overall prognosis is dependent on the severity of glomerular lesions.[2]

There is an increasing trend for the use of ACE inhibitors in elderly patients with hypertension or cardiac failure, especially in general practice. The cutaneous reactions with captopril have usually been macropapular or urticarial, often progressing to severe erythroderma;[3] various other lesions, including pemphigus and lupus-like eruptions, have been reported. Enalapril was said to be freer of cutaneous adverse effects because, unlike captopril, it does not contain the thiol group that is thought to be responsible: but skin eruptions have been described.[4,5] In our case with SHP, the favourable response to steroid therapy suggests that early immunosuppressive therapy is beneficial.

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1. Habib R, Cameron JS. Schönlein-Henoch purpura in the kidney. In: Bacon PA, Hadler N, London M, eds. Rheumatic diseases. London: Butterworth, 1982: 178–201.
2. Cameron JS. The nephritis of Schonlein-Henoch purpura: current problems. In: Kincaid Smith P, d'Apice AF, Atkins R, Wiley C, eds Progress in glomerulonephritis. New York: Wiley, 1979: 283–89
3 Goodfield MJ, Millard LG. Severe cutaneous reactions to captopril. *BMJ* 1985; **290:** 1111–12.
4. Furness PN, Goodfield MJ, MacLennan KA, Stevens A, Millard LG. Severe cutaneous reactions to captopril and enalapril: histological studies and comparison with mycosis fungoides. *J Clin Pathol* 1986; **39:** 902–07.
5 Barnes JN, Davies ES, Gent CB. Rash, eosinophilia, and hyperkalaemia associated with enalapril. *Lancet* 1983; i: 41–42.

Hyperpyrexia induced by 3,4-methylenedioxyamphetamine ("Eve")

SIR,—Illicit use of amphetamine derivatives is increasing, and there have been 8 reported deaths from hyperpyrexia associated with the use of 3,4-methylenedioxymethamphetamine (MDMA, "ecstasy") in the past year.[1-4] We report a case of hyperpyrexia due to a different derivative, 3,4-methylenedioxyamphetamine (MDA, "Eve") which illustrates important features of treatment.

A 20-year-old man was admitted from a "rave" party having taken five tablets of a drug sold as ecstasy 2 h previously. He was said to have been convulsing for 1 h. He was in Glasgow coma scale 3, with widely dilated pupils and an opisthotonic posture due to pronounced generalised muscle rigidity. He was sweating profusely and rectal temperature of 40°C on admission rose rapidly to 41·4°C. Heart rate was 170 per min with sinus rhythm and blood pressure was 110/70 mm Hg, which fell to 80/30 within 30 min. Investigations showed metabolic acidosis (pH 7·18, PaO_2 22·6 kPa, $PaCO_2$ 3·06 kPa) and increased creatine phosphokinase (CPK) of 774 U/l. Coagulation times and platelet count were normal.

Resuscitation commenced immediately with intravenous fluids and dantrolene 1 mg/kg. Shortly after admission he had a generalised tonic-clonic convulsion, which was ended by intravenous diazepam. He was intubated, ventilated and given activated charcoal. Further intravenous fluid administration was guided by central venous pressure readings; 3·5 l of fluid were required in the first 2 h. 12 h after admission CPK was 15 700 U/l and serum creatinine had fallen from 173 μmol/l to 124 μmol/l. Toxicological analysis of serum showed MDA 1·51 mg/l and ethanol 0·2 g/l. Urine showed MDA 48·6 mg/l and MDMA 0·5 mg/l. He recovered and was discharged at 72 h.

In the UK, most tablets sold as "ecstasy" contain MDMA as the active agent, although some may contain lysergic acid diethylamide (LSD), amphetamine sulphate, or MDA. Our case-report suggests that MDA (the major metabolite of MDMA) has a similar pattern of acute toxicity to MDMA. The small amount of MDMA recovered is probably a synthetic byproduct. The mechanism by which these agents produce hyperpyrexia is not clear but is likely to be a central effect. In rats, MDMA-induced hyperpyrexia is mediated via stimulation of serotonergic pathways causing an elevation of metabolic rate.[5] It may be possible to attenuate the hyperthermic response by blocking 5-HT2 receptors.[6]

This case illustrates the need to start treatment rapidly in view of the precipitous rise in core temperature. Vigorous fluid replacement is necessary to correct the substantial volume depletion that may occur and to facilitate thermoregulation by sweating. Maintaining a high urine output will help prevent acute renal failure secondary to the myoglobinuria produced by rhabdomyolysis. Skeletal muscle will remain a substantial source of heat production, which may be diminished by a peripherally acting agent such as dantrolene.

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1. Screaton GR, Singer M, Cairns HS, Thrasher A, Cohen SL. Hyperpyrexia and rhabdomyolysis after MDMA ("ecstasy") abuse. *Lancet* 1992; **339:** 677–78.
2. Chadwick IS, Curry PD, Linsley A, Freemont AJ, Doran B. Ecstasy, 3-4 methylenedioxymethamphetamine (MDMA), a fatality associated with coagulopathy and hyperthermia. *J R Soc Med* 1991; **84:** 371.
3. Campkin NTA, Davies UM. Another death from Ecstasy. *J R Soc Med* 1992; **85:** 61.
4. Henry JA, Jeffreys KJ, Dawling S. Toxicity and deaths from 3,4-methylenedioxymethamphetamine ("ecstasy"). *Lancet* (in press).
5. Gordon CJ, Watkinson WP, O'Callaghan JP, Miller DB. Effects of 3,4-methylenedioxymethamphetamine on thermoregulatory responses of the rat. *Pharmacol Biochem Behav* 1991; **38:** 339–44.
6. Schmidt CJ, Black CK, Abbate GM, Taylor VL. Methylenedioxymethamphetamine-induced hyperthermia and neurotoxicity are independently mediated by 5-HT2 receptors. *Brain Res* 1990; **529:** 85–90.

Transmission of hepatitis C with pasteurised factor VIII

SIR,—We report a case of hepatitis C virus (HCV) transmission via a pasteurised plasma product. The patient is white, born in 1985, and has severe haemophilia A. He has only received substitution of factor VIII with Haemate P (HS) (Behringwerke). He has never had surgery or received other blood products. The family lives in a remote village in northern Sweden, and they have not travelled abroad with him. No other cases of hepatitis C have lately been diagnosed in his village or at the local hospital, where he sometimes receives the infusions. His family members have remained anti-HCV negative. Other factor VIII products have not been delivered to the pharmacy of the local hospital during the past few years.

On routine testing for antibodies against HCV he was negative in May, 1991, and, retrospectively, also in May, 1989. But he became positive in October, 1991 (Abbott second generation anti-HCV EIA), a finding confirmed with 4-RIBA (Ortho Diagnostic Systems), which was positive for C22 and C33, and had a positive test for HCV-RNA by the polymerase chain reaction (PCR).[1] In addition aminotransferase concentrations were raised for the first time: asparate 10·07 and alanine 11·92 μkat/l (normal $<0·80$). 2 weeks later, these enzymes were 21 and 30 μkat/l, respectively. The boy was more tired than usual and had a poor appetite for a few weeks, but he was never jaundiced.

During the year before seroconversion this patient had received Haemate P from five different batches. We have analysed these batches for HCV-RNA with PCR, using primers from the 5′ non-coding region,[1] and two batches were positive. PCR has been routine at the laboratory for more than 6 months, and no contamination has been observed. Every second sample has been a negative control.

Intensive follow-up of two groups of patients with haemophilia A or von Willebrand's disease exclusively treated with this concentrate did not reveal any case of hepatitis.[2,3] Isolated reports of the occurrence of hepatitis after treatment with pasteurised factor VIII[4,5] have, according to manufacturer, subsequently turned out to be questionable cases, who erroneously also might have received other plasma products. After thorough investigation we could exclude the transfusion of any other plasma products to our patient. The demonstration of HCV-RNA in the product does not prove that it is infectious, since viral RNA might have survived the pasteurisation despite destruction of virions, but it supports our suspicion. Thus, our case demonstrates that a factor VIII concentrate, which has undergone viral inactivation through pasteurisation (60°C for 10 h in solution), may still occasionally transmit HCV. That this is a rare complication is illustrated by the fact that no other case of HCV transmission has been definitely connected to Haemate P over 10 years. Moreover, 7 of our patients, who have been treated only with Haemate P and who have received material from at least one of the two positive batches half a year ago