Cutter Lecture in Harvard nearly 30 years ago on the experimental method in medicine and cited Willis, the president of the New England Antivivisection Society presented me with a copy. When I taxed Lewis with the tract he casually asked if I thought it very bad; I told him I thought it outrageous that a popular theologian with so wide an audience as he had should descend to such ill-informed polemics.

It was a pity that so superb a literary critic as Lewis was should write ignorantly about important questions. *The Problem of Pain* (1940) showed his failure to appreciate simple physiological principles. When he was writing the book I lent him Adrian's *The Basis of Sensation*, but he admitted he had only read a couple of pages. Dr Livesley might better have chosen a much greater Oxford man who like Willis splendidly advanced medicine by using the experimental method: Sherrington summarised the usefulness of pain by: "My aching tooth drives me to the dentist as if it motivated me thither," and "pain is a silver penny bringing a golden groat."

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Response of antidiuretic hormone to chlorpropamide

SIR,—We have read with great interest the report of Dr M C Champion and others concerning the response of antidiuretic hormone to chlorpropamide during chronic treatment (6 September, p 645). They used a fixed dosage and, as they stated, the question of a possible acute effect of chlorpropamide on antidiuretic hormone release remains unanswered.

We have studied the effect of chlorpropamide on antidiuretic hormone levels under both chronic[1] and acute[2] administration. In 25 patients with diabetes mellitus having long-term treatment with a wide chlorpropamide dosage range (from 125 mg to 750 mg daily) the mean fasting antidiuretic hormone concentration did not differ significantly from that of an equally large control group (table). Furthermore, we could not demonstrate any significant correlation between chlorpropamide and antidiuretic hormone levels.

Antidiuretic hormone concentrations (percentage means and SDs) in chlorpropamide group and controls*

	Control group %	Chlorpropamide group %
Chronic administration:		
basal	100 ± 56·7 (n = 25)	109 ± 46·4 (n = 25)
Acute administration:		
basal	100 ± 17·2 (n = 9)	107 ± 27·6 (n = 9)
at the time of maximal rise	117 ± 37·9 (n = 9)	124 ± 31·0 (n = 9)

*The basal values of the control groups are set as 100%.

In the acute study we administered 62·5 mg chlorpropamide intravenously as a quick bolus injection to nine fasting volunteers. We were unable to record a significant rise in antidiuretic hormone levels during an eight-hour observation period by comparison with the effect of an equal volume of saline given intravenously (table). Although the chlorpropamide dose administered was lower than during conventional oral therapy we must remember that the drug was given directly into the central compartment in the absence of the water load, the physiological inhibitor of antidiuretic hormone release.

Antidiuretic hormone was measured by radioimmunoassay[3] and the absolute basal values during the acute study were 3·1 ±0·8 ng/l (chlorpropamide) and 2·9 ±0·5 ng/l (saline).

Thus our findings basically confirm the results of Dr Champion and others and are in agreement with an earlier study made with diabetes insipidus patients.[4] In view of this, mechanisms other than augmented antidiuretic hormone release are likely to be responsible for the effect of chlorpropamide on water metabolism.

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Lymphocyte sensitisation in nifedipine-induced hepatitis

SIR,—I read with interest the article by Dr Heschi H Rotmensch and his colleagues from Israel concerning nifedipine-induced hepatitis (11 October, p 976). Recently I looked after a similar case: he was a 59-year-old man who had drunk alcohol excessively in the past. He presented with a five-year history of mild angina. Nifedipine was used because of increasing angina, and about two weeks after starting this therapy he felt generally unwell and he noticed sweating and shaking chills usually immediately after taking the nifedipine.

The only abnormal physical finding was mild jaundice. Laboratory results included a bilirubin of 52 μmol/l (3 mg/100 ml), alkaline phosphatase 20 KA units, γ-glutamyl transferase 1592 IU/l, and alanine aminotransferase 49 IU/l. Liver biopsy showed subacute hepatitis on a background of alcoholic liver disease. The nifedipine was discontinued, and immediately the patient's symptoms settled and he felt better. The liver function tests returned to normal. A repeat liver biopsy six months after stopping therapy showed no hepatitis.

Although this patient had underlying alcoholic liver disease, there was a definite hepatatic illness which occurred while on nifedipine therapy and resolved when the drug was withdrawn. Therefore, this case adds further weight to the suggestion that nifedipine should be added to the list of drugs causing allergic hepatitis.

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Hearing impairment in the elderly

SIR,—I read the article "Hearing impairment and mental state in the elderly living at home" by Kétia Gilhome Herbst and Charlotte Humphrey (4 October, p 903) with great interest as my observations are complementary to their findings. I carried out routine auriscopy on 59 consecutive hospital admissions to a geriatric unit irrespective of their hearing defect. Wax in the ears was present in 43 patients (73%), to a considerable amount in 10 of them (23%) causing gross hearing impairment with marked improvement in their hearing simply following wax removal.

From these results it can be recommended that all elderly should have routine auriscopy as part of their clinical examination to exclude presence of wax prior to their referral to ear, nose, and throat specialists or audiometricians for evaluation of hearing defects.

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Following up patients with rheumatoid arthritis

SIR,—I have been reading with great interest all the recent communications on this subject including the latest one from Dr D R L Newton (11 October, p 1007). I would wholeheartedly like to support the view expressed so clearly by Dr Newton.

I started in my new job about two years ago and already the outpatient clinic waiting time has lengthened to even five and six months in some of the clinics. This kind of waiting time is quite embarrassing and for any consultant working singlehanded extremely difficult to cope with.

A situation of this type should certainly put some responsibility on to the shoulders of the Royal College of General Practitioners. Patients are sometimes referred to the consultants for even simple procedures like local steroid injections for "tennis elbow." In such cases can any consultant be expected to entrust the follow up of cases for rheumatoid arthritis to the respective family doctor? I am sure something can be done and should be done to rectify these deficiencies in areas where they exist.

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A better system for polio vaccination in developing countries?

SIR,—Dr W Ehrengut (11 October, p 1004) has already corrected Dr Dion Bell's statement (20 September, p 810) that freezing oral poliomyelitis vaccine is harmful but further comment is required. Both the British and European pharmacopoeias recommend that oral poliomyelitis vaccines be stored at −20°C or below, or between 0° and 4°C. Since many users do not have facilities for storage at −20°C the vaccine is issued with expiry dates based on storage at 0°-4°C. The precaution "do not freeze" is misleading and is being removed.

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Treatment of axillary hyperhidrosis

SIR,—We are writing to take issue with a number of points raised in your columns concerning solutions of aluminium chloride hexahydrate in ethanol used for the treatment of hyperhidrosis (6 September, p 683).

While we agree that dissolution of this salt in absolute ethanol is a lengthy procedure, we wonder at this choice of solvent. Our results suggest that the solvent composition is fairly unimportant provided that a reasonable dissolution time and drying rate are achieved. This