thorough cleaning to remove that contamination, sterility cannot be guaranteed. This could be crucial if, for example, the next patient required an episotomy while in the pool.

We argued that there was a duty of care to ensure that the pools users (and their babies) were exposed to the least risk possible of acquiring a blood-borne virus infection from a previous user. These concerns have recently found substance in the transmission of hepatitis B in an unexpected situation.[1] We considered that a birthing pool is likely to be heavily contaminated with infectious material from the mother. In the case of an HIV-infected mother, the water might pose a risk to mother and infant if the infant is also HIV-infected. Where the infant is not infected, it seems illogical to allow the baby to be delivered in the potentially infectious water of a pool. We also wished to protect midwives and other attendants from blood-borne virus infection. It seemed that handling sharp instruments in the environment of the birthing pool would be hazardous and that the central dogma of infection control of blood-borne viruses (containment of blood loss) could not be achieved in practice.

Screening for blood-borne viruses, for purposes of infection control, is not without precedent. Maintenance haemodialysis and organ and tissue donation are prominent examples. Nevertheless we are being criticised for this policy in the professional and lay press and by midwives, patients and pressure groups. The matter is scheduled for discussion by the national Expert Advisory Group on AIDS. The time has come for an open debate.

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Near-fatal hyponatraemic coma due to vasopressin over-secretion after "ecstasy" (3,4-MDMA)

SIR—A 20-year-old woman took an ecstasy tablet at a "rave" party. Previously she had used ecstasy without serious adverse effects. She experienced some unpleasant alterations of perception and was encouraged to drink a large quantity of water. She subsequently became stuporose and was incontinent of urine. She may have had a fit. She was taken to hospital. On examination 9 hours after ingestion of the tablet, she was talking unintelligibly and lying prostrate or in a Buddha-like posture with little response to pain or commands. Her pupils were dilated with a diminished light reflex. Pulse, temperature, and blood pressure were normal. Serum sodium concentration was 112 mmol/L (normal range 135–145 mmol/L). Serum osmolality was 238 mosmol/kg. Urine sodium was 112 mmol/L. Urine osmolality was 256 mosmol/kg. Plasma arginine vasopressin was 4·5 pmol/L, which is diagnostic of vasopressin excess in this context. Computed tomographic brain scan showed cerebral oedema. 3,4-methylenedioxymetamphetamine (ecstasy) was detected in urine. The patient was thought to be at imminent risk of coning. She was ventilated to decrease cerebral oedema and given mannitol and dexamethasone intravenously. 24 hours later, serum sodium was 120 mmol/L and she was extubated. By day 4, serum sodium had risen to 131 mmol/L and she had largely recovered. On the follow up 2 months later, she reported unpleasant flashbacks and anxiety symptoms but was otherwise well.

The arginine vasopressin and sodium/osmolality levels indicate that vasopressin release was the primary pathogenetic mechanism rather than water intoxication, but it is likely that excess water ingestion, a common practice at raves, contributed to its severity.[1] Four less severe cases of ecstasy-induced hyponatraemia have been reported in the UK.[2-4] Arginine vasopressin was not measured in these cases. Otherwise we have found no evidence that drugs of the amphetamine class can cause inappropriate vasopressin secretion in previous reports. It is curious that no cases have been reported in other countries. The effect may be direct or mediated by some other mechanism. Experimental research on the effects of ecstasy is legally and ethically constrained by drug toxicity. Although this may be an unusual reaction, we emphasise the importance of urgent electrolyte estimation together with attention to fluid balance in persons presenting unwell with a history of ecstasy ingestion in order to detect either this or the more common hyperpyrexia syndrome.[5] If in doubt, an urgent computed tomogram scan of the head should be done. Hyponatraemia after ecstasy use requires early recognition as the vasopressin level may remain high for some time after ingestion of the drug, leading to worsening hyponatraemia and possible death.

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Gas cooking and respiratory health in women

SIR—Jarvis and co-workers (Feb 17, p 426),[1] report that in young women, but not in men, the use of gas for cooking is associated with an increased risk of respiratory symptoms and impaired lung function. We have also addressed this issue with data for 441 men and 506 women aged 20–44 years, from the Paris and Montpellier centres of the European Community Respiratory Health Survey (ECRHS). We also found no significant relation between gas cooking and respiratory symptoms in men, whereas in women gas cooking was associated ($p<0·05$) with an increased risk of awakening with shortness of breath), with having asthma, with taking asthma medication (odds ratios 2·1, 3·3, and 4·9 respectively, after adjustment for age, smoking, and town of residence). There was no association between gas cooking and allergy (atopy, IgE, nasal allergies), and the relations between gas cooking and symptoms were similar in atopic and non-atopic women.

Jarvis and co-workers did not report any findings about usual cough or phlegm. In our study population, among women gas cooking was associated with increased frequency of usual morning phlegm (OR=1·9, $p=0·04$). The relation with usual morning cough was not significant (OR=1·5,