serum-protein-bound iodine 19.0 $\mu\text{g.}/100\text{ ml.}$

The baby was started on L-thyroxine sodium 0.01 mg./day and ampicillin. The day after admission he suddenly collapsed and died.

On necropsy, the lungs were pinkish, but showed numerous areas of subpleural haemorrhage over all surfaces. There was no evidence of atelectasis. The thyroid was many times larger than normal, the enlargement being symmetrical, and the cut surface had a translucent appearance. Section: hypertrophic picture; the acini often tube-shaped, and tipped by tall epithelium. They contained scant or no colloid. There were fairly extensive areas of subarachnoid haemorrhage extending over an area from the anterior part of the parietal region to the occipital lobe; all on the lateral surface of the brain. Serial sections through the brain showed no gross abnormality. All other systems appeared normal.

Though the infant's death was primarily due to subarachnoid haemorrhage, post-mortem examination gave us an opportunity to observe the histology of the thyroid. It is unusual that an infant shows signs of cretinism at or shortly after birth. It seems probable that this is the result of maternal thyroxine passing across the placenta, which accounts for the euthyroid appearance in those infants who subsequently become cretinous in appearance.¹ Morgans and Trotter² described a baby who appeared cretinous at birth and whose mother had taken a proprietary asthma compound during pregnancy, the infant having a goitre at birth. The goitre disappeared spontaneously.

The infant described provides further evidence of the goitrogenic effects and dangers to the fetus of the antenatal consumption of proprietary asthma cures by the expectant mother.—I am, etc.,

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Steroids and Acute Pyogenic Meningitis

SIR,—Your leading article (3 January, p. 6) on pyogenic meningitis and steroid treatment prompts us to make some comments.

We have investigated¹ the results of steroid treatment in pneumococcal meningitis in 81 patients. The results are given in Table I. More patients in the steroid-treated

TABLE I.—*Pneumococcal Meningitis: Incidence of Factors Related to a Poor Prognosis in the Two Treatment Groups*

Conditions on admission	Controls		Steroid-treated group	
	No. of patients	No. of deaths	No. of patients	No. of deaths
Age over 60	9	6	7	1
Coma	6	3	13	4
Convulsions	7	3	7	1
Pneumonia	4	3	8	2
Leucocyte count in spinal fluid less than 1000/cu. mm.	10	4	14	3
Bacteraemia	23	7	21	1
Total no. of patients	37	10 (27%)	44	5 (11.4%)

group suffered from coma and pneumonia at admission. It is seen that among patients with bacteraemia the fatality rate was considerably higher in the control group. This corresponds to the experience gained from animal experiments, in which early treatment with hydrocortisone in pneumococcal bacteraemia revealed a favourable effect.²

It is stressed that the steroid treatment in our material was initiated at an early stage of the infection. No side-effects were observed and the incidence of sequelae was similar in the two groups (Table II).

TABLE II.—*Pneumococcal Meningitis: Incidence of Sequelae in the Two Treatment Groups*

	Controls	Steroid-treated group
No. of patients	37	44
No. of patients discharged	27	39
Slight sequelae (deafness, headache, facial paresis)	6	5
Severe sequelae (paresis, epilepsy, mental disturbance)	3	3

Though the difference in mortality between the two groups is not significantly different at the 5% level, our findings might suggest a beneficial effect of steroid treatment in pneumococcal meningitis.—We are, etc.,

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Pharmacological Aid for Faecal Incontinence

SIR,—The difficulties in keeping incontinent patients clean are great. Children particularly live in constant fear of an "accident," of the odour involved, of the shame, and of becoming a social outcast. We have not found the simple recommendations of the Spina Bifida Study Group¹ helpful in the treatment of our twenty incontinent children who wish to go to school, to social events, and to work. Enemata are not a good solution.^{2,3}

We have had remarkable successes with the following method of bowel-training, which invokes a conditioned reflex. This can be reinforced by the gastro-colic reflex, by stimulation of intestinal motility with neostigmine, and also by suppositories which irritate the rectal mucosa. The programme must of course be adapted to the patient's usual daily schedule, and the advantages of completing this duty early in the morning have to be weighed against the early hour at which the two-hour programme must be commenced.

The patient is given a tablet of neostigmine 15 mg. (up to two tablets if one is inadequate) at a fixed hour, preferably before a meal, on two or three days a week depending on the

frequency of the patient's earlier bowel habits. One-and-a-half hours later, or soon after the meal, the patient is given two glycerin suppositories per rectum. If these cannot be retained, the larger glycerin ovule, ordinarily for vaginal use, will usually remain in situ.

Twenty minutes after insertion of the suppository, the patient is seated on the toilet for ten minutes. If there is a small result, or no result, at the end of this time, an enema is given immediately. The need for this has been found to diminish progressively. Rectal (digital) examination is performed after the bowel action to confirm adequate emptying, especially in the early stages of training. Complete emptying of the rectum is essential, and if necessary the enema is repeated immediately.

By frequent trial over the weeks or months, the neostigmine dose is gradually reduced, and finally eliminated. Suppositories are continued at the scheduled time. Our physiotherapists have helped with some patients by education of the abdominal muscles.

Success with this regimen has enabled us to discharge a resident patient for fourteen years from all care except three-monthly supervision. Now aged 23, she had been admitted for meningo-myelocoele with complete incontinence, loss of sensation of perineum and feet, and partial paralysis of both legs. Braces enabled her to walk on crutches; she had for a long time managed her ileal conduit with complete independence, but it was only after the need for enemata was eliminated that she became confident of being able to cope at home.

The failures so far have been in younger children, probably because they are too young to collaborate actively.—I am, etc.,

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Appendicitis in Umbilical Hernial Sac

SIR,—Although acutely inflamed appendices have been found in inguinal hernias¹ and even in femoral hernias,² I could find only one reference to an appendix being found in an umbilical hernia. From Russia, Bulgakov reported an acutely inflamed appendix in a strangulated umbilical hernia of a ten-month-old baby.³ The rarity of the finding is probably due to the fact that the appendix is seldom found in the proximity of the umbilicus. It would, therefore, appear worthwhile to report the occurrence of a chronically inflamed appendix in an umbilical hernia of an adult.

The patient, a 63-year-old man, was first seen in the outpatient department in January, 1969 complaining of a red and painful swelling in the umbilical region. He had first noticed this swelling three months previously. It had subsided in a few days but had recurred one month later. His own doctor had treated him with tetracycline. He had never vomited, his bowel action was normal, and at no time had he lost his appetite. In the past he had had bilateral femoral

hernias repaired. On examination the lump was found to be closely associated with and deep to the umbilicus, of a cystic consistency, but did not give a cough impulse nor was it reducible into the peritoneal cavity.

At operation, the swelling was found to communicate with the peritoneal cavity. It contained an appendix, the tip of which was chronically inflamed and adherent to the under surface of the umbilical skin. Routine appendicectomy was carried out and no other abnormality was noted.

Photograph of specimen with umbilicus and attached appendix.

The specimen (Fig.) was sectioned and microscopic appearances were that of a mild interstitial inflammation of the appendix. Postoperatively he did well and was discharged home two weeks after his operation. At follow-up one month later he was well.—I am, etc.,

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Deafness after Topical Neomycin

SIR,—Your leading article on neomycin ototoxicity (25 October, 1969, p. 181) following the topical use of this antibiotic has produced surprisingly little comment: may I raise a number of points?

First, we were originally led to believe that despite its small molecule (M.W. about 1,500) it was not absorbed via the gut or other routes. As you note, one had to find out during the last ten years from personal experience or occasional case reports¹ that this was not true.

Second, your leader omits to mention that neomycin is a popular and successful constituent of many proprietary ear drops today. Once upon a time chloramphenicol (M.W.273) was in this position, but neomycin displaced it, perhaps largely because of the much lower incidence and severity of sensitivity reactions causing otitis externa. Only lately have much more sinister side-effects been suggested in man,² and as the result of guinea-pig experiments,³ where topical chloramphenicol at the round window appears to cause deafness by direct absorption. One wonders whether the

skin sensitivity may have been a blessing in disguise and prevented some similar toxic effects in our patients with chronic suppurative otitis media. However, chloramphenicol was in disrepute otologically long before such suggestions were made.

I now wonder whether we are running risks in using any parenterally ototoxic antibiotic in the middle ear, although this would affect also the use of framycetin (M.W. about 600), and seriously reduce our choice of topical treatment for ear infections.

In six years' experience here, where suppurative otitis media and its complications are rife, I have seen several cases of sensorineural deafness in old cases where curious remedies had been instilled into ears having perforated tympanic membranes—for example, "Streptomycin drops" (unknown composition—streptomycin M.W. 1,257.5). Folk-remedies here such as kerosene seem to give a "dead" ear quite frequently. In neither instance has the history been consistent with a suppurative labyrinthitis, and it is tempting to assume absorption by diffusion at the round window (stapes footplate being intact). Even in the United Kingdom I noted many mixed deafnesses in children with chronic suppurative otitis media and much exposure to ear drops containing chloramphenicol or neomycin, without suppurative labyrinthitis or a history of mumps, and at that time defying rational explanation.

Perhaps you will agree that more basic research in this matter is required. Meanwhile clinical expediency dictates the continued use of neomycin and framycetin on a massive scale in ears, but with what ultimate result?—I am, etc.,

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Serratia Septicaemia

SIR,—Your recent leading article (27 December 1969, p. 756) described reports from the United States that *Serratia marcescens* sometimes causes a septicaemia. A recent case showed that *Serratia* is no less pathogenic on our side of the Atlantic.

A man aged 54, known to have chronic renal failure and essential hypertension, underwent mucosal antrectomy for a gastric ulcer on 17 April 1969. After the operation congestive cardiac failure developed and his plasma urea rose. On 21 April he was transferred to the Hammersmith Hospital under Dr. O. M. Wrong with a plasma urea of 340 mg./100ml., and peritoneal dialysis was started. His general condition gradually improved although he continued to require dialysis. Prophylactic oral and intraperitoneal ampicillin were given from 28 April to 8 May. From 8 to 10 May he was given oral

nystatin and intraperitoneal amphotericin B because of oral thrush and a growth of *Candida albicans* in the peritoneal dialysate culture.

On 11 May he developed fever for the first time and suddenly became unconscious. He was hypotensive, with head and eyes deviated to the left and bilateral extensor plantar responses. His plasma urea was 148 mg./100 ml., sodium 123 mEq/l., potassium 4.3 mEq/l., bicarbonate 25 mEq/l., and plasma cortisol more than 100 µg./100ml. A blood culture grew *Serratia marcescens* from all four bottles. Hydrocortisone, cephaloridine, and kanamycin were given, but the patient died eight hours later. Subsequent tests showed that the organism was sensitive to kanamycin but resistant to ampicillin and cephaloridine.

Necropsy showed bilateral lobar pneumonia, erosive oesophagitis, patchy purulent peritonitis, left ventricular hypertrophy with old and recent myocardial infarction, and small kidneys with renal arteriosclerosis, but no evidence of glomerulonephritis or tubular necrosis. There was no focal cerebral lesion.

The source of the patient's terminal septicaemia is unclear: the *Serratia marcescens* might have reached the blood stream from the oesophagus, peritoneum, or lungs. This experience confirms that *Serratia marcescens* may be a serious pathogen in a debilitated patient.—We are, etc.,

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Post-vagotomy Diarrhoea?

SIR,—Mr. R. H. Franklin (14 February, p. 412) rightly stresses the importance of considering coeliac disease in the differential diagnoses of post-vagotomy or post-gastrectomy diarrhoea, as this disease may become unmasked by a gastric operation.

It should also be emphasized that consideration be given to the possibility of exocrine pancreatic insufficiency due to chronic pancreatitis as a cause of steatorrhoea and diarrhoea following partial gastrectomy. Examples of this are well documented.^{1,2}

A suggestive history, radiographic demonstration of pancreatic calcification, diabetes mellitus, and the finding microscopically of undigested meat fibres in the faeces are the main criteria for establishing such a diagnosis. This deficiency state, if recognized, can be remedied by replacement therapy with pancreatic extract, resulting in an alleviation of the diarrhoea.—I am, etc.,

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