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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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.,

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B. M. WRIGHT

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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.,

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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.,

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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.