Fainting during General Anaesthesia in Supine Dental Patients

SIR,—I have repeatedly suggested[1-3] that though an emotional faint can occur in the horizontal position fainting would not be a hazard to life if dental patients were anaesthetized lying down. There are, however, disturbing reports suggesting that even in this position should fainting happen to coincide with the administration of an intravenous agent such as methohexitone or diazepam (Valium) there may be a collapse so severe as to threaten life. In one reported case,[4] for example, a boy aged 11 given only 20 mg of methohexitone instantly collapsed, was pulseless, and stopped breathing. And a similar collapse was reported[5] in a youth given 7·5 mg of diazepam. His life was feared for at first and it was 15 minutes before consciousness returned. I have received reports of other dental cases in which there was a similar and most alarming collapse in the horizontal position with intravenous agents.

In the cases so far reported the collapse has been treated with the patient remaining horizontal. Sharpey-Schafer suggested[6] that an emotional faint resulted from the pooling of blood in the splanchnic region. If this is so the logical treatment would be to place the entire patient in a steepish head-down tilt, which should improve the return of blood to the heart and so restore the blood pressure.—I am, etc.,

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2 Bourne, J. G., *Anaesthesia*, 1970, **25**, 473.
3 Bourne, J. G., *Lancet*, 1973, **1**, 35.
4 Shafto, C. E., *British Journal of Anaesthesia*, 1969, **41**, 407.
5 Verrill, P. J., *British Medical Journal*, 1970, **4**, 348.
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Who is the Dental Anaesthetist of the Future?

SIR,—No one can deny that a fully anaesthetized patient needs the services of a separate person specially devoted to safeguarding his welfare, but does this per se make anaesthesia safer? Deaths have occurred with an operator/anaesthetist in action and also with a separate anaesthetist of all three groups—dentist, general medical practitioner, and specialist anaesthetist. A statistical analysis to prove whether one group has more (or fewer) deaths than the others would serve no purpose other than to arouse emotional reaction. So long as man remains imperfect deaths under anaesthesia will continue to occur.

Surely the rational approach towards prevention of mishaps is to give more training both at undergraduate and postgraduate levels. The constructive plan of Professor P. A. Bramley and his colleagues (4 May, p. 270) seems to have fallen on stony ground. I would have expected a massive response in your columns from teaching institutions throughout the country, but a great silence so far is the only outcome.—I am, etc.,

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Biofeedback

SIR,—While I must commend the scientific caution with which your leading article on biofeedback (17 August, p. 427) approaches its subject, I would suggest not only that you failed to give proper recognition to the range and extent of the present literature on the subject in both experimental and clinical psychology but also that you did not inform your readers of the rigorous and careful experimental design which characterizes studies in this new and important field of behaviour science.

As you rightly state, it is highly dangerous to extrapolate from experimental work with curarized rats to a human clinical population. Psychologists have long experience of these inherent dangers and have already shown that it may not be possible to generalize from one particular strain of rats to another[1] let alone from one species to another. Yet generalization has to be attempted if experience gained in the psychological laboratory is to be of benefit to human populations. It can reasonably be argued that undue caution on the part of traditional elements in psychology and in medicine delayed the application of the laboratory-based principles of learning theory to psychiatric disorders in the form now recognized as behaviour therapy.

The question whether control of autonomic functions by conditioning is direct or is mediated is one which experimental psychologists such as Miller have obviously been concerned with from the very onset of biofeedback research, hence his use of curarization.[2] The evidence to date indicates that control is almost certainly direct rather than mediated, and indeed some studies go further by providing evidence of the probable physiological mechanisms underlying such control.[3]

When using any laboratory-based techniques with a human population it must always be considered to what extent the results obtained are attributable to the technique and to what extent they are due to placebo effects, anticipatory attitudes, expectation, and instruction.[4] All too little attention has often been paid to these important human factors in medical research and, in particular, in drug studies. Considerable attention has, however, usually been paid to them in psychological research, including biofeedback.[5-7] As with any clinical method, such factors are likely to play a part but there is certainly no indication that the results of biofeedback can largely be explained in terms of them. In my own research I was able to show a mean heart rate increase of 23 b.p.m. in five experimental subjects while five control subjects showed no change over baseline heart rates. Each group received exactly the same instruction and the same amount of reinforcement. The only difference between the two groups was that the control group received visual pseudobiofeedback (that is, feedback was derived from the subject's yoked control rather than from his own physiological responses).

The first paragraph of your leading article seems to imply a dichotomy between clinicians and psychologists. In reality, of course, there are psychologists who are primarily experimentalists and others who are primarily clinicians. The latter have been interested in the clinical use of biofeedback for a number of years already.[8] While scientific caution is always commendable, I would suggest that biofeedback is now established and founded on adequate experimental evidence from both animal and human research to the point that caution should not be permitted to hold back the benefits to patients that can be obtained from its clinical use now.—I am, etc.,

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1 Broadhurst, P. L., and Levine, S., *British Journal of Psychology*, 1963, **54**, 121.
2 Miller, N. E., and Dicara, L., *Journal of Comparative and Physiological Psychology*, 1967, **63**, 12.
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Care for the Mentally Handicapped

SIR,—The addresses of Professors A. D. B. Clarke and J. Tizard at the B.M.A.'s Annual Scientific Meeting (27 July, p. 240) again highlight the profession's own confusion on the subject of its place in this field. Were the professors concerned with the efforts of psychology, sociology, education, or medicine, or did they assume, as so many do, that all have much the same contribution to make? Unfortunately this confusion has tarnished our image.

The fact that intelligence (however defined), and consequently its relative lack, is a sociological phenomenon and that a subnormal population (however defined) is homogeneous only in terms of intelligence renders such information almost totally irrelevant in terms of physical or psychological treatment needs. Subnormality of itself is not and never has been a medical problem. The involvement of medicine generally and psychiatry particularly in the field of subnormality is precisely that of the paediatrician who is not primarily concerned with the phenomenon of childhood as such but with the varieties of disease which accompany it. The psychiatrist (and the paediatrician) is involved in subnormality only to the extent which disease or pathology accompany it. Intelligence, like childhood, is important only in so far as it effects communication and the expression of disease and as it affects the choice of or the response to treatment, or both. Lack of intelligence of itself is untreatable in medicopsychiatric terms.

Dependence attributable to intellectual deficit alone is the concern of the sociopolitical agencies but not of medicine. Dependence attributable to primary or intercurrent physical or psychiatric pathology is the concern of the medical profession, since much of it is treatable. Once it is realized that most pathology occurs in the context of subnormality and is not causally related to it the medical profession can do much to lighten the burden of dependence.

The recognition of disease in the subnormal is complicated by the dual difficulties of communication and expression. As the intellectual deficit increases words become less valuable as communicators and are replaced by a variety of non-verbal motor behaviours, which may function as communicators but often merely as expressors highlighting the problems of failure of communication

as a cause, a correlate, or a consequence of mental illness. The difficulties inherent in this situation, the therapeutic nihilism which can be induced by it, and the futility of therapeutic effort directed towards intelligence rather than pathology have led to an almost total devaluation of the therapeutic processes. This has led to replacement of treatment by institutionalization and custodial care. Because these are social rather than medical or psychiatric techniques they have failed in the hands of doctors and nurses whose roles in subnormality have become undervalued by their own professions and by others. As a result, both physical and psychiatric illness has gone unidentified, therefore untreated, and the symptomatology attributed directly to the intellectual deficit.

Thus as a profession we must hasten to clarify our role in subnormality—that is, the identification and treatment of the physical and psychiatric illnesses and disturbances commonly accompanying intellectual deficit. Intelligence must be removed rapidly from its predominant position relative to treatment and seen for what it is—merely one of many factors influencing variably the response to and the choice of a spectrum of therapeutic techniques now available. We must also learn to identify pathology expressed and communicated atypically and at apparently inappropriate developmental levels in order to treat it vigorously and individually, so casting off the mantles of institutionalization and custodial care, for they have restricted our therapeutic sensibilities and smothered our skills. This task completed the profession will be able to evaluate its own effort in subnormality directed towards treatment modified by the requirements of the individual, whatever his handicap. As a result it will recognize that the effort on behalf of the intellectually handicapped individual, as distinct from "the subnormal," is as worthwhile to those individuals as it is to their intellectually normal fellows. Gratitude and pleasure (in Suttie's sense) do not cease suddenly at an I.Q. of 70.—I am, etc.,

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Tests of Hearing in School

SIR,—Dr. L. Fisch (10 August, p. 409) has perhaps missed the point of my original article.[1] I agree with him that the sweep test could have been set at 20 decibels but, as he states, there is wide variation throughout the country in the methods employed. The suggestion that impedance audiometry might replace sweep testing is based on the assumption that there were more adequate preschool screening services at all ages from birth up to school entry. I realize that this assumption may not be correct for certain parts of the country, and difficulties may be experienced in implementing full-scale audiometric services until there is a reasonably captive population at the age of five. It is assumed that the sensorineural defects would be detected before school entrance. Unfortunately there is still a dichotomy in thinking between the preschool service and the school service, which it is hoped will disappear.

However, assuming that the preschool service is of high quality, I think that there is a case for examining whether impedance audiometry ought to be given more prominence at school entry. I cannot agree that the time needed for 40-60 pupils using impedance audiometry would be so long as 10 hours. This has certainly not been our finding, and Denzil Brooks[2] is of the opinion that less skill is needed for the impedance audiometry test than for sweep testing, provided there is an adequate means of reporting.—I am, etc.,

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1 Ferrer, H. P., *Public Health*, 1974, **88**, 153.
2 Brooks, D. N., *Hearing*, 1971, **26**, 250.

Nephrotic Syndrome with Oat-cell Carcinoma

SIR,—I read with interest the paper by Dr. M. R. Higgins and others (17 August, p. 450) describing a case of nephrotic syndrome with oat-cell carcinoma. Their findings of large quantities of extracellular DNA in necrotic tumour tissue and Feulgen-positive deposits within glomerular basement membrane are not surprising. Haematoxyphil change around blood vessels (fig. 1) in oat-cell carcinoma has been shown to be due to the deposition of Feulgen-positive material.[1] Similar vascular change has been described in retinoblastomas and a possible immune response suggested.[2]

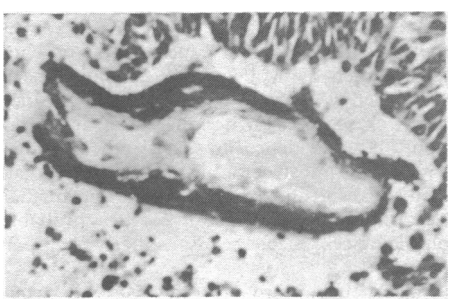

FIG. 1—Haematoxyphil sheath around a vessel and tumour necrosis. (× 209)

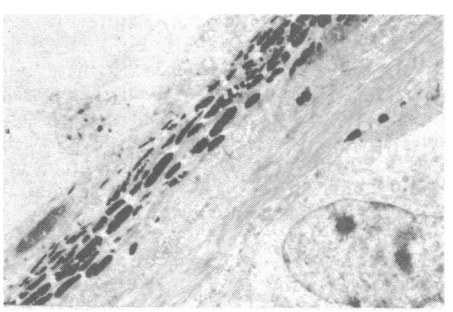

FIG. 2—Electron-dense fragments in the subendothelial region. (× 3,190)

I have recenly described the ultrastructural appearances of such vessels in an oat-cell carcinoma.[3] The deposits consisted of fragments of nuclear material trapped in the subendothelial region (fig. 2). Similar nuclear fragments were present in necrotic areas of the tumour. An interesting finding was the presence of fine fibrillar material resembling antigen-antibody complexes surrounding some of the nuclear fragments. This feature leads me to suggest the possibility of an immune process existing in some patients with oat-cell carcinoma. A case of nephrotic syndrome with oat-cell carcinoma appears to support this suggestion. The estimation of anti-DNA antibodies in oat-cell carcinoma patients should be of interest.—I am, etc.,

A. AHMED

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1 Azzopardi, J. G., *Journal of Pathology and Bacteriology*, 1959, **78**, 513.
2 Mullaney, J., *Lancet*, 1968, **2**, 918.
3 Ahmed, A., *Journal of Pathology*, 1974, **112**, 1.

SIR,—The medical memorandum by Dr. M. R. Higgins and others (17 August, p. 450), reporting a case of nephrotic syndrome with oat-cell carcinoma, suggests the possibility of antibody formation against nuclear antigen, possibly DNA, released by tumour necrosis.

I showed[1] that much of the basophilic deposition in retinoblastoma previously attributed to focal calcification was in fact due to DNA precipitation. I postulated that these deposits might be a manifestation of some form of tumour activity. Dr. A. Ahmed quoted[2] my work and with electron microscope studies on cases of oat-cell carcinoma of the lung found a similarity between this vascular DNA deposition and similar fibrillary material considered to be antigen-antibody complexes in dermal lesions in disseminated lupus erythematosus.[3]

I suggested[4] doing serological studies in retinoblastoma cases to determine if anti-DNA antibodies might be present, particularly in relation to the occasional spontaneous retrogression of these growths. I would also like to offer a theory that the demyelinating phenomena which can occur in oat-cell lung cancer patients could be due to localizing antigen-antibody complexes in the central nervous system, such as may be happening in the kidney in the nephrotic syndrome, and that a DNA product may act as the antigen.—I am, etc.,

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1 Mullaney, J. M., *Archives of Ophthalmology*, 1969, **82**, 454.
2 Ahmed, A., *Journal of Pathology*, 1974, **112**, 1.
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Intrathoracic Foregut Cysts

SIR,—I was interested to read the leading article on this subject (20 April, p. 132), and I regret that my absence from the U.K. has delayed my comments. It is also unfortunate that I am away from most books of reference as well as my own clinical records. What is not stressed in the article is that such lesions may be manifestations of a much more extensive "spinal dysraphism." This syndrome can include mesenteric cysts and intestinal duplications, intrathoracic cysts, and also intraspinal lesions—the so-called enterogenous cysts. Any of these lesions may be associated with a wide variety of axial skeletal anomalies, which include hemivertebra, fusion of vertebral bodies or of laminae, bifid vertebral bodies, and fused or forked ribs. In one article in the *Proceedings of the Staff Meetings of the Mayo Clinic*, on intestinal anomalies, these changes were referred to as "scrambled spine." There may also be anomalies of the spinal cord.

Some years ago Mr. R. A. C. Jones, of