reabsorption either in their patient with primary aldosteronism or in their earlier studies on the effect of aldosterone in normal subjects.²

At the present time it is believed, on evidence which is mainly derived from animal experiments, that potassium secretion into the colon is passive and occurs as a result of the negative intraluminal potential created by active absorption of sodium ion. If this view is correct the primary action of aldosterone is likely to be on sodium, as appears to be the case in all other tissues, including the renal tubule,⁴ in which aldosterone influences electrolyte transport. The fact that Levitan and Ingelfinger,⁵ using essentially the same method as that employed by Mr. Shields and his colleagues, found an effect on sodium but not on potassium suggests that small differences in experimental technique have a profound effect on the results of this type of experiment.

The accompanying Figure, from an experiment which Dr. R. B. I. Morrison, Miss Ann Metcalfe-Gibson, and myself performed in 1962, confirms that aldosterone indeed has an effect on net sodium absorption from the human colon.

The patient was a 52-year-old woman who had had a mid-transverse colostomy for diverticulitis performed three months earlier. At zero time in each experiment the loop of defunctioned colon, which had previously been repeatedly washed out, was infused from the proximal opening with 600 ml. of physiological saline, and 5-ml. samples were thereafter removed at 30-minute intervals through a soft rubber catheter for analysis. In the two control experiments (days one and ten) no more fluid could be obtained after eight hours. Although up to that time sodium and potassium concentrations showed reciprocal changes, it is clear from the disappearance of the infused solution that net sodium reabsorption was greater than net potassium secretion. Aldosterone 0.5 mg. eight-hourly was given intramuscularly from days two to five inclusive, and two more studies, otherwise identical, were performed on days three and five. Sodium concentrations now fell much more rapidly, with reciprocal changes in potassium concentration. It was not possible to aspirate more fluid after five and a half hours, showing that water had also been absorbed more rapidly as a result of the increased net sodium absorption.

Patients with a loop of defunctioned colon are not common, but do provide an opportunity for electrolyte studies which can be performed with much less disturbance than the wash-through technique used by Mr. Shields and his colleagues. Our patient agreed to this procedure, knowing that it was

not necessary for her treatment, and was so little upset that we were able to repeat the investigation several times. Where information about net transport, rather than flux, is required (as in this study) there is no need for radioactive isotopes or even venepuncture, and I would encourage Mr. Shields to study this sort of patient in the belief that they can provide information which may not be yielded by more complicated experiments.—I am, etc.,

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REFERENCES

- 1 Wrong, O., and Metcalfe-Gibson, A., *Proc. roy. Soc. Med.*, 1965, 58, 1007.
- 2 Shields, R., Mulholland, A. T., and Elmslie, R. G., *Gut*, 1966, 7, 686.
- 3 Wilson, T. H., 1962, *Intestinal Absorption*. Philadelphia.
- 4 Berliner, R. W., in *Diseases of the Kidney*, 1963, ed. M. B. Strauss and L. G. Welt. Boston.
- 5 Levitan, R., and Ingelfinger, F. J., *J. clin. Invest.*, 1965, 44, 801.
- 6 Levitan, R., *J. Lab. clin. Med.*, 1967, 69, 558.

Lysergic Acid Diethylamide Intoxication

SIR,—The case reported by Dr. R. Clayton (20 January, p. 163), occupying a whole page of your valuable space, purports to describe "the idiosyncratic psychological and physical ways in which L.S.D. intoxication may present." It is unfortunate that the role of other drugs in this case is not discussed, as the evidence incriminating L.S.D. is extremely slender. The patient took about 350 µg. of L.S.D., an amount within the therapeutic range,¹ and a small dose for him, as he had previously taken "1,000 µg. in one dose"; he then took other drugs later in the day and retired to bed 12 hours after ingestion of the L.S.D. He was admitted to hospital unconscious 19 hours after taking the L.S.D.

The effects of L.S.D. are usually of rapid onset and noted within half an hour. A "trip" does not include loss of consciousness, and, after a dose of 350 µg., regardless of tolerance, lasts for no longer than 12 hours.²

The published information regarding the psychological effects of L.S.D. is disturbingly confused. It would be a pity if the effects of intoxication by the drug were to become equally confused.—I am, etc.,

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REFERENCES

- 1 *J. Amer. med. Ass.*, 1967, 202, 141.
- 2 Hoffer, A., *Clin. Pharmacol. Ther.*, 1965, 6, 183.

Alcoholism and Drug Dependence in Doctors and Nurses

SIR,—The proportion of addicts in the medical profession in Great Britain has greatly decreased compared with 40 to 50 years ago.¹⁻³ But even today drug dependence among doctors is by no means uncommon, as suggested in your leading article (9 December, p. 567) and borne out by our experience. Drug dependence among doctors, dental surgeons, pharmacists, nurses, etc., is no doubt to a certain extent attributable to the ease of access. This factor, however, cannot be the explanation for the relatively high proportion of doctors

among alcoholics. In the series of M. F. A'Brook *et al.*⁴—referred to in your leading article—out of 63 "addicts" 24 were dependent on alcohol alone, 30 on (other) drugs, and nine on alcohol and other drugs. Among approximately 290 male alcoholic in-patients treated at Warlingham Park Hospital (1952-7) there were 11 doctors (3.8%), of whom five also took amphetamines or barbiturates or both; one (non-alcoholic) doctor was a morphinist. This relative preponderance among doctors of alcoholics over addicts dependent on other drugs occurred constantly among our more recent dependent doctor patients. Among approximately 640 male alcoholics admitted to St. Bernard's Hospital (1963-7) there were 19 doctors (3.0%), of whom seven also took barbiturates and one glutethimide; two (non-alcoholic) doctors were pethidine addicts. (Incidentally, during this period three dental surgeons and one veterinary surgeon were admitted for alcoholism, and one dental surgeon for barbiturate dependence.) Among approximately 100 alcoholics seen during the past year outside hospital there were five doctors. The impression of a high incidence of alcohol abuse among doctors is supported to some extent by the findings of an above average standard mortality rate from liver cirrhosis among medical (and dental) practitioners (S.M.R. 240 and 250 respectively),⁵ and by the relatively high proportion of drunk-in-charge cases appearing before the General Medical Council.⁶

Your leading article makes a passing reference to nurses. However, the official report quoted, that doctors made up 81% of all known "professional addicts" (in the United Kingdom), "including nurses," refers to Dangerous Drugs Acts addicts only, and in our experience dependence on alcohol and "soft" drugs seems to be no less common among nurses than among doctors. Thus out of 450 women alcoholics admitted to St. Bernard's Hospital (1958-67) 21 (4.7%) were trained nurses and 14 untrained (assistant) nurses; of 113 women dependent on drugs other than alcohol and older than 20 years 14 (including three who also took alcohol to excess) were trained nurses (12%) and 12 (including five who also took alcohol) were "assistant nurses." (The "new wave" of female heroin addicts, etc., aged 20 years and less admitted during the same period—29 patients—did not include any nurses.) The drugs involved were mainly amphetamine and barbiturates, rarely non-barbiturate hypnotics and chlorodyne. Among the 560 women alcoholics and addicts over 20 there were three alcoholic doctors, one drug-dependent doctor, and eight doctors' wives (three alcoholics, five amphetamine and/or barbiturate dependent). Similarly, among women admitted to Spelthorne St. Mary Home (1965-7), of 87 alcoholics 9 (4.4%) were nurses, of 60 addicts older than 20 years 7 (11.7%).

One point referred to in your leading article is the finding repeatedly mentioned in the literature⁴ of the "exceptional difficulties" encountered when treating doctors. However, when treated within the framework of a therapeutic community and therapeutic groups such doctor-patients often found that they had a great deal to give to other patients and to the community, a finding which often proved very helpful to their own self-respect and to the progress of the whole group.⁷ However, in view of the difficulties in treating alcohol- and drug-dependent patients

prophylaxis is all-important, and here doctors and nurses should obviously occupy leading roles. The finding that so many members of the medical and nursing professions themselves become victims of drug-dependence and alcoholism once more illustrates the urgent need for much better coverage of these subjects in the medical and nursing curriculum.—I am, etc.,

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REFERENCES

- Glatt, M. M., Pittman, D. J., Gillespie, D. G., and Hills, D. R., *The Drug Scene in Great Britain*, London, 1967.
- Lewin, L., *Phantastica, Narcotic and Stimulating Drugs*, London, 1964, p. 54.
- Carver, A. E., *Brit. J. Ineb.*, 1934, 31, 73.
- Brook, M. F., Hailstone, J. D., and McLaughlan, I. E. J., *Brit. J. Psychiat.*, 1967, 113, 1013.
- General register office, *Dec. Suppl. Engl. and Wales*, 1951, 1957. *Occup. Mortal. Part II. Tables H.M.S.O.* London.
- Glatt, M. M., in *Alcohol and Road Traffic*, Proc. 3rd Internat. Confer., ed. J. D. J. Havard, B.M.A., London, 1963, p. 99.
- "Lyn" *Brit. med. J.*, 1956, 2, 101.

Psychotropic Drugs

SIR,—Recently I was called urgently to see a young woman complaining of severe headache. She had been on a monoamine oxidase inhibitor drug for a considerable time, and was under consultant psychiatric supervision for a moderately severe depression.

During the previous night she had been unable to sleep as a result of an irritating cough. In scientific parlance, she took 30 ml. of a proprietary children's linctus, freely available over the counter, and thereby ingested a total of 9 mg. of the antihistaminic agent promethazine hydrochloride. Shortly afterwards she suffered the typical pounding occipital headache of the M.A.O.I. adverse reaction.

In lay language, here was a young woman who while taking a drug for her depression at the same time took a moderate dose of a children's cough mixture, as a result of which she could in theory have died from a brain haemorrhage, leaving a motherless young family. I make no apology for the dramatic nature of the last sentence. Fortunately, she recovered fully, and I should also point out that she had been well warned about the usual agents likely to cause this reaction. I would be the first person to agree with Dr. William Sargent (27 January, p. 249) that the antidepressants have a specific action, and are not mere placebos supported by the therapeutic personality of the physician. However, from the above experience, I would suggest two things.

First of all there would appear to be a case for regarding the M.A.O.I. group as potentially very dangerous drugs, to be used solely on consultant psychiatric advice. I would suggest that the vast majority of depressive states met with in general practice can be successfully and safely treated with the tricyclic compounds such as amitriptyline and imipramine.

Next, it should behave all practitioners prescribing the M.A.O.I. drugs to impress upon their patients the need not only to avoid the well-known adverse reactors, such as cheese, broad-beans, and Marmite, but to eschew every drug and proprietary medicine

unless previously approved by their physician.—I am, etc.,

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LOGAN MITCHELL.

SIR,—Psychotherapists should be able to take heart from Dr. William Sargent's letter (27 January, p. 249). The value of their work has often been criticized because they failed to supply statistics convincing enough to support their claims. But now we are told that even the "most refined statistics" can equally prove or disprove the alleged efficacy of a particular therapy. This being so, psychotherapists like pharmacotherapists can argue their case on the strength of bedside experience, and similarly claim gratifying results in selected patients, without bothering too much about statistics.—I am, etc.,

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Diphenhydramine in the Treatment of Oculogyric Crisis

SIR,—The recent interest in your correspondence columns (2 December, p. 554) in the bizarre manifestations of oculogyric crises following phenothiazine therapy prompts us to record the following unusual instance in a child, and its treatment with the antihistamine diphenhydramine.

The patient, a boy aged 2½ years, was seen two hours after the ingestion of about 6 ml. of a film cement containing as a solvent 80% ethylene chlorhydrin and 20% acetone. His stomach was washed, but he continued to vomit and became increasingly drowsy and shocked. In addition to the usual resuscitative therapy amphetamine sulphate 2.5 mg. was administered orally as a stimulant, followed by a similar dose four hours later, together with an intramuscular injection of 1 mg. because of profuse vomiting. There followed an increase in respiratory and pulse rates, and a rise in blood pressure, which were attributed to the effect of amphetamine. Twenty-four hours after ingestion episodes of upward rolling of the eyes with a fixed gaze,

opisthotonos, and gustatory movements in the mouth were noticed. Neurological examination, liver-function tests, serum electrolytes, and blood urea were normal. The attacks were initially thought to be a form of petit mal (there was no previous or family history of this), and two doses of phenobarbitone 15 mg. each were administered at 12-hourly intervals. The patient had a total of six attacks and a few milder ones in the next 24 hours, when it was realized that they were in fact "oculogyric crises." Oral diphenhydramine 10 mg. six-hourly was then started, and no significant attacks followed, other than two mild ones, within the following 24 hours.

No similar neurological disturbance is known to follow amphetamine¹ or ethylene chlorhydrin. Toxic effects of the latter² include central nervous system depression, peripheral circulatory and respiratory failure, and impairment of liver and kidney functions. There was no evidence that the small dose ingested resulted in any residual deleterious effects, but its association with the oculogyric crisis is very strong. This was also the case with the striking improvement following the administration of diphenhydramine.

A similarly dramatic response was witnessed by one of us (J. A.) in a 10-year-old girl with Riley-Day syndrome (familial dysautonomia) who developed oculogyric crisis following a relatively small dose of chlorpromazine. Besides the well-known effect of the solanaceous drugs in the treatment of oculogyric crisis attention is drawn here to diphenhydramine, which is also useful in the treatment of other extrapyramidal manifestations of phenothiazine toxicity.³—We are, etc.,

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REFERENCES

- Wallace, H. E., Neumayer, F., and Gutch, C. F., *Amer. J. Dis. Child.*, 1964, 108, 657.
- Dreisbach, R. H., *Handbook of Poisoning*, 1966, 5th ed. Blackwell Scientific Publications, Oxford.
- Martindale's *Extra Pharmacopoeia*, edited by R. G. Todd, 1967, 25th ed. The Pharmaceutical Press, London.

Gynaecomastia in Renal Failure

SIR,—We quite agree with the conclusions of Dr. R. M. Lindsay and others that "gynaecomastia may occur not uncommonly in male patients with chronic renal failure during the early stages of regular dialysis treatment" (30 December, p. 779).

Gynaecomastia has been documented in eight of our 22 patients receiving maintenance haemodialysis. On occasions, breast enlarge-

ment may be extreme, as shown below. Since reporting this "complication" of haemodialysis to the American College of Physicians in April 1967¹ we have learned of over 25 additional cases. In addition, lactation in women has apparently been noted following haemodialysis.²

We are also unaware of any reports specifically dealing with gynaecomastia in