Butadiene workers do indeed show a deficit of mortality for all cancer sites combined but this outcome does not provide an appropriate evaluation of butadiene risk because smoking-induced lung cancer, the commonest cause of cancer in males, is less common in exposed workers than in the general population. Safety requirements severely restrict smoking for these workers. In addition, epidemiological inquiry has focused on wartime butadiene production. Assessment of the human risk should concentrate on those cancers which best reflect results in animals. There is a remarkable correspondence between human cancer mortality and the increases in leukaemia/lymphoma observed in mice.[3,8] Rates of mortality from lymphosarcoma and reticulosarcoma were increased 5-6-fold in one butadiene manufacturing plant; a similar increase for lymphopoietic cancers and leukaemia was reported among black production workers in styrene-butadiene rubber plants; a 6-fold excess for lymphohaemopoietic cancers was noted in one part of tyre manufacturing plants where butadiene was present, and in a case-control study, a large excess of leukaemia (odds ratio 9·4, 95% confidence interval 2·1–22·9) was associated with exposure to butadiene and not to styrene.

An International Agency for Cancer Research expert panel has concluded that butadiene is probably carcinogenic to man.[9] Responding to these same findings, the US Occupational Safety and Health Administration proposes a lowering of the occupational exposure standard from 1000 ppm to 2 ppm and the UK limit of butadiene is expected to be re-evaluated in 1996. Such actions are warranted as an important step in cancer prevention and we disagree with the view that animal data on butadiene should be ignored.

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Signs of falanga torture

SIR,—In falanga, the soles of the feet are beaten. It is practised world wide especially in the Middle-East.[1,2] We have observed clinical signs not previously ascribed to this form of torture but of importance in the diagnosis of sequelae. We examined 25 men and 5 women who claimed to have been beaten this way.

The padding of the balls of the heels and the medial and lateral anterior balls were examined. A ball was registered as "smashed" when palpation with a finger demonstrated loss of tissue between the skin and underlying bones (ie, the tuber of the calcaneus and the bases of the first and fifth proximal phalanges). Jumping or running can injure the ball of the heel.[3,4] Experiments on cadavers have confirmed these clinical findings.[5] During falanga, the soles are exposed to blunt trauma repeatedly and with extreme force, leading to damage of both the anterior and the posterior balls of the feet. We found such damage in 57% (17/30) of the victims. We have not found any descriptions of "smashed" anterior feet balls, and this sign may therefore be specific for falanga. However, since only half the victims had "smashed" foot balls, the absence of this sign cannot be used as a criterion of exclusion in cases requiring legal documentation.

A passive dorsal flexion of more than 70° in the metatarso-phalangeal joint of the big toe was interpreted as a lesion to the distal attachment of the aponeurosis into the deep transverse ligament.[6] Tearing of the fibres that hold the aponeurosis to the skin and to the heads of the metatarsal bones via the deep transverse ligament is a possible pathophysiological sequela of the oedema following falanga.[6] We found this sign in 53% (16/30), bilaterally in half the cases.

Fixation or instability of the tarsus, the tibio-fibular joints, and the interosseous ligament was demonstrated by evaluation of the passive translatoral movement in each joint separately. The elasticity of the interosseous ligaments was evaluated by moving the fibula anterior and posterior in relation to the tibia. Blows to the soles can affect the tarsal joints and the joints between the tibia and the fibula, leading to bindings and/or instability. We saw involvement of the tarsus in 25 cases (83%), and of the tibiofibular joint in 19 (63%).

Our study showed a high frequency of pathological findings in the clinical examination of victims who claimed exposure to falanga. Specific clinical examination makes the diagnosis of sequelae after falanga possible. However, the lack of pathological conditions does not exclude exposure to falanga since 4 subjects had none of the described findings.

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Complications of "ecstasy" misuse

SIR,—Dr Henry and colleagues' (Aug 15, p 384) account of the complications of "ecstasy" (3,4-methylenedioxymethamphetamine, MDMA) ingestion should alarm all anaesthetists. They will no doubt recognise the similar manifestations of (fatal) ecstasy ingestion and severe hyperthermia: muscle rigidity, trismus, hyperthermia, sinus tachycardia, sweating, cardiac arrhythmias, cardiac arrest, tachypnoea, cyanosis, metabolic acidosis, rhabdomyolysis, myoglobinuria, and disseminated intravascular coagulation. It is especially worrying that signs can develop several hours after ecstasy ingestion. These patients may come under the emergency care of anaesthetists either for resuscitation or for anaesthesia for trauma surgery. The complications seem to occur unpredictably and will need prompt action, with a treatment plan that is similar to that adopted by anaesthetists for treating severe hyperthermia, including the use of dantrolene.[1]

We are concerned, however, that in the absence of a history or biochemical evidence of ecstasy ingestion a diagnosis of severe hyperthermia will be wrongly made, with all its adverse consequences on future management of anaesthesia in such patients, and on their close relatives. There are medicolegal implications for the anaesthetist should the patient die; anaesthesia will probably be blamed. We therefore recommend that serum MDMA concentrations are measured in the admission blood sample of young adults who develop hyperthermia during anaesthesia. We are also concerned that hepatitis due to unknown ecstasy ingestion may be attributed to anaesthetic agents, especially halothane. Perhaps anaesthetic vapours should be avoided for patients on ecstasy and total intravenous anaesthesia used instead. It remains to be seen whether patients who have complications after

ecstasy use are also at higher risk than usual of severe hyperthermia from anaesthetic agents.

Although cardiac arrhythmias might be caused by ecstasy itself we think arterial blood should be immediately analysed to exclude hyperkalaemia and metabolic acidosis, and that high-percentage supplementary oxygen should be given on admission. Physicians and anaesthetists should work closely together in the management of patients presenting with acute severe complications of ecstasy misuse.

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SIR,—Dr Henry and colleagues provide some interesting case reports of deaths partly attributable to 3,4-methylenedioxymethamphetamine (MDMA, "ecstasy") and excessive exertion. However, they failed to give any estimate of how dangerous MDMA use is. In addition, the article and, especially, the subsequent media coverage were judgmental and scaremongering. Furthermore, use of the term "misuse" to describe use of MDMA throughout the article was inappropriate and suggestive of the motivation behind the article. The table of five accident victims in whom MDMA was identified was meaningless, especially since blood ethanol concentrations were not reported; high values of which are a far more important cause of accidents. How could one of the victims have contributed to the accident, since he was a passenger?

My main criticism of the article is that it fails to give an estimate of MDMA use and so the seven deaths cannot be seen in context. From the data presented one cannot determine if MDMA is any more dangerous than aspirin. For the public to make rational decisions about recreational drug use the incidence of death and serious morbidity needs to be known. When medical scientists allow their data to be uncritically used they reduce the chances of more serious messages being listened to.

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SIR,—Dr Woods and Dr Henry's report (Aug 1, p 305) of hyperpyrexia induced by 3,4-methylenedioxyamphetamine (MDA, "eve") raises an interesting point about the mechanism of thermogenesis caused by this agent and the related compound MDMA. Hyperpyrexia is ascribed to a central action at serotonin ($5HT_2$) receptors, but is treated (apparently successfully) with dantrolene, an agent with no central action. Although sweating is common after ingestion of MDMA,[1] severe heatstroke-like reactions (hyperpyrexia, rhabdomyolysis, myoglobinuria, and disseminated intravascular coagulation), although increasingly recognised, are rare and unpredictable.[2] Because hyperpyrexia may be an initiating factor for these complications,[3] understanding its pathogenesis is especially important for appropriate therapy.

Evidence that individuals with exertional heatstroke may have an underlying abnormality of muscle biochemistry, causing deregulation of myoplasmic calcium-ion homoeostasis similar to that in severe hyperthermia,[4] prompts the speculation that individuals with MDMA/MDA-induced hyperpyrexia may be similarly predisposed.[5] The genetic predispostion to severe hyperthermia has a heterogeneous basis, but mutations at the ryanodine receptor gene, *ryr1*, which codes for the calcium-ion release channel of skeletal muscle sarcoplasmic reticulum, underlie at least some cases.[6] Family studies of the inheritance of polymorphisms in *ryr1*, with the use of DNA markers, combined with standardised in-vitro muscle biopsy contracture tests, might be informative of MDMA susceptibility. Evidence for a genetically inherited metabolic deregulation of muscle calcium homoeostasis, which might be triggered by raised metabolic rate or raised ambient temperature, would endorse a policy of treatment of MDMA/MDA-induced hyperpyrexia with dantrolene.

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Serum cardiac troponin T in polymyositis/dermatomyositis

SIR,—Cardiac involvement has been reported in patients with polymyositis/dermatomyositis (PM/DM),[1] including congestive heart failure, pericarditis, myocarditis, and arrhythmia. As many as 30–50% of PM/DM patients have cardiac manifestations that resemble the degenerative and inflammatory changes present in skeletal muscles in postmortem examinations.[2] However, cardiac involvement is seldom clinically symptomatic,[2] and is not always detected by electrocardiogram (ECG). Moreover, the concentration of creatine kinase (CK) from skeletal muscle surpasses and masks that from myocardial CK; therefore, cardiac involvement should be assessed only from the ratio of CK-MB/CK.

To demonstrate cardiac involvement in PM/DM patients, we measured serum cardiac troponin T, which has been reported to be increased during the course of myocardial infarction, with an immunometric one-step sandwich assay.[3] The upper limit of normal was 0·25 ng/ml in the serum of 30 healthy volunteers (mean + 3 SD). Among the randomly selected sera from 30 PM/DM patients, 8 (27%) had troponin T above normal (mean [SD] 4·76 [4·86], range 0·38–27·25), whereas none among 25 rheumatoid arthritis patients, 28 systemic lupus erythematosus patients, or 30 healthy controls was positive for troponin T. The concentration of troponin T was not correlated with CK ($r = 0·254$). In the 8 patients, arrhythmia was detected in 1 patient only. No symptoms and/or findings of cardiac involvement were detected in the remaining patients. In 1 patient with 15 ng/ml troponin T, although no abnormal findings were detected by ECG and ultrasound, decreased thallium-201 accumulation in the left ventricular wall was detected, which became normal after high-dose prednisolone with a simultaneous decrease in serum troponin T to zero.

The cardiac involvement has a prognostic implication since congestive heart failure has been reported in 25–45% of patients with this disease.[1,2,4] Furthermore, no correlation of overall severity of the disease with the presence or absence of active myocarditis has been reported.[2] The assay of non-invasive and myocardium-specific troponin T, which indicates early cardiac involvement and dysfunction will, therefore, be helpful in management.

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