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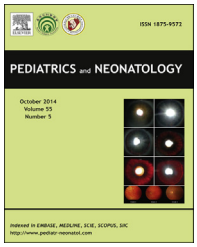

INVITED REVIEW ARTICLE

Gastric Residual Evaluation in Preterm Neonates: A Useful Monitoring Technique or a Hindrance?

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It is routine practice in most neonatal intensive care units to measure the volume and color of gastric residuals (GRs) prior to enteral bolus feedings in preterm very low birth weight infants. However, there is paucity of evidence supporting the routine use of this technique. Moreover, owing to the lack of uniform standards in the management of GRs, wide variations exist as to what constitutes significant GR volume, the importance of GR color and frequency of GR evaluation, and the color or volume standards that dictate discarding or returning GRs. The presence of large GR volumes or green-colored residuals prior to feeding often prompts subsequent feedings to be withheld or reduced because of possible necrotizing enterocolitis resulting in delays in enteral feeding. Cessation or delays in enteral feeding may result in extrauterine growth restriction, a known risk factor for poor neurodevelopmental and growth outcomes in preterm very low birth weight infants. Although some neonatal intensive care units are abandoning the practice of routine GR evaluation, little evidence exists to support the discontinuation or continuation of this practice. This review summarizes the current state of GR evaluation and underlines the need for a scientific basis to either support or refute the routine evaluation of GRs.
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1. Introduction

Gastric residuals (GRs) are often evaluated in preterm infants who are being fed via an orogastric (OG) or nasogastric (NG) tube as a putative indicator of feeding intolerance (FI) or as an early symptom of necrotizing enterocolitis (NEC).[1,2] Although GR measurement prior to feeding is routinely used to guide subsequent feeding advancement,[3,4] little scientific evidence exists to justify this practice.[5] Standards for the management of GRs are lacking, and reports in the literature indicate a wide variation in practice regarding the evaluation of feeding tube position, frequency of GR evaluation, standards that dictate the discarding or returning of GRs, and even what constitutes "significant" GR volume and/or quality.[2,6] This lack of uniform standards[7] often leads to a discontinuation or delays in the advancement of enteral feedings, which in turn may lead to an unnecessary prolongation of intravenous nutrition, increased risk of late onset sepsis, and extrauterine growth restriction.[8] Because researchers now question the utility of routine GR evaluation,[9] this study assessed this potentially unnecessary procedure and reviewed current literature regarding routine GR evaluations to underscore the need for additional research.

2. Gastric emptying: correlates and influence on GR

Evaluation of GRs is used in the neonatal intensive care to measure the volume of milk remaining in the stomach at a variable time after a feeding, and as an indicator of gastric emptying (GE).[10] Compared to term infants, preterm infants have slower GE owing to intrinsic immaturities of the gastrointestinal (GI) tract,[11] including suck-swallowing coordination, immature lower esophageal tone and function, low percentage of gastric electrical slow wave, and slower intestinal transit.[12] Furthermore, intestinal motor patterns during fasting and feeding are immature in preterm infants. Motor patterns are characterized by short episodes of quiescence alternating with irregular contractions without clear migrating motor complexes.[13] During fasting, the cluster amplitude and mean duration of the duodenal motor activity are lower in preterm than in term infants, whereas cluster frequency is higher in preterm infants.[14] These physiologic characteristics are intrinsic factors responsible for delayed GE and increased GRs in preterm infants.

Many extrinsic factors such as hormonal input, drug administration, and nutritional management can also influence GE by accelerating GI development and increasing GE, whereas other factors can delay GE and lead to a larger GR volume. For example, antenatal steroid therapy stimulates fetal gastrin secretion, thereby increasing neonatal gastrin level after birth, which in turn strengthens antral contractions against the pylorus, and relaxes the pyloric sphincter, thereby stimulating GE.[15] It also induces the release of intestinal mucosal enzymes and promotes gut development.[16] However, formula milk has been shown to empty half as fast as expressed breast milk, which also has important implications for preterm infants with FI due to delayed GE.[17]

Similarly, other drugs can also impact GE by regulating GI function. Mydriatics, or drugs routinely used for retinopathy of prematurity screening in preterm infants, can cause delayed GE by inhibiting duodenal motor activity.[18] Theophylline can delay GE by its action on cyclic adenosine monophosphate, calcium influx, or potassium-induced membrane depolarization.[19] Gastroprokinetic agents such as domperidone significantly increase GE and may prove to be a useful agent for infants with FI.[20] However, its safety still requires investigation because of a possible QT prolongation in infants >32 weeks in gestation.[21] Another prokinetic agent, erythromycin, may also improve GE and feeding tolerance,[22] but there is still insufficient evidence to recommend its routine use in preterm infants at risk of FI.[23] As a result, it is currently recommended that erythromycin be used cautiously and selectively in preterm infants with moderately severe GI dysmotility.[24] Lastly, another prokinetic, cisapride, is currently not used in the United States owing to reports of an associated long QT syndrome that predisposes infant to arrhythmias.[25]

Studies suggest that certain supplements such as probiotics may increase GE, improve feeding tolerance, and promote gut maturation.[26] Preterm newborns receiving *Lactobacillus reuteri* showed a significant decrease in regurgitation and mean daily crying time, and a larger number of stools compared with those given placebo. The GE rate was significantly increased and the fasting antral area was significantly reduced in both the newborns receiving *L. reuteri* and breast-fed newborns compared to placebo. There is currently no conclusive evidence to recommend routine probiotic supplementation in preterm infants.[27]

The timing of initiation, type of enteral feeding, and mode of administration may also influence GE. Early enteral nutrition hastens the maturation of motor function, as demonstrated by enhanced duodenal motor activity,[28] whereas the administration of minimal enteral feedings (feedings <24 mL/kg/day provided for intestinal maturation and protection rather than nutrition) has been shown to induce the appearance of mature migrating motor activity and promote GE.[29]

Decreased osmolality combined with an increased feeding volume has also been shown to increase GE.[30] Compared to formula, human milk has been shown to result in a more rapid GE in premature infants.[31] However, it is unclear whether the use of human milk fortification influences GE. A study by Ewer and Yu[32] on the effect of human milk fortifier on GE in preterm infants reported that human milk fortifier may slow GE owing to an increased osmolality and a change in milk composition. However, Gathwala et al[33] found no change in feeding tolerance when human milk was fortified.

The mode of enteral feeding administration may also influence GE. Compared to bolus feedings, feedings provided by continuous infusion enhance duodenal motor responses and hasten GE.[34] However, a recent Cochrane review found insufficient evidence to support the use of continuous enteral versus bolus feedings.[35]

Several diseases can also influence GE in preterm infants. Infants with severe hypoxemia often have significantly decreased GI blood flow and tissue oxygenation, which may lead to decreased GE and result increased gastric residual

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