Thiopentone in Status Epilepticus

Sir,—In their paper (7 January, p. 27) on status epilepticus treated by intravenous infusion of thiopentone sodium Drs. Alan Brown and Jean Horton express a confidence about the efficacy and certainty of the method which is not in agreement with our experience. They also comment that not all patients can be controlled adequately by intermittent pressure ventilation (I.P.V.), a statement which we find hard to explain.

Working in a neurosurgical department, they have much more experience with this problem than most anaesthetists, averaging about seven per year over 16 years without failure. Of the 350 cases treated by members of the staff of the intensive respiratory care unit at the Royal Victoria Hospital, Belfast, six have suffered from epilepsy which did not respond to "conventional treatment." In at least two of these this included generous amounts of intravenous thiopentone. One patient with epilepsy of traumatic origin is noteworthy in that over 4 g. of the drug had been given, and when admitted the patient showed all the signs of barbiturate overdosage but was still convulsing. These, combined with the barbiturate-induced respiratory depression and hypotension, led to moderately severe hypoxia with metabolic acidosis, a spiny epileptic fit occurring 24 hours later. Curarization, endotracheal intubation, and I.P.V. were life saving in this instance. Tracheotomy was performed 24 hours later, and curarization was needed for 12 days before the fit stopped, the drug being stopped periodically to assess the need for its continuation. Another young patient, seen in a medical ward, had been given almost 1 g. of thiopentone, and in addition to the above had a very "wet" chest. Endotracheal intubation and tracheobronchial suction showed evidence of aspiration of gastric contents. Fortunately, this complication was noted in time, and vigorous treatment with corticosteroids, antibiotics, humidification, and suction prevented a catastrophe.

Five of our six patients have required curarization, and this has always been successful in controlling fits—in fact it is difficult to see how it could be otherwise if an adequate amount of thiopentone is given. We agree with Brown and Horton that this regimen requires special supervision and may mask the progress of neurological disease, but it may be the only effective method for treatment of some cases. In some instances a single dose of suxamethonium will be adequate, permitting intubation, tracheal suction, and good oxygenation. If followed by N2O, O2, and halothane for six to eight hours, during which adequate therapy with parenteral pheno- barbitone and anticonvulsants is given, there is a good chance of the convulsions subsiding, thus obviating the need for tracheotomy and continued ventilation.

With dosage recommended thiopentone is a comparatively safe drug and will be effective in the large majority of epileptics, but not all, although in the hands of competent anaesthetists much larger doses could be given without danger. However, if injected extra- venously (as is possible in a patient with convulsions) it can cause tissue damage, which does not appear to follow methohexi- tone. It can lead to hypoxia and hypercarbia, which are likely to aggravate the convulsions, and it carries the risk of inhalation of vomitus if given indiscriminately or where facilities for resuscitation are not available. Some years ago it was found that infusions of thiopentone were successful in controlling eclamptic convulsions, but many patients died from the effects of the barbiturate. A note of caution is necessary, lest in unskilled hands Brown and Horton’s recommendations could lead to this occurring again.—We are, etc.,

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Alternative Discriminant in Breast Cancer

Sir,—Dr. H. Miller and others have reported (21 January, p. 147) on the ratios of the excretions of two groups of steroids in the urine of healthy women and women with breast cancer. They found that, as a rule, a critical value for the ratio 11-deoxy-17-oxosteroids (mg. per 24 hours)/17-hydroxy-17-oxosteroids (mg. per 24 hours) of approximately 0.13 in healthy women and 0.16 in the cancer group reproduced fairly well the dichotomy of the cases which is produced by Spicer’s discriminant 80-80 (17/17-hydroxy-17-oxosteroids) (mg./24 hours), which has a critical value nought. They remark that in some cases the discriminants gave opposite classifications, and that in all their cases of this kind the 17-oxosteroid excretion was less than 30% of the total 11-deoxy-17-oxo- steroid excretion.

Before much effort is expended on seeking a prognostic significance in this 30% it may be noted that the existence of a maximal percent of this kind is a mathematically inevitable consequence when the discriminants disagree in the way they found. If, for example, a patient A excretes 2.25 mg. of 17-hydroxy-17-oxosteroids per 24 hours, of aetiocholanolone 337.5 µg. of 11-deoxy-17-oxosteroids then she is precisely at the critical value of Spicer’s discriminant, and gives a value of 0.15 for the alternative discriminant, and her aetiocholanolone excretion is approximately 29.7% of her total 11-deoxy-17-oxosteroid excretion. If a patient B has the same 17-hydroxy-17-oxosteroids excretion as A but has a negative Spicer discriminant (that is, <100 µg. aetiocholanolone) and a positive alternative discriminant (that is, >337.5 µg. 11-deoxy-17-oxosteroids) then her aetiocholanolone percentage must be less than 29.7%.

The maximal percentage of aetiocholanolo- lone which is imposed by this kind of discordance almost certainly, when the 17-hydroxy-17-oxosteroids excretion is greater, though it cannot exceed about 53%. Although this mathematical phenomenon may not in fact explain the reported experience, it should not be overlooked.

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Dependence on Dextromoramide

Sir,—The paper by Dr. R. Seymour-Shove and Professor C. W. M. Wilson on dependence on dextromoramide (14 January, p. 162) is an important study which should be read by all those concerned with the treatment of patients suffering from drug addiction. The authors state that dependence on dextromoramide has not been previously reported, and that all the patients who were dependent on this drug had first been addicted to dihydrocodeine. The evidence for this conclusion is weak, however, and the authors have failed to explain why dihydrocodeine should be more habit-forming than dextromoramide. In view of the extensive use of dihydrocodeine, the present paper is an important one, and the authors have made a valuable contribution to the study of addiction.

Various combinations of other drugs were tried with little success from the point of view of lessening his dependence on the dextromoramide. Substitution with dihydrocodeine was attempted, but after only one day's trial the patient reported with the word "frantic" with the pain and threatened suicide. At this juncture levorphanol was tried at the dosage of 1.5 mg. q.d.i. This, in addition to dihydrocodeine, chlorpromazine, and amitriptyline seemed to be controlling his clouded consciousness and in April 1966 at 635, 362 of 11 February 1967. Downloaded from http://www.bmj.com/ on 18 April 2021 by guest. Protected by copyright.