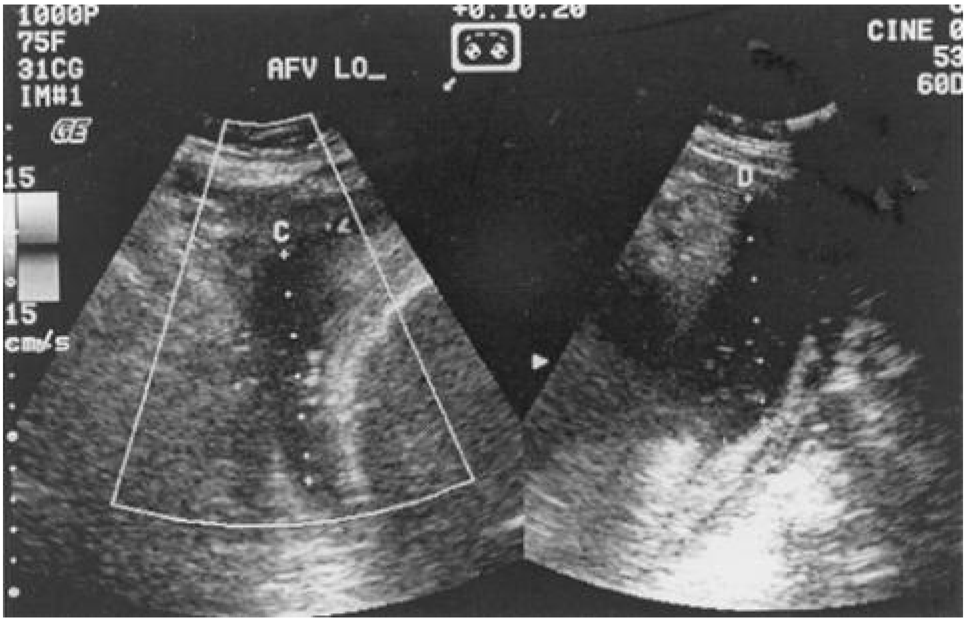

Fig 5b. Lower quadrant. Click for larger image.

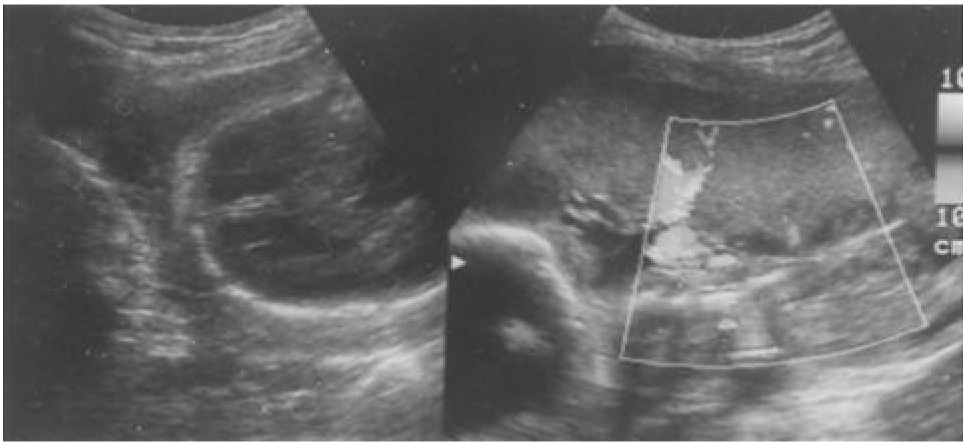

Fig 6a. An amniotic fluid index of 4.2 cm, indicating oligohydramnios. a) there is an absence of amniotic fluid in the upper quadrants. The color box to the right of the image indicates the presence of umbilical cord. Click for larger image.

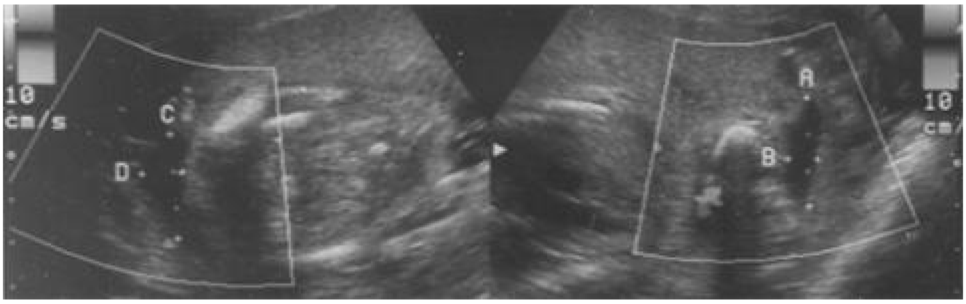

Fig 6b. There is a 2.5 cm (C) and 1.8 cm (A) pocket of fluid in the lower uterine quadrants. Click for larger image.

Moore and Cayle (1990) obtained AFI's in 791 normal pregnancies. They defined oligohydramnios as an AFI below the 5th percentile for gestational age. This value varied between 7.9 cm at 16 weeks and 6.3 cm at 40 weeks' gestation. Although an AFI of <5cm would include <1% of term gestations, this AFI definition of oligohydramnios is the one that is most commonly quoted. When evaluating low AFI's, the intra-observer variation is higher. It is, therefore, recommended that 3 values be obtained and averaged. Color Doppler is also helpful in evaluating a decreased amount of amniotic fluid (Bianco, 1999).

The 2 cm x 2 cm pocket definition (Magann, 1999a) and an AFI <5 cm (Horsager, 1994) were compared to the actual amniotic fluid volume as measured by a dye-dilution technique. The single 2 cm pocket had a sensitivity of 9.5% and an AFI <5.0 cm had a sensitivity of 18% for the detection of oligohydramnios.

It is not surprising that with different sonographic definitions, the prevalence of oligohydramnios varies from study to study. Magann, (2000) found that 8% of their study population had an amniotic fluid index <5cm. Only 1% of the same population had a single pocket of amniotic fluid <2cm. While an AFI <5cm may be more sensitive in the detection of oligohydramnios, it also has a higher false positive rate.

ETIOLOGY OF OLIGOHYDRAMNIOS

Oligohydramnios is commonly caused by one of the following:

1. intrauterine growth restriction
2. post-term pregnancies
3. preterm rupture of the membranes
4. fetal anomalies and/or aneuploidy
5. iatrogenic

INTRAUTERINE GROWTH RESTRICTION (IUGR)

Historically, the association between IUGR and oligohydramnios has been attributed to decreased fetal urine production. However, recent animal studies have not found that hypoxemia significantly effects urine formation. It has been proposed that a reversal of intramembranous flow (i.e., into the fetal circulation from the amniotic cavity) is responsible for oligohydramnios (Cock, 1997).

There is a direct relationship between decreased amniotic fluid volume and the prevalence of IUGR. When a single pocket of amniotic fluid is >2cm, between 1 and 2 cm and <1 cm, the prevalence of IUGR is 5%, 20% and 37%, respectively (Chamberlain, 1984).

POST-TERM PREGNANCIES

There is an acknowledged relationship between prolonged pregnancies (> 42 weeks) and an increase in perinatal morbidity and mortality (Chamberlain, 1984; Eden, 1987). Over 40 years ago a relationship was noted between the diminishing placental function of post-maturity and oligohydramnios (Elliot, 1961). As a result, the ultrasonic assessment of amniotic fluid volume has been used extensively in the antepartum testing of post-date pregnancies. Shime (1984) reported a 79% false positive rate and a 0.4% false negative rate using the biophysical profile score to assess the post-date pregnancy. A majority of post-date fetuses, therefore, are neither dysmature nor hypoxic and will have a benign perinatal course. To date, we have been unable to distinguish between normal and compromised post-term pregnancies with oligohydramnios.

PRETERM RUPTURE OF THE MEMBRANES

Preterm rupture of the membranes is defined as rupture of the membranes prior to 38 weeks' gestation. Cox (1988) has reported that spontaneous rupture of the membranes between 24 and 34 weeks' gestation occurs in 1.7% of pregnancies, but accounts for 20% of perinatal deaths. Second trimester oligohydramnios has a particularly poor prognosis with an approximate survival rate of 10% (Shipp, 1996).

FETAL ANOMALIES AND/OR ANEUPLOIDY

The majority of congenital anomalies associated with oligohydramnios involves the urinary tract (Figure 3). The prevalence of congenital anomalies and aneuploidy varies between 4.5%-37% and 0-4.4%, respectively. (Hill, 1983; Nicolaides 1991; Shipp, 1996).

Bilateral renal agenesis (Figure 7A and B), multicystic dysplastic kidneys and posterior urethral valves

(Figure 8A and B) can all result in oligohydramnios. The reduction in amniotic fluid volume makes an assessment of fetal anatomy more difficult. Transvaginal sonography (Hill, 1991) and color or power Doppler (DeVore, 1995) can be used to confirm the presence or absence of the kidneys and renal arteries, respectively. Early symmetric intrauterine growth restriction and oligohydramnios should suggest a possible karyotypic abnormality (Figure 9) (Nicolaides, 1986).

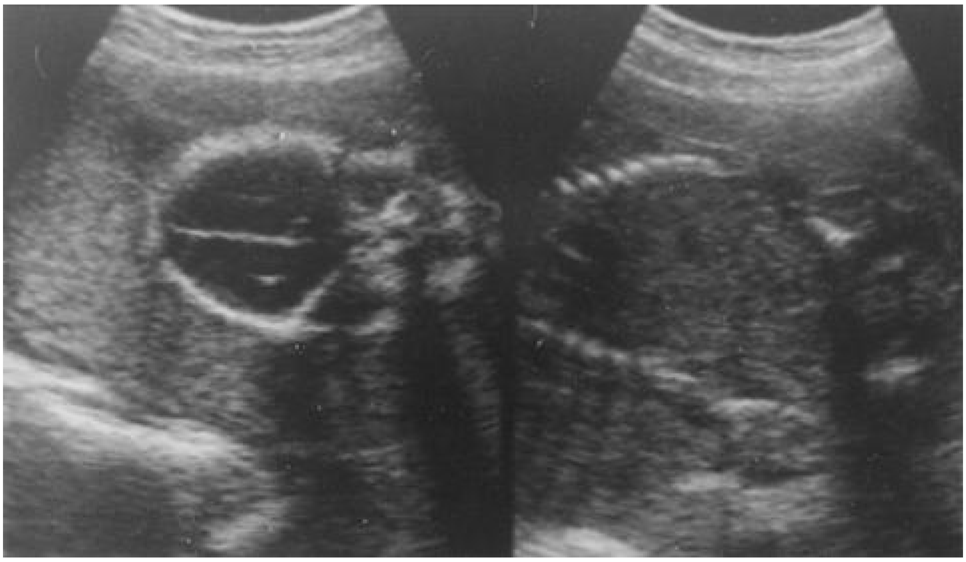

Fig 7a. Oligohydramnios due to bilateral renal agenesis. a. Transabdominal ultrasound at 20 weeks' gestation. The fetus is in breech presentation. There is an absence of amniotic fluid. Click for larger image.

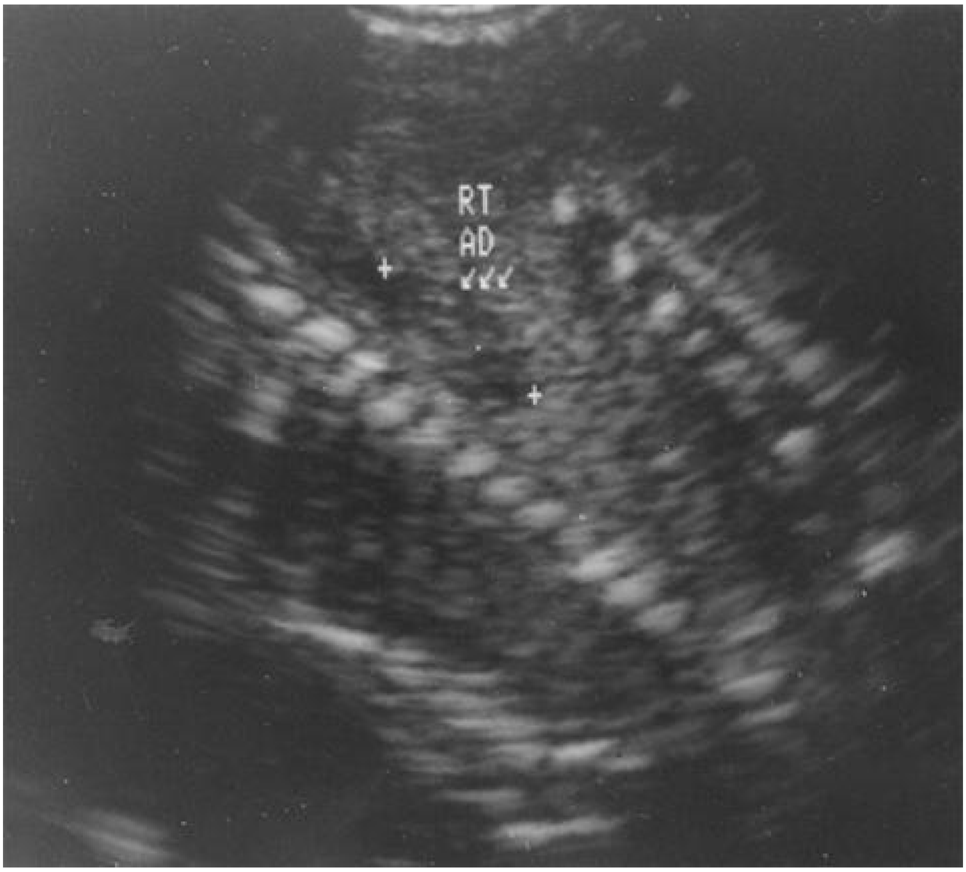

Fig 7b. Transvaginal ultrasound of the right adrenal (RT AD). The right kidney is not present within the renal fossa. Click for larger image.

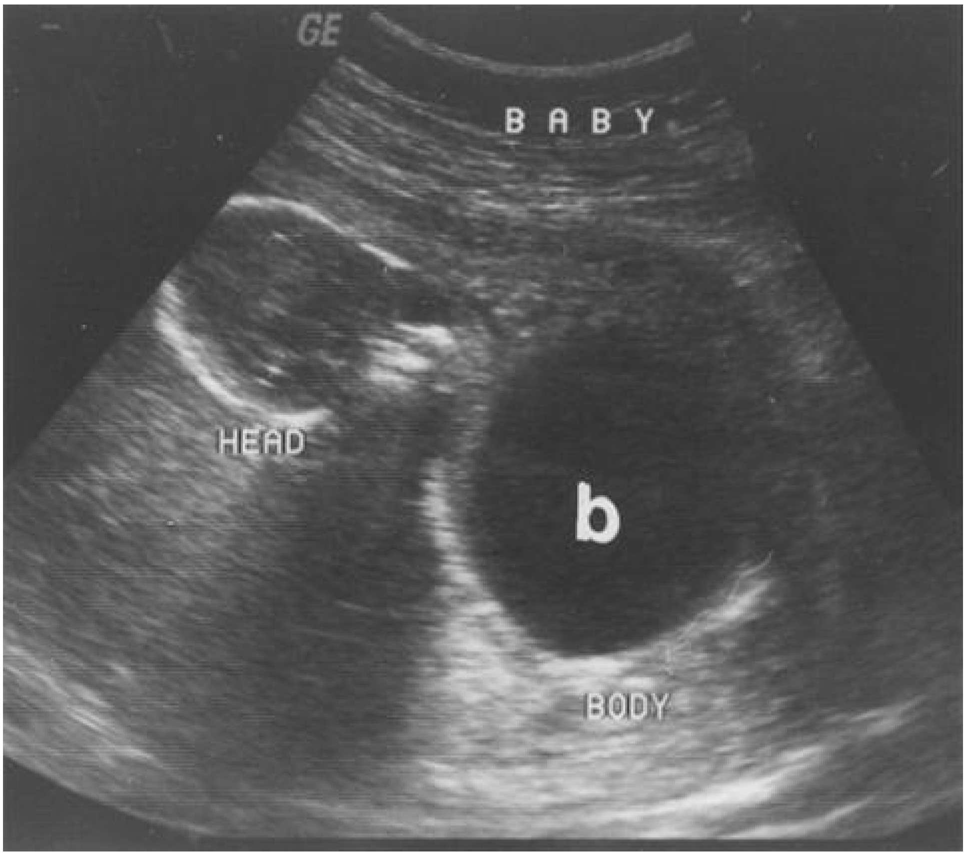

Fig 8a. 15 week fetus with posterior urethral valves. a. The fetus is in breech presentation. The bladder (b) is massively distended. Click for larger image.

Fig 8b. Enlarged "key-hole" bladder associated with posterior urethral valves. Click for larger image.

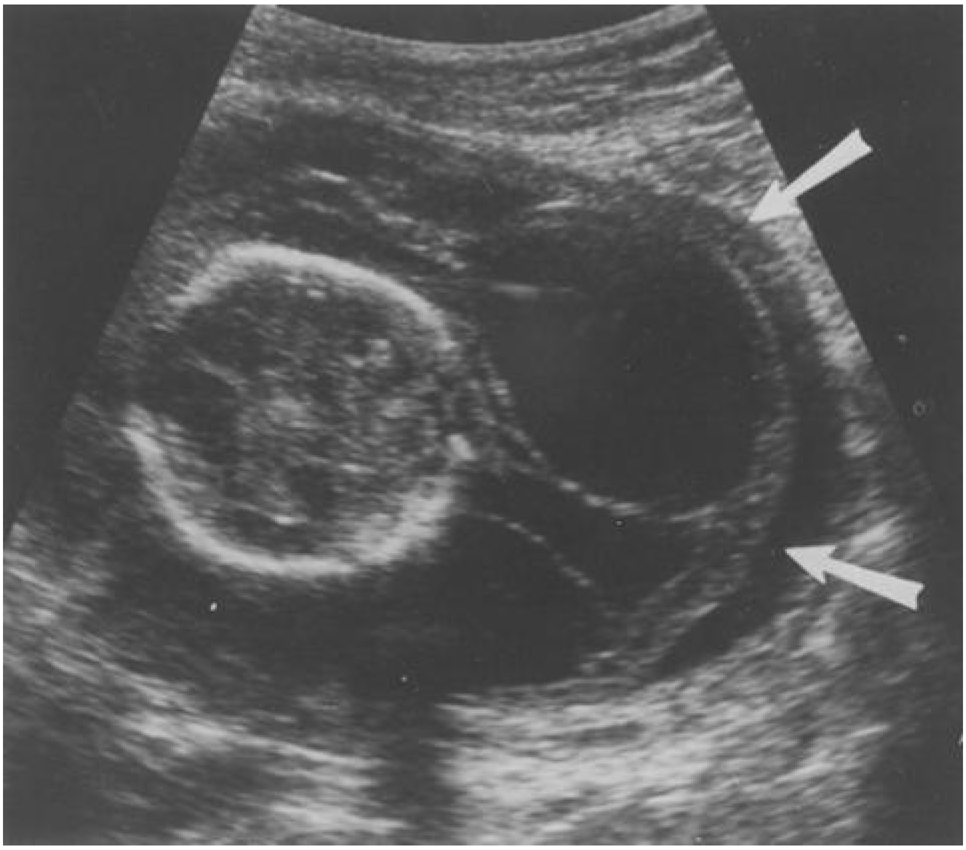

Fig 9. 19 week fetus with Turner's syndrome, cystic hygroma (arrows) and oligohydramnios. Click for larger image.

IATROGENIC

Non-steroidal prostaglandin synthetase inhibitors inhibit renal vascular flow and thereby reduce amniotic fluid volume (Hill, 1996). When the prostaglandin synthesis inhibitor is discontinued, the amniotic fluid volume gradually reaccumulates.

Oligohydramnios is an acknowledged complication of first trimester chorionic villus sampling and second trimester genetic amniocentesis. If the amniotic fluid volume subsequently returns to normal, the neonatal outcome is generally good (Shipp, 1996; Bronshtein, 1991).

SEQUELAE OF CHRONIC OLIGOHYDRAMNIOS

The reported complications associated with oligohydramnios include:

1. fetal demise
2. pulmonary hypoplasia
3. facial deformities
4. skeletal deformities

As previously discussed, the reduction in amniotic fluid pressure with oligohydramnios results in a net egress of fluid from the lungs and subsequent pulmonary hypoplasia (Nicolini, 1989). Facial and skeletal deformities are due to the restriction of fetal movement with oligohydramnios.

NEONATAL PROGNOSIS

Pregnancies with oligohydramnios have a bimodal distribution. Patients with second trimester oligohydramnios have a higher prevalence of congenital anomalies (50.7% vs. 22.1%) and a lower survival rate (10.2% vs. 85.3%) then those women with oligohydramnios in the third trimester (Shipp, 1996).

Perinatal mortality is directly related, not only to gestational age at presentation, but also to the severity of oligohydramnios. The perinatal mortality associated with a single pocket of amniotic fluid measure, <1.0 cm, 1.0cm-2.0cm, and from 2.0cm-8.0cm is 109.7, 37.7, and 1.97/1,000, respectively (Chamberlain, 1984).

MANAGEMENT

The obstetrical management of oligohydramnios is determined by its etiology. A careful assessment of both the mother and fetus is necessary. With treatment of the primary disease process (i.e., placement of a bladder-amniotic shunt for posterior urethral valves, discontinuing a prostaglandin synthatase inhibitor, etc.), the amniotic fluid volume will return to normal.

Intrauterine growth restriction is managed with appropriate antepartum testing and determining the optimal time for delivery. Antibiotics and corticosteroids may be utilized with preterm premature rupture of the membranes at a gestational age of <32 weeks (Vermillion, 2000). It should be remembered that isolated third trimester oligohydramnios is not necessarily associated with poor perinatal outcome (Magann, 1999b).

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