A survey of specimens submitted to five English laboratories² showed that the most important contribution of the results to patient management was that they helped to exclude particular possible diagnoses. Urine examinations are a substantial proportion of the work of any microbiology laboratory, and it is clearly unacceptable that the reports on up to 20% of such specimens may be misleading. With the help and encouragement of our clinical colleagues we are reviewing both the rationale and the cost effectiveness of microbiological investigation generally. While the major areas of concern are "routine" screening tests, duplicated specimens on the same patient, and out-of-hours requests, substantial savings in time, money, and temper can clearly be made by avoiding self-inflicted clinical confusion like that described here. Reports on other specimens too may be misleading but faults are not generally as easily demonstrable as in urine.

¹ Sandler G. Cost of unnecessary tests. *Br Med J* 1979;ii:21-4.

² Spencely M, Parker KJ, Dewar RAD, Millar DL. The clinical value of microbiological laboratory investigations. *Journal of Infection* 1979; 1:23-36.

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Sjögren's syndrome treated with bromhexine: a reassessment

Frost-Larsen *et al*¹ reported that bromhexine was effective in the treatment of Sjögren's syndrome. At a dose of 48 mg daily lacrimal secretion significantly increased when measured by the Schirmer test. Salivary flow did not improve, but the authors conceded that their methods for assessing salivary function were crude and of doubtful value. Nevertheless, they concluded that bromhexine was the drug of first choice in treating Sjögren's syndrome.

Patients, methods, and results

Using objective tests we investigated whether bromhexine significantly altered lacrimal and salivary function in 14 patients with Sjögren's syndrome,² including five with sicca syndrome, and in 11 healthy volunteers. Each patient or volunteer was randomly allocated either bromhexine hydrochloride 16 mg or an identical placebo to be taken by mouth four times daily for two weeks. After a further week without treatment patients and volunteers took the alternative preparation four times daily for a further two weeks. Resting and stimulated whole and parotid salivary flow rates were measured. Lacrimal function was assessed by the Schirmer test and lacrimal lysozyme concentration.³

There was no difference in the incidence of side effects reported by subjects when taking active or placebo preparations. Three patients thought that one or more of their symptoms improved while taking bromhexine and four while taking the placebo. There were significant ($p < 0.05$) increases in whole stimulated salivary flow rates in both Sjögren's patients and volunteers after treatment with bromhexine and placebo compared with initial flow rates (fig). Improvement was greater with placebo than with bromhexine in the patients, but the difference was not significant. No significant increases in either Schirmer test values or lysozyme concentrations were detected in response to bromhexine treatment. Seven of the nine patients with Sjögren's syndrome had higher salivary flow rates and seven higher Schirmer-test values with placebo. The patients with sicca syndrome did not show this trend.

Comment

The significant increase in whole stimulated salivary flow rates after bromhexine treatment in Sjögren's patients and healthy volunteers was no more than after the placebo, and is unlikely to represent a pharmacological effect on salivary secretion. Frost-Larsen *et al*¹ and Nahir *et al*⁴ were also unable to show that bromhexine increased the production of saliva. Similarly, no subjective improvement attributable to bromhexine occurred in our patients or those of Frost-Larsen *et al*.¹ We have been unable to confirm that lacrimal flow in Sjögren's syndrome is significantly enhanced by bromhexine. In Frost-Larsen's¹ series lacrimal fluid production rose particularly with bromhexine in nine out of 11 patients with Sjögren's syndrome secondary to rheumatoid arthritis or lupus erythematosus. In our series brom-

Salivary flow rates in patients and volunteer controls at initial examination and after bromhexine or placebo.

hexine showed no such effect; while the placebo was more commonly beneficial among patients with Sjögren's syndrome than those with sicca syndrome.

We have not found that bromhexine is effective in the treatment of Sjögren's syndrome, but harmless placebos may have a valuable place in its management.

¹ Frost-Larsen K, Isager H, Manthorpe R. Sjögren's syndrome treated with bromhexine: a randomised clinical study. *Br med J* 1978;ii:1579-81.

² Bloch KJ, Buchanan WW, Wohl MJ, Bunim JJ. Sjögren's syndrome—a clinical, pathological and serological study of sixty-two cases. *Medicine* 1965;44:187-231.

³ Mackie IA, Seal DV. Quantitative tear lysozyme assay in units of activity per microlitre. *Br J Ophthalmol* 1976;60:70-4.

⁴ Nahir AM, Ben Aryeh H, Szargel R, *et al*. Sialochemistry in evaluating bromhexine treatment of Sjögren's syndrome. *Br Med J* 1979;ii:833.

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Parenteral nutrition at home in management of intestinal failure

Parenteral nutrition can now be continued for long periods and patients can be taught to continue treatment at home. In most cases the inadequacy of the gut is only temporary and feeding by mouth can be started again when the bowel has adapted. Rarely treatment has to be continued indefinitely. Several reports on home parenteral nutrition have come from North America and Europe¹⁻⁵ but not from the United Kingdom, although a few centres practise it sporadically. We have treated five cases of intestinal failure of varied aetiology by parenteral nutrition at home. The place of this expensive form of treatment in a society where health care is being subjected to tight cash limits requires careful evaluation.

Patients, methods, and results

Details of the five patients are shown in the table. The indication for prolonged parenteral nutrition was inability to maintain weight and normal

Details of five patients on home parenteral nutrition for intestinal failure

Case No	Age (years)	Sex	Disease and nutritional problem	Time on parenteral nutrition (months)	Time at home on parenteral nutrition (months)	Weight at start of treatment (kg)	Weight at end of treatment (kg)
1	18	F	Polyarteritis nodosa. Mesenteric vascular occlusion. Short-bowel syndrome	5½	3	28	39
2	21	M	Intestinal motility disorder. Small and large bowel resection. Short-bowel syndrome	13*	9*	45	56
3	36	M	Crohn's disease, multiple entero-cutaneous fistulae	10	3	25	47
4	49	M	Mesenteric ischaemia, intestinal fistula, short-bowel syndrome	5	1½	44	52.5
5	34	M	Crohn's disease, multiple small bowel resection, short-bowel syndrome	5	1½	45	57

*Parenteral nutrition continuing.

biochemistry whenever feeding by mouth was reintroduced. All five patients were referred to the hospital. Treatment began with a vigorous programme of resuscitation followed by parenteral nutrition, initially through a silicone elastomer catheter inserted into the right atrium by infraclavicular puncture of the subclavian vein. The feeding regimen was calculated in standard fashion from the patient's measured nitrogen losses and estimated energy requirements. As soon as long-term parenteral nutrition appeared to be necessary the subclavian catheter was changed to one more suited for prolonged intermittent feeding. We have found the Broviac catheter (Evergreen Medical Products, USA) the most suitable. It is inserted into the cephalic vein in the deltopectoral groove and the tip positioned in the right atrium. The proximal end of the catheter is tunnelled beneath the skin to emerge below and medial to the ipsilateral nipple, where the hub is easily accessible to the patient. Once nitrogen and energy requirements have stabilised the infusion system is changed to the "big bag" technique. In this the day's nutrients are placed under sterile conditions into a three-litre bag (Viaflex, Travenol Ltd) and given over the whole or part of the day.

The patients were taught to infuse the nutrients themselves and (between infusions) to fill the catheter with heparinised saline (Heparin lock). When home parenteral nutrition was expected to be for only a few weeks the patients were not taught to use an infusion pump. Instead they were taught to rely on overnight gravity infusion from the three-litre bag controlled by a metal gate clamp. When treatment seemed likely to be needed for several months patients were taught to use a constant infusion pump (Imed 922H, Tekmar Medical Ltd). We gave them a manual, written by ourselves, which reminded them of the technique and how to minimise complications. The staff of the departments of medicine and surgery provided 24-hour cover for emergencies on a rota basis. Complications were remarkably few. Catheter sepsis caused septicaemia in two patients. No case of catheter sepsis occurred in a patient at home. Two Broviac catheters that fractured were repaired successfully with the manufacturer's repair kit.

All the patients lived and four of the five returned to feeding by mouth and are in good health. The results are summarised in the table. A period of 15 weeks off parenteral nutrition in one patient was associated with a profound fall in weight and the onset of listlessness. He rapidly improved when home parenteral nutrition was reintroduced. He will probably have to remain on it indefinitely.

Comment

Only a few patients with nutritional problems require this type of treatment, and hardly ever permanently. The main indications are the short-bowel syndrome resulting from extensive resection or severe inflammatory bowel disease, complicated in some cases by fistula. In the former the residual bowel usually compensates enough for feeding by mouth to be resumed, while in the latter the disease often subsides with bowel rest and appropriate medication. Parenteral nutrition at home adds a new dimension to the treatment of these patients. It is analogous to the early days of home renal dialysis for renal failure. Our results show that the treatment is effective. The problem is the cost, which we calculate exceeds £15 000 a year for each patient. Nevertheless, we believe that it should be available in a few centres.

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Salivary duct calculi presenting as trismus in a child

The submandibular gland and duct are the commonest sites for salivary calculi, which are usually found in adults with good oral hygiene. The condition is rare in children and thus may be overlooked. The diagnosis can usually be made on the typical history of painful swelling in the submandibular region during eating. I report a case in a young girl with salivary duct calculi who presented with acute trismus, making clinical assessment difficult.

Case report

A 9-year-old Caucasian girl presented with a 24-hour history of pain in the right side of the neck and a visible swelling in the same region. On the day of admission to hospital she had not spoken and had been unable to drink, holding her mouth slightly open and dribbling saliva. She was afebrile and had oedema, without erythema, of the right side of the face and neck. Below the angle of the mandible on the right side a hard, fixed, acutely tender swelling was palpable. Examination of the mouth was extremely difficult but suggested there was a calculus in the region of the right submandibular duct orifice. Haemoglobin concentration was 12.9 g/dl and white cell count $16.9 \times 10^9/l$ ($16\,900/mm^3$) with a 90% polymorphonuclear neutrophil leucocytosis. X-ray films of the submandibular area, taken with great difficulty, showed a small (3 mm diameter) calculus in the region of the right submandibular duct orifice. Examination under anaesthesia confirmed that there was a calculus 5 mm proximal to the right submandibular duct orifice. A direct incision into the duct released the calculus and a small amount of sterile pus. Over the next 48 hours the trismus and oedema resolved completely and the right submandibular gland became palpable.

Comment

Submandibular calculi are very rare in children. Only a handful of cases have been reported since 1895. Doku and Berkman¹ reviewed the first major series of 11 cases in children under the age of 16 years and added one case. Several cases have been described since in otherwise healthy children.²⁻⁴ Stones are thought to form because of deposits of calcium salts around a central nidus, such as desquamated epithelial cells, bacteria, or a foreign body. Perhaps surprisingly, only one case has been reported in a child with cystic fibrosis, where there is apt to be increased calculus formation owing to a high ratio of calcium to phosphorus in the saliva and increased turbidity of the saliva with a tendency to form crystals.⁵

Although the condition is unusual in children, there is often a history of intermittent, submandibular swelling, but the diagnosis is often overlooked.² There have been no reports of children with submandibular calculi presenting acutely with cervicofacial swelling