multisystem involvement, some of which may be explained by the effects of shock, but the skin changes, the central nervous system involvement (with adenovirus cultured from the cerebrospinal fluid), the skeletal muscle involvement (with a creatine phosphokinase of 8130 U/l), and the myocarditis cannot be attributed to hypotension alone. We therefore believe that she had disseminated adenovirus infection producing a clinical picture identical to that of staphylococcal toxic shock syndrome.

Toxic shock-like syndrome has been described with several infectious agents, including staphylococcus,³ streptococcus,⁴ and pseudomonas⁵. The toxins which cause staphylococcal and streptococcal toxic shock syndrome have a common mode of action, binding to specific V β bearing T cells and stimulating T cell proliferation and cytokine release.⁶ It is possible that similar immune events were induced by adenovirus infection in this child.

We conclude that this case with multiorgan

involvement producing a clinical picture of toxic shock syndrome was due to infection with adenovirus. We therefore recommend that clinicians consider adenovirus infection in the differential diagnosis of toxic shock syndrome in children.

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Accidental ingestion of 'Ecstasy' (3,4-methylenedioxymethylamphetamine)

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Abstract

There is no report of the effects of 'Ecstasy' (3,4-methylenedioxymethylamphetamine) poisoning in childhood. The case of a 13 month old boy who ingested one capsule of Ecstasy is reported. Neurological and cardiovascular side effects predominated, which responded well to treatment with a chlormethiazole infusion.

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Recreational use of 'Ecstasy' or 'E' is common particularly within the confines of acid house parties. Chemically Ecstasy is 3,4-methylenedioxymethylamphetamine (MDMA). MDMA is a psychoactive phenylisopropylamine, chemically related to both amphetamine-like stimulants and hallucinogens such as mescaline, and is reputedly safe. The usual dosage is 50-150 mg, and the duration of action is 4-6 hours. The drug reportedly induces a state of enhanced self awareness and ability to communicate, without distorting normal thought processes.¹ Increasing use by young people has been documented,² but our report and those of fatal or serious side effects in teenagers and adults³⁻⁵ must caution against the belief that this drug is safe.

Case report

A 13 month old boy was admitted with a history of ingesting a capsule of Ecstasy found on the

floor 30 minutes previously. He was in the care of his grandmother at her home while his mother was at work. His uncle, who was present, freely admitted to recreational use of the drug in this home where he also lived, but not on the evening in question.

The clinical features are summarised in table 1. The convulsion was unresponsive for 60 minutes to a total of 2.5 mg/kg diazepam and 0.4 mg/kg of haloperidol, both given intravenously. A chlormethiazole infusion (10 mg/kg/hour) was commenced and there was a dramatic response with both the convulsion and the cardiovascular instability resolving within 5-10 minutes. A further self limiting episode of hypertension (180/70 mm Hg) and tachyarrhythmia (170 beats/min) occurred five hours after admission. All abnormal signs had resolved by seven hours so the chlormethiazole infusion rate was halved and finally stopped at 16 hours.

No haematological or biochemical abnormalities were observed. Results of toxicology are shown in table 2.

The possibility of non-accidental ingestion of

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Table 1 Clinical features
attributed to MDMA

<i>Initial findings</i>
Generalised shaking
Dilated pupils
Hypertonia
Writhing and jerking movements
<i>Within one hour</i>
Convulsion
Tachyarrhythmia (70-170 beats/min)
Hypertension (maximum 120 mm Hg systolic pressure)
Fever (maximum temperature 38.5°C per axilla)

Table 2 Toxicology on admission. Time represents interval since ingestion

Serum (at 90 minutes)	MDMA	0.7 mg/l
	MDA	<0.1 mg/l
Urine (at 2 hours)	MDMA	Detected
	MDA	Detected
	Chlormethiazole metabolites	Detected
	Other basic or neutral drugs	Nil

MDMA was a foremost consideration and social services staff were therefore involved at an early stage. Prompt presentation in the early evening and a clear, consistent history from carers who themselves had no signs of MDMA ingestion resulted in the conclusion that the episode was accidental.

The child recovered completely and was discharged four days after admission to the care of his mother, with social service follow up and strong advice regarding the safety of drugs in the home. He had no short term neurological deficit but was subsequently lost to medical follow up.

Discussion

Animal studies show that MDMA reduces 5-hydroxytryptamine and 5-hydroxy indoleacetic acid concentrations at nerve terminals, and the metabolite 3,4-methylenedioxymphetamine (MDA) selectively destroys 5-hydroxytryptamine nerve terminals. MDMA also increases dopamine turnover, this being implicated in the long term neurotoxicity of MDMA use. The dopamine receptor antagonist haloperidol and some 5-hydroxytryptamine antagonists are effective in blocking these actions,¹ hence the recommendation of haloperidol in the treatment of side effects.

Four fatalities in adults related to Ecstasy abuse have been reported, with recorded MDMA concentrations between 0.2 mg/l and 1.1 mg/l; one patient survived with a concentration of 7.0 mg/l.³⁻⁵ Fatalities have been associated with cardiovascular autonomic derangement and in one case coagulopathy and hyperthermia. The exact cause of the latter complications is unknown, but MDMA induced 'oxidative stress' may play a part.¹ Alternatively coagulopathy

and hyperthermia may be linked to the circumstances of the ingestion, be idiosyncratic reactions, or the result of unidentified impurities in the manufacturing process.

In addition, the effects of amphetamine induced hyperthermia are similar to the effects of heatstroke and include derangements in coagulation.⁶ Rapid control of the rising temperature in our case may have prevented coagulopathy occurring by a similar mechanism.

Controlling the seizure proved the major problem in this child. Chlormethiazole infusion brought about rapid control not only of the convulsion, but also of tachyarrhythmia and hypertension. Chlormethiazole may have acted by potentiating the effects of the inhibitory neurotransmitters γ -aminobutyric acid and glycine. Serotonergic mechanisms and dopamine inhibition are possibly secondary.

In conclusion Ecstasy is not a 'safe' drug. It is a drug of abuse and must at least be stored safely away from children. We suggest that chlormethiazole may have a role in controlling toxicity from MDMA.

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