

# **Aberrant liquor volume**

## **Amniotic fluid**

By 12 weeks gestation, the amnion comes into contact with the inner surface of the chorion and the two membranes become adherent, but never intimately fuse. Neither the amnion nor the chorion contains vessels or nerves, but both do contain a significant quantity of phospholipids as well as enzymes involved in phospholipid hydrolysis. Choriodecidual function is thought to play a pivotal role in the initiation of labour through the production of prostaglandins E<sub>2</sub> and F<sub>2a</sub>.

The amniotic fluid is initially secreted by the amnion, but by the 10th week it is mainly a transudate of the fetal serum via the skin and umbilical cord.

From 16 weeks gestation, the fetal skin becomes impermeable to water and the net increase in amniotic fluid is through a small imbalance between the contributions of fluid through the kidneys and lung fluids and removal by fetal swallowing.

# Daily amniotic fluid dynamics in human fetus

Inflow	Outflow
Urine flow (1000-1200 ml)	Swallowing (500-1000 ml)
Lung fluid (340 ml)	Intramembranous (200-500 ml)
Pharyngeal fluid (10 ml)	Transmembranous (10 ml)

Amniotic fluid volume increases progressively:

(10 weeks: 30 mL; 20 weeks : 300 ml;  
30 weeks: 600 mL; 38 weeks: 1000 ml),

but from term there is a rapid fall in volume (40 weeks: 800 mL; 42 weeks: 350 mL). The reason for the late reduction has not been explained.

# The function of the amniotic fluid is to:

- protect the fetus from mechanical injury;
- permit movement of the fetus while preventing limb contracture.
- prevent adhesions between fetus and amnion.
- permit fetal lung development in which there is two-way movement of fluid into the fetal bronchioles; absence of amniotic fluid in the second trimester is associated with pulmonary hypoplasia.

# Assessment of amniotic fluid:

Because the clinical assessment of the amniotic fluid is unreliable, the objective assessment of the abnormalities of amniotic fluid volume (AFV) depends on ultrasound.

A reduction in amniotic fluid volume is referred to as 'oligohydramnios' and an excess is referred to as 'polyhydramnios'.

Definitions of oligohydramnios & polyhydramnios are based on sonographic criteria.

Two ultrasound measurement approaches gives an indication of amniotic fluid volume.

These are :

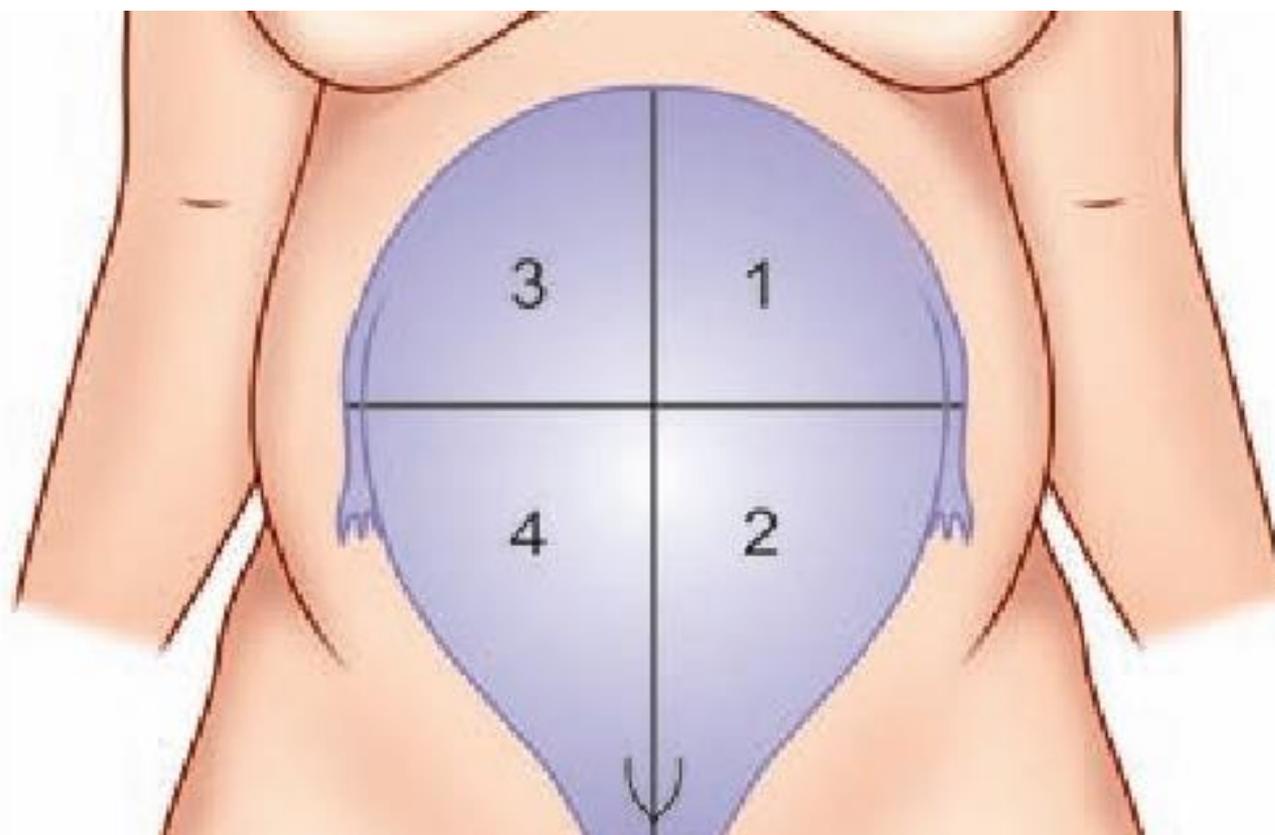
**Maximum vertical pool** and  
**Amniotic fluid index.**

The maximum vertical pool is measured after a general survey of the uterine contents.

Measurements of less than 2 cm suggest oligohydramnios, and measurement of greater than 8 cm suggest polyhydramnios.

The Amniotic Fluid Index (AFI) is measured by dividing the uterus into four 'ultrasound' quadrants. A vertical measurement is taken of the deepest cord free pool in each quadrant and the results summated.

The AFI alters throughout gestation, but in the third trimester it should be between 10 and 25 cm; values below 10 cm indicate a reduced volume and those below 5 cm indicate oligohydramnios, while values above 25 cm indicate polyhydramnios.





**oligohydramnios**

**Oligohydramnios (reduction in the amount of liquor) is defined as deepest vertical pocket of less than 2cm.**

**Incidence it complicates about 3.9 per cent of pregnancies.**

**Oligohydramnios is associated with poor perinatal outcome with perinatal mortality rate reaching 90%. (related to the underlying cause).**

# Causes of oligohydramnios:

## Too little production

## Diagnosed by

Renal agenesis

Ultrasound: no renal tissue, no bladder

Multicystic kidneys

Ultrasound: enlarged kidneys with multiple cysts, no visible bladder

Urinary tract abnormality/  
obstruction

Ultrasound: kidneys may be present, but urinary tract dilatation

FGR and placental insufficiency

Clinical: reduced SFH, reduced fetal movements, possibly abnormal CTG

Ultrasound: FGR, abnormal fetal Dopplers

Maternal drugs (e.g NSAIDs)

Withholding NSAIDs may allow amniotic fluid to re-accumulate

## Post-dates pregnancy

Leakage

## Diagnosed by

PPROM

Speculum examination: pool of amniotic fluid on posterior blade

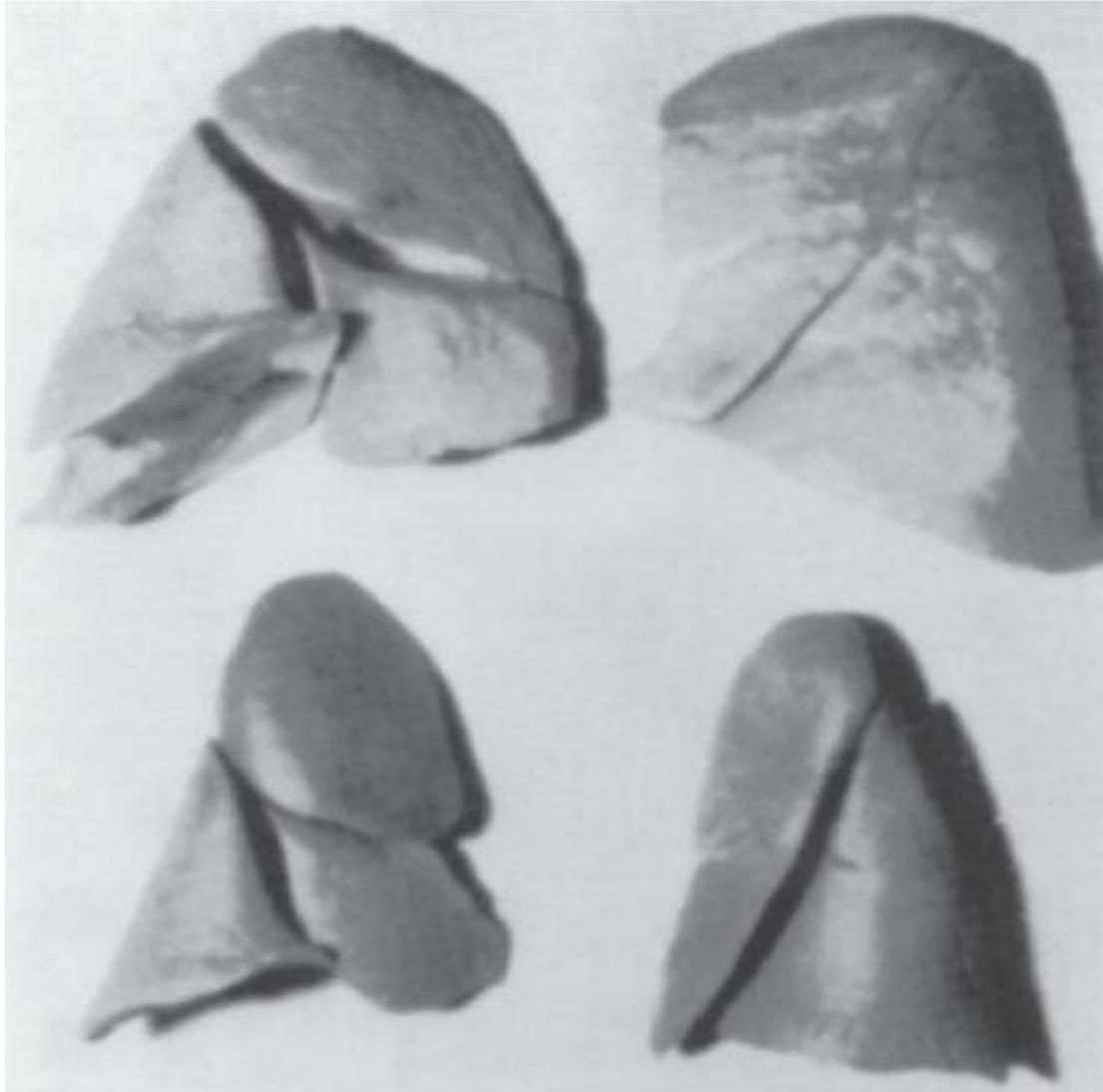
# Fetal risk:

The main fetal risks are:

- \* Pulmonary hypoplasia.
- \* Chorioamnionitis subsequent to PPROM .
- \* Prematurity.

Perinatal mortality are mainly due to prematurity and congenital malformations. (the sequelae of oligohydramnios flattened facies, postural abnormalities and pulmonary hypoplasia are referred to as Potter syndrome)

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# Management:

The main management objective is to confirm aetiology of oligohydramnios and to define the prognosis.

PPROM is often apparent from the history and examination.

The earlier it occurs the higher the incidence of pulmonary hypoplasia.

Clinical examination may reveal the presence of chronic hypertension or pre-eclampsia and/or symphysis-

Fundal height that is small for gestation.

Subsequent US biometry may reveal intrauterine growth restriction or structural anomalies.

A combination of biophysical studies and Doppler waveform analysis (of both uterine and umbilical circulations) may be used to assess fetal well-being and as indirect measure of placental function.

Exclusion of congenital anomalies is mandatory.

( renal agenesis, urinary tract outflow obstruction)

# Polyhydramnios

Definition and incidence:

**Polyhydramnios** is usually defined as DVP > 8 cm or AFI above 95<sup>th</sup> centile for gestational age.

**Incidence:** 1-3.5% of all pregnancies.

# Causes of polyhydramnios:

## **Idiopathic (the most common)**

### **Maternal**

- Diabetes

### **Placental**

- Chorioangioma
- Arterio-venous fistula

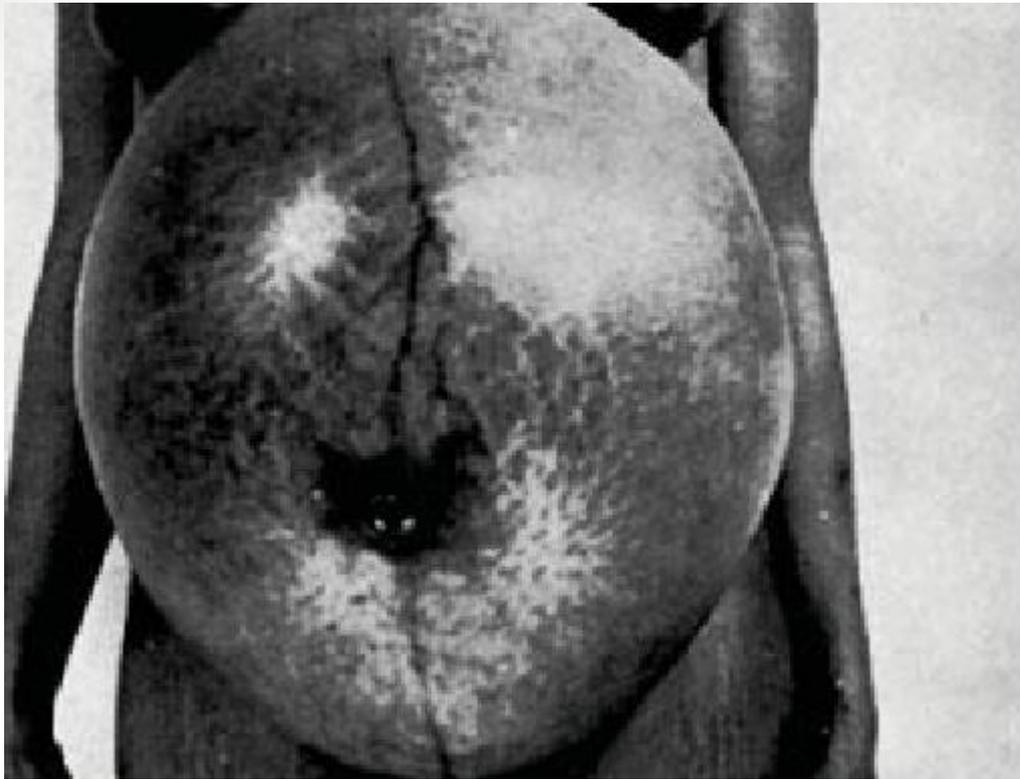
# Fetal

- Multiple gestation (in monochorionic twins, it may be twin-to-twin transfusion syndrome)
- Oesophageal atresia/tracheo-oesophageal fistula
- Duodenal atresia
- Neuromuscular fetal condition (preventing swallowing)
- Anencephaly.
- \* Hydropes fetalis (due to Rh isoimmunization).

# Fetal and maternal risk:

**Maternal complications** are related to overdistension of the uterus and include preterm labour, abdominal discomfort and uterine atony postpartum.

Unstable lie, placental abruption and increased incidence of caesarean section are also results of severe polyhydramnios.





**FIGURE 11-3** Sonogram of severe hydramnios at 35 weeks in a pregnancy complicated by fetal aqueductal stenosis. This pocket of amniotic fluid measures more than 15 cm, and the amniotic fluid index measured nearly 50 cm.

Perinatal mortality in case of polyhydramnios is increased by 10-30% and is secondary to presence of congenital abnormalities and preterm labour.

## Management:

It is important to evaluate each case thoroughly in a systemic manner. A careful history , with attention to maternal symptoms, diseases such as diabetes mellitus or red cell alloimmunization or recent viral infection is important.

High resolution ultrasound should be performed to assess the degree of polyhydramnios, identify multiple pregnancy if present, and target assessment of fetal anomalies.

Karyotyping should be offered, particularly in association with structural anomalies.

If viral infection is suspected, appropriate fetal and maternal samples should be obtained.

If the excess liquor is associated with fetal anaemia, the fetus is almost always hydropic. Assessing the fetal middle cerebral artery peak systolic velocimetry helps to identify fetal anaemia with 100% accuracy .



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13.8cm / 11Hz

Tis 0.7

23.09.2010

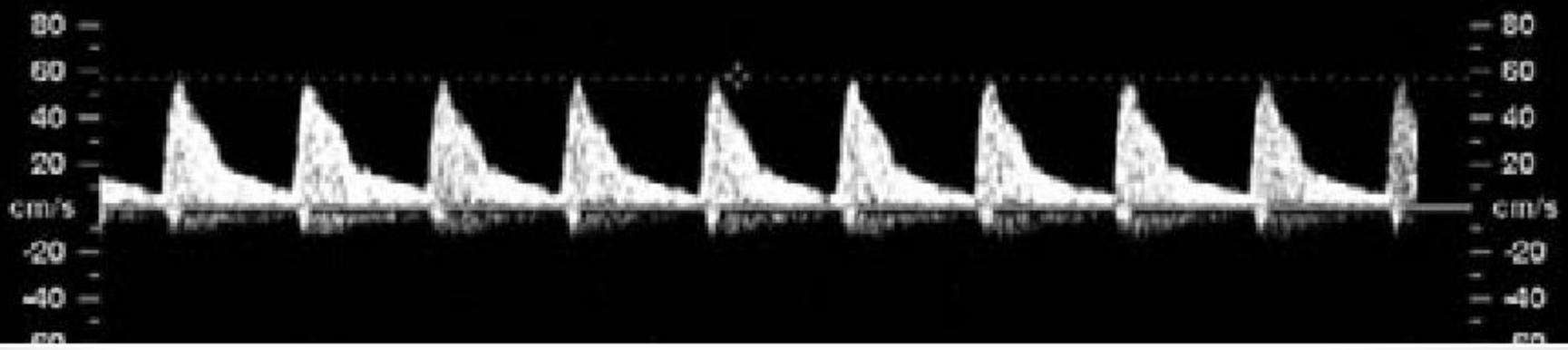
16:02:48

Pwr 100  18cm/s  
 Gn -1  
 WMF 120 Hz  
 SV Angle -16  
 size 3.0mm  
 Frq mid  
 PRF 6.6kHz



Rt MCA-P3 56.89cm/s  
 Har-mid  
 Pwr 97 %  
 Gn 0  
 G7 / M5  
 P3 / E3  
 SRI II 3  
 REC

Pwr 100 %  
 Gn -3.4  
 Frq mid  
 Qual norm  
 WMF low1  
 PRF 1.3kHz



Polyhydramnios due to maternal diabetes needs urgent investigation, as it often suggests high maternal blood glucose levels.

In this context, polyhydramnios should correct itself when the mother's glycaemic control is optimized.

A major management aim is to reduce maternal discomfort and prolong the pregnancy.

Treatment is only indicated when there is severe polyhydramnios (DVP > 12 cm; AFI > 40 cm).

# Treatment options include:

Pharmacological management with COX inhibitors (indomethacin and sulindac), these act by decreasing fetal urine output but have a lot of serious fetal side effects including renal failure in neonates and premature closure of fetal ductus arteriosus resulting in perinatal mortality.

There are also reports of necrotizing enterocolitis and intracranial haemorrhage in infants.

Serial amnioreduction (risk of preterm labour and rapid re-accumulation of amniotic fluid).