Letters to the Editor

Paracetamol cardiotoxicity

Sir,

I read with some interest the case report of a 29 year old man with evidence of paracetamol-induced cardiotoxicity in the absence of hepatic encephalopathy reported by A. Armour and S.D. Slater.¹ It has not been our routine practice to perform electrocardiograph recordings of patients having overdosed on paracetamol alone but we will now include this as part of our initial investigations.

I was, however, surprised that treatment with acetylcysteine was withheld from this man because he had taken the overdose 23 hours previously. There is now evidence that acetylcysteine can be safely given up to 24 hours post-ingestion² and in some cases it has been given up to 36 hours post-ingestion.³ In my experience, these patients are usually fairly unreliable historians with a view to the time of ingestion and it is probably best to err on the side of treatment if in doubt. One group of people who seem to show increased susceptibility to the toxic effects of paracetamol are those patients with increased hepatic enzyme induction, for example, chronic alcoholics and patients on anti-convulsant therapy. Interestingly, the British National Formulary recommends treating these patients at serum paracetamol levels at 50% of those recommended for standard treatment with acetylcysteine.

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Intratracheal antibiotics for broncho-pneumonia

Sir,

Both the leading article on 'The role of tracheostomy in the adult intensive care unit'¹ and the article by R.N. Gunawardana² refer to the difficulty in treating broncho-pneumonia which complicates tracheostomy. When working on tetanus neonatorum treated by curarization and intermittent positive pressure respiration via tracheostomy tube, a secondary broncho-pneumonia was a fatal complication in these infants irrespective of what or how these antibiotics were given until a 4 ml aqueous solution of penicillin and colistin was instilled 4 hourly down the tracheostomy tube. The result was dramatic with an immediate and maintained drop in overall mortality from 25% to 3%.

The efficacy and wider application of this method of topical application of antibiotics was emphasized subse-

quently when an undernourished 5 year old boy who had measles was admitted with severe laryngeal obstruction associated with bilateral cavital pneumonia. Staphylococci were isolated from the copious pus aspirated at tracheostomy. Antibiotics in aqueous solution instilled 4 hourly down the tracheostomy tube together with systemic antibiotics resulted in recovery except for a number of residual thin walled cysts due to the staphylococcal abscesses in the lungs.

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Maternal paroxysmal supraventricular tachycardia treated with adenosine

Sir,

A 30 year old woman at 20 weeks gestation presented with a 6 hour history of palpitations. She had a 30 minute episode the previous day which had terminated spontaneously. She felt well generally and could still feel the baby kicking.

On examination, she appeared comfortable with a pulse rate of 140 b.p.m. and a BP 110/70 mmHg. There were no signs of cardiac failure. Electrocardiograph confirmed an atrioventricular nodal re-entry tachycardia (AVNRT). This did not respond to vagotonic manoeuvres and intravenous adenosine was administered as a 9 mg i.v. bolus of adenosine through a large cannula, flushed with normal saline. The arrhythmia resolved within 20 seconds and she remained in sinus rhythm. She was discharged the following day and the rest of the pregnancy was unremarkable.

Adenosine is an endogenous purine nucleoside found in all cells of the body. It is a potent inhibitor of atrioventricular (AV) nodal conduction, and is useful in the termination of supraventricular tachycardia (SVT).¹ SVT during pregnancy is not uncommon, and may even be increased.² In view of its short half-life (0.6–1.5 seconds),¹ the use of adenosine in pregnancy for the treatment of SVT is theoretically attractive. If the patient is severely haemodynamically compromised, synchronized DC cardioversion is the treatment of choice. However, the majority of SVTs are well tolerated and require some form of pharmacological intervention.

Verapamil, a calcium channel blocker, has been widely used in the acute management of SVT. Its main problems

are exacerbation of systemic hypotension, worsening of cardiac failure, serious bradyarrhythmias (especially when used in conjunction with beta-blockers) and when given inappropriately for broad-complex tachycardias which are ventricular in origin.³ These can be further compounded by its relatively long half-life, compared to adenosine, of approximately 30 minutes. The drug also crosses the placenta and can result in fetal bradycardia. It has been incriminated in sudden intrauterine fetal death.⁴ Beta-blockers are less successful at terminating SVTs, and can be associated with systemic hypotension, transient neonatal hypoglycaemia and hypotonia.⁴

Adenosine acts rapidly, is short acting and the common side effects (of flushing, dyspnoea, chest discomfort and bradyarrhythmias) usually resolve quickly. The use of adenosine during pregnancy needs further assessment, particularly with respect to its effects on the fetus. Our report suggests that adenosine is effective in pregnancy, but we have no information on the cardiovascular response to treatment in the fetus. We suggest that during treatment of an arrhythmia in a pregnant woman, monitoring of the fetal heart rate is important, if available and when the situation is not immediately life-threatening to either mother or child.

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Further information may be obtained from Mrs J.M. Coops, Fellowship of Postgraduate Medicine, 6 St Andrew's Place, London NW1 4LB (Tel: 071-935 5556), to whom applications should be made, giving a concise summary of the research project, the sum requested and a curriculum vitae of the applicant.

Closing date for applications: 30 August 1993