Physiology of amniotic fluid volume regulation

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INTRODUCTION

Amniotic fluid (AF) is the liquid that surrounds the fetus after the first few weeks of gestation. During much of pregnancy, AF is derived almost entirely from the fetus and has a number of functions that are essential for normal growth and development [1]:

- It helps to protect the fetus from trauma to the maternal abdomen.
- It cushions the umbilical cord from compression between the fetus and uterus.
- It has antibacterial properties that provide some protection from infection.
- It serves as a reservoir of fluid and nutrients for the fetus.
- It provides the necessary fluid, space, and growth factors to permit normal development of the fetal lungs and musculoskeletal and gastrointestinal systems.

Aberrations in amniotic fluid volume (AFV), both low (oligohydramnios) and high (polyhydramnios), are associated with multiple adverse pregnancy outcomes. For example, a study that examined pregnancy outcomes of pregnancies with oligohydramnios (AF index ≤5 cm) compared with those with AF index >5 cm found that oligohydramnios was associated with increased rates of labor induction (42 versus 18 percent), stillbirth (1.4 versus 0.3 percent), nonreassuring fetal heart rate pattern (48 versus 39 percent), admission to the neonatal intensive care nursery (7 versus 2 percent), meconium aspiration syndrome (1.0 versus 0.1 percent), and neonatal death (5.0 versus 0.3 percent) [2]. A similar study that assessed the adverse effects of idiopathic polyhydramnios reported increased rates of

obstetric complications (eg, preterm birth, abnormal fetal presentation, fetal anomalies) and a two- to fivefold increase in perinatal mortality [3].

This topic will review basic physiologic mechanisms responsible for both AFV and AF composition. Detailed discussions of assessment of AFV, oligohydramnios, and polyhydramnios can be found separately.

- (See "Assessment of amniotic fluid volume".)
- (See "Oligohydramnios: Etiology, diagnosis, and management in singleton gestations".)
- (See "Polyhydramnios: Etiology, diagnosis, and management".)

OVERVIEW

Understanding the sources of AF is required for understanding the causes and consequences of AFV disorders and developing effective management strategies for pregnancies with these disorders. Briefly:

- The major inputs to the AF are fetal urine and fetal lung secretions.
- The major physiologic pathway for removal of AF from the amniotic cavity is fetal swallowing, but intramembranous flow (ie, across the amnion and into the fetal vasculature at the placental surface) likely plays a role as well.
- In the absence of ruptured membranes, oligohydramnios in the second half of pregnancy is most often a result of decreased fetal urine flow; possible causes include obstruction of the urinary tract and decreased fetal renal blood flow related to fetal growth restriction.
- Maternal dehydration can lead to a reduction in placental water flow and, in turn, decreased AFV.
- Polyhydramnios may be associated with increased fetal urine flow due to fetal tachyarrhythmia with heart failure, severe anemia, maternal diabetes, and fetal renal abnormalities, among others.
- A reduction in fetal swallowing due to a high gastrointestinal obstruction or fetal neurologic abnormalities may be associated with severe polyhydramnios.

SOURCES AND COMPOSITION OF AMNIOTIC FLUID

Sources of amniotic fluid — There are several sources of AF; the relative contribution from each source changes across gestation.

Embryonic period — The embryo resides within two fluid-filled sacs: an outer exocoelomic cavity and an inner amniotic cavity. These sacs contain large amounts of liquid relative to the size of the embryo. Because both coelomic fluid and AF are present in anembryonic pregnancies, the embryo is probably not the primary fluid source in the embryonic period.

- **Exocoelomic cavity –** Beginning at approximately the 7th week of gestation, coelomic fluid fills the exocoelomic cavity (ie, space between the developing amnion and chorion). It reaches maximum volume around the 10th week, and then decreases until completely disappearing at 12 to 14 weeks of gestation, thus enabling fusion of the amniotic and chorionic membranes. The mechanism for the disappearance of coelomic fluid is unknown, but it is likely that both its liquid and solutes cross the amnion into the amniotic cavity.

  The composition of coelomic fluid is similar to maternal plasma and different from AF, suggesting that maternal plasma may be its source, although a pathway for movement of fluid from maternal plasma to the exocoelomic cavity has not been defined. Endometrial gland secretions may be another source.

- **Amniotic cavity –** The amniotic cavity contains AF. The volume of AF increases prior to the transition from the embryonic to fetal period at 10 weeks of gestation. Early gestational AF is likely derived from three sources:

  - The embryonic surface of the placenta
  - Transport from the maternal compartment across the amnion (transmembranous pathway)
  - Secretions from the surface of the body of the embryo

  The relative contributions of these potential sources of AF are unknown.

Early fetal period — In the early fetal period, fetal urine begins to enter the amniotic cavity, and the fetus begins to swallow AF, although the daily volume flows are quite small at this time [4,5]. The fetal lungs also begin to secrete liquid into the amniotic cavity at this time.

Mid and late fetal periods

Synopsis of production and clearance — Sonography and animal models have helped inform the source and composition of AF during the latter half of gestation.

The sources of AF production are:

- Major – Fetal urine and fetal lung liquid
- Minor – Secretions from the fetal oral and nasal cavities

The sources of AF clearance are:

- Major – Fetal swallowing and the intramembranous pathway
- Minor – Transmembranous pathway

Both the transmembranous and intramembranous pathways permit flow of water and solutes into and out of the amniotic cavity (ie, osmotic flow of water and diffusion of solutes), while the other pathways only allow flow of water and solutes in one direction (ie, bulk flow) [1].

Daily amniotic volume flows in the near term fetus are estimated to be [6]:

- Fetal urine production – 800 to 1200 mL/day
- Fetal lung liquid secretion – 170 mL/day
- Fetal swallowing – 500 to 1000 mL/day
- Intramembranous flow – 200 to 400 mL/day
- Oral-nasal secretions – 25 mL/day
- Transmembranous flow – 10 mL/day

These values are experimentally derived, in many cases using animal models. Specific sources of production and clearance are reviewed below.

Fetal urination — The daily volume of fetal urine excreted is approximately 30 percent of fetal body weight [1]. Hourly flow rates progressively increase from 2 to 5 mL at 22 weeks of gestation to 30 to 50 mL at 40 weeks [7,8]. The reduction in maternal plasma sodium concentration (approximately 5 mEq/L) during pregnancy may increase the fetal urine output and contribute to AF formation by enhancing osmotic flow of water across the placenta [9].

There are major differences in the magnitude of estimates of human fetal urine production [10-12]. Variations may be partially explained by maternal position when the measurement is obtained; maternal rest in the left lateral decubitus position markedly increases fetal urine production [13]. Another factor affecting flow rate is the time before onset of labor; hourly fetal urine production is reduced in the 14 days prior to birth [14]. Technique of measurement also appears to play a role; ultrasound assessments of fetal urine production rates may be best performed with three-dimensional ultrasound [15].

Fetal urinary flow rates decrease with conditions associated with placental insufficiency (eg, preeclampsia, fetal growth restriction) and increase with conditions associated with heart failure (eg, fetal anemia, supraventricular tachycardia, twin-twin transfusion syndrome). Fetal urination (and therefore AFV) is also adversely affected by obstructions in the fetal urinary tract (eg, posterior urethral valve).

Fetal swallowing — Fetal swallowing increases throughout gestation. Although swallowing-type movements can be noted on ultrasound late in the first trimester, these movements do not become well-coordinated until the third trimester [16,17].

Direct and indirect measurements of ovine fetal swallowing suggest that the fetus swallows AF equivalent to 20 to 25 percent of body weight [1] The volumes swallowed are significantly greater than in adults, relative to body weight [18]. Low rates of swallowing compared with urination in early and midpregnancy account for the gradual increase in AFV during this period [18]. By contrast, increased swallowing near term, and especially postterm, may contribute to the fall in AFV at the end of pregnancy.

Fetal swallowing is decreased with a consequent increase in AFV in some fetuses with neurologic abnormalities, such as anencephaly. Fetal swallowing may also be impaired by esophageal or duodenal obstruction.

Fetal lung secretions — The fetal lung secretes 100 times as much fluid as is needed to expand the developing lungs to facilitate their growth. The excess fluid exits the trachea, primarily during episodes of fetal breathing [19]. Approximately 50 percent of this fluid (170 mL/day near term) is swallowed, and the remainder enters the amniotic cavity, which was the basis for fetal lung maturity testing [20,21]. (See "Assessment of fetal lung maturity".)

Lung fluid secretion is reduced during periods of fetal asphyxia and during labor [1]. Residual fluid is absorbed into the pulmonary lymphatics.

There are no disease states in which fetal lung fluid secretion is increased.

Intramembranous flow — The intramembranous pathway refers to water and solute exchange that occurs directly between AF and fetal blood [1,22,23]. This occurs primarily across microscopic fetal vessels present on the fetal surface of the placenta in primates and over the whole membrane surface in species with vascularized membranes. Smaller contributions may occur across the umbilical cord and fetal skin; however, transcutaneous flow ceases with skin keratinization at 22 to 24 weeks of gestation. Aquaporins (cell membrane water channel proteins) in the chorioamniotic membrane and placenta may play a role in intramembranous fluid resorption [24,25].

The normal intramembranous flow rate has been calculated only in fetal sheep [22,26]. Changes in AF osmolality were measured over time in sheep with tracheoesophageal occlusion and continuous urinary drainage [26]. This preparation eliminated all major flows into and out of the amniotic cavity, except for the intramembranous path. Half of the normal osmolality gradient between AF and fetal blood disappeared in eight hours, suggesting that 400 mL of water per day was absorbed via this route.

Factors likely to affect the functioning of the intramembranous pathway, and thus fluid volume, include fluid osmolality and the concentrations of sodium and chloride.

- **Osmolality –** Fetal and maternal blood osmolalities are nearly equivalent across gestation. In contrast, starting in the early fetal period, AF osmolality is slightly lower

than that of fetal blood and decreases further as the pregnancy progresses. AF osmolality averages 260 mOsm/kg at term compared with a blood osmolality of 280 mOsm/kg. Fetal urine osmolality is usually 50 to 60 percent of AF osmolality, although the near-term fetus can alter its urine volume and osmolality in response to fetal hormonal signals such as vasopressin [27]. Lung liquid osmolality is similar to that of fetal plasma and is not altered by hormonal stimuli.

- **Sodium –** The AF sodium concentration is much lower than that of fetal blood. The fetal urinary sodium concentration is low, averaging 20 to 40 percent of AF concentrations. By comparison, fetal lung liquid sodium concentration is only modestly lower than blood sodium concentration and thus is significantly higher than that in the AF.

- **Chloride –** Lung liquid chloride concentration is almost twice the AF chloride concentration because lung secretion is driven by the active secretion of chloride into the future airways [28]. Fetal urinary chloride concentration is very low: 10 to 20 percent of the AF chloride concentration.

Transmembranous flow — The transmembranous pathway refers to water and solute exchange between maternal blood and AF across the decidua and myometrium. In contrast to the intramembranous pathway, which occurs from one fetal compartment to another (amniotic cavity to fetal vasculature), transmembranous flow takes place between maternal and fetal compartments.

Transmembranous water and solute fluxes are immeasurably small, although these exchanges were once considered major contributors to AFV and composition [1].

Fetal oral and nasal secretions — These pathways account for small volumes of fluid exchange. They are difficult to measure and have not been studied [1,23].

Composition of amniotic fluid — The composition of AF reflects a combination of fetal lung liquid and fetal urine, both of which have properties different from fetal plasma. The composition of AF is distinctively different from that of all other fetal and maternal fluids.

AF is 98 to 99 percent water and 1 to 2 percent solids, including proteins, carbohydrates, chemical substances (eg, urea, uric acid, creatinine, electrolytes), lipids and phospholipids, enzymes, hormones, and pigments [29]. Lung liquid contributes phospholipids and surfactant, while urine contributes urea. Exposed peritoneum (eg, omphalocele) and neural tube defects, when present, allow increased passage of alpha-fetoprotein into the AF.

In addition, AF contains a variety of types of fetal cells (eg, skin, respiratory, intestinal, urinary tract, and stem), as well as hair, blood cells, cells shed from the amnion, and sometimes meconium [30].

REGULATION OF AMNIOTIC FLUID VOLUME

Fetal urination, lung liquid secretion, fetal swallowing, and intramembranous absorption make significant contributions to fluid movements into and out of the amniotic cavity in late gestation, as discussed above. The integration of all fluid inflows and outflows determines the ultimate volume of AF. Although many reviews have addressed the topic of AFV regulation, no study has proven whether and how this occurs [1,22,23]. For AFV self-regulation to occur, there must be a feedback mechanism to detect aberrations in AFV and return volume toward normal. These issues will be addressed below by first considering normal AFV turnover and then discussing possible volume regulatory mechanisms.

The AFV does not change significantly from day to day, but the AF itself is completely replaced over 24 hours. In the third trimester 1000 mL of fluid flows into and out of the amniotic cavity daily so that even small changes in one of the paths of fluid migration could rapidly affect AFV [1]. Conversely, regulation of AFV must be precise to maintain normal volumes with such high flow rates. If AFV is self-regulated in the latter half of gestation, then this must occur through adjustment of one or more of the four primary AF inflows and outflows. (See 'Mid and late fetal periods' above.)

Variations in fetal homeostasis affect the volume of fetal urine production, swallowing, and lung liquid secretion. In this respect, the AFV is a passive reflector of the fetal condition. Examples of this are the fetus with decreased renal blood flow, which leads to decreased urine production and oligohydramnios, and the anomalous fetus that cannot swallow due to gut atresia, resulting in polyhydramnios [31,32].

Maternal disorders also affect fetal homeostasis, and, in turn, AFV. Maternal dehydration increases maternal osmolality, favoring transfer of water from the fetus to the mother, which, in turn, promotes transfer of water from the AF to the fetus, presumably via the intramembranous pathway [33]. Likewise, maternal hyperglycemia can lead to fetal hyperglycemia, increased fetal urine output, and polyhydramnios. These types of alterations in AFV are presumably pathologic, and these mechanisms are not likely to be involved in normal fluid homeostasis.

Changes in intramembranous flow are thought to be a key factor in AFV homeostasis, but the mechanism(s) for regulating intramembranous flow is largely unknown [34]. One possibility involves aquaporins (AQPs), which are a family of cell membrane water channels found ubiquitously in tissues. Multiple AQPs have been identified: 1, 3, 8, 9, and 11 have been described in the placenta and fetal membranes of humans, sheep, and mice [35-37].

> In an ovine model, AQP1 increased in the amnion of late-gestation fetal sheep under experimental conditions that increased intramembranous flow, suggesting that AQP1

could be involved in regulating intramembranous flow and therefore AFV [38]. In humans, one report described increased expression of AQP8 in the placenta and membranes of fetuses with polyhydramnios [39]. Another report described increased expression of AQP8 and AQP9 in the amnion but decreased expression in the placenta in patients with idiopathic polyhydramnios [40]. In fetuses with oligohydramnios, decreased expression of AQP1 and AQP3 in the membranes and increased AQP3 in the placenta have been observed [41].

NORMAL AMNIOTIC FLUID VOLUME ACROSS GESTATION

Few studies that have measured AFV across gestation; however, the available data describe a characteristic pattern of AFV change (figure 1).

- In a review of 705 normal pregnancies from 12 studies between 1962 and 1977, AFV was observed to increase progressively from 8 weeks of gestation, peak at 34 weeks (at over 800 mL), and decline thereafter [42]. Early in pregnancy, fetal urine and lung fluid production exceed the fetal volume swallowed, resulting in the gradual increase in AFV. Late in pregnancy, fetal swallowed volume exceeds fetal fluid production, accounting for the decrease in AFV. The largest variability in AFV occurred in the third trimester, probably due to greater variation in fetal urination and swallowing later in pregnancy.

- A subsequent study of 144 pregnancies performed all of the fluid evaluations using the same dye-dilution technique and a single laboratory for the calculation of the AFV [43]. AFV increased well into the third trimester of pregnancy, with the most significant increase after 30 weeks and until term.

- In a review of four studies with 1095 total patients evaluated by direct evaluation of the AFV, the maximum AFV was at 30 to 31 weeks [44].

The two earlier studies [42,43] found that AFV at 36 weeks of gestation was approximately 800 mL, but they differed as to whether the volume remained stable [43] or decreased [42] at term. Potential selection bias, methodologic differences, and limited numbers of patients studied prevent a firm conclusion regarding the normal change in AFV between 30 and 40 weeks. Nevertheless, beyond 40 weeks it is likely that mean AFV decreases, with a marked reduction in postterm patients.

SUMMARY AND RECOMMENDATIONS

- **Background –** Amniotic fluid (AF) is crucial to normal fetal growth and development. Abnormalities in amniotic fluid volume (AFV; eg, oligohydramnios and polyhydramnios),

even when not accompanied by other fetal abnormalities, are associated with a poorer perinatal outcome. (See 'Introduction' above.)

- **AFV across gestation** – AFV increases in human gestation until 34 to 36 weeks of gestation after which the volume decreases, resulting in reduced AFV postterm (figure 1). (See 'Normal amniotic fluid volume across gestation' above.)

- **Production and resorption**

  - In the second half of pregnancy, AF is produced by the fetus as urine and lung liquid and resorbed by fetal swallowing (see 'Mid and late fetal periods' above). An additional pathway for AF resorption is the intramembranous pathway, which is fluid movement across the amnion into fetal vessels on the surface of the placenta. The rate of fluid resorption through the intramembranous pathway adapts to maintain normal AFV in experimental models; mechanisms for this change are speculative. (See 'Intramembranous flow' above.)

  - A variety of fetal and maternal conditions can cause variations in fetal homeostasis and affect the AFV by altering fetal urine production, swallowing, and (possibly) lung liquid secretion. Thus, AFV is a passive reflector of the fetal condition. (See 'Regulation of amniotic fluid volume' above.)

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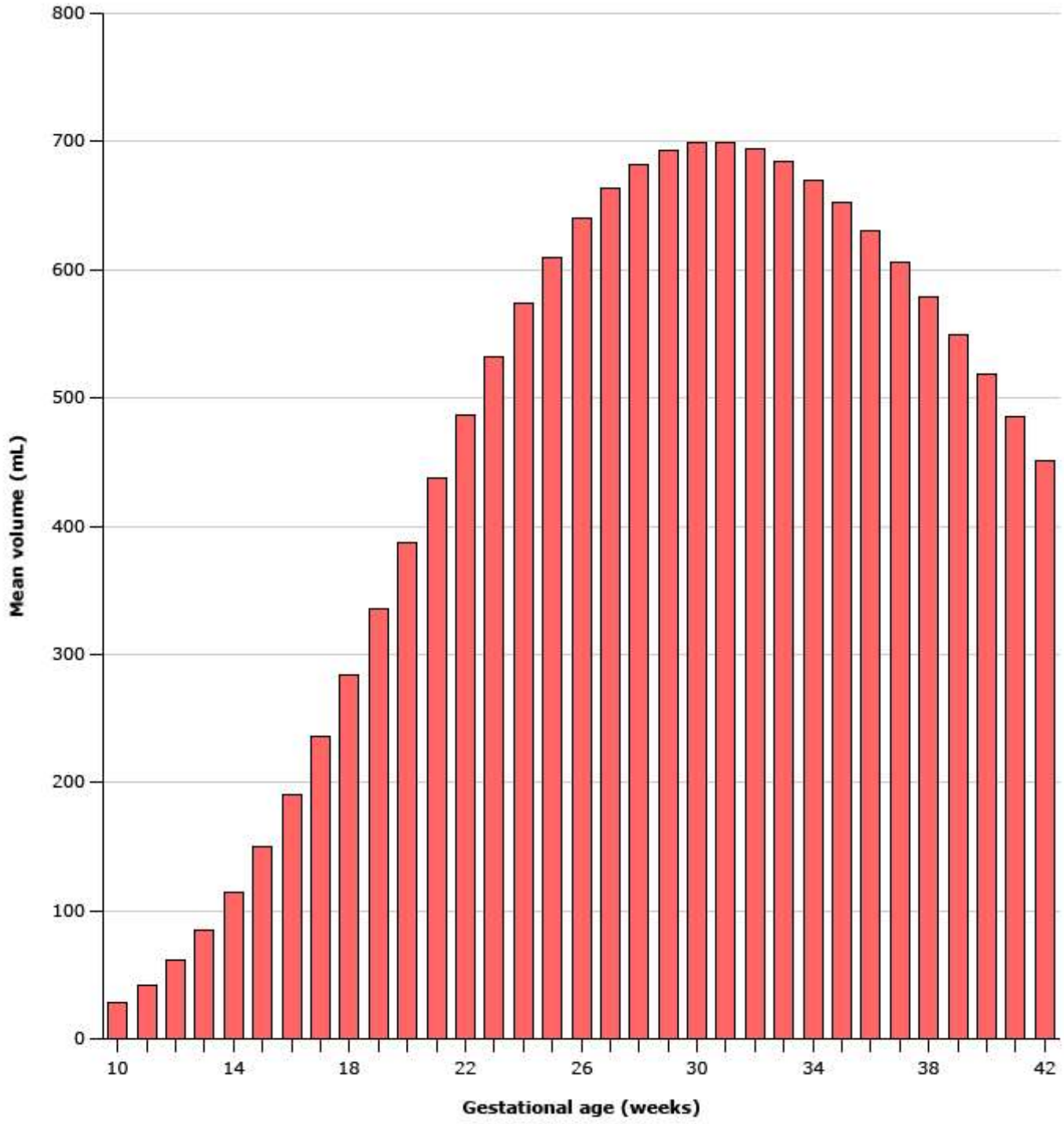
GRAPHICS

Mean amniotic fluid volume across normal pregnancy



These values represent the 50th percentile. There is considerable variability around the mean. The 5th, 50th, and 95th percentiles at 30 weeks of gestation are approximately 260, 700, and 1900 mL, respectively.

Data from: Ounpraseuth ST, Magann EF, Spencer HJ, Rabie NZ, Sandlin AT Normal amniotic fluid volume across gestation: Comparison of statistical approaches in 1190 normal amniotic fluid volumes. J Obstet Gynaecol Res; 2017: 43:1122.

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Contributor Disclosures

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