In answer to Dr. R. M. Robertson (7 December, p. 642) 8 out of 48 patients (16.7%) who were not known diabetics showed glycosuria at some time prior to operation. However, Jackson *et al.*[8] have reported that "glycosuria is commonly intermittent, even in untreated diabetics, is often absent when hyperglycaemia is present and is not a good screening test even after a glucose load." It was for this reason that we have not stressed glycosuria data. Since the data from this study became available it has increasingly become the clinical practice of the urological surgeons of this hospital to perform a glucose-tolerance test in the preoperative assessment of patients with prostatism.

The clinical observation that cardiovascular disorders are frequently associated with prostatic hyperplasia was made in the last century by Sir Benjamin Brodie,[9] who wrote: "When the hair becomes grey and scanty, when specks of earthy matter begin to be deposited in the tunics of the artery, and when a white zone is formed at the margin of the cornea, at this same period the prostate gland usually—I might perhaps say invariably—becomes increased in size."—We are, etc.,

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Rejection on Medical Grounds

SIR,—I recently saw a man who had been promised a job "subject to medical examination." After the examination he was told that the firm was extremely sorry that he could not be appointed because his blood pressure was above the limit laid down. He had never been ill, was symptom-free, had no cardiac enlargement, and his blood pressure varied between 180/105 and 160/95. He had set his heart on this job and was profoundly despondent about his future, fearing that as one firm had turned him down because of his blood pressure other firms would do the same.

I have advanced the view[1] that leaving aside those such as bus drivers whose illnesses may endanger others—this kind of medical rejection is a monstrous injustice and should be forbidden by law. Those who are rejected because of creed, colour, race, or sex can at least appeal to some organization; those rejected medically are helpless.

As a profession we often complain about bureaucratic interference in the doctor–patient relationship. Yet some of us so distort that relationship that we harm the patient in the supposed interests of a prospective employer. Those who do so appear to observe the following modification of the Hippocratic Oath, "The regimen I adopt shall be for the benefit of my patients according to my ability and judgment, and not for their hurt or for any wrong, unless I am examining them on behalf of an employer, when the welfare of the patient shall count for nothing, and the interests of the employer shall be my sole concern."—I am, etc.,

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Prevention of Lumbar Puncture Headache

SIR,—I note that Dr. H. Aziz and others (December 14, p. 677) have not found vasopressin to be effective prophylactically in post-lumbar puncture headache. If I recollect correctly, it has been advocated that after lumbar puncture patients should lie prone for at least 24 hours, with one flat head pillow. The rationale appears to be that the spinal extension so produced helps to close the thecal puncture hole, while flexion tends to keep it open. I cannot quote my authority for this simple, safe, and rational suggestion, and would welcome any references.—I am, etc.,

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Neurogenic Intermittent Claudication

SIR,—"Intermittent claudication simply means intermittent limping, but it has become a term specifically applied to the interference with exercise caused by pain from an ischaemic muscle. Therefore one can designate a pain as intermittent claudication only if it is produced by exercise and relieved by rest."

This is the opening paragraph of an article in "Medicine Today" (7 December, p. 630). It implies that occlusive arterial disease of the legs is the sole cause of intermittent claudication. There is no reference to the neurogenic causes of intermittent claudication through narrowing of the lumbar spinal canal by a chronic disc prolapse or spondylitic bar. These have been so well described in your own, as well as in other journals, and can mimic the syndrome produced by chronic arterial disease so closely, that the differential diagnosis may depend upon myelography as much as upon aortography.[1–6] When correctly diagnosed, neurogenic intermittent claudication can be completely relieved by laminectomy. Why therefore ignore it?—I am, etc.,

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Hyperpyrexia during Anaesthesia

SIR,—It has been suggested by Dr. P. J. Horsey (28 September, p. 803) that malignant postoperative hyperpyrexia is related to postoperative halothane shivering, and indeed both conditions are associated with muscular rigidity of unknown cause and with anaesthesia. Furthermore, the degree of muscular rigidity occurring after halothane anaesthesia may be extreme.

We have made some observations on postoperative halothane shivering and agree with Dr. Michael Johnstone (19 October, p. 184) that methylphenidate (Ritalin) is effective in suppressing shivering occurring during emergence from a halothane anaesthetic.

In 1967 we were looking for some means of controlling the latter condition, and at that time the weight of the evidence seemed to support an association between a fall in body temperature during anaesthesia and the occurrence of shivering during the recovery phase.[1] The evidence for this was not conclusive, but we conducted a pilot trial using intravenous ethyl alcohol, which we have found effective in producing vasodilatation and suppressing shivering during controlled hypothermia.

We found alcohol of no benefit as a treatment given as a 15% solution in normal saline intravenously in doses of 40–50 ml. in six cases of established shivering; nor was it of benefit prophylactically. Of 16 patients given the same dose of alcohol as an intravenous infusion in the immediate postoperative period five developed marked shivering.

It has been reported that methylphenidate is effective in suppression of shivering from many causes.[2] We gave it in a dose of 20 mg. intravenously to 34 patients who had developed shivering following a halothane anaesthetic. It was effective within two minutes in every case. Other effects noted included flushing, a rise in pulse and respiratory rate, occasional slight rises in blood pressure, arousal, and two instances of vomiting. The last effect had been related to speed of injection.[2]

The effect of methylphenidate on post-halothane muscular rigidity is so dramatic that it would be of interest to study its action in cases of malignant hyperpyrexia.—We are, etc.,

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Eating and Corticosteroid Levels

SIR,—I have found that the concentration of 11-hydroxycorticosteroids (11-OHCS) in plasma of healthy men and women is usually quickly increased to up to 300% of its initial level by lunch. Less pronounced increases were found after glucose, alcohol, and unsweetened white coffee. Intravenous infusion of ethyl alcohol was recently reported to increase the plasma 11-OHCS (29 June, p. 804).

Blood was taken from 11 healthy subjects a few minutes before and one hour after they had started to eat their usual (unstandardized) lunch. 11-OHCS were estimated in plasma by a method similar to that of Spencer-Peet *et al.*[1] and were expressed as μg. cortisol/100 ml. The mean plasma 11-OHCS concentration before lunch was 11.8 μg./100 ml. (range 7.4–19.5), and after the meal was 18.7 μg./100 ml. (range 13.4–23.5). The increase depended on the initial plasma level, being greatest when the plasma concentration was lowest (Fig. 1). There was

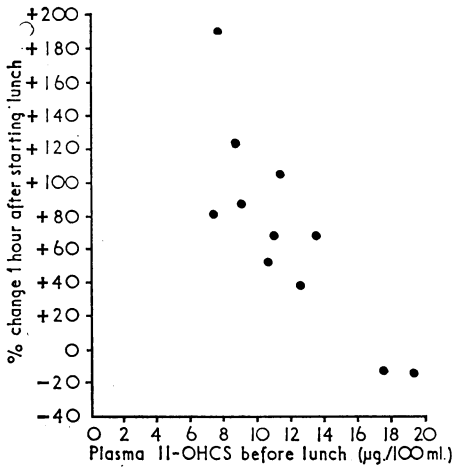

FIG. 1.—The percentage change of concentration of 11-OHCS in plasma one hour after lunch, plotted against the concentration before lunch (μg. cortisol/100 ml.).

a slight fall in two of the subjects whose initial levels were above 17 μg./100 ml. In a patient with Addison's disease who had a cortisol level of 3.0 μg./100 ml. there was no change. In some individuals, serial samples of plasma were taken at intervals after eating. Slight rises were found after 30 minutes. The level at 1½ hours was lower than at 1 hour (Fig. 2) and at 2½ hours the plasma concentration was below its initial level. Ingestion of 50 g. of glucose at 11 a.m. or of two cups of unsweetened white coffee each produced smaller increases of plasma 11-OHCS—up to 80% after 1 hour. A rise was noticed as soon as 20 minutes after ingestion. Increases produced by drinking 15–20 ml. of 50% ethanol at 11 a.m. or at 12.30 p.m. were small after 1 hour (up to 40%), but were up to 100% after 2 hours. The exertion of walking twice down and up four flights of stairs to and from the refectory did not alter plasma 11-OHCS.

Using pooled plasma, thin layer chromatography was carried out on silica gel impregnated with fluorescein. The silica gel was eluted with methanol and methylene chloride, 2 ml. of water was added, and the estimations for 11-OHCS carried out as usual. The fluorescein did not interfere with the method. No evidence was obtained that the after-lunch plasma was qualitatively different from usual. Urine samples were collected during one-hour periods and analysed for 11-OHCS. The method was the same as for plasma except that the 10 ml. methylene chloride extract was washed once with 2.5 ml. of 0.1 N-NaOH. The mean concentration of 11-OHCS, unaffected by lunch, was 69.8 μg. cortisol/g. of creatinine (range 46.4–92.6), or 3.14 μg. cortisol/hour (range 1.63–4.48). During the hour starting half an hour after lunch began, excretion rose to a mean value of 113 μg./g. creatinine (range 55.1–174) or 6.10 μg./hour (range 3.80–9.60). Levels were usually less high during the subsequent hour (Fig. 2), and the increase was always smaller when it was expressed in terms of creatinine.

The above changes of plasma 11-OHCS concentration and of urinary excretion are quite distinct from the slow variations which constitute the diurnal rhythm. Their mechanism is not known. Hypoglycaemia stimulates the secretion of cortisol, and it may be surprising that ingestion of carbohydrate should do the same. There appears to be a critical concentration of cortisol—about 16 μg./100 ml.—above which food is ineffective. The increase is produced more quickly by food than by alcohol, in spite of the more rapid absorption of the latter. Its time course may parallel that of some aspect of digestion with which it might be concerned, such as, for example, transfer of glucose or fatty acid across a membrane.

Breakfast too might interfere with tests of pituitary and adrenocortical functioning in cases where either gland is hypoactive.

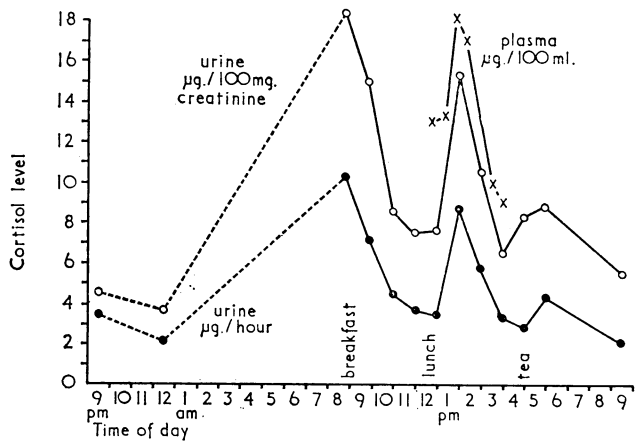

FIG. 2.—The concentrations of 11-OHCS in plasma and in one-hour samples of urine at different times of day. The curves for urine have been drawn through the middle of each collecting period.

I am grateful to Dr. E. J. Ross and volunteers who gave blood for these experiments.

—I am, etc.,

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Damages against Doctors

SIR,—It is disturbing to find your correspondents Mr. G. H. Alabaster (30 November, p. 576) and Professor M. F. A. Woodruff (7 December, p. 643) basing their criticisms of the law relating to professional negligence on what appears to me to be a misunderstanding of the position.

Professor Woodruff refers to a test of negligence which is "grossly unfair to doctors." As you, Sir, pointed out in a leading article earlier this year (18 May, p. 381), the standard of care required is that of a doctor of ordinary competence, of equivalent qualifications and pretensions to skill. Professor Woodruff's claim that the standard is that of "a doctor who never becomes fatigued or makes mistakes, and who is not only better informed than most of us can ever hope to be but never seems to forget what he has learned" cannot be substantiated. Perhaps two of the many dicta of judges in actions for negligence will show that the courts are aware of the difficulties inherent in practising medicine. Lord Justice Scott said, in *Mahon* v. *Osborn*,[1] "It is not every slip or mistake which imports negligence and, in applying the duty of care to the case of a surgeon, it is particularly necessary to have regard to the different kinds of circumstances that may present themselves for urgent attention." The Court of Appeal in *Williams* v. *North Liverpool H.M.C. and Others*[2] approved Mr. Justice Elwes's dictum that "There are risks inherent in most forms of medical treatment. . . . All one can ask of a practitioner is that he should keep these risks to a minimum. . . . If he does this, no injury which occurs, however serious, is actionable."

Admittedly there are decisions (such as *Hucks* v. *Cole*,[3] which you castigated in the leading article already mentioned) which appear to misapply the test in relation to the facts, but this works as often in favour of the defendant as against him.

Mr. Alabaster writes from South Africa about, *inter alia*, the iniquities of juries. In England jury actions in negligence cases have become rarer since 1934,[4] and where personal injuries are concerned are practically obsolete.[5] As to the worry caused by unfounded allegations, a doctor is in no worse position than anyone else against whom an unfounded claim is made. The law can help only by dismissing the action.

It is ironical that Mr. Alabaster should refer to the Medical Defence Union immediately before speaking of "a legal penalty of possibly obliterative dimensions" which may be the lot of a negligent practitioner. Is it not to indemnify doctors against such catastrophes that the defence organizations exist?

The function of the law is not only to protect the public from negligent treatment, it also protects the medical profession from lowering its standards. "The ordinary skill of an ordinary competent [medical] man"[6] should be attainable by most doctors: is Mr. Alabaster asking that the profession should tolerate something less than this?—I am, etc.,

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