ENFLURANE SEIZURE: A CASE REPORT

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Enflurane, a useful alternative to fluothane for repeated procedures is notable for its cerebral irritability, exhibiting both latent and frank seizure activity in animals and man. This effect is dose related and may be augmented by other agents and by hypocarbia. The predisposition of patients to seizure is often an unknown quantity and this report confirms the stability of two other volatile anaesthetics under comparable conditions.

CASE REPORTS

A.D. a 40 year old married woman was admitted to hospital with a diagnosis of stage 1 carcinoma of the cervix. She was in excellent health with no medical history and subsequent enquiry revealed no personal or family record of fits. On 17th December, she received a general anaesthetic for a diagnostic D and C. No premedication was given and induction was obtained with thiopentone 300 mg and maintained with nitrous oxide, oxygen and fluothane, for a procedure lasting 15 minutes. Her recovery from anaesthesia was uneventful and she was transferred to the Christie Hospital for radiotherapy. On 31st December the patient was premedicated with lorazepam 2 mg orally two hours before presenting in theatre for the insertion of a radio-active source in the cervix. She was awake and cooperative and was placed in the knee-chest position before anaesthesia was induced with thiopentone 200 mg and maintained using nitrous oxide, oxygen and trichlorethylene via a face mask and Magill circuit. The anaesthetic lasted for 10 minutes and recovery was again quite normal. On 7th January she attended theatre for further radium insertion and once more received lorazepam 2 mg orally for premedication. She was induced with thiopentone 400 mg and was maintained with nitrous oxide, oxygen and enflurane administered as a 5% concentration for the first 5 minutes reducing to 3% for the subsequent 10 minutes of the procedure. Recovery of consciousness was speedy and uneventful and the patient was returned to the ward. One hour after the end of the anaesthetic, whilst lying quietly in bed she suffered a generalised tonic/clonic convulsion. The seizure activity started in her face and rapidly spread to the whole body, subsiding spontaneously after 2 minutes. The anaesthetist was called but no treatment was required and the patient regained consciousness in approximately 15 minutes though she remained very drowsy for the following 10 hours. A full neurological examination revealed no abnormality and biochemical investigations were normal. An electroencephalogram (EEG) performed five days later displayed medium voltage symmetrical alpha rhythm at 8-10 Hz in central and post central areas and diffuse low voltage beta activity at 16-20 Hz, which was not altered by hyperventilation. This EEG was considered to be within normal limits. Although her general condition remained satisfactory she required a further radium insertion and was prescribed phenytoin 300 mg daily for the two days prior to her final operation. On January 14th the patient again received lorazepam premedication and was induced with thiopentone 400 mg and maintained with nitrous oxide, oxygen and trichlorethylene. No abnormal muscular activity was observed either during or subsequent to the anaesthetic. The patient has attended the regular follow-up clinic and has remained in good health showing no evidence of convulsions or central nervous abnormality.

COMMENTS
Although Oshima, Urabe, Shingu et al\(^1\), suggest that enflurane does not exacerbate pre-existing epileptic fits, the incidence of electroencephalographic abnormalities both during and subsequent to the administration of enflurane is well documented and indeed such changes have been noted to persist in man for up to 30 days\(^2\). Manifest seizure however, has only been noted infrequently during enflurane anaesthesia. Linde, Lamb, Quimby et al\(^3\), exploring the predisposing causes of the excitability, demonstrated mild seizures in three patients receiving high concentrations of the vapour and Sprague and Wolf\(^4\) reported two cases in whom concomitant medication with amitriptyline may have been a predisposing factor, whilst Kainuma and Suzuki\(^5\) showed an immediate onset of clonic activity when adrenaline was infiltrated during enflurane anaesthesia in babies. In the immediate postoperative period generalised muscle contractions have been reported\(^6-8\) whilst a delayed convulsion at one hour has also been noted\(^9\). Fahy\(^10\) and Nicoll\(^11\) both report seizures occurring 5 to 17 hours after the anaesthetic and Ohm, Cullen and Amory et al\(^12\), presented two cases of late onset attacks at 6 and 8 days. All but three of the reported cases had received enflurane under controlled ventilation and it has been noted that hypocarbia levels of 34 torr or less p\(\text{a CO}_2\) increase the cerebral irritability associated with this agent\(^2,13\). In our patient, spontaneous respiration in the knee-chest position would tend to raise the arterial carbon dioxide level, discounting the role of hypocarbia in this incident. However, animal studies indicate that other predisposing factors may have included the use of a diazepine premedicant\(^14\) and the augmentation of seizure activity by thiopentone\(^15\). However, the case under discussion is unique in providing her own control in that she received essentially similar treatments involving two alternative volatile agents, ethrane and fluothane, within a period of 4 weeks from which she suffered no sequelae. The brief exposure time to enflurane and the postoperative onset of seizure may warrant a review of our current practice of using this agent for repeated anaesthetics at short intervals.

REFERENCES