strains and 11 antibiotics, with the findings of Dr. Stokes's and Professor Mary Barber's departments. In all, the participating laboratories reported on 3,386 tests, and only 114 of their results were classified as errors. On this basis, 97% of their reports were "correct". The clinicians who depend on us for vitally important information may find this figure more reassuring.—I am, etc.,

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¹ Association of Clinical Pathologists Bacteriology Committee, *Journal of Clinical Pathology*, 1965, 18, 1.

Drink, Drugs, and Driving

SIR,—It has recently been reported¹ that the number of road traffic accidents involving charges being preferred as regards excessive alcohol levels have increased substantially. However, it may well be that this represents the tip of the iceberg as regards people whose driving is impaired; not only by alcohol, but by drugs, mainly psychotropic agents.

Generally speaking, it is only the patient and his family doctor who are aware of those driving under the influence of drugs (which is an offence at law). Many doctors advise their patients not to drive within the few hours after taking these agents.

It would seem that steps should be taken not only to dissuade people from driving under the influence of drugs, but to bring existing legislation into effect by requiring family doctors to provide details of current therapy of their patients involved in road traffic accidents.—I am, etc.,

T. C. MAYER

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¹ *Birmingham Post*, 17 June, p. 2.

Pericarditis after Myocardial Infarction

SIR,—I read with great interest the paper by Dr. U. Thadani and others (17 April, p. 135). I have studied a series of 195 patients with myocardial infarction treated in the coronary care unit during the course of a year. A pericardial friction rub was heard in 22 of the patients. In most of them the rub appeared on the second, third, and fourth day after the onset of symptoms and in three soon after cardiac resuscitation. It remained audible for one to three days in most of the patients, but in very ill patients it persisted for three to seven days. In none of the patients the rub recurred, so that none developed the postmyocardial infarction syndrome.¹ The site of infarction was anterior in 12, posterior in four, and anterior and posterior in six. The rest of the most important clinical and laboratory findings are given in the Table. As a group the 22 patients with pericarditis compared with 50 consecutively admitted patients without pericarditis had a worse prognosis, but this was due to the extensive infarction per se and not to pericarditis.

The interesting finding of this study is that postmortem examination revealed a considerable amount of haemorrhagic fluid²⁻⁴ in two patients who were receiving anticoagu-

	With Pericarditis (22 patients)	Without Pericarditis (50 patients)
	No. of Patients	No. of Patients
Anticoagulants ..	13	35
Arrhythmias and heart block* ..	18	34
Heart Block* (2nd and 3rd degree)	12	4
Severe Cardiac Failure ..	14	10
Cardiogenic Shock	6	1
Haemorrhagic Pericardial Fluid	2	0
Abnormal Chest x-ray ..	13	16
S.G.O.T. (units/ml. normal <100 units) ..	150-500 Mean 333	160-330 Mean 180
Mortality ..	4	6
Days of Hospitalization	46	28

There was no significant difference in admission delay, sex and age, and site of infarction between the two groups of patients.

*Most patients developed more than one arrhythmia.

lants (heparin and warfarin), and had died in cardiogenic shock. Both clinicians and pathologists agreed that the effusion had attributed to the development of the shock. It is suggested therefore that anticoagulants should be discontinued after the development of a pericardial friction rub in patients with myocardial infarction.

I am grateful to Dr. C. S. McKendrick for permission to report the results of this study.

—I am, etc.,

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¹ Dressler, W., *Archives of Internal Medicine*, 1959, 103, 28.

² Goldstein, R., and Wolff, L., *Journal of the American Medical Association*, 1951, 146, 616.

³ Fell, S. C., Rubin, I. L., Enselberg, C. D., and Hurwitz, E. S., *New England Journal of Medicine*, 1965, 272, 670.

⁴ Miller, R. L., *Journal of the American Medical Association*, 1969, 209, 1362.

Marihuana Withdrawal Symptoms

SIR,—There are few references in the literature to reported cases of physical withdrawal symptoms from marihuana,¹⁻⁴ while others have an open mind as to their existence.⁵⁻⁸ However, the literature is full of the mistaken impressions that they do not occur at all and hence I feel obliged to report my experiences in this regard.

During the winter of 1970, I accompanied a large group of men and women to an area of the South-west African desert. Here I encountered symptoms among three males and two females, aged 19-29 years, which I believe were indicative of marihuana abstinence.

The smoking of "pot" was prevalent among the younger adults in the camp and as supplies diminished I was presented with patients who had anxiety symptoms and restlessness coupled with acute abdominal cramps, nausea, sweating, increased pulse rate but no rise in temperature, low blood

pressure, and muscular aches. There was no loss of appetite, in fact the contrary was the case, with a craving for sweets and particularly chocolate. There was no enteritis which might have been due to dietary changes or local brackish water supplies, and no other illnesses. There were no viral infections in the camp; this was not a malarial district nor were there tick bites. The abdominal cramps did not appear to me to suggest a diagnosis of acute abdomen, or gynaecological or urinary infections or conditions, or spasm of biliary or urinary tracts.

I was unable to make a diagnosis at the time and the patients were treated with antispasmodics and bed rest. The symptoms persisted from one to three days. A short while later, on looking back, I realized that the disappearance of the symptoms had coincided with the arrival of a courier from the nearest sea-port who had brought with him a new supply of marihuana.

Some weeks later, at another area, I saw one more severe case in a 20-year-old woman with symptoms of similar pattern. She admitted that she had been partaking recently of too much "pot" and as she was proceeding home she had decided to make a complete break and thus had suddenly stopped taking it. I did not see her again, but learned from her friends some ten days later that her symptoms had disappeared, she was no longer taking marihuana, and in fact was extremely well. There was yet another much milder case which followed a like pattern.

I was satisfied that none of these patients had been on other drugs that might have given withdrawal symptoms. Hence it was not unreasonable that I came to the conclusion that these seven patients had in fact been experiencing acute physical withdrawal symptoms from the smoking of what is recognized in our country as a more potent type of cannabis than usually encountered in Europe or America.—I am, etc.,

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¹ Williams, E. G., Himmelsbach, C. K., Wikler, A., Ruble, D. C., and Lloyd, B. J., *Public Health Reports*, 1946, 61, 1059.

² Bouquet, J., *Journal of the American Medical Association*, 1944, 124, 1010.

³ Fraser, J. D., *Lancet*, 1949, 2, 747.

⁴ Gaskill, H. S., *American Journal of Psychiatry*, 1945, 102, 202.

⁵ Bergel, F., and Davies, D. R. A., *All About Drugs*, p. 54. London. Nelson, 1970.

⁶ Cameron, D. C., *World Health*, 1971, April, p. 4.

⁷ Abdulla, A., *Schweizer medizinische Wochenschrift*, 1953, 83, 541.

⁸ Watt, J. M., *Bulletin on Narcotics*, 1961, 13, No. 3, 9.

Trauma to the Urinary Tract

SIR,—I should like to congratulate Mr. J. P. Mitchell on this excellent article (5 June, p. 567), with which I entirely agree. There is, however, one small point with regard to complete rupture of the posterior urethra which I feel he does not stress sufficiently and I hope he will not mind my drawing attention to it.

When repairing a rupture of the posterior urethra it is extremely important to approximate the two ends as nearly as possible by one or more sutures running from the prostatic capsule on either side to the triangular ligament below, so as to hold the

prostate down on to the triangular ligament. The catheter itself is not sufficient to do this, and if one relies on the catheter alone there is usually a long gap between the two ends which has to heal by fibrous tissue and this inevitably leads to an extremely difficult stricture. I know that it is impossible to stitch the two ends of the urethra itself as they are inaccessible, but, though difficult, it is not impossible for those used to operating in the retropubic space to fix the prostate and therefore the prostatic urethra down to the triangular ligament and therefore the membranous urethra with the stitches I have described.

It is a great pity that this point is not stressed more in the routine textbooks of surgery for I think most surgeons and many urologists rely on the indwelling catheter to approximate the two ends. I am quite sure that if this amount of tension is put on a Foley catheter further damage can occur and in any event, it would be unlikely to bring the two ends anywhere near together.—I am, etc.,

S. H. C. CLARKE

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Surgery for Rectal Prolapse

SIR,—In reply to Mr. R. S. Lawson (24 April, p. 224) I think it is important to provide some background to the paper in which I reported the results of rectosigmoidectomy for rectal prolapse at St. Mark's Hospital.

The staff of this hospital were unhappy about the results of a number of operations carried out for this distressing complaint, and I was given the task of reviewing all cases of rectal prolapse admitted to St. Mark's Hospital between the years 1948 and 1960 inclusive. The total number of cases reviewed was 536. Of these, 142 were treated by rectosigmoidectomy. Thirty-two (22%) were untraced. Full details were available on the remaining 110 cases. Of these, 50% developed a complete recurrent prolapse postoperatively and 8% a mucosal recurrence. Half of the complete recurrences occurred within three years of the operation. Admittedly the criterion adopted for assessment of incontinence in this study is exacting in that we define it as the uncontrolled passage of solid or liquid faeces. Attempts to quantify it in any other way make nonsense of statistical evaluation. A knowledge of anorectal physiology makes the poor continence rate understandable in that this operation anastomoses a highly active segment of colon—namely, the sigmoid—to the top of the anal canal, which in rectal prolapse has defective sphincters. Add to this the loss of rectal sensation concomitant upon excision of the rectum and these patients are left with very little of the normal physiological requirements for faecal continence, so that when they do develop recurrent prolapse they present a very difficult salvage problem indeed. Further amputation is not a proper solution.

The follow-up and postoperative management of these cases was thorough, and they were all taught sphincter exercises and a number of them were given courses of pelvic faradism, which does not appear in itself to improve muscle function but it does help to

re-educate the patient in the use of his pelvic muscles. This is important as investigation by electromyography shows that many of these patients have lost their pelvic postural reflex and the faculty of conscious proprioception where their pelvic muscles are concerned.

In spite of these measures this very large number of recurrences occurred—surely because the operation of rectosigmoidectomy is not based on either sound physiological or aetiological principles. One has great respect for Ernest Miles and his works but at the time he developed this operation for rectal prolapse very little was known about the physiology of the anus and rectum and, in all fairness, one should recognize Aufret's original contribution of first performing the operation for a gangrenous rectal prolapse in 1886.

With regard to Mr. Lawson's other points, we did not find the obesity had any significant bearing on success or failure with this operation.

His final point in regard to avoidance of alcohol and heavy beer drinking puzzles me. The predominant incidence of complete rectal prolapse in this country occurs in women from the age of 40 upwards with a peak in the seventh decade. I can only conclude that the incidence of rectal prolapse in Melbourne with regard to sex and age differs from that in this country, or maybe the drinking habits do.—I am, etc.,

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Sussex

Hormones in Advanced Cancer

SIR,—In the article on hormones in advanced cancer (26 June, p. 760) the following statement occurs. "Larger doses of L-thyroxine (0.4 mg daily) and tri-iodothyronine (200 mg daily) may control recurrent disease and should be tried after other methods of treatment have failed, regardless of the histological type of the tumour."

I know of no evidence that this treatment has any beneficial effect in undifferentiated tumours of the thyroid and to prescribe these hormones "regardless of the histological type of the tumour" is incorrect, unless the patient is hypothyroid.—I am, etc.,

RAYMOND GREENE

London W.1

Iatrogenic Raynaud's Phenomenon

SIR,—We would like to report the development of Raynaud's phenomenon in a patient treated with the recently introduced anti-hypertensive agent clonidine.

The patient is a 28-year-old man with severe hypertension owing to chronic proliferative glomerulonephritis of five years' duration. His renal function has remained relatively stable over this period (creatinine clearance 56 ml/min/1.73 m²) but on recent hospital admission his blood pressure was found to be 200/140 mm Hg, despite methyldopa and bethanidine therapy. On this occasion clonidine was gradually substituted for the above therapy to a dosage of 0.9 mg daily with initial good control of his hypertension. At the end of a four-week period on

this dosage he presented with dry mouth, drowsiness, complete impotence, a dry scaly rash of the dorsal aspect of the hands beginning with bulla formation, and arthralgia. More striking was his complaint of recent onset of Raynaud's phenomenon de novo of one week's duration, which had become so troublesome that he requested withdrawal of the drug. Withdrawal resulted in gradual cessation of his symptoms over a period of three days, with no recurrence over the last four months. There was no evidence of cervical rib; lupus erythematosus, latex, and antinuclear factors were negative; erythrocyte sedimentation rate and immunoglobulin levels showed no change from previous figures; there was no evidence of eosinophilia nor other haematological abnormality.

The symptoms suggested a syndrome resembling hydralazine-induced systemic lupus erythematosus but evidence for this was not forthcoming. Skin eruptions have been reported,¹ and five patients (one of whom had transient Raynaud's phenomenon which disappeared during continuation of the drug) were reported in discussion at a symposium on clonidine.¹ The drug is a peripheral vasoconstrictor due to its alpha-adrenergic action when given intravenously,² but when given orally its hypotensive action is thought to be mediated by reduced vascular smooth muscle response to catecholamines and angiotensin.³

It is interesting that Raynaud's phenomenon can develop with such diverse peripheral actions of the drug and we wonder if other workers have knowledge of similar phenomena.—We are, etc.,

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¹ *Catastrophes in Hypertension: A Symposium* held at the Royal College of Surgeons of England, March, 1968. Ed. M. E. Connolly, p. 213-215, London, Butterworths.

² Barnett, A. J., and Cantor, S., *Medical Journal of Australia*, 1968, 1, 87.

³ Zaimis, E., and Hanington, E., *Lancet*, 1969, 2, 298.

Sniffing Syndrome

SIR,—The correspondence (19 June, p. 708) on the use of medicated aerosols by asthmatics in response to your leading article on the "Sniffing Syndrome" (24 April, p. 183) has prompted us to give this brief account of experiments we have undertaken to assess the risks that may be associated with using isoprenaline aerosols propelled by fluorocarbons.

In common with other workers, such as Reinhardt *et al.*,¹ we have found that serious cardiac arrhythmias can be produced under certain severe experimental conditions by the rapid intravenous injection of a large dose of adrenaline during the inhalation of the fluorocarbon propellants by conscious dogs. In contrast to Taylor and Harris,² however, we found this cardiac sensitization to be only temporary, since an injection of adrenaline a few minutes after cessation of exposure never resulted in arrhythmias.

In similar experiments, using isoprenaline in place of adrenaline, we were unable to produce cardiac arrhythmias during fluorocarbon inhalation. Furthermore, when we tried to simulate the use of a pressurized