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Water balance in the fetus and neonate

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A B S T R A C T

Fetal water balance is dependent prenatally on the placental transfer of water from maternal to fetal circulation. Adequate amniotic fluid volume is one indicator of stable fetal status and development. Excessive or less than expected amniotic fluid volume may be a precursor to postnatal morbidity and mortality. Postnatal transition is marked by predictable changes in body water including contraction of extracellular volume and insensible fluid loss, primarily across the skin barrier. The degree to which these occur is determined by gestational and postnatal age. Neonatal complications and clinical conditions associated with either retention or excessive loss of body water can occur. Fluid therapy in the neonatal intensive care unit may be guided using three clinical indicators: change in body weight, serum sodium concentration, and urine output.

1. Fetal water balance

1.1. Amniotic fluid physiology

Amniotic fluid water balance is critical to fetal health. Amniotic fluid supports normal fetal development and allows for unrestricted fetal movement. Inadequate or excessive amniotic fluid volume is associated with fetal and neonatal conditions associated with morbidity and mortality.

During fetal life, water is divided between the fetus, placenta, chorionic and amniotic membranes, and amniotic fluid. Amniotic fluid may be conceptualized as a fetal fluid compartment. It is formed from either a transudate of fetal plasma through non-keratinized skin or from maternal plasma across the uterine decidua or placental surface. At term, the average fetus contains ~3000 mL of water, 350 mL of which is in the vascular compartment [1].

The major sources of amniotic fluid water are fetal urine and fetal lung fluid. Amniotic fluid is absorbed via fetal swallowing and intramembranous flow [2]. During the first trimester, amniotic fluid is isotonic with maternal plasma, but contains minimal protein [3]. As gestation advances, the osmolality and sodium content of amniotic fluid both decrease. This is due to fetal production of dilute urine and isotonic fetal lung fluid secretion. By term the amniotic fluid osmolality is about 85–90% of maternal serum osmolality. In contrast, fetal urinary byproducts in amniotic fluid (urea, creatinine, and uric acid) increase during the second half of gestation to concentrations two to threefold higher than concentrations found in fetal plasma [4].

The amniotic fluid volume at term may vary from 500 to >1200 mL [5] (Fig. 1).

Body water in the fetus may be conceptualized by dividing it into the intracellular and extracellular compartments. Intracellular water is defined as water present within cells. Extracellular water is divided between the blood plasma, the interstitial space, and in relatively smaller amounts bone, dense connective tissue, and epithelial-lined spaces (transcellular fluid) including cerebrospinal fluid, synovial fluid, and pleural fluid.

At 24 weeks of gestation fetal total body water is ~86% of body weight, of which the majority (60%) is in the extracellular compartment. At term, total body water is ~78% of body weight with 44% in the extracellular compartment, 34% in the intracellular compartment, and the remainder (22%) as solid body mass [6].

1.2. Homeostatic mechanisms

Under the current understanding of fetal water flow, water moves from maternal to fetal circulation through the placenta, possibly by formation of an osmotic gradient created by the active transport of sodium. Transplacental water flow to the fetus occurs through aquaporin water channels. The expression of these proteins changes as gestation advances and with pathologic states such as polyhydramnios and fetal acidosis [1].

Homeostatic control of amniotic fluid volume is thought to be

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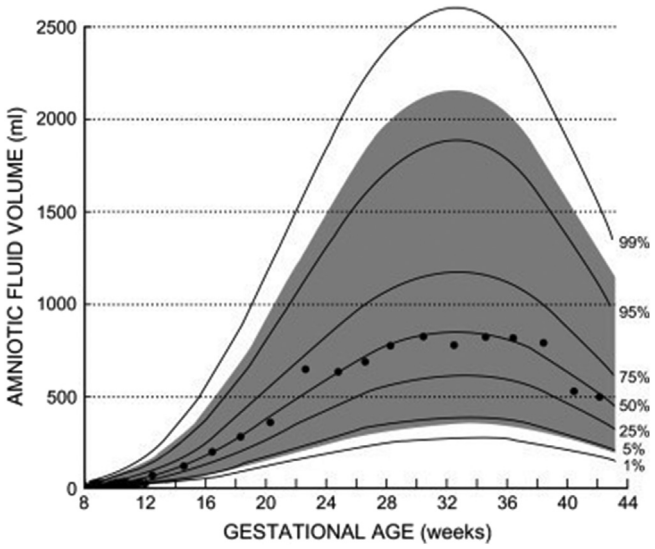

Fig. 1. Black dots refer to the mean amniotic fluid volume at a given gestational age between 8 and 44 weeks. Percentiles along the distribution are shown on the right vertical axis, with amniotic fluid volume in milliliters shown on the left vertical axis. Grey shading indicates 2 standard deviations above and below the mean. Reproduced with permission from Beall et al. [5].

mediated, at least in part, by small (28–30 kDa) membrane proteins known as aquaporins. First discovered by Abre, these proteins allow water molecules to pass across lipid membranes at a faster rate than would be possible by diffusion alone, while excluding most other molecules [7]. Thirteen aquaporins have been described, numbered AQP0-12 [8]. Aquaporins contain four subunits, each with an hourglass-shaped channel lined with charged chemical groups. The charged groups interact with polar water molecules to allow passage through the channel (Fig. 2). It has been shown that five of these aquaporins (Aquaporins 1, 3, 8, 9, and 11) are expressed

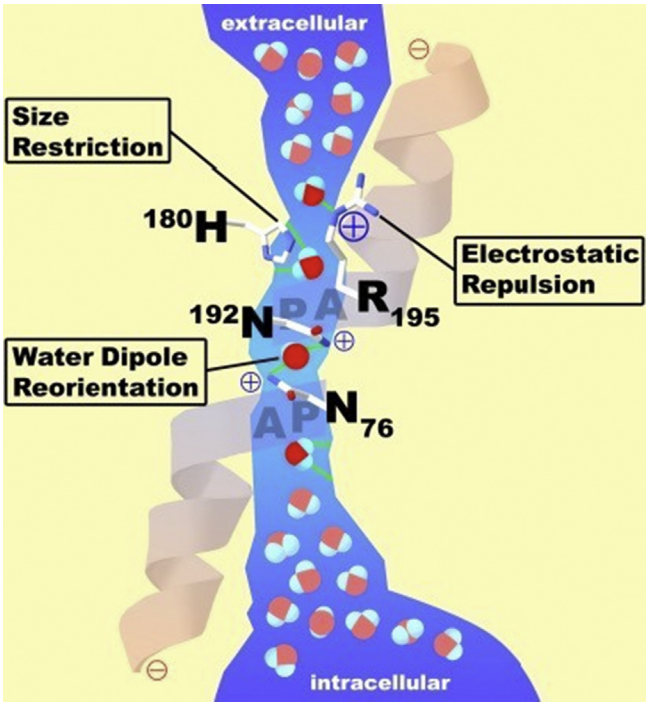

Fig. 2. Aquaporin 1 (AQP1) structure. Reproduced with permission from Abre [8].

in the human fetal–maternal membranes (amnion and chorion) and are differentially expressed throughout gestation [9] (Fig. 3).

Pathologic states are associated with changes in aquaporin expression on the membrane surface. For instance, in the setting of polyhydramnios, Aquaporin 1 expression is markedly increased (>30-fold), suggesting a regulatory function of these proteins [10].

1.3. Clinical relevance

Oligohydramnios is defined as amniotic fluid volume that is less than expected for gestational age. This finding may be idiopathic, a result of premature and/or prolonged rupture of membranes, twin–twin transfusion syndrome (donor) or secondary to decreased fetal urine production or flow. This condition may cause fetal deformation, umbilical cord compression, and death [11].

Polyhydramnios is defined as an excessive volume of amniotic fluid. This condition should be suspected when uterine size is large for gestational age. It may also be idiopathic, a result of twin–twin transfusion syndrome (recipient), maternal diabetes, fetal gastrointestinal tract obstruction, neural tube defect, or genetic conditions such as trisomies. Even a relatively minor increase in daily fetal urine production or decrease in fetal swallowing may result in a marked increase in amniotic fluid volume [12]. This condition carries a higher incidence of adverse perinatal outcomes, even when cases of congenital anomalies are excluded [13].

Maternal dehydration with increased maternal osmolality (excessive water loss from hot weather or diabetes insipidus, for example) may be associated with reversible decreased fetal compartment water and oligohydramnios [1].

Maternal overhydration ('water intoxication') during labor may cause dangerous hyponatremia in both the mother and neonate through transfer of water from hyponatremic maternal blood to the fetus, who also develops hyponatremia. In severe cases this condition may cause maternal and neonatal seizures [14].

Intrauterine growth restriction (IUGR) is associated with a relatively higher proportion of total body water at birth as compared to neonatal body composition resulting from expected fetal growth. This is due to a decrease in body solids in growth-restricted fetuses rather than an excess of water production or retention [15].

2. Neonatal water balance

2.1. Neonatal transition

At birth, neonates must rapidly adapt to the extrauterine environment. Body water content changes gradually from fetal to neonatal life. Both total body water and extracellular fluid compartments decrease proportionately over time. Conversely, intracellular fluid percentage increases at birth and peaks at three months' postnatal age (Fig. 4).

After birth, notable changes in body water and composition take place. Contraction of extracellular water occurs, with a corresponding weight loss of between 7% and 15% of body weight by the end of the first week. The degree of contraction of the extracellular water compartment is inversely proportional to gestational age. Term infants may exhibit a 5–7% weight loss during the first week whereas preterm infants with birth weight <1500 g may exhibit a 10–15% weight loss [6].

2.2. Conditions determining water balance in the neonatal period

Gestational age, postnatal age, IUGR, prenatal steroid exposure, total body surface area, ambient temperature and humidity, type of heat source, and the neonate's activity all affect postnatal water