

for veterinary and human use. Use human Lethidrone (veterinary Lethidrone contains 20 mg nalorphine/ml). (iii) If neither Narcan nor Lethidrone is available inject 0.1 ml of the appropriate veterinary antagonist (small/large animal Revivon) intramuscularly, or if the actual dose of Immobilon is known a similar volume of Revivon should be given; repeat the dose if respiratory depression is not reversed.

IT IS VITAL THAT ADEQUATE RESPIRATION AND HEART BEAT BE MAINTAINED UNTIL MEDICAL HELP ARRIVES. IF NECESSARY APPLY ARTIFICIAL RESPIRATION AND EXTERNAL HEART MASSAGE.

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¹ Ministry of Agriculture, Fisheries and Food, *Veterinary Record*, 1976, **98**, 514.
² Firm, S, *Lancet*, 1973, **2**, 95.
³ Firm, S, *Lancet*, 1974, **1**, 577.
⁴ Vaudrey, J C, *Veterinary Record*, 1974, **94**, 52.
⁵ Unpublished reports to Reckitt and Colman Pharmaceuticals.

Enterotoxinogenic bacteria in Africa

SIR,—We read with interest the letter from Dr T Wadström and others (5 June, p 1401) on enterotoxinogenic bacteria in Ethiopian children. This is, however, not the first report on enterotoxin-producing bacteria in an African community. In April 1975 we reported the isolation of three stable toxin (ST)-producing *Escherichia coli* strains from 101 Black infants with acute gastroenteritis in Baragwanath Hospital, South Africa.¹

We have since completed a microbiological investigation of acute sporadic summer gastroenteritis in 36 Black infants under two years of age admitted to Kalafong Hospital near Pretoria. Enterotoxin production was assessed by the Chinese hamster ovary and the suckling mouse techniques for labile (LT) and stable (ST) toxins respectively.

A total of 16 enterotoxinogenic bacteria were found in 15 out of 36 patients (42%), comprising *E coli* 9 (56%), klebsiella 4 (25%), enterobacter 2 (12%), and proteus 1 (6%). In one of the patients two different enterotoxinogenic species, an *E coli* and a klebsiella, were isolated. No invasive strains of *E coli* were found with the Serény guinea-pig keratoconjunctivitis test. By means of negative staining electron microscopy rotaviruses were detected in only 2 (5%) patients. We feel, however, that this figure is unusually low since most patients are admitted to hospital at a relatively late stage in their illness when the excretion of rotavirus particles is too low for detection by electron microscopy.²

We fully endorse the view of Dr Wadström and his colleagues that testing for enterotoxin should be performed before the isolated strains are speciated, as enterotoxinogenicity is clearly not confined to *E coli*. Indeed, in future diagnostic laboratories may well dispense with the species determination of isolated bacteria in cases of sporadic gastroenteritis and merely report on whether enterotoxinogenic or invasive bacteria were isolated. In addition, recently described simpler techniques for the diagnosis of rotavirus infections, such as reverse complement fixation³ and counter-immunoelectro-osmophoresis,⁴ should be

added to the routine tests of the clinical microbiology laboratory, especially where an electron microscope is not readily available.

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² Schoub, B D, *et al*, *South African Medical Journal*, 1976, **50**, 1124.
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⁴ Middleton, P J, *et al*, *Journal of Clinical Pathology*, 1976, **29**, 191.

Raised alpha-fetoprotein levels and congenital defect

SIR,—The observation (3 July, p 22) of a raised amniotic alpha-fetoprotein (α -FP) level in association with a minor congenital defect, postanal dimple, is disquieting. To date a raised amniotic α -FP level has indicated in the fetus either an open neural tube defect¹ or other major congenital abnormalities, namely, oesophageal atresia,² congenital nephrosis,³ Turner's syndrome,⁴ and omphalo-coele.⁵ Although the explanation for the raised amniotic fluid α -FP is not apparent, it is unlikely to be related to the postanal dimple.

We have monitored four pregnancies with normal amniotic α -FP levels which resulted in infants with postanal pits or sinuses (table). We would agree that so far as possible additional investigations should be used to confirm the abnormality in the fetus before the pregnancy is terminated.

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¹ Brock, D J H, and Scrimgeour, J B, *Lancet*, 1972, **2**, 1252.
² Seppälä, M, *Obstetrics and Gynecology*, 1973, **42**, 613.
³ De Bruijn, H W A, and Huisjes, H J, *Lancet*, 1975, **1**, 525.
⁴ Seller, M J, *et al*, *British Medical Journal*, 1974, **2**, 524.
⁵ Nevin, N C, and Armstrong, M J, *Journal of Obstetrics and Gynaecology of the British Commonwealth*, 1975, **82**, 826.

Case	Obstetric history	Amniocentesis			Outcome of pregnancy
		Indication	Gestation	Amniotic α -FP (μ g/ml)	
1	Para 4	Previous anencephaly	17	18.4 (16.02; 30.38)	Female with postanal pit
2	Para 2	Rhesus isoimmunisation	17	11.0 (16.02; 30.38)	Female with neurodermal sinus lower lumbar area; x-ray no abnormality of spine; oval soft tissue shadow posterior to L4
3	Para 2 + 1	Previous infant with spina bifida	15	15.0 (18.46; 32.8)	Male with hairy area lower spine with a postanal sinus; x-ray normal
4	Para 2 + 1	Previous spina bifida infant	16	19.2 (17.08; 32.20)	Male with small postanal pilonidal sinus

Figures in brackets: mean and 95th percentile for amniotic fluid α -FP for this gestation.

Dosage of neomycin sulphate

SIR,—With reference to the letter from Mr M G Thuse and Mr D P Morgan (31 July, p 303) neomycin sulphate tablets contain 350 000 units of neomycin activity, but they are actually marketed as tablets of neomycin sulphate 500 mg and hence it seems convenient for users to have the dose expressed in weight rather than potency units. The dose given in the *British Pharmaceutical Codex* is that recommended by its actions and uses subcommittee and appears to be in accordance with established usage. The dose recommended by the British Pharmacopoeia Commission is somewhat higher, but no doubt both authorities will be reviewing these doses in the light of the discrepancy to which attention has been drawn.

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Early gastric cancer

SIR,—Your leading article (24 July, p 198) implies that it is highly desirable to make a diagnosis of "early" gastric cancer in a greater number of cases, as at that stage of the disease there is a greatly improved prognosis (95% 5-year survival). With that implication we wholeheartedly concur, but feel that there is not sufficient clarity on exactly how this is to be achieved.

Cancer of the stomach (as cancer of many other internal organs) gives specific symptoms only late in its life cycle, and the first point needing emphasis is that earlier diagnosis in significant numbers will be achieved only by testing individuals in an "asymptomatic" phase. The Japanese have been able to produce their large series of "early" gastric cancer by screening an asymptomatic population by means of the double contrast barium meal technique, the gastric camera, and endoscopy, combined with greater use of wash and brush cytology and microbiopsy histology.¹

We in this country have a low incidence of cancer of the stomach compared with the Japanese or some Central European and Scandinavian countries, but the high risk groups of chronic atrophic gastritis, gastric ulcer, polyps, pernicious anaemia, and some familial conditions have been recognised for many years.² It is also becoming apparent that cytological examination of all patients attending a gastroenterological clinic (with or without gastroscopy) will produce a number of latent cancers, as was found in the Massachusetts General Hospital, where early cancer (undiscovered by other diagnostic tests) was