that compression of the oesophagus by the rigid aorta was the cause of her symptoms.

A man of 68 had noticed for a month that after the first few mouthfuls of each meal he had a vague epigastric sensation and then vomited the food without any acid taste. The remainder of the meal could then be swallowed normally. He had not had any difficulty in swallowing liquids. The "ultrasound swallow" gave a long, steady, squirting noise, easily imaginable as a narrow stream under pressure. He is well three months after resection of an oesophageal carcinoma.

A group of patients with symptoms such as sticking of food, retrosternal pain on swallowing, or acid regurgitation have been examined. Many showed swallowing patterns which were irregular in the number, timing, and volume of the pulses and in these patients the symptoms cleared spontaneously, which suggests that the upset was functional. These patients, who are usually rather anxious, have been reassured to hear the evidence that the water reaches the abdomen. This technique may provide reliable positive evidence of oesophageal incoordination and save some patients the ordeal of a barium swallow.

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Naming of drugs

SIR,—I refer to the letter from Dr G A MacGregor (18 November, p 1433) in which he points out that the same preparations (drugs) may be marketed under different names. You comment that his letter favoured the use of BPC approved names.

I have recently examined several aspects of the question of availability of drugs in third world countries. Another phenomenon of the naming of drugs has come to light. The same proprietary preparations made by the same manufacturers may have significantly different compositions depending on whether they are marketed in Britain or in Africa. Sonalgin in Britain contains paracetamol; in Africa this is replaced by phenacetin. Again, the paracetamol in Veganin in Britain is replaced by phenacetin in Africa. Saridone in the United Kingdom contains propyphenazone, but in Africa this is replaced by isopropylantipyrine. Are these compounds the same or different? The busy prescriber will have to find out for himself.

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Frusemide and renal enzyme excretion

SIR,—It was with interest that we read the recent letter from Drs S M Harding and A J Munro (18 November, p 1431) reporting the effect of frusemide on urinary *N*-acetyl- β -glucosaminidase (NAG) excretion. This has not been our experience in similar unpublished work in mice or man.

We have given 80 mg of frusemide at zero

time (9 am) to six healthy male volunteers aged 22-31. The subsequent mean urinary volumes and NHG content are shown in the table. Our results do not show any significant difference ($P < 0.1$) in outflow of urinary NAG in response to oral frusemide. We feel it is therefore unlikely that the increased urinary NAG found by Drs Harding and Munro can be explained by the normal tubular action of frusemide.

It is our conclusion that random urinary NAG estimation remains a sensitive indicator of renal tubular cell damage even after consideration of drug therapy.

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Nitrites—to ban or not to ban?

SIR,—Many thanks for publishing the article by Miss Barbara J Culliton and Mr Wallace K Waterfall (9 December, p 1613) on the banning of nitrites. The antics of the American Food and Drug Administration are frequently good for a laugh—or would be were it not for the possibility of the formation of a pressure group in this country to follow its example; witness the absurd banning of cyclamates.

There are thousands of angina sufferers the world over who in addition to their regular daily dose of nitrites in vegetables (80%) and smoked meats (20%) could easily consume as much again and more in the form of tablets to relieve their pain. So far as I am aware, although they have been doing this for over 50 years, there has been no suggestion that they are more liable to lymphomas or lymphoreticular changes than anybody else. Will someone with access to a sufficient number of cases please take the trouble to confirm (or refute) this before we get the formation of an NBG (nitrite banning group)?

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Car doors

SIR,—The illustration (2 December, p 1552) of a cyclist swerving to avoid an opening car door depicts a hazard all too familiar, but please do not put all the blame on the motorist. Through no fault of his own it takes quite a long time for the less agile driver to climb out *after* he has made certain that nothing is about to overtake him. Car body designers appear to ignore ease of entry and exit, while most of the test drivers of motor journals are young and active and seldom mention such mundane matters, though hailing the cutting of 0.1 s from the 0 to 60 mph acceleration time as a major advance.

I personally have driven since the end of the first world war (certainly over half a million miles) and until I had recently to sell an old Mark VI Bentley I and any passengers had no difficulty in getting out of either side of the car.

The floors were flat and unobstructed and no one had to alight in the traffic stream. Two developments now prevent this. Firstly, it became illegal to fit rear-hinged doors to avoid any risk of their being blown open if they were not properly closed and the safety catches were not functioning. This was a good idea but made a rapid exit more difficult for the unathletic, especially in two-door models. The second and more dangerous trend is to divide a car into four rigid compartments by a high transmission tunnel often mated with an instrument console beneath the dashboard. This combination with gear and brake levers makes it virtually impossible for the driver to slide over and emerge from the near side on to the pavement and quite difficult for the off-side rear passenger to do the same. Anyway, why do front-wheel-drive cars have a tunnel at all? If there is no prop shaft a vestigial hump would be all that is necessary to convey rods, wiring, and pipelines.

What would we think of London Transport if buses were introduced which decanted their passengers into passing traffic instead of on to the pavement?

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Children with small stature

SIR,—We applaud Dr R R Gordon's plea (18 November, p 1434) for more height measurements on children, but we find his other remarks somewhat puzzling in the context of this plea.

We know of no evidence that parents of children with Turner's syndrome or growth hormone deficiency are short. However, the correlation between parental heights and those of their children with Turner's syndrome is maintained¹ so that affected children of short parents will certainly become apparent earlier.

In investigating short stature, as Dr Gordon implies, the crucial measurement is that of height velocity. If this is normal over a period of a year further investigations are unnecessary and likely not to be helpful, regardless of the height of the child on a height distance chart. Conversely, children with low growth velocities may well be within the centiles at the time of referral yet have a diagnosable condition requiring investigation and treatment. A single height measurement hardly ever constitutes an indication for investigation.

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¹ Brook, C G D, *et al*, *Archives of Disease in Childhood*, 1974, **49**, 789.

General practice records

SIR,—Your leading article on this subject (9 December, p 1587) is almost defeatist in tone. That good medical records contribute to good standards of care is a widely held belief in all branches of clinical medicine; it is more than an assumption. General practitioners have far too long laboured under the constraints of an out-of-date and outmoded official records system. It is greatly to their credit that an increasing number of GPs are breaking away from these constraints and introducing A4 records at their own expense.

Mean (± 1 SD) urinary volume (ml) and NAG excretion (mIU/h/mg urinary creatine) following 80 mg oral frusemide

Time (h)	Control (2 h)	0-2	2-4	24-26	26-28
Urine volume	114 $\pm$ 52	1377 $\pm$ 308	596 $\pm$ 178	45 $\pm$ 21	38 $\pm$ 41
NAG excretion	71 $\pm$ 12	65 $\pm$ 28	106 $\pm$ 70	58 $\pm$ 28	70 $\pm$ 44