

IF, WHEN AND HOW TO TREAT GASTRO-OESOPHAGEAL REFLUX — THE NEONATOLOGIST'S DILEMMA

In the introduction to their comprehensive review,¹ Rode *et al.* reference several statements, *inter alia* (i) despite the oesophagus being structurally and functionally intact from an early age (33 weeks' gestation), synchronous peristaltic activity is present in less than 60% of newborns, and approximately 40% of the peristaltic waves are incomplete or retrograde; (ii) particularly controversial is the role of the medical treatment of persistent occult gastro-oesophageal reflux; (iii) persistence with ineffective long-term medical therapy may unnecessarily place the infant at risk; and (iv) the prevalence of pathological reflux among low-birth-weight infants is 3 - 10%, with symptoms of irritability, apnoea, bradycardia, vomiting and deterioration of bronchopulmonary disease.

From this the reader could easily formulate an opinion along the following lines: neonates (and, more likely, premature neonates) are predisposed to reflux; there might be adverse consequences of not intervening surgically in the neonate or infant in whom motility does not normalise; and it is relatively easy to identify candidates for such intervention on the basis of symptomatology. However, neonatologists and paediatricians dealing with premature and full-term infants would have a very different reaction to these statements, recognising that they are perhaps opening the door to unnecessarily aggressive treatment, both medical and surgical.

Recent reviews on the subject indicate that virtually all infants have some degree of reflux in the newborn period, while approximately 50% of healthy infants still have



symptomatic reflux at the age of 2 months.^{2,3} However, only a small proportion require consultation with a medical practitioner, and by 1 year of age the symptoms have resolved in the vast majority. This occurs with or without therapy, making it difficult for those dealing with neonates and infants to identify the candidates who justify the statement made by Rode *et al.*: 'if therapy is started early, 90% will have resolved by 10 - 12 months of age'.

In the case of premature infants, while the clinical signs mentioned by Rode *et al.* (apnoea, irritability, bradycardia, vomiting and deterioration of bronchopulmonary disease) are observed in infants with reflux, they are also extremely common in infants without reflux. The important issue is once again how to identify those patients in whom the symptoms are actually due to the reflux. Most commonly, apnoea and bradycardia will be the clinical signs that manifest in the premature infant during the first few weeks of life, and the usual therapeutic response will be to ensure that there is no hypoxaemia, low-grade nosocomial infection, or metabolic abnormality. In the absence of any of the latter, a respiratory stimulant such as theophylline will be considered. Persistent symptoms, particularly if temporally associated with regurgitation or vomiting, will alert the attending practitioner to the possibility of gastro-oesophageal reflux requiring more specific interventions, ranging from confirmation of the problem by means of simple barium studies, to management which might involve positioning of the infant, drug therapy, and occasionally surgery.

As pointed out by Rode *et al.*, the prone sleeping position has in recent years been associated with an increased risk of sudden infant death syndrome (SIDS).⁴ Prone positioning of premature infants while still in hospital, where they can be carefully monitored, remains common practice due to the beneficial effects on gastric emptying and respiratory function.⁵ However, such infants should be changed to the supine sleeping position before hospital discharge. Medical practitioners should be very circumspect about recommending prone positioning of any infant, with or without the reversed Trendelenberg position, during the first 6 - 9 months of age due to the SIDS risk, and it should only be considered in those infants with severe symptomatic reflux.

While Rode *et al.* are critical of the ESPGAN guidelines,⁶ these are nevertheless of value in that there is a clear progression from 'commonsense' interventions such as feed-thickening, through drug therapy to definitive surgery. However, what the guidelines do not do, and what Rode *et al.* have done to some extent, is highlight the risks of the various therapeutic options. For example, in the premature infant, the hazards of a usually benign antacid have been pointed out in terms of the risk of sodium overload resulting from immature renal function. Their comments on the risks of exposing infants to drugs which have become commonplace in paediatric practice need to be emphasised. Bethanecol commonly causes

cramping and diarrhoea,⁷ metoclopramide may cause extrapyramidal reactions,⁸ and cisapride is now contraindicated in South Africa during the first 3 months of the life in infants born at a gestational age of less than 36 weeks due to the risks of cardiac arrhythmias.⁹

Yes, in dealing with reflux in the infant and young child one must resort to aggressive therapy and to surgery when confronted by severe disease, and especially when the infant's life is threatened. Those requiring medical and surgical treatment are usually older infants and young children who commonly have neurological handicap and/or severe congenital abnormalities,² but in the usual management of this common disorder of neonates and infants, the oft-quoted watchwords must always be remembered: '*Primum non nocere*'.

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