Fatty Acid	Day 3	Day 7	Four Months Later	Normal Adults (9) (mean ± S.E.M.)
14:0 Myristic	1·3%	*	1·9%	1·5 ± 0·1%
16:0 Palmitic	27·9%	30·0%	26·6%	23·5 ± 0·5%
16:1 Palmitoleic ..	9·7%	11·5%	3·5%	5·3 ± 0·2%
18:0 Stearic	7·5%	8·4%	7·0%	9·5 ± 0·3%
18:1 Oleic	44·0%	38·7%	25·6%	29·3 ± 0·5%
18:2 Linoleic	2·5%	2·0%	19·5%	27·0 ± 0·9%
20:4 Arachidonic ..	1·7%	*	6·5%	Others 3·9 ± 0·4%
Total	3158 mg/l	3262 mg/l	3570 mg/l	Approx. 3000-4500 mg/l

*Insufficient to estimate.

Deficiency symptoms have previously been reported in experimental conditions in human infants fed on a milk formula containing only traces of linoleic acid. The features which occurred included dry, scaly, thickened skin which became red and desquamated; perianal irritation; loose, dark green, watery stools; weight loss; and increased susceptibility to chest infections. Prompt recovery occurred when E.F.A. were restored to the diet. The authors of that paper found average blood values of dienoic acids to be about 5·6% of total fatty acids in deficient infants, and stated that 12·9% ± 2·6% was the minimum level found in infants fed on formulae with acceptable linoleic acid content. They noted that the skin changes were the most characteristic deficiency symptom.[2]

Dermatological features suggestive of E.F.A. deficiency were seen in a girl of 4½ years treated with a low-fat diet for chylous ascites.[3] Apart from the infants used in the dietary experiments referred to above, no other instances of E.F.A. deficiency induced by artificial diet seem to have been recorded in man. However, the condition has been well documented in patients maintained on prolonged intravenous nutrition and deprived of fat, both infants[4 5] and adults,[6] and recently it was reported in three adult patients with malabsorption secondary to intestinal resection.[7]

Considerable interest is currently being shown in the use of the artificial diet in cystic fibrosis. It may be used as a supplement to ordinary food or to supply all the nutritional needs of the child.[1] In the latter event we believe that it is particularly important to emphasize to the parents the need for an additional source of dietary fat. Clinical evidence and animal experiments suggest that infants, because of their rapid growth rate, are particularly at risk and that in the absence of dietary E.F.A. they will develop deficiency symptoms in about two months.[2]

A recent paper speculates that E.F.A. deficiency may be responsible for some of the clinical features in cystic fibrosis,[8] but the above case suggests that when it does occur it produces a characterisic acute illness which should be easily recognized.

J.G.Y. is supported by a research grant from the Cystic Fibrosis Trust.

—We are, etc.,

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Beta-blockade and Myocardial Infarction

SIR,—In reply to the letter from Dr. I. M. Graham and his colleagues (15 March, p. 627) we should like to make the following comments.

(1) They suggest that our study (18 January, p. 117) could not be accurately described as "prospective." We stated in our introduction that the study was prospective with respect to our previous retrospective observations on the behaviour of patients who had previously entered our coronary care unit. A prospective study of the type they suggest—that is, that "patient matching and follow-up should have started before the initiation of beta-blockade"—though desirable, would not in our opinion be either ethical or practical, since the beneficial effects of beta-blockade for stable and unstable angina are now well proved and such a study would mean withholding this form of therapy from half the patients. Furthermore, it would require a large number of patients and would predictably take a long time to complete within one cardiac department.

(2) They question the comparability of our control and beta-blocker groups. In our protocol we stated that both groups had a similar "cardiac history." They were, in fact, comparable in terms of previous angina, myocardial infarction, and incidence of hypertension. We did not record smoking habits. To the best of our knowledge there is no evidence that smoking influences hospital mortality after acute myocardial infarction in coronary care units.

(3) They question the term "myocardial necrosis without infarction" and consider coronary insufficiency as "too 'soft' an endpoint" to be acceptable in the study. In our criteria for selection it was stated that all patients had a three months' history of symptoms of coronary artery disease and were admitted with a prolonged attack of ischaemic pain. The mean duration of chest pain for the two groups was similar (4·75 and 4·5 hours); likewise the mean duration of chest pain of those patients diagnosed as having coronary insufficiency (4·5 hours) was not significantly different from those with myocardial infarction (4·75 hours). It cannot be accepted therefore that coronary insufficiency was "too 'soft' an end point." While we agree that the diagnostic term "acute coronary insufficiency" is frequently used, the definition of this syndrome varies throughout the literature. The term "myocardial necrosis" was used in preference to subendocardial infarction since the E.C.G. criteria for this diagnosis are often uncertain and inaccurate; furthermore, it is possible for patchy microscopic myocardial necrosis to promote elevated myocardial enzyme levels without a characteristic accompanying E.C.G. pattern.

(4) They suggest that our consecutive selection of controls had prejudiced the results of our study. We do not concede this since there were only 122 out of the 251 remaining patients who could have been selected as matching controls. Subsequent analysis of the 32 other possible matching control patients reveals that they behaved in a similar manner to the published control group (see table).

(5) Our statistician has rechecked our results and we apologize for the error pointed out with regard to late cardiac failure. He also discovered that the total incidence of ventricular arrhythmias was not statistically greater in the control group as we had stated. Since we drew no conclusions from these figures they do not influence our final comments. The statistician has confirmed that all other statistical values are correct. The superficially impressive difference in mortality between the control and beta-blockade groups of patients following acute myocardial infarction (presented by Dr. Graham and his colleagues) is not supported statistically ($P > 0·2$). The finding that four of the patients in the beta-blockade group and six of the controls who died had sustained previous myocardial infarcts and that the mean peak SGOT levels of the patients who died were similar and exceedingly high in both groups (423 ± 118 IU for the beta-blocker group and 438 ± 109 IU for the control group) is in accordance with our suggestion "that a myocardial infarction sufficiently extensive to be fatal was not prevented by beta blockade."

We do not, on the basis of our observations alone, advise the routine use of prophylactic beta-blockers but believe that our data warrant us to report that "established

Incidence of Myocardial Infarction, Myocardial Necrosis without Infarction, and Coronary Insufficiency in the 251 Patients not on Beta-blockers Admitted with Prolonged Ischaemic Myocardial Pain. Results are Numbers of Patients

Group	Myocardial Infarction	Myocardial Necrosis without Infarction	Coronary Insufficiency	Total
Published controls	62	14	14	90
Patients without past history of three months' coronary artery disease	94	15	20	129
Outstanding possible controls with suitable past cardiac history	20	7	5	32
Total	176	36	39	251

beta blockade . . . in no way prejudices the outcome of patients admitted to hospital with prolonged ischaemic pain. On the contrary, it may protect some patients from the development of a myocardial infarction and so enhance their long-term prognosis."—We are, etc.,

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Battered Babies

SIR,—Throughout the country committees are being set up (and perhaps a central index) to investigate parents who may be harming their children. Recent reports by the National Society for the Prevention of Cruelty to Children and others show the alarming situation that may exist in this respect. These committees may well be not only helpful but essential, but they pose problems of principle that need to be ventilated and agreed.

They are self-appointed committees (composed basically of local doctors and health officials with power to co-opt the police and N.S.P.C.C.) with no authority from Parliament. No one doubts their good intentions; the members, however, have no special training in these delicate matters and it could be argued that these investigations are better left to the police, who have such training and know their responsibilities and their position in common law. These committees work in secret and the parents need not know they are under investigation (though eventually the parents and the neighbourhood are likely to find out—to the distress of both if the suspicions are unfounded—and can they claim redress?). Anybody can be reported to this committee by any person, be he an official, a doctor, or a neighbour. The potential for excessive keenness in investigation is alarming, and there is always a remote risk that such investigation could have a mischievous background.

If this principle is accepted, however, it could expand to other groups. Why not similar committees to investigate wife-battering, drivers with suspected drink or drug problems, officials in power with suspected psychotic disorders, etc.? Furthermore, what is the ethical position of the doctor to whom parents have come for help when he refers their case to this committee without their knowledge? I think we all accept that there will be cases in which the child clearly takes priority, but there are many borderline cases which are less easy to resolve. We could reach the stage where any mother seen pulling an unwilling child out shopping could find her name, unknown to her, on a computerized index of potential batterers.—I am, etc.,

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Impaired Colour Vision in Diagnosis of Digitalis Intoxication

SIR,—I am indebted to Mr. H. P. Williams and Miss Janet Silver for their kind letter (22 March, p. 682), but I should not like them to remain in the belief that the Pickford anomaloscope is a research instrument only. Far from it; it has been employed for a routine colour clinic for schoolchildren in this unit since 1965 and has also been operated by two orthoptists in examining over 1500 schoolchildren in Kilmarnock and Ayr. Naturally it, like all instruments, requires a short period of training to familiarize oneself with the technique and it is necessary to establish a set of norms on a random population, but after this is done it is quite within the competence of a trained technician. I feel that every major eye unit should operate a colour clinic, not only for the diagnosis of congenital defects but in acquired diseases such as the subject of this correspondence.—I am, etc.,

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Better Medical Writing

SIR,—In the bibliography to your leading article (12 April, p. 56) you quote six books by medical or scientific authors on writing but none by non-medical authors. Is Fowler of no account? Or Partridge? Or Gowers? Do you really think that, outside the small range of literary matters peculiar to their disciplines, doctors and scientists know better than these giants how to teach people to write good English—in so far as it ever can be taught?

A much more important comment, however, concerns your discussion of how medical writing might be improved: your recipe boils down to "teaching postgraduates the elements of clear writing." You surely cannot believe such an attempt would achieve very much. Even undergraduate medical students, who in your dispensation would not—because of "the already overloaded curriculum"—be given any guidance at all on how to write, are made or broken English-wise long before they enter medical school. Why are they? And with what is the undergraduate curriculum overloaded? You give the answers yourself in your earlier leading article "Open Minds for Open Medicine" (12 April, p. 54): the overloading is to make them competent for "the inefficiency of much expensive health care" and is a sign of the "stagnation in medical educational methods." And they are already broken English-wise because of "the ever-increasing demands for higher A-level grades"—which prepare and condition them, of course, for the undergraduate overloading. Schoolchildren who have to concentrate exclusively on high science/maths A-level grades have no time to read, say, Miss Muriel Spark or Mr. Ted Hughes, or Tolkien, T. S. Eliot, or Ivy Compton-Burnett, let alone Swift or Sir Thomas Browne; and there now you have a septet that *might* teach the receptive young how, among much else, to write good modern English, of which the medical sort is but a tiny derivative, and to learn it by the best possible means, which is through the pores—in from much reading, and out as the sweat of much writing—and not from any pedagogic "core" course, to use the current jargon, inflicted in early middle-age.

There was once a tradition that doctors were men of culture (and that they were the better doctors for it); and in particular were somewhat acquainted with the classics and with the correct, pleasing, and effective use of their own language. It is now, of course, in good part gone. We live in a world consisting mainly, not of two cultures, but of two second-rate barbarisms—one of ever-narrower technologists, commonly quite lacking culture, and the other of meretricious entertainers or "educators" who are very often ignorant of the technologies that enable them to spread their small messages and often not wholly at ease in culture proper. But I do not think one should connive at its extension by encouraging doctors to sink ever deeper into their particular acultural quagmire. Or do you not now have a sinking feeling?—I am, etc.,

JOHN S. BRADSHAW

How Caple, Hereford

SIR,—It is an unhappy coincidence that the very issue of the *B.M.J.* which contained your leading article on "Better Medical Writing" (12 April, p. 56) should also have had a leading article (p. 52) which began: "Recent neurophysiological and hormonal research is supporting the concept that primary anorexia nervosa reflects a psychological avoidance-response to the maturational demands of adolescence." As near as I can make out, this means that these patients wish to avoid the problem of growing up. Physician heal thyself!—I am, etc.,

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Drug Combinations for Anaesthesia

SIR,—As Dr. J. P. Alexander (15 March, p. 626) has coupled my name with the use of intravenous methohexitone and intravenous diazepam for anaesthesia in patients requiring endotracheal intubation I wish to make it quite clear that the method which he describes and which he used in his experiments differs in important respects from the method I have used successfully in over 500 cases.

All my patients are given an oxygen-enriched mixture by a mask applied as soon as the patient becomes unconscious during the injection of methohexitone. The patient then saturates his blood with oxygen while he is still breathing and before the addition of diazepam. Intubation is usually carried out while the methohexitone effect is at its peak. Difficulties are liable to arise if this moment is lost and the methohexitone has to be replaced, some minutes later, by halothane anaesthesia.

My original comments (9 November 1974, p. 345) were made because my personal experience over the past few years suggests that the combination of these two drugs can be of great value and is no more lethal, though probably not less lethal, than any other combination of similar drugs. The efficiency of methohexitone, with the dose suitably adjusted to suit the patient and gain the end in view, plus not more than 10 mg of diazepam, can be gauged by the fact that only once in over 500 cases was it necessary to use suxamethonium as well to obtain conditions necessary for atraumatic intubation. If conditions similar to those produced by suxamethonium are deliberately sought it follows that the same stringent precautions in regard to adequate oxygenation also