References

1 Price DK, Turnbull GJ, Gregory RP, *et al.* Neuroleptic malignant syndrome in a case of post-partum psychosis. *Br J Psychiatry* 1989;**155**:849-52
2 Castillo E, Rubin RT, Holsboer-Trachsler E. Clinical differentiation between lethal catatonia
3 Gelenberg AJ. The catatonic syndrome. *Lancet* 1976; i:1339-41
4 Gjessing LR. A review of periodic catatonia. *Biol Psychiatry* 1974;**8**:23-45
5 Berrios GE. Stupor Revisited. *Compr Psychiatry* 1981; **22**:466-89
6 Penn H, Ravy J, Lapham L, Mandel M, Sandt J. Catatonic behaviour, viral encephalopathy, and death. *Arch Gen Psychiatry* 1972;**27**:758-61

Landmarks for insertion of oesophageal endoprosthesis

I read the article by Beynon *et al.* (August 1991 *JRSM*, p 479) with interest. I would like to suggest an alternative but simpler and perhaps more applicable method to identify the upper level of malignant stricture during insertion of oesophageal endoprosthesis. The medial border of the diaphragm is relatively fixed which can be easily visualized during fluoroscopy. The upper margin of the neoplasm can be easily 'marked' against the vertebra adjacent to the tip of the endoscope. This level can then be judged with reference to the medial border of the left diaphragm and memorized. The satisfactory positioning of the endoprosthesis is therefore easily achieved by advancing the tube until its funnelled proximal part just sits above the marked vertebra. Movement of the patient's gown must be minimal while the procedure is being carried out under general anaesthesia. However, there is now increasing preference to insert oesophageal endoprosthesis under conscious sedation for obvious reasons (fewer medical personnel involved, more cost effective, less tedious and probably safer in the frail and elderly patients). In this case, using artery forceps as the 'landmarks' against the top tumour level may not be reliable because patient movements can disturb the position of the artery forceps and hence the landmarks. The method that I have described here by the 'eye-balling' technique has been used successfully without misjudging the top tumour level in my last 20 attempts. I would strongly recommend the same technique for aiding oesophageal endoprosthesis insertion.

C K CHING
Department of Medicine,
Nottingham City Hospital,
Hucknall Road, Nottingham NG5 1PB

Another death from Ecstasy

Chadwick *et al.* (June 1991 *JRSM*, p 371) recently reported a fatality associated with coagulopathy and hyperthermia due to ingestion of Ecstasy 3,4-methylenedioxymetamphetamine (MDMA). This is claimed to be the first reported death resulting from abuse of this hallucinogenic recreational drug in the United Kingdom; at least four deaths have been reported from the USA[1,2]. Since reading this report we have seen another death from Ecstasy abuse with similar clinical manifestations.

A previously fit 18-year-old male was admitted to casualty deeply unconscious and convulsing. He was hypotensive, with a systolic blood pressure of 70 mmHg and a tachycardia of 180/min, peripherally cyanosed and sweating with an axillary temperature of 42°C. He was treated with intravenous Diazemuls, which controlled the convulsions, and transferred to the intensive care unit. There he was intubated and ventilated; resuscitation was commenced with cold intravenous fluids, glucose, bicarbonate, dopamine and dobutamine. Intravenous dantrolene (1 mg/kg, repeated) and mannitol were administered. Despite these measures he remained hypotensive, oliguric and hyperpyrexial. A grossly elevated serum creatine kinase (9000 U/l) suggested the occurrence of rhabdomyolysis.

Within 3 hours of admission he began bleeding steadily from puncture sites, oropharynx and gastrointestinal tract. Disseminated intravascular coagulation was diagnosed with prolonged clotting times, hypofibrinogenaemia, elevated fibrin degradation products and thrombocytopaenia. Despite treatment with whole blood, platelets and fresh frozen plasma, bleeding continued. The patient's condition rapidly deteriorated and he died 5 hours after admission.

Enquiries after death from police and friends established that he had used Ecstasy in the past and had ingested three tablets of Ecstasy that evening. Serum MDMA concentration on admission was 1.26 mg/l; no other drugs were detected. MDA, an active metabolite of MDMA, was not detected.

The clinical features of this case, with hyperthermia, rhabdomyolysis and coagulopathy are similar to those reported by Chadwick *et al.* and to a non-fatal case reported by Brown and Osterloh from the USA[3]. The serum MDMA concentrations in these cases were 0.424 mg/l and 7.0 mg/l respectively. MDMA has been shown to produce hyperthermia, which may be mediated by 5-HT2 receptors, in rats[4]. Other reports of fatalities associated with Ecstasy abuse in the USA have recorded death due to cardiac complications or accidents. Recent reports indicate that abuse of this drug is increasing - it appears that it is far from benign and that unpredictable, fulminating and fatal hypermetabolic reactions may occur after its use.

N T A CAMPKIN
U M DAVIES
Department of Anaesthesia,
Westminster Hospital,
Dean Ryle Street, Horseferry Road,
London SW1P 2AP

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References

1 Dowling GP, McDonough ET, Bost RO. 'Eve' and 'Ecstasy'. A report of five deaths associated with the use of MDEA and MDMA. *JAMA* 1987;**257**:1615-17
2 Suarez RV, Reimersma R. 'Ecstasy' and sudden cardiac death. *Am J Forensic Med Pathol* 1988;**9**:339-41
3 Brown C, Osterloh J. Multiple severe complications from recreational ingestion of MDMA ('Ecstasy'). (letter) *JAMA* 1987;**258**:780-1
4 Schmidt CJ, Black CK, Abbate GM, Taylor VL. MDMA-induced hyperthermia and neurotoxicity are independently mediated by 5-HT2 receptors. *Brain Res* 1990;**529**: 85-90