ous pneumomediastinum was caused by repeated Valsalva type manoeuvres that resulted in episodes of increased intra-alveolar pressure. This occurred while the patient was repeatedly blowing a whistle during an eight hour dancing session. The use of a stimulant drug aids this physical overexertion. Vomiting resulting in an increased intrathoracic pressure has been proposed as a mechanism for spontaneous pneumomediastinum following Ecstasy ingestion, but was not reported by our patient.¹⁰

We feel that a young person presenting with chest pain following ingestion of Ecstasy should have a chest x ray to exclude a spontaneous pneumomediastinum. It is generally agreed that spontaneous pneumomediastinum can be managed conservatively and does not require hospital admission, but more serious

causes such as oesophageal perforation must be excluded.

- 1 Abolnik I, Lossos IS, Breuer R. Spontaneous pneumomediastinum. A report of 25 cases. *Chest* 1991;100:93-5.
- 2 Hamman I. Spontaneous mediastinum emphysema. *Bull Johns Hopkins Hosp* 1939;64:1-21.
- 3 Macklin CC. Transport of air along sheaths of pulmonary blood vessels from alveoli to mediastinum. *Arch Intern Med* 1939;64:913-26.
- 4 Panacek EA, Singer AJ, Sherman BW, Precott A, Rutherford WF. Spontaneous pneumomediastinum: clinical and natural history. *Ann Emerg Med* 1992;21:1222-7.
- 5 Fajardo LL. Association of spontaneous pneumomediastinum with substance abuse. *West J Med* 1990;152:301-4.
- 6 Millar NW, Spiekeman PE, Hepper NG. Pneumomediastinum resulting from performing Valsalva manoeuvres during marijuana smoking. *Chest* 1972;62:233.
- 7 Mir BJA, Galvete VJ, Plaza VM, Ingles AMJ, Alonso OE, Navarro LF. Spontaneous pneumomediastinum after cocaine inhalation. *Respiration* 1986;50:230-2.
- 8 Brody SL, Anderson GV, Gutman JB. Pneumomediastinum as a complication of "Crack" smoking. *Am J Emerg Med* 1988;6:341-3.
- 9 Barrett PJ, Taylor GT. "Ecstasy" ingestion: a case report of severe complications. *J R Soc Med* 1993;86:233-4.
- 10 Levine AJ, Drew S, Rees GM. "Ecstasy" induced pneumomediastinum. *J R Soc Med* 1993;86:232-3.

MDMA induced hyperthermia: a survivor with an initial body temperature of 42.9°C

A Mallick, A R Bodenham

Abstract

A young male survived hyperpyrexia (42.9°C) following MDMA ("Ecstasy") ingestion. He developed convulsions, rhabdomyolysis, metabolic acidosis, and respiratory failure. This was successfully managed by assisted ventilation, aggressive fluid therapy, and the early administration of dantrolene, in addition to cooling measures. This is the first report of a survivor with such a severe hyperpyrexia.

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Keywords: MDMA; Ecstasy; hyperthermia; dantrolene

In recent years the use of 3,4-methylenedioxymethamphetamine (MDMA, "Ecstasy") has increased in the USA, the United Kingdom, and continental Europe.¹ Severe and sometimes fatal adverse reactions are increasingly encountered in various accident and emergency (A&E) departments. These include hyperthermia, metabolic derangements, seizures, hypertensive crises, cardiac dysrhythmias, disseminated intravascular coagulation (DIC), rhabdomyolysis, acute renal failure, hepatic toxicity, cerebrovascular accidents, and psychiatric disturbances.^{2,3} Survival with a core temperature greater than 42°C is rare.³

Case history

A 19 year old male presented after collapsing in a local night club. The patient was reported to have ingested three tablets of Ecstasy four

hours previously. At presentation, he was obtunded, with a Glasgow coma score of 7 (eye opening 2, vocalisation 2, motor response 3). He was sweating profusely, with a rectal temperature of 42.9°C, central cyanosis, and a respiratory rate of 44 breaths/min. A narrow complex tachycardia of 192 beats/min was present but blood pressure was 150/90 mm Hg. His pupils were widely dilated.

The patient arrived in the A&E department with seizures, which were controlled with 10 mg diazepam intravenously. In view of the seizures and the marked hyperpyrexia, he was anaesthetised using thiopentone sodium and suxamethonium, and his airway was secured by tracheal intubation. Anaesthesia was maintained with increments of midazolam, and muscle relaxation was continued with an infusion of atracurium. Hyperthermia was treated with cooling measures (moistened towels, ice packs, fanning, and body exposure). At the same time, dantrolene was given intravenously as 60 mg boluses at 10 minute intervals to a total of 180 mg until the body temperature was brought down to 38°C.

The patient was transferred to the intensive care unit. Sedation was maintained with infusions of alfentanil and propofol. From the time of admission he required a total of 6 litres of crystalloid over a period of six hours for resuscitation, with continuous monitoring of central venous pressure. Over the ensuing few hours he became progressively hypotensive despite adequate central venous pressure. A pulmonary artery catheter was inserted and the wedge pressure was found to be adequate. He

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Table 1 Blood gases, acid–base status, temperature, and ventilation over 24 hours after admission

	Time (h)					
	1	2	3	6	12	24
Pao ₂ (kPa)	12.04	9.16	11.5	6.9	10.5	15.6
Paco ₂ (kPa)	6.56	5.2	4.1	4.2	3.6	3.8
pH	7.101	7.165	7.233	7.32	7.36	7.43
Na ⁺ (mmol/l)	146	143	147	144	141	145
K ⁺ (mmol/l)	5.2	5.9		5.1	4.1	4.4
Base excess (mmol/l)	−17.4	−11.5	−8.7	−7.2	−8.6	−4.1
Fio ₂	1.0	1.0	1.0	1.0	0.8	0.4
Temperature (°C)	42.9	37.8	37.3	36.9	37.6	36.9
Ventilation	CMV	CMV	CMV	CMV	SIMV	SIMV
MV (l/min)	14	15	15	12	15	12
PEEP (cm H ₂ O)	0	0	5	10	10	5
CVP (mm Hg)		4	8	13	14	12
PAWP (mm Hg)				9	10	11
iv NaHCO ₃ (mmol)		50	50			
Blood urea (mmol/l)		17		6.0		5.7
Serum creatinine (μmol/l)		109		98		91
INR			1.3		1.5	1.2

CMV, controlled mechanical ventilation; PEEP, positive end expiratory pressure; SIMV, synchronised intermittent mandatory ventilation; MV, minute ventilation; CVP, central venous pressure; PAWP, pulmonary artery wedge pressure; iv, intravenous; INR, international normalised ratio.
Dantrolene: protocol for administration
Each vial containing 20 mg of dantrolene should be reconstituted by adding 60 ml of water and shaking until the solution is clear. The dose is 1 mg/kg intravenously and may be repeated up to a cumulative dose of 10 mg/kg.

needed an adrenaline infusion at 0.1 μg/kg/min to maintain his circulation. Aliquots of 50 mmol sodium bicarbonate were given twice to correct a metabolic acidosis found on repeated arterial blood gas analyses. Urine output was maintained at more than 2 ml/kg/h. There were no further seizures in the intensive care unit.

From the time of presentation to A&E department, normal oxygenation proved difficult to achieve. Blood gases are shown in table 1. Despite the application of positive end expiratory pressure of up to 10 cm H₂O, Pao₂ was 6.9 kPa on an Fio₂ of 1.0. Secretions were minimal and there was no evidence of aspiration of gastric contents. Chest x ray showed bilaterally increased vascular markings. Hypoxia resolved spontaneously over the next 24 hours, and the patient was extubated on day 2. There were no detected residual deficits on a neurological assessment and he was discharged home on day 6.

Full blood count, coagulation profile, and blood biochemistry were all within normal limits. Serum sodium concentration was within normal range. Creatine kinase reached a maximum of 42 120 IU/l, falling to 6000 IU/l after five days. Serum aspartate transaminase (AST) rose to 1400 IU/l, returning to normal after five days. MDMA was detected both in blood and urine. Urine examination revealed myoglobinuria of more than 100 mg/dl. Despite myoglobinuria, this patient did not show any signs of renal impairment, as blood urea and serum creatinine remained within normal range.

On follow up at six weeks, the patient had not taken any more MDMA and there was no abnormality on gross neurological evaluation.

Discussion

MDMA is commonly used in the “rave” scene, where strenuous exertion and subsequent dehydration are generally thought to be partly responsible for the hyperthermic syndrome. MDMA-induced hyperthermia, as suggested from animal evidence, is due to an acute

increase in central 5-hydroxytryptamine (5-HT) and dopamine levels, and their synergistic action on hypothalamic centres.⁴ The severity and duration of hyperthermia appear to be important prognostic indicators in this condition.³ Complications seem to be related to duration and degree of hyperthermia and they increase in number and severity if not treated promptly.³ The concentration of circulating endotoxins and cytokines, tumour necrosis factor α (TNF-α), interleukin-1 (IL-1), and interleukin-6 (IL-6) are increased in the presence of very high body temperature and they can lead to multisystem organ failures.⁵

The patient in this report had a peak temperature of 42.9°C which was quickly brought down, within an hour of arrival, with both intravenous dantrolene and cooling measures. We attribute the survival of our patient to prompt and rapid control of hyperthermia in the A&E department. It would not have been possible to bring down the body temperature within an hour with cooling measures alone. Moreover, external cooling can lead to peripheral vasoconstriction and a raised core temperature.

As the patient was hypoxaemic and had a full stomach, we used suxamethonium to secure the airway quickly, although its known side effects of hyperkalaemia and precipitation of malignant hyperpyrexia are of potential concern in these group of patients. In vitro muscle testing for malignant hyperpyrexia in one patient did not substantiate this.⁶ Non-depolarising relaxants such as atracurium or the more rapid acting rocuronium may be suitable alternatives in such a situation.

Dehydration was corrected by aggressive fluid therapy with isotonic 0.9% saline over a period of six hours, with central venous pressure and pulmonary artery wedge pressure monitoring as a guide. There have been reports of three deaths due to dilutional hyponatraemia and acute water intoxication following MDMA ingestion.⁷ This may result from drinking excessively large volumes of water

throughout the event in rave parties, following advice from drug misuse agencies, and the release of antidiuretic hormone by MDMA. Urgent estimation of serum sodium is therefore indicated in this situation.

There is no available antidote for the management of this condition. Supportive treatment starting in the A&E department and continuing in an intensive care unit is essential. We think that dantrolene should be used early, in addition to cooling measures, if the temperature is greater than 39°C or rapidly rising.⁸ As the number of such patients is increasing, we suggest that departments should have an appropriate protocol for cooling these patients, including administration of dantrolene sodium.

- 1 Cuomo MJ, Dyment PG, Gammino VM. Increasing use of "Ecstasy" (MDMA) and other hallucinogens on a college campus. *J Am Coll Health* 1994;42:271-4.
- 2 Henry JA, Jeffreys KJ, Dawling S. Toxicity and deaths from 3,4-methylenedioxymethamphetamine ("ecstasy"). *Lancet* 1992;340:384-7.
- 3 O'Connor B. Hazards associated with the recreational drug 'ecstasy'. *Br J Hosp Med* 1994;52:507-14.
- 4 Gordon CJ, Watkinson WP, O'Callaghan JP, Miller DB. Effects of 3,4-methylenedioxymethamphetamine on autonomic thermoregulatory responses of the rat. *Pharmacol Biochem Behav* 1991;38:339-44.
- 5 Bouchama A. Heat stroke: a new look at an ancient disease. *Intensive Care Med* 1995;21:623-5.
- 6 Tehan B, Hardern R, Bodenham A. Hyperthermia associated with 3,4-methylenedioxymethamphetamine ('Eve'). *Anaesthesia* 1993;48:507-10.
- 7 Walking on the moon [editorial]. *Lancet* 1996;347:207.
- 8 Bodenham AR, Mallick A. New dimensions in toxicology: hyperthermic syndrome following amphetamine derivatives. *Intensive Care Med* 1996;22:622-4.

Seat belt injuries and sigmoid colon trauma

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Abstract

Colonic seat belt injuries are rare but carry higher mortality rates than small bowel injuries. The case of a 44 year old man is described who had severe sigmoid colon compression injury from his seat belt a few days after a road traffic accident.

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Keywords: seat belt; sigmoid compression

Case report

A 44 year old male driver with no significant past medical history presented to the accident

and emergency department two days after a road traffic accident with increasing abdominal pain and constipation.

He was febrile, dehydrated, and abdominal examination showed severe peritonitis. There was a faint resolving bruise over the lateral margin of the left iliac fossa. Digital rectal examination was normal.

He was the driver in a road traffic accident when he skidded and hit a lamp post. The front passenger died of severe head injury and two of the three back seat passengers sustained variable physical injuries. He was treated in another city hospital for minor facial wounds

Figure 1 Radiological appearances: (A) erect; (B) supine.

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