from the figure that the rise in the inhibitory activity of the whole serum is the result of increased linoleic acid-like activity, the inhibitory activity of the protein fraction remaining unaffected.

Clearly, in applying the analytical methods of a modern biochemical approach, it is important to realize the frequently multifactorial origin of an end effect and assess or attempt to assess individual influences. In this case the activities of PUFA and linoleic acid-like activity appear disjunct, but this may not always be so.

Heavy animal fat meal at beginning of experiment. Serum separated into ether-soluble (linoleic acid-containing) and ether-soluble (L.D.F.-containing) fractions. 1 in 60 whole serum or fractions added to the in vitro test system in the M.E.M. test. The ordinate shows the percentage reduction in lymphocyte-antigen interaction. The rise in effect of whole serum is entirely due to the linoleic acid-like activity in the ether-soluble fraction, the L.D.F. fraction remaining unchanged.

The illustrative figure is part of a study designed to evaluate the effect which saturated fat may have on the absorption of a dose of linoleic acid. At zero time in the figure a heavy animal fat meal was taken followed immediately by the unsaturated acid. For many years it has been urged that cutting down animal fat intake may have a beneficial effect upon the course of multiple sclerosis.⁷ Clearly, before embarking upon a prolonged double-blind study of the effect of linoleic acid supplementation of the diet in multiple sclerosis (where it has been reported to be beneficial,⁸) it is important to know whether reduction in the ingestion of saturated fats might facilitate the development and maintenance of high linoleic acid-like activity in serum. Our first results do in fact show that the presence of animal fats does delay and diminish the plateau of linoleic acid in blood.—We are, etc.,

E. J. FIELD
B. K. SHENTON

Institute of Pathology,
Newcastle General Hospital,
Newcastle upon Tyne

- Field, E. J., and Caspary, E. A., *Lancet*, 1971, 2, 95.
- Cooperband, S. R., Bondevik, H., Schmid, K., and Mannick, J. A., *Science*, 1968, 159, 1243.
- Ford, W. H., Caspary, E. A., and Shenton, B. K., *Clinical and Experimental Immunology*, 1973, 15, 169.
- Field, E. J., and Caspary, E. A., *British Medical Journal*, 1971, 4, 529.
- Field, E. J., and Caspary, E. A., *British Journal of Cancer*, 1972, 26, 164.
- Caspary, E. A., and Field, E. J., *British Medical Journal*, 1971, 2, 613.
- Swank, R. L., *American Journal of the Medical Sciences*, 1950, 220, 421.
- Millar, J. H. D., et al., *British Medical Journal*, 1973, 1, 765.

Return of the Foster-child

SIR,—Like a large number of medical men I have been distressed to read the recurring accounts of children weeping bitterly when taken from foster-parents and returned to parents they have often not seen for years. This has culminated in the tragic case of Maria Colwell and no doubt there are many similar ones which have not been given publicity.

It seems to me that this is a matter which cannot be properly investigated by social workers, who have so often failed in the past. The judges and magistrates are often ignorant of psychiatry and paediatrics so they cannot have a proper understanding of the traumatic results of tearing a child away from someone it has learned to love and putting it into the care of those it considers strangers—even if they are close blood-relations. Surely this matter rests to some extent on the medical profession and we should make ourselves heard regarding it. Might I suggest that we should state loudly and clearly that children should not be separated from those they love unless it has been shown without doubt that they are in some sort of moral or psychiatric danger, and if separation is considered necessary by the courts their decision should be confirmed by psychiatric opinion.

Those of us who have practised psychiatry are only too well aware of the neurosis, criminality, and psychological instability which occur later on as the result of such cases.—I am, etc.,

CLIFFORD ALLEN

Mold, Flintshire

Deliberate Injury of Children

SIR,—Your leading article on deliberate abuse of children (13 October, p. 61) draws attention only to the effects of physical battering, which are for the most part visible. It makes no mention of the other more subtle and sinister form of battering—namely, psychological abuse.

This form of injury is just as real and probably just as prevalent as its physical counterpart. Some parents, who cannot vent their feelings in physical violence, can often execute a vicious campaign of psychological attack performed in the name of discipline, good behaviour, or proper upbringing. We find it hard enough to convince ourselves that a child is being physically abused, though our standards of physical behaviour towards children have evolved considerably in the past century. We are much less expert at spotting a situation in which the emotional growth of the child is being wilfully distorted. After all, who are we to decide what kind of behaviour is appropriate for our children? Where does deliberate neglect become distinct from ignorance? When has an authoritarian upbringing given way to primitive excesses?

For the children with physical abuse we can at least mobilize the social agencies and call on the law when necessary. It is far more difficult to be persuaded in cases of psychological abuse; the idea of this type of battering is so novel that we still seem to demand tangible evidence before being able to take legal action. And yet the consequences of this kind of wilful abuse may be longer lasting than some of those associated with

physical damage. The emotional turmoil produced by a vindictive parent may persist into adult life long after the trauma has occurred.

This letter is a plea that we become aware of this problem and that we try to explore the psychological aspects of child abuse in order to develop a better medical, social, and legal system for handling such cases.—I am, etc.,

NEIL R. M. BUIST

Department of Pediatrics,
University of Oregon Medical School,
Portland, Oregon

I.Q. of Parents of Battered Babies

SIR,—In a recent report by Dr. Selwyn M. Smith and others on "Parents of Battered Babies: A Controlled Study" (17 November, p. 388) they report that nearly half of the mothers were "of borderline subnormality or below." They base this claim on the averaged scores of four of the subtests from the Wechsler Adult Intelligence Scale (W.A.I.S.). I would suggest that this is not a permissible way to use the subtest scores. Either the subtests maximizing factor differences should be used or a full-scale test carried out in the usual way.

The failure to distinguish between verbal and non-verbal abilities has bedevilled even such little research of this kind as has been carried out into the intellectual functioning of battering parents. It prolongs the myth that simple stupidity explains their behaviour. In a recent study (to be published), in which a short form of the W.A.I.S. was used, I found no differences in the battering and control parents' spatial-perceptual abilities. It was in their ability to use verbal concepts that differences emerged. This verbal inability was not, however, nearly as gross as that reported by Dr. Smith and his colleagues and appears to be part of a consistent personality pattern in the batterers reflecting withdrawal, depression, and non-communicativeness. This was borne out by their scores on Cattell's 16 PF test.

While my findings on the mothers' youth and psychological immaturity confirm those of Dr. Smith and his colleagues, I believe that it would be mistaken to take their findings on global I.Q. at face value, based as they are on a statistical misunderstanding of the test construct validity.—I am, etc.,

CLARE A. HYMAN

Consultant Psychologist,
N.S.P.C.C. Battered Child Research Department

Department of Biological Sciences,
University of Surrey,
Guildford

Glucagon and Duodenal Ulcer

SIR,—Your leading article on "Glucagon Therapy in Acute Pancreatitis" (1 December, p. 503) reflects the increasing interest in the use of pancreatic glucagon among clinicians, and the encouraging reduction of mortality reported by Condon *et al.*,¹ albeit in a small and uncontrolled study, will no doubt add to this interest. It is most important that further clinical assessment of this promising treatment should be undertaken, but it may be salutary to bear in mind that the actions of glucagon are multiple and could lead to undesirable complications in some situations. In particular its powerful action as a

duodenal relaxant may increase the incidence of duodenal ileus after pancreatitis.

As stated in your article the mortality in acute pancreatitis is approximately 25%. Twelve per cent of patients admitted with acute pancreatitis will develop one of the major complications, and when this occurs the mortality may rise to over 50%. Trapnell² described 70 patients who developed a major complication. Ten of these had prolonged duodenal ileus, four of them dying.

We have recently treated a 67-year-old man who was admitted to hospital with a pancreatic pseudocyst and copious vomiting two weeks after a severe attack of pancreatitis. He had an epigastric mass and signs of spreading peritonitis and was markedly alkalotic. He continued to deteriorate despite all efforts with conservative treatment, and laparotomy, drainage, and cystogastrostomy were performed. Postoperatively, treatment with glucagon was begun and was continued for seven days. Very high gastric aspirates consistent with duodenal ileus persisted for five days and began to improve only with metoclopramide treatment and after a Gastrografin swallow, which showed some contrast medium reaching the proximal jejunum. Unfortunately he developed further complications and died 10 days after admission. At postmortem the pancreas was completely necrotic. There was extensive abscess formation extending to the right subphrenic space, massive pulmonary embolism, and terminal haemorrhage into the stomach in the absence of demonstrable ulceration. The gall bladder and biliary tree were normal.

It is likely that the role of glucagon will prove to be in reducing the severity of the initial attack of pancreatitis. This should lead to a reduction in the incidence of major complications and therefore further reduce the overall mortality. Duodenal ileus is believed by Trapnell to be a consequence of continuing pancreatic destruction rather than a mechanical obstruction by a swollen and oedematous pancreatic head. If this is so then glucagon may reduce the frequency and severity of continuing pancreatic destruction and consequently of duodenal ileus. In the presence of established duodenal ileus, however, the relaxation of duodenal musculature that glucagon causes may worsen an already dangerous situation.—I am, etc.,

C. E. NEWMAN

St. Chad's Hospital,
Birmingham

- 1 Condon, J. R., Knight, M., and Day, J. L., *British Journal of Surgery*, 1973, **60**, 509.
- 2 Trapnell, J. E., *Annals of the Royal College of Surgeons of England*, 1971, **49**, 361.

Blood Carboxyhaemoglobin Levels in Smokers

SIR,—In view of the current interest in the carbon monoxide content of cigarette smoke,^{1,2} the following results appear to be relevant. We have measured the rise in carboxyhaemoglobin (COHb) in smokers smoking a single low-, intermediate-, or high-nicotine cigarette. The cigarettes used were of familiar size, weight, and density and contained the same blends of tobacco. However, the filter efficiency for nicotine differed in such a way that if the cigarettes were smoked in a standard manner in a smoking machine the mainstream smoke contained 1, 2.1, and 2.4 mg of nicotine in the low-, intermediate-, and high-nicotine cigarettes respectively, but the carbon monoxide yield did not differ. The smokers smoked in their normal manner at rest and all except two

Blood COHb level, Puffing Rate, and Nicotine delivered to Smoker in Subjects smoking low-, high and intermediate-nicotine Cigarettes

	Low-nicotine			Intermediate-nicotine			High-nicotine			Analysis of Variance	
	Mean	S.D.	n	Mean	S.D.	n	Mean	S.D.	n	F	Significance
COHb %											
Before smoking	2.3	1.1	10	2.6	1.5	8	1.9	1.0	10		
After smoking	4.8*†	0.8	10	3.8*	2.0	8	3.1†	1.5	10		
Difference	2.5*†	1.1	10	1.2*	0.7	8	1.1†	1.0	10	3.9710	P<0.05
Puffs/min	1.8	0.7	10	2.2	1.2	8	1.6	0.6	9	1.1547	n.s.
Nicotine delivered to smoker (mg)	1.2†	0.3	12	1.3‡	0.5	8	1.8†‡	0.4	9	5.7538	P<0.01

Symbols refer to significant differences between types of cigarettes: * = low-v. intermediate-nicotine; † = low-v. high-nicotine; ‡ = high-v. intermediate-nicotine (t tests; P<0.01 in all cases).

of the cigarettes (both intermediate-nicotine) were smoked as the first cigarette of the day. COHb was measured from finger-prick samples before and immediately after smoking by the method of Commings and Lawther.³ The puffing rate and the amount of nicotine delivered to the smoker from each cigarette were also estimated.

The rise in COHb during smoking was greatest for the low- and least for the high-nicotine cigarettes (see table). This difference was accompanied by a tendency towards a lower puffing rate for the high-nicotine cigarettes, though more nicotine was in fact delivered. Thus the subjects were able to obtain a relatively high dose of nicotine with a smaller increase in COHb from the high- as compared with the low-nicotine cigarettes. The intermediate-nicotine cigarettes occupied an intermediate position. These findings appear to be in general agreement with those of the longer-term study by Russell *et al.*,² in which it was shown that cigarette consumption and COHb rise diminished in subjects changing from their usual brand to high-nicotine cigarettes over five-hour periods. In Russell's study, unlike the present one, the carbon monoxide yield of the different types of cigarette differed owing to differences in size and weight of the cigarettes. Nevertheless, our findings agree with the suggestion that a "safer" cigarette might be one which combined a low yield of carbon monoxide and tar with a relatively high yield of nicotine.

We have also followed some short-term changes in the blood level of COHb in a few subjects. The rise while smoking a single cigarette of the subject's own brand in four smokers with different initial levels of COHb is shown in fig. 1. The rate of rise

FIG. 1—Changes in blood COHb levels in four subjects while smoking one cigarette.

was greatest at the start of the cigarette followed by a levelling off or even a fall of COHb towards the end. This plateau effect may be related to the fact that smokers tend to take fewer puffs towards the end of a cigarette than at the beginning, possibly because the nicotine in the stub becomes more concentrated as the cigarette burns down. The fall in COHb levels immediately after smoking in six smokers at rest (one smoking a small cigar) is shown in fig. 2.

FIG. 2—Change in blood COHb level in six smokers after smoking. Subject B smoked (and inhaled) a small cigar; the other subjects smoked a cigarette. (O = blood samples taken from antecubital vein; all other blood samples taken by finger prick).

In all subjects there was a relatively rapid fall (1-2.5%) of COHb in the first five minutes after smoking, followed by a levelling off to a very slow rate of decline, about 0.5% between five and 25 minutes. The cause of the initial sharp fall of COHb in the first five minutes is not clear. It does not appear to be due to an artefact due to unequal distribution of carbon monoxide in the circulation, since in one subject blood samples taken simultaneously from the right antecubital vein and the left middle finger tip gave identical results. Possibly binding with other molecules such as myoglobin is involved. Extrapolation of the curves in fig. 2 gives a half life for COHb of approximately 2-2½ hours, agreeing with the findings of Russell *et al.*²—We are, etc.,

HEATHER ASHTON
ROSEMARY TELFORD

Clinical Pharmacology Unit,
The University,
Newcastle upon Tyne

- 1 Russell, M. A. H., Wilson, C., Cole, P. V., Idle, M., and Feyerabend, C., *Lancet*, 1973, **2**, 687.
- 2 Russell, M. A. H., Wilson, C., Patel, U. A., Cole, P. V., and Feyerabend, C., *British Medical Journal*, 1973, **4**, 512.
- 3 Commings, B. T., and Lawther, P. J., *British Journal of Industrial Medicine*, 1965, **22**, 139.