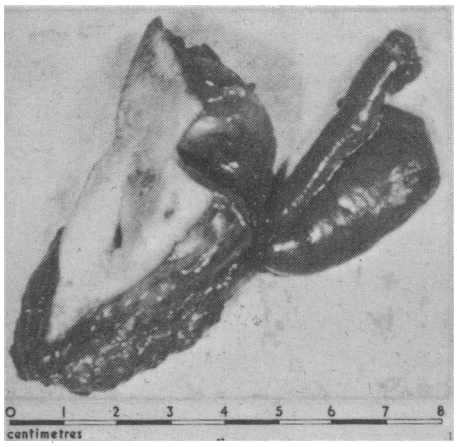
hernias repaired. On examination the lump was found to be closely associated with and deep to the umbilicus, of a cystic consistency, but did not give a cough impulse nor was it reducible into the peritoneal cavity.

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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REFERENCES

1 Shepherd, J. A., *Lancet*, 1954, 2, 299.
2 Mitchell, J. E., *British Journal of Clinical Practice*, 1964, 18, 419.
3 Bulgakov, P. P., *Pediatriya*, 1965, 44, 91.

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 reports[1] 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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REFERENCES

1 Last, P. M., and Sherlock, S., *New England Journal of Medicine*, 1960, 282, 385.
2 Rasmussen, P. E., *Year Book of the Ear, Nose and Throat*, 1967-68, p. 71.
3 D'Angelo, E. P., Patterson, W. C., and Morrow, R. C., *Archives of Otolaryngology*, 1967, 85, 682.

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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REFERENCES

1 Wilson, D. R., Loach, F. W., and Bogoch, A., *Canadian Medical Association Journal*, 1962, 87, 9.
2 Herner, B., and Ysander, L., *Acta Medica Scandinavica*, 1960, 166, 395.