

tions to alter medical practice. These have included the suggestion that neonatal intensive care should be stopped and telling surgeons not to do certain operations on children. The auditor should have confined the report to aspects of structure and process without reporting on areas of clinical judgment, medical guidelines, or protocols. We share the concerns of our colleagues about the dangers of protocols.

If similar audits are usefully to be conducted across Britain we advocate thorough research into the local situation and medical practices and full medical consultation.

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1 Dillner L. Children poorly cared for in hospitals. *BMJ* 1993;306:354. (6 February.)

2 Audit Commission. *Children first: a study of hospital services*. London: HMSO, 1993.

Drug smuggler's delirium

EDITOR,—Further to Punit S Ramrakha and Ian Barton's editorial about drug smuggler's delirium we wish to report a case and further clues to diagnosis and treatment.

A 31 year old woman was admitted in deep coma (Glasgow coma score 3/15) complicated by a generalised tonic-clonic seizure and ventricular tachycardia. She was intubated and mechanically ventilated. There were no signs of intravenous drug misuse. A nasogastric tube was inserted and activated charcoal given. Toxicological screening showed cannabinoids and cocaine (Abbott's ADx fluorescence polarisation assay). Confirmation and quantification of cocaine and its major inactive metabolite benzoylecgonine was done by gas chromatography and mass spectrometry (table).²

Cocaine and benzoylecgonine concentrations (mg/l) in urine and plasma samples

Hours after admission	Cocaine	Benzoylecgonine	Benzoylecgonine: cocaine
<i>Urine</i>			
	2030	2220	1.09
5	5920	9150	1.55
24	580	7070	12.18
72	ND	10.5	>100
5 days	ND	1.5	>100
6 days	ND	0.5	>100
<i>Plasma</i>			
	42.8	8.2	
2	20.4	4.4	
15	1.2	2.4	
24	ND	1.3	
36	ND	0.6	
48	ND	0.3	

ND=Not detectable.

High plasma cocaine concentrations, as in our patient, are rarely seen because of cocaine's short half life. Concentrations above 1 mg/l are usually associated with toxicity, although great variety has been noted in fatal overdoses, ranging from 0.1 to 20.9 mg/l.³ As cocaine exhibits first order kinetics over a wide dose range the time since the last dose can be roughly determined by examination of urinary ratios of benzoylecgonine to cocaine. A ratio of less than 100 in urine suggests that cocaine was ingested less than 10 hours before the sample was collected.⁴ The fact that this ratio was less than 100 after 24 hours in our patient raised suspicion of a body package.

A history taken from a relative indicated that the patient had been travelling regularly to South America for two years; when she was about to be apprehended by the customs authorities she

ingested a condom at Heathrow airport. Abdominal x ray examination, ultrasonography, and contrast study of the bowel failed to show a drug condom. Enteral feeding was started within 24 hours. After 68 hours an intact condom containing 4 g of cocaine was found in the stool. The patient's condition improved, and she was discharged after two months. Neurological sequelae were still present one year after diagnosis—namely, oligophrenia, ataxia, and slurred speech.

In contrast to body packers, body stuffers hurriedly ingest packages to conceal an illicit drug and avoid arrest. These packages are especially prone to leakage or rupture.^{5,6} Urinary drug concentrations and the ratio of benzoylecgonine to cocaine can be helpful.^{4,5} Recent reports also show the benefit of alkalinisation of gastric fluids to enhance hydrolysis to benzoylecgonine.⁷ In addition to activated charcoal whole body irrigation with polyethylene glycol-electrolyte lavage solution or enteral feeding has been used successfully.^{5,7} Sodium bicarbonate and aluminium hydroxide-magnesium hydroxide can be used to achieve further alkalinisation of gastric fluids.⁷ The type of material and method of wrapping also determine the risk of liberation of cocaine and subsequent toxicity.² This emphasises the importance of obtaining a detailed history.

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1 Ramrakha PS, Barton I. Drug smuggler's delirium. *BMJ* 1993;306:470-1. (20 February.)

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EDITOR,—Punit S Ramrakha and Ian Barton discuss cocaine intoxication in people who swallow packages of drugs or conceal them in the rectum or vagina to smuggle them.¹ Cocaine has effects on sodium channels that result in its anaesthetic action. The systemic effects of cocaine are due to reduced reuptake of catecholamines at peripheral sympathetic nerve endings and at central pre-synaptic sites. The picture of toxicity is therefore one of sympathetic overactivity. Lignocaine is an illogical choice as an antiarrhythmic in cocaine intoxication. As another local anaesthetic it will potentiate the lowering of the seizure threshold and may precipitate convulsions. Propranolol is also an illogical choice: β blockade alone may result in unopposed α activity with worsening of coronary vasoconstriction.² Myocardial ischaemia and infarction during cocaine intoxication are not confined to patients with known coronary artery disease.³

Two logical choices are available for controlling the cardiovascular effects of cocaine toxicity. Labetalol, with its combined α and β blockade, has been shown to be of use.⁴ Secondly, esmolol, a β_1 selective (cardioselective) adrenergic blocker with a rapid onset and short duration of action (elimination half life roughly nine minutes), has also been used with success.⁵

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EDITOR,—It is all very well to suspect cocaine intoxication in travellers with fever and a bizarre mental state (or coma) and to follow the excellent advice given by Punit S Ramrakha and Ian Barton,¹ but please make blood films and check the blood glucose concentration and while waiting for the results start giving intravenous quinine in glucose 5%. The relatives of a patient who dies of missed cerebral malaria are much more likely to sue for negligence than a drug smuggler is likely to complain of having received unnecessary quinine. Patients from west Africa may well have cerebral malaria and so, I suspect, may those from South America.

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Measuring outcome of perinatal care

EDITOR,—Hart points out the lack of adequate measurement of the outcome of perinatal care, quoting the example of cerebral palsy and suggesting that variations in rates of cerebral palsy could become an important measure of the quality of obstetric services.¹ This approach has several difficulties, not least that only a small proportion of cerebral palsy originates in the perinatal period. In a prospective study of 43 437 full term infants Naeye *et al* estimated that only 14% of quadriplegic cerebral palsy was attributable to birth asphyxia.² There is also no nationally agreed definition of cerebral palsy.

A more useful measure of intrapartum care is the incidence of neonatal hypoxic-ischaemic encephalopathy (a clearly defined clinical syndrome)^{3,4} or the presence of neonatal seizures in the first 48 hours in term infants.⁵ While these conditions are not always associated with other evidence of intrapartum "asphyxia," they seem to provide a better indicator of perinatal care than later outcomes such as cerebral palsy. A recent study documented a significant fall in the incidence of hypoxic-ischaemic encephalopathy over 13 years in a large district general hospital maternity unit, which was accompanied by falls in both the stillbirth rate and neonatal mortality.⁶

Another approach to monitoring perinatal care has been advocated recently by the Audit Commission in *Children First*, a study of hospital services.⁷ A set of outcome measures is recommended for all surviving very low birthweight babies as part of a national data collection exercise. The seven indicators recommended are cerebral palsy, developmental delay, impairment of vision, impairment of hearing, growth, epilepsy, and other serious physical conditions. While these conditions are not necessarily directly attributable to perinatal events, they have an increased prevalence in surviving very low birthweight infants.

Until there is a nationally agreed set of measures