after 1952; some major factor other than bread consumption must therefore be involved. Neither can the downturn in mortality beginning about 1935-6 be explained by the rise in flour fibre content starting six years later in 1942.—We are, etc.,

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Clofibrate and Fat Embolism

SIR,—We were not surprised to learn from the report of Dr. W. G. Cole (16 October 1971, p. 148) that clofibrate did not significantly improve the morbidity of patients who developed fat emboli. The effect of clofibrate on blood lipids does not reach its maximum in rats until one or two weeks of treatment¹ and apparently even longer in man. Therefore, it would appear the only patients which one would expect to be aided by clofibrate would be those maintained on the drug for its hypolipaeic effect and who subsequently sustained a serious accident. Baker *et al.*² reported a study in animals which is open to the same question because their animals were treated only 24 hours before the production of the fat emboli, and because the drug was given intravenously, a route which could cause clofibrate embolism since the drug is water insoluble. Both these studies conclude that clofibrate is an ineffective drug for treatment of fat emboli.

We have conducted a study which overcomes these objections. Rats were fed clofibrate for three weeks before the production of fat emboli by the method of Baker *et al.*² using both triolein free of fatty acids³ and oleic acid (U.S.P.). Blood, obtained from the tip of the tail, was analysed for cholesterol⁴ and total lipids⁵ to demonstrate that the clofibrate had lowered the blood lipids in the treated group. Cholesterol was 153 ± 32 (S.D.) mg/100 ml in the controls, compared with 85 ± 17 (S.D.) mg/100 ml in the treated group ($P < 0.01$) and total blood lipid levels were 664 ± 135 (S.D.) mg/100 ml in controls compared with 430 ± 79 (S.D.) mg/100 ml in the treated ($P < 0.001$). The animals that died in the first few hours after the intravenous lipid were necropsied within one to two hours after death, and all animals

Lipid Injected	Groups	No. of animals	Cholesterol mg/100 ml Mean $\pm$ S.D.	Total Lipid mg/100 ml Mean $\pm$ S.D.	Pulmonary Surfactant Mean $\pm$ S.D.	Deaths	Haemorrhagic Lungs at 48 hr	Total with Proven Fat Emboli
Triolein	Control	10	161 $\pm$ 35	706 $\pm$ 144	0.570 $\pm$ 0.101	4 (40%)	1 (10%)	50%
	Clofibrate	11	86* $\pm$ 16	436* $\pm$ 86	0.613 $\pm$ 0.139	4 (36%)	1 (9%)	45%
Oleic Acid	Control	7	142 $\pm$ 26	604 $\pm$ 97	0.484 $\pm$ 0.076	2 (28%)	1 (14%)	42%
	Clofibrate	7	84* $\pm$ 19	421* $\pm$ 73	0.567 $\pm$ 0.094	3 (43%)	2 (28%)	71%

*Significantly different at $P < 0.001$ level from the control group.

alive after 48 hours were killed and searched for evidence of grossly haemorrhagic lungs, which was taken as positive evidence of marked fat embolism. Lung biopsies from all animals of both groups were analysed for pulmonary surfactant.⁶ The results of this experiment are as in the above table.

Our results confirm in a more realistic and therapeutic situation the previous findings that clofibrate cannot be expected to improve the morbidity or mortality of patients who develop fat emboli following trauma. We also find that clofibrate treatment does not significantly alter pulmonary surfactant in the rat and that there is no difference of significance in rats sustaining fat emboli.

We wish to thank Mr. Marion Johnson and Dr. J. Noble, Ayerst Laboratories, for the supply of Atomid-S and the National Institutes of Health, U.S.P.H., Bethesda, Maryland, for grants Nos. HE-11782 and HE-05432.

—We are, etc.,

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Wetting and Soiling

SIR,—We would like to comment on some of the points raised by Dr. A. C. Woodmansey (15 July, p. 161).

Firstly, in discussing "reactive incontinence," Dr. Woodmansey makes the common assumption that the reported association of incontinence with psychiatric symptoms is indicative of causation. It is, however, equally in accord with the evidence to regard the association as indicative solely of the expected coincidence of disorders similarly distributed in the population, or to regard the accompanying psychiatric disturbance as in a large part secondary to incontinence.¹ It is certainly not our experience to find a high proportion of emotional disorder in the enuretic or encopretic children who attend for treatment.

Dr. Woodmansey describes the enuresis alarm as "contrived" and "even traumatic," and considers that its aim "ought to be attainable by more appropriate and less dis-

treasing means." We absolutely disagree that the use of an alarm is traumatic or distressing, the number of cases in which the child fears an alarm after proper demonstration being virtually negligible. The vast majority of our patients, and their parents, are only too delighted to be relieved of so distressing a problem as bedwetting by such simple means. Dr. Woodmansey states that "many a child has suffered years of distressing tests and treatments . . . to appease a parent's unrealistic expectation of early sphincter control, or because his . . . wetting or soiling was not recognized as the 'presenting symptom' of his parent's own emotional disorder." We feel, however, that many patients could have been relieved of the constant distress of bedwetting had those they consulted been more ready to use the enuresis alarm.

Postulating that "in many cases incontinence appears to be a more indirect result of a specific fear—namely, that of actually being incontinent," Dr. Woodmansey claims that "the specific treatment of sleep wetting is to emancipate the child from his dread of waking in a wet bed." Unfortunately, there are no data supplied to indicate that such treatment may be comparable in success to treatment by an enuresis alarm, which apparently fails to satisfy the criterion of treatment by "improving relationships." Where "advice" fails, Dr. Woodmansey recommends the use of "psychotherapeutic skill to overcome their [the parents'] anxious doubts, or to alleviate a parental disturbance whose existence the child's symptoms (though undoubtedly important in themselves) have served to bring to light." However, while the success of conditioning treatment has been well demonstrated,² it has yet to be shown that psychotherapy is of significant value in the treatment of enuresis.

From the patient's point of view, rejection of conditioning treatment in consequence of a therapeutic reformulation unsupported by data relating to any more effective form of treatment cannot be acceptable. We strongly recommend the wider adoption of conditioning treatment as the most effective presently available treatment for childhood enuresis.—We are, etc.,

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Puzzling Cases of Secondary Syphilis

SIR,—I was interested in your leading article on "Puzzling Cases of Secondary Syphilis" (8 July, p. 66).

One of the most important diagnostic hall