Table Data on stool frequency, weight, and water content in normal infants at different ages

	1st week	8-28 days	1-12 months	13-24 months
No. of infants	16	11	11	17
No. of stools	278	139	88	136
Stool frequency				
Mean interval between stools (hours) $\pm$ 1 SD	5.2 $\pm$ 1.9	9.9 $\pm$ 6.5	13.2 $\pm$ 9.2	14.9 $\pm$ 8.3
Range (hours)	0.5-22	0.7-38.5	0.5-53	2-120
No. of stools per 24 h $\pm$ 1 SD	4 $\pm$ 1.8	2.2 $\pm$ 1.6	1.8 $\pm$ 1.2	1.7 $\pm$ 0.6
Range	1-12	0-6	0-5	0-3
Weight of individual stools (g)				
Mean	4.3	11.0	17	35
Range	0.5-48	0.3-40	2-98	4-180
Stool water content (%)				
Mean $\pm$ 1 SD	72.8 $\pm$ 5.0	73.3 $\pm$ 2.7	75.0 $\pm$ 3.0	73.8 $\pm$ 3.2
Range	65-84	72-77	72-80	66-81

641 stools were examined. As expected there was a wide range of individual variation in stool weight and frequency, but the general trend of an increase in weight and decrease in frequency with age is apparent. However, even in the second year it was common for the children to pass more than one stool a day, although some children went for 2-3 days without defecation in the absence of clinical 'constipation'. Stool water content hardly varied from the first week to the second year and had a remarkably small SD. There was no correlation between stool frequency and stool weight.

Comment

The only published information on defecation patterns

in infancy is by Colón and Jacob (1977) who investigated the subject by using questionnaires. The parents in their study reported that more than half the children passed one stool daily after 18 months. These findings seem on the optimistic side for British children who on average continue to pass more than one stool per 24 hours until 2 years of age. Colón and Jacob (1977) obtained no data on stool weights.

The surprising finding in this investigation was the constancy of stool water content, apparently finely controlled to maintain an even stool consistency throughout the age range. Since stool water is easy to measure and remains so constant in health, it could be an unnecessarily neglected marker of gastrointestinal disease and deserves future study.

We intend that the range of variation in stool weight and frequency given in the Table should give some long-needed guidance for child health workers as to what constitutes 'normality'. The study also confirms the effectiveness of napkin liners.

We acknowledge the invaluable help of Mrs M. Lewis in the data collection.

References

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Infantile hypertrophic pyloric stenosis—unusual familial incidence

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SUMMARY A man, who had been treated for infantile hypertrophic pyloric stenosis (PS), was found to have 3 sons with the same condition. A genealogical table of his family showed that both his parents had relatives with PS. So far as it was known, none of his wife's relatives had the condition. It is suggested that the man's parents carried genes predisposing to PS and transmitted these to him in such quantity that he and all his children acquired the condition.

The aetiology of infantile hypertrophic pyloric stenosis (PS) is largely unknown. It has long been

recognised that there is an increased preponderance of the condition in the relatives of patients. This familial predisposition is shown very clearly in the family to be described.

A patient (IV₄ in the Figure) had an operation at this hospital some years ago for PS. On further investigation it was found that both his brothers (IV₃ and IV₆) and his father (III₆) had been treated for the same condition. The main features of their case histories are summarised in the Table.

A genealogical table stretching over four generations was assembled for this family (Figure). The patient's mother had no known relatives with PS, but both his paternal grandfather and grandmother (II₃

Figure Genealogical table of family with infantile hypertrophic pyloric stenosis. Roman numerals indicate generation. Arabic numbers indicate number in generation. Figures in brackets indicate the number of unaffected individuals.

Table Main features of the case histories of Case III₆ and his 3 sons IV₃, IV₄, and IV₅

Case	Gestation at birth (weeks)	Start of projectile vomiting	Clinical findings	Treatment	Outcome
III ₆	40	3rd week	Case notes lost	Spasmolytics and frequent small feeds	Projectile vomiting ceased after few weeks. Uneventful recovery
IV ₃	40	2nd day	Tumour palpated. Hypertrophy on x-ray	Pyloromyotomy at 4 days	Reoperated next day because of gastric retention. Diaphragm in pyloric region excised. Uneventful recovery
IV ₄	38	3rd week	Tumour palpated. Visible peristalsis. X-ray normal	Pyloromyotomy at 3 weeks	Uneventful recovery
IV ₅	40	3rd day	First x-ray normal. Hypertrophy on x-ray after 10 days	Pyloromyotomy at 2 weeks	Uneventful recovery

and II₄) had. Cases III₂ and IV₈ had both had pyloromyotomies performed, and III₁ had died on his way to hospital when he was 7 weeks, after suffering projectile vomiting and progressive weight loss from age 2 weeks.

There was no history of marriage between relatives.

Discussion

Although PS is found in infants it is not congenital in the sense that it is present at birth. The symptoms usually present between ages 3 and 6 weeks, and only rarely during the first week. All reported series show a pronounced preponderance of male to female patients, and it has long been noted that there is an increased incidence of PS in the relatives of patients who have the condition. Carter and Evans (1969) studied 813 patients treated by Ramstedt's pyloromyotomy between 1920 and 1949; 20% of the sons and 7% of the daughters of their female patients had developed PS, but only 5% of the sons and 2.5% of the daughters of the male patients had developed the condition. They estimated the incidence in the general population as being 0.5% for liveborn boys and 0.1% for liveborn girls. They concluded that the hereditary background to PS is polygenic.

It would appear that there are a number of genes in different locations which may predispose for PS, and that it is necessary for a certain number of these

genes to code for the condition for it to develop. The female phenotype seems to be protected to some degree so that a greater genetic load is required for girls to acquire the disease. This greater genetic load explains why the children of female patients are more likely to develop PS rather than those of male patients.

It is interesting to note that Cases IV₃ and IV₅, who were born at term, developed the symptoms at an exceptionally early age. There was little time for any postnatal environmental factors to influence the development of the condition in them. Their brother, Case IV₄, was born 2 weeks preterm and PS was diagnosed at age 3 weeks. All 3 brothers, therefore, developed the condition at about the same time after conception.

It is not certain from whom Cases III₁, III₂, and IV₈ acquired their predisposition for PS. However, finding the condition in these patients increases the likelihood of Cases II₃ and II₄ carrying genes predisposing to PS, although not to such a degree that they developed the condition. These genes were inherited by their son (Case III₆) in such quantity, that not only did he develop PS, but he transferred the condition to all his 3 children.

Reference

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