Simple Oxygen-failure Safety Device

SIR,—I was concerned to note that the oxygen-failure safety device reported by Drs. H. T. Davenport and B. M. Wright (31 August, p. 570) had a serious defect. Any safety device must confer added safety under all conditions of use; otherwise the device becomes an unexpected danger. The designers of this device do not state that if the patient is apnoeic and being ventilated with 100% oxygen, then the whistle will not sound if the supply should fail. The use of almost pure oxygen in apnoeic patients is not at all uncommon either during anaesthesia¹ or at its termination or during therapeutic artificial ventilation of the lungs. The device could then be a danger if the user were unaware of this defect.—I am, etc.,

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Birmingham

¹ Robinson, J. S., *International Anesthesiology Clinics*, 1967, 5, 467

** We showed this letter to Drs. Davenport and Wright, whose reply is printed below.—Ed., B.M.J.

SIR,—Professor Robinson is, of course, quite right. We did point out in our preliminary letter (10 November, p. 357), which is referred to in our paper, that our device will not protect a paralysed patient on pure oxygen, but we should have mentioned it again in our paper and should also have pointed out that an apnoeic patient on a semiclosed circuit is also at risk, as the nitrous oxide flow may be insufficient to operate the alarm whistle. We would like, however, to make four points.

(1) We feel that any anaesthetist who wishes to rely on a safety device should familiarize himself with its properties and suitability for the apparatus and technique that he is using and should check that it will in fact operate satisfactorily.

(2) Our primary object was to protect the spontaneously breathing patient. The fact that it was easily possible under most circumstances to get an alarm signal also, even with an apnoeic patient, was an unexpected bonus.

(3) Prolonged ventilation with pure oxygen is becoming rare because of the increased recognition of its hazards. Its use during termination of anaesthesia is, of course, almost universal, but this is for only a short time and the patient is usually under continuous observation.

(4) We do not think it is reasonable to reject a device just because it will not work under absolutely all conditions, especially when, as in this case, its failure under certain circumstances is not "unexpected" but easily predictable. Nevertheless, we recognize that anaesthetists are human and machines are fallible and it must not be automatically assumed that the use of a safety device, especially an alarm, is necessarily a good thing. The history of the Bosun (Dr. R. Parfitt, 22 September 1973, p. 635; Dr. R. L. Wynne, 13 October 1973, p. 105) shows that the fitting of an alarm can cause changes in the behaviour of anaesthetists (medicolegal report, 4 August 1973, p. 302) and lead to accidents that

might not have occurred if the alarm had not been fitted.—We are, etc.,

H. T. DAVENPORT
B. M. WRIGHT

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Clinical Research Centre,
Harrow, Middlesex

Haemodialysis and Forced Diuresis for Tricyclic Antidepressant Poisoning

SIR,—We were interested by the experience of Dr. R. R. Bailey and others (26 October, p. 230) with haemodialysis in tricyclic antidepressant poisoning. However, their claim that their experience encourages them to "give early consideration to this form of therapy" cannot go unchallenged.

Death from tricyclic antidepressant poisoning is relatively uncommon. There was no evidence that their patient was in immediate danger of death and her clinical course was similar to that of other patients.¹ Their own data show that haemodialysis did not alter the plasma decay curve, so whether any drug was extracted in the dialysate or on the polyvinyl chloride tubing is immaterial. There is therefore no case for haemodialysis.

The authors recommend forced diuresis because a patient who had taken a "massive" overdose excreted the equivalent of 1½ tablets in 23,370 ml of urine. This amount hardly seems sufficient to justify their claims, and in less experienced hands such a diuresis regimen would probably result in pulmonary oedema.—We are, etc.,

N. WRIGHT
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Dudley Road Hospital,
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¹ Matthew, H., and Lawson, A., *The Treatment of Common Acute Poisonings*, p. 67. Edinburgh and London, Churchill Livingstone, 1972.

Diagnostic Test for Multiple Sclerosis

SIR,—Encouraged by our success in confirming lymphocyte sensitization to encephalitogenic factor in both human malignant disease and in multiple sclerosis (M.S.) and other neurological disease (O.N.D.),¹ as described by Field and Caspary² and Caspary and Field,³ we have studied the inhibition by linoleic acid of the response of lymphocytes to antigens. A specific increase in this inhibition in M.S. was reported by Field *et al.*⁴ and simultaneously denied by Foster *et al.*⁵ The latter do not give technical details but we have followed the method of Field *et al.*⁴ in using thyroid antigen in both the macrophage electrophoretic mobility test and the modification introduced by Pritchard *et al.*⁶

In a study of 10 M.S. subjects, 10 patients with O.N.D., and 10 normal subjects in which the observer did not know from which

patients his "scrambled" specimens had been taken we were able to show that the lymphocytes of the first group were very clearly more susceptible to depression of reactivity to thyroid antigen by linoleic acid (0.08 mg/ml) than were those of the second and third groups (see table).

While there was some indication that O.N.D. cells were suppressed by linoleic acid to a greater degree than were those from normal subjects we are aware of deficiencies in our experimental technique, and our present expertise (in a first group of subjects) does not allow of a clear differentiation between these two groups. However, the M.S. patients stand apart from the O.N.D. and normal groups, and the test as carried out in the manner recommended by Field *et al.*⁴ does appear in our hands to be specific for multiple sclerosis.—We are, etc.,

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- 1 Meyer-Rienecker, H., *et al.*, *Klinische Wochenschrift*, 1974, 52, 288.
- 2 Field, E. J., and Caspary, E. A., *Lancet*, 1970, 2, 1337.
- 3 Caspary, E. A., and Field, E. J., *European Neurology*, 1970, 4, 257.
- 4 Field, E. J., Shenton, B. K., and Joyce, G., *British Medical Journal*, 1974, 1, 412.
- 5 Foster, J. B., Mertin, J., and Thomson, A. M., *British Medical Journal*, 1974, 1, 452.
- 6 Pritchard, J. A. V., *et al.*, *Lancet*, 1972, 2, 627.

Caecal Rupture in Psychotic Patients

SIR,—I suspect that this condition is far more often of iatrogenic origin than psychiatric. For example, in Dr. M. McCormack's second case (12 October, p. 82) the patient had a 13-year history of depression requiring treatment. Though the drug history is not given, I am pretty confident that in these days such a patient would not escape prolonged treatment with tricyclic drugs. These do, of course, have often quite profound anticholinergic activity—quite sufficient, in my experience, in a susceptible patient to require admission to hospital for subacute obstruction. In an elderly patient this can quite easily progress to caecal rupture.—I am, etc.,

R. D. LAST

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Psychiatric Manifestations of Giardiasis

SIR,—Your leading article on giardiasis (18 May, p. 347) did not mention that *Giardia lamblia* can cause severe behaviour disorder with E.E.G. changes in children.

Percentage Inhibition of Lymphocyte Response to Thyroid (F1) Antigen in Presence of Linoleic Acid (LA) (Numbers of Subjects Shown in Parentheses).

Group	(A) % Slowing with Thyroid		(B) % Slowing with Thyroid + LA		(C) % Inhibition by LA*	
	Mean ± S.D.	Range	Mean ± S.D.	Range	Mean ± S.D.	Range
Normal (10)	22.34 ± 2.72	18.4 – 26.35	10.61 ± 2.46	7.0 – 14.33	52.84 ± 6.88	44.0 – 63.5
O.N.D. (10)	20.44 ± 3.61	14.8 – 24.92	10.10 ± 1.60	7.72 – 12.75	48.77 ± 10.16	27.8 – 64.5
M.S. (10)	23.07 ± 2.35	17.8 – 26.5	1.98 ± 1.49	0.0 – 3.99	91.69 ± 5.88	84.55 – 100.0

*C = $\frac{A - B}{A} \times 100$ Significance: M.S. v. O.N.D., $P < 0.001$; M.S. v. normal, $P < 0.001$; O.N.D. v. normal, not significant.

In 1969, during an outbreak of giardiasis in a children's psychiatric hospital, six children aged from 2 to 10 years suddenly developed bizarre behaviour after infestation. In each case after treatment with metronidazole and when the stools had become free from cysts the child's behaviour returned to its previous pattern. The E.E.G. in one mentally retarded patient aged 5 while infested with *G. lamblia* showed generalized, non-classical spike and wave complexes suggesting a cerebral lesion, probably at a subcortical level, or a metabolic disorder. At the same time he was withdrawn and irritable, with loose, offensive stools. After eradication of the infestation the E.E.G. became normal and the child became active and took an interest in his surroundings. The E.E.G. of the other five children showed minor abnormalities before infestation and it was not repeated.

Children returning from abroad are often referred for child guidance therapy for behaviour disorders. Giardiasis should be borne in mind in these cases. A stool examination might save months of psychological investigations. *Entamoeba histolytica* is known to cause meningoencephalitis.^{1,2} The infection may be acquired from swimming in fresh water in hot weather, and the infection of the olfactory lobes of the brain together with inflammation and ulceration of the nasal mucosa points to the cribriform plate as the most likely portal of entry.³ Probably a similar chain of events may occur in giardiasis.—I am, etc.,

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Bothwell, Lanarkshire

¹ Culbertson, C. G., *American Journal of Clinical Pathology*, 1961, 35, 1952.

² Symmers, W. St. C., *British Medical Journal*, 1969, 4, 449.

³ *British Medical Journal*, 1970, 1, 581.

Deaths during Dentistry

SIR,—After referring to four deaths that had occurred with operator anaesthetists Dr. P. J. Tomlin (2 November, p. 288) continues: "There may well have been a fifth case involving a 10-year-old boy in Lancashire who died after nitrous oxide, oxygen, and halothane anaesthesia." For the record, the anaesthetic in this case was in fact given by a consultant anaesthetist.

In Dr. Tomlin's final case, that of a 13-year-old boy in Croydon who died during recovery from a brief administration of nitrous oxide, oxygen, and halothane, the anaesthetic was given, as Dr. Tomlin correctly states, by a "separate anaesthetist." This was in fact a medical practitioner who since the war has practised dental anaesthesia to the exclusion of all other work and estimates his experience at about 175,000 administrations. Dr. Tomlin attributes this death to pulmonary oedema. I gave evidence at the inquest and Dr. Tomlin must surely have noted from the details the coroner sent him that the anaesthetist, the pathologist, and I were agreed that the death was almost certainly caused by fainting.—I am, etc.,

J. G. BOURNE

Salisbury, Wilts

Delirium after Surgery

SIR,—We wish to correct some important misconceptions arising from your leading article on this subject (21 September, p. 702).

Most of the data presented on cardiac

surgery were from a paper published by Tufo *et al.* in 1970.¹ The early operative mortality in this series was 15% and, more significantly, abnormal neurological signs were detected in no fewer than 44% of the 85 survivors; 15% of these still had neurological abnormalities at the time of discharge from hospital. No mention was made of Branthwaite's more recent study, which reported an incidence of neurological dysfunction of 19% in 417 patients surviving open heart surgery, of whom only 4% had residual neurological disability on discharge.²

We believe that in 1974 the incidence of postoperative neuropsychiatric complications in most cardiac surgical units has been still further reduced. This is due largely to improvement in perfusion techniques directed towards eliminating microembolism during cardiopulmonary bypass. Our own hospital mortality for 166 open heart operations during the year between 1 September 1973 and 31 August 1974 was 3.6%. Eight patients showed postoperative neurological or psychiatric abnormalities and none of these were permanent.—We are, etc.,

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Papworth Hospital,
Cambridge

¹ Tufo, H. M., Ostfeld, A. M., and Shekelle, R., *Journal of the American Medical Association*, 1970, 212, 1333.

² Branthwaite, M. A., *Thorax*, 1972, 27, 748.

Management of Lithium Treatment

SIR,—Dr. S. P. Tyrer and others (12 October, p. 108) miss two main points of our paper (14 September, p. 650). We reported that when a patient takes Priadel once a day the plasma lithium varies two- to four-fold in the course of 24 hours. We also showed that oral propantheline bromide, known to delay stomach emptying, lowers this plasma swing when taken with the Priadel. Therefore gastrointestinal function to some extent determines plasma lithium level. How tablets release lithium into solutions of different acidity in the laboratory is a secondary matter which may or may not be relevant to the clinical finding.

Our lithium tolerance test is no more than a name for the practice of measuring the plasma lithium at several times on the same day after a routine dose (instead of only once) to see how much diurnal swing in plasma lithium is taking place in the course of treatment. This is quite different from the renal lithium clearance test, mentioned by Dr. Tyrer and his colleagues, which is a set of measurements at the start of treatment so as to calculate the dose needed to establish in that patient any given basal plasma lithium level—on the assumption that renal and gastrointestinal functions remain constant. They also draw attention to their finding that subjects taking slow-release lithium tablets may have 4% or more of the lithium dose in their stools (up to 7 mEq/24 hours). We originally thought this might be so and set out to look for it in a number of patients without, however, finding it, as we noted. I hope they will publish their results in more detail, showing the psychiatric natures of the subjects, their previous medical histories, the periods

during the course of lithium treatment when these positive findings in the stools were obtained, and so on, so that we can judge whether lithium in the stools is a general or a particular phenomenon and why there is this discrepancy between our findings.

In this connexion I should mention that we have so far failed to confirm another finding from Dr. Hullin's laboratory—that patients taken off lithium after a long course of treatment continue to excrete small amounts of lithium in the urine intermittently for months thereafter. In our metabolic ward, long-established and run by two experienced sisters and two experienced night nurses, we thought at first we had confirmed the finding. All our seven patients have single rooms and either excrete in their rooms or in a toilet for which a nurse holds the key. In spite of these arrangements we eventually discovered a patient who was not only incapable or unwilling in keeping her faeces uncontaminated by her urine but who also managed to introduce her lithium-containing urine into the specimens produced by patients off lithium treatment and also into the urine from patients who had never had lithium. Since her exclusion from the ward we have had no further positive findings.

I think, therefore, when reporting lithium in stools or urine it is necessary also to report the metabolic ward conditions under which specimens were obtained. In the end these depend on co-operation from subjects, and some are by personality or illness incapable of co-operating consistently. They also depend on capable, experienced staff, but it is necessary to check that each nursing shift in fact had such a nurse on it. Everybody has days off, holidays, sickness, attendance at hospital meetings which may take them off the ward at crucial times.

Dr. E. H. Bennie (26 October, p. 232) raises the question of what constitutes a toxic plasma level of lithium but fails to consider either how such levels can be discovered or that the duration, and possibly the degree of intermittency, of a particular lithium level is important as well as its height. Most of the advice on toxic levels derives from seriously-ill or fatally toxic cases where it is not known for how many days or weeks beforehand the level has been over 2 mEq/l., and advice is given on the assumption that patients will be seen only infrequently. My experience is that short periods of high lithium levels are not injurious and are permissible when the patient is under very close supervision with laboratory support. However, as our paper tried to show, there is as yet no evidence that such high lithium levels have any therapeutic value.—I am, etc.,

JOHN CRAMMER

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Uterine Contractions due to Heparin

SIR,—We would like to report an unusual side effect of intravenous heparin.

A 32-year-old multigravida in her thirty-second week of pregnancy was admitted to hospital suffering from pain in her right calf muscles of 24 hours duration. Examination revealed deep vein thrombosis of her right leg. She was started on 10,000 IU of heparin intravenously. Twenty minutes later she had severe abdominal pain