but found that the phenomenon was still present nine to 12 months after the end of treatment.

The optimal treatment regimen has yet to be established, but this may now be possible as synthetic growth hormone becomes more widely available.

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Diagnosis of rotavirus gastroenteritis by smell

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SUMMARY Clinical features cannot differentiate rotavirus gastroenteritis from other types of diarrhoea. Sixty eight stool specimens were examined by nurses on an infant gastroenteritis ward. Of these, 69% were correctly classified by smell alone. The results are significant (p=0·009) and support the suggestion that rotavirus stools have a characteristic smell.

The appearance and smell of stools have always interested physicians,[1] and the smell of the stool has provided an important clue in establishing the original link between enteropathogenic *Escherichia coli* and neonatal gastroenteritis.[2] The present study was undertaken because it was noticed that the nurses on a paediatric infectious disease ward usually diagnosed rotavirus gastroenteritis correctly before the results of culture were available. They considered the appearance and smell of the stools to be of diagnostic value. Our aim was to test the hypotheses that rotavirus gastroenteritis could be diagnosed by either the smell of the stool alone, or a combination of smell and appearance.

Patients and methods

Overnight stools were collected from 23 babies with diarrhoea, aged 0–18 months (mean 4·8 months), and coded by night staff. Table 1 shows the causes of the diarrhoea. The stools were randomised and examined by day staff at 9.00 am the next morning.

Each stool was examined in two ways. The first way by smell alone, the specimen pot being wrapped in a paper towel, and the nurse keeping her eyes closed. She was asked to classify it as rotavirus or not rotavirus; 'don't know' was not allowed. For the second examination the paper towel was removed and she was asked to inspect the stool and given the opportunity to change her opinion. Seven nurses made 68 examinations of 33 stool specimens over three months.

The ages of the children, the feeds given, and the ages of the stools were comparable.

Table 1 *Diagnoses in 23 babies studied*

Diagnosis	*No of babies*
Positive for rotavirus	10
Rotavirus only	8
Rotavirus and adenovirus	1
Rotavirus and enteropathogenic *E. coli*	1
Not positive for rotavirus	13
Adenovirus	2
Campylobacter	1
Respiratory infections	4
Cystic fibrosis	2
No organism isolated	4

Table 2 *Correlation of diagnosis by smell of stool and by culture*

Diagnosis from culture	*Nurse's diagnosis by smell of stool*		*Total*
	No positive for rotavirus (%)	*No negative for rotavirus (%)*	
Positive for rotavirus	10 (38)	16 (62)	26
Negative for rotavirus	5 (12)	37 (88)	42
Total	15 (22)	53 (78)	68

Results

Table 2 shows the results of examination of stools by smell alone. Sixty nine per cent of stools were classified correctly. The sensitivity was 38% (χ^2 using Fisher's exact test; p=0·01). When the variation between nurses was taken into account the significance was unchanged (Cochrane's test; p=0·008). The information about stool appearance did not materially affect the results.

Discussion

Experienced nurses classified most of the stools by smell alone; it seems likely, therefore, that stools infected by rotavirus have a distinctive smell. As stools infected with enteropathogenic *E coli* also have a characteristic smell, it may be that other organisms causing diarrhoea may also be identified by smell.

None of the clinical features of rotavirus diarrhoea distinguishes it clearly from diarrhoea caused by other organisms, though studies of inpatients have suggested that fever, frequent vomiting, isotonic dehydration, and lack of white cells in the stools are more commonly associated with diarrhoea caused by rotavirus.[3 4] The smell of the stool could, therefore, be a useful aid in diagnosis.

Only 38% of the stools containing rotavirus were correctly identified, so the sensitivity of smell alone as a diagnostic test is low. These results might be improved if the nurses were told the results of culture at the time they made their diagnosis, and allowing them to change their minds in cases of uncertainty.

It is not clear what causes the distinctive smell; rotavirus damages the mucosa of the small intestine directly, not by producing a toxin. Its effect on stool composition, therefore, is probably indirect disruption of normal gut function, or a change in the normal gut flora.

Damage to the brush border reduces disaccharide concentration both in man and animals, and endopeptidases are probably also affected in this way.[5] Disaccharides and peptides may, therefore, be present in the stools in increased concentrations. Derivatives, such as lactic acid, are volatile and pungent, and could affect the smell of the stools.

Any change in the composition of colonic fluid will unbalance the normal flora which carry out many metabolic processes. Such change could affect the relative concentrations of pungent products, including short chain fatty acids and indoles, thereby altering the smell.

Disturbances in the fatty acid and bile acid concentrations in stools have been shown in patients with chronic diarrhoea by chromatography, and this might also be a useful technique for diagnosing acute gastroenteritis in children.[6]

We conclude that the smell of the stools of infants with acute gastroenteritis may help to diagnose rotavirus and possibly, other infections.

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