screening for amoebiasis in patients from endemic areas who may require systemic therapy with corticosteroids. The difficulties in screening such patients, especially those who are asymptomatic, have been emphasised recently.¹ This man's serology was positive for *E histolytica* just four days after the onset of symptoms of perforation, implying an immune response which antedated the acute event. We therefore suggest that screening of both faeces and blood for evidence of amoebiasis is desirable in this group of patients.

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¹ Krogstad, D J, Spencer, H C, jun, and Healy, G R,
New England Journal of Medicine, 1978, **298**, 262.

Homes for tuberculous homeless alcoholics

SIR,—It was a relief, for once, to see your publication drawing attention to the plight of the single homeless. All too often they are callously treated, or ignored altogether. Take, for instance, the Housing (Homeless Persons) Act of 1977, under which they are not considered a priority, and therefore are entitled only to "advice" (sic).

Your leading article (21 October, p 1107) notes the difficulties of men discharged to common lodging houses where heavy drinking is prevalent and inmates are obliged to walk the streets for much of the day. Voluntary organisations such as the Cyrenians are setting up small projects where these men may live together in a "family" (and dry) environment—or at least trying to. The Cyrenians and other groups are continually being frustrated in their attempts to help the single homeless by the refusal of local councils to grant planning permission. Exeter and Cardiff City Councils are two recent examples of apparently caring groups whose caring stops at the single homeless person. I'm glad that the *BMJ* is not another.

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Salicylate poisoning in dermatological therapy

SIR,—We would like to report a case of salicylate poisoning from the percutaneous absorption of salicylic acid. A boy aged 12 years was admitted to the paediatric unit of this hospital on 15 September because of severe ichthyosis and asthma. On this occasion he was admitted because of exacerbation of his dermatitis of two weeks' duration. On admission he was started on salicylic acid, 2% in simple cream, applied to all areas of the body twice daily. On the third day in hospital it was increased to 5%, and on the fifth day to 10% concentration. He developed symptoms of salicylate toxicity three days after starting the 10% salicylic acid. The plasma salicylate level was 3.3 mmol/l (46.0 mg/100 ml). The salicylate therapy was immediately stopped and he was started on intravenous fluids and bicarbonate, and complete clinical and biochemical recovery was obtained within 48 hours of the diagnosis being made and

salicylate therapy being stopped. We suggest, therefore, that salicylic acid may cause systemic poisoning when applied to large areas of the skin and that this possibility should be kept in mind when treating cutaneous disorders. As far as we can ascertain, the British medical literature contains no description of poisoning attributed to salicylic acid used on skin. We wonder whether others have seen similar cases.

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The telephone in general practice

SIR,—In a leading article on this subject (21 October, p 1106) you suggest fuller use of the telephone for consultation. Might I suggest that one reason why this is avoided is the doctor's fear of overuse by the patient, particularly the neurotic patient. In a study of 143 patients after myocardial infarction for the purpose of evaluating rehabilitative methods, essential clinic or home visit appointments were made following hospital discharge and patients knew clearly when these would be. The telephone number of the clinic was given to the patient and his wife for interim direct contact with the doctor or nurse, but the telephone was used infrequently and at no time unnecessarily. Patients admitted that the knowledge that direct contact was available was often sufficient in itself to relieve anxiety.

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Current vital statistics

SIR,—Dr A M Adelstein in his article "Current vital statistics: methods and interpretation" (7 October, p 983) notes the paucity of international data relating stillbirths and infant mortality to birth weight. He has a table which shows, by birth weight, neonatal death rates for England and early neonatal rates for Sweden, but he also states that no more comparable statistics are available. This is not strictly true, because early neonatal mortality rates are available for both England and Sweden and neonatal rates also are available for Denmark and Poland.

International comparisons of mortality among low-birth-weight infants (<2500 g)

	England and Wales ¹	Mortality rate			
		Sweden ²	Denmark ³	Poland ⁴	
1974					
Stillbirth	95.6	83.7	52.0		
Perinatal	180.1	178.0	128.6		
Early neonatal	93.4	103.0	80.8		
Neonatal	101.4	Not available	91.7		
1975					
Stillbirth	87.2	Not yet available			68.2
Perinatal	170.4				222.2
Early neonatal	91.2				165.2
Neonatal	98.6				190.1
1976					
Stillbirth	85.3	Not yet available			65.9
Perinatal	156.6				213.6
Early neonatal	77.9				166.8
Neonatal	86.5				192.4

*Stillbirth and perinatal rates per 1000 total births; early neonatal and neonatal rates per 1000 live births.

¹ DHSS Annual Returns LHS 27/1.

² Statistiska Meddelanden Series HS.

³ Medicinsk Fødselsstatistik.

⁴ Rocznik Demograficzny.

The table below indicates some comparisons that are possible from routinely published data, but it is most important that the data be interpreted with caution because of international differences in definition and customary practice in birth and death registration. This applies particularly to infants of very low birth weight.

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Side effects of colapsase

SIR,—We have recently treated a patient with the regimen of sequential colapsase (asparaginase) and high-dose methotrexate for refractory acute leukaemia, as reported by Boh-Seng Yap and others (16 September, p 791). We encountered complications which are not mentioned in their report, but which have appeared in previous publications on the use of colapsase and should be borne in mind when using this regimen.

The patient was a 30-year-old Caucasian male with acute lymphatic leukaemia who first presented in October 1976 and was admitted on 4 October 1978, having relapsed for the third time. There were 89% lymphoblasts in the bone marrow. At the time of admission investigations revealed the following: haemoglobin 11.1 g/dl, white cell count $103.4 \times 10^9/l$ (blasts 60%, smear cells 22%), platelets $103 \times 10^9/l$, sodium 140 mmol(mEq)/l, potassium 4.1 mmol(mEq)/l, bicarbonate 29 mmol(mEq)/l, urea 3.8 mmol/l (22.9 mg/100 ml), creatinine 125 $\mu\text{mol/l}$ (1.4 mg/100 ml), urate 0.88 mmol/l (14.8 mg/100 ml). There was no glycosuria.

On 4 October he was started on allopurinol 300 mg a day; and on 6 October he was given methotrexate, 100 mg intravenously, followed three hours later by colapsase derived from *Escherichia coli* (Crasnitin), at a dose of 40 000 units intravenously. There was little initial reaction to the treatment but throughout the following night he vomited persistently, had diarrhoea, and became dyspnoeic and oliguric. On examination he was dehydrated and had Kussmaul respiration. The results of our investigations on 8 October were: haemoglobin 8.4 g/dl, white cell count $68 \times 10^9/l$, platelets $27 \times 10^9/l$, sodium 117 mmol(mEq)/l, potassium 8.9 mmol(mEq)/l, bicarbonate 13 mmol(mEq)/l, urea 42 mmol/l (253 mg/100 ml), creatinine 710 $\mu\text{mol/l}$ (8.03 mg/100 ml), glucose 42.6 mmol/l (768 mg/100 ml). He was treated with continuous infusion of soluble insulin, intermittent intravenous sodium bicarbonate, rehydration, and blood transfusion. On 10 October sodium was 131 mmol/l(mEq)/l, potassium 5.0 mmol(mEq)/l,