for investigation of palpitations and postural hypotension. He also suffered from Parkinson’s disease but was well controlled on treatment.

The positive findings on examination included the classical features of Parkinson’s disease and a postural drop in blood pressure. The latter quickly resolved on withdrawal of his diuretic therapy. Two days after admission he started vomiting, initially four times on one day and then intermittently over the following 10 days. This usually occurred in the mornings. He was not receiving any new medication.

Fibreoptic gastroscopy showed multiple erosions in the lower part of his stomach and first part of the duodenum. His oesophagus appeared normal. Histology confirmed an active atrophic gastritis with many helicobacter-like organisms present. A full blood count, biochemical screen and liver function tests were all normal. It transpired that every morning he washed himself on the ward with Hydrex. However, he also used this preparation as a mouth wash and then swallowed it. He said that he felt nauseated each time he did this.

He was advised to discontinue this practice and was given ranitidine. He made a full recovery with no further vomiting and repeat endoscopy after 6 weeks revealed resolution of his mucosal erosions with some mild residual antral gastritis.

Chlorhexidine is a bisbiguanide disinfectant which is effective against a wide range of vegetative Gram-positive and Gram-negative bacteria. It is most active at a neutral or slightly acidic pH. Hydrex is a 4% solution of chlorhexidine gluconate in detergent that is used in pre-operative skin preparation and hand washing. Skin sensitivity, transient taste disturbance and oral desquamation have occasionally been reported with a variety of preparations of chlorhexidine.

There has been one report of an acute hepatitis and liver necrosis following ingestion of chlorhexidine gluconate in a suicide attempt. Because it is irritant, it is recommended that it should not be used on the brain, meninges, eyes, middle ear or other sensitive tissues.

There have been no previous reports of chlorhexidine-induced gastritis. This is presumably because the taste and consistency of concentrated preparations of chlorhexidine render them sufficiently unpalatable to discourage ingestion in the majority of cases. We have informed the manufacturers of Hydrex of our unusual observation in the hope that a recurrence of this problem can be avoided by providing a more visible written warning on the container.

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References

The cardiovascular effects of beta adrenergic agonist drugs administered by nebulization

Sir,

I read with interest the report of Flatt et al.1 reporting the cardiovascular effects of inhaled bronchodilators. They clearly demonstrate the greater cardiovascular effects of fenoterol 5 mg compared with equal doses of salbutamol and terbutaline. They conclude that this is due to ‘greater beta1 adrenoceptor stimulation following fenoterol’. However, I think a more satisfactory explanation of their findings is that fenoterol has a greater systemic bioavailability than salbutamol or terbutaline (i.e. more fenoterol is absorbed).

The authors have assumed that the tachycardia and positive inotropic effects of the drugs are primarily due to cardiac beta1 adrenoceptor stimulation. In studies of human subjects and tissues, the chronotropic and inotropic effects of salbutamol have been shown to be purely due to cardiac beta2 adrenoceptor stimulation. Consequently, all the cardiovascular effects in the study of Flatt et al. could be attributable to cardiac beta2 adrenoceptor stimulation and the greater responses seen after fenoterol could be due to greater absorption. Conclusive evidence of greater absorption of fenoterol comes from a previous study published by this group of workers,1 where fenoterol by metered dose inhaler produced more hypokalaemia than equal doses of salbutamol. Hypokalaemia induced by beta-agonists is purely due to beta2 adrenoceptor stimulation.

The references cited by Flatt et al. to show beta1-mediated effects of fenoterol also assume that chronotropic and inotropic effects are not mediated by beta2 adrenoceptors whereas evidence exists to disprove that assumption in the species studied, i.e. in man and guinea pigs.

Although the findings of differential cardiovascular effects of equal doses of these commonly used bronchodilators are important I think it should be recognized that probably all the cardiovascular effects can be attributed to beta2 adrenoceptor stimulation. Consequently in order to avert these important side effects of bronchodilator treatment it would not be necessary to develop an agent with ‘greater beta2 selectivity’ but to develop a beta2 agonist which is less well absorbed.

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References
Acute tubular necrosis induced by coronary thrombolytic therapy

Sir,
It is well recognized that streptokinase infusion may be accompanied by systemic hypotension, and indeed, occasionally cardiovascular collapse. Cases of renal failure associated with the use of thrombolytic drugs can thus be anticipated. A 74 year old lady presented with an acute inferior myocardial infarction. She had a blood pressure of 110/70 mmHg, 1st degree heart block with a cardiac rate of 96/min and there was no evidence of left ventricular failure. Her renal biochemistry was within normal limits and her only medication was tranilcypromine. Streptokinase infusion (1 mega-unit over 1 hour) was commenced on the coronary care unit but was accompanied by severe hypotension (40 mmHg systolic) after 10 minutes, followed by a grand mal convulsion. Thrombolytic therapy was discontinued immediately and dobutamine infused, initially at a dose of 20 μg/kg/min. Despite inotropic support the patient remained significantly hypotensive for 2 hours although the cardiac rhythm remained unchanged.

Acute oliguric renal failure developed over the following 48 hours and this necessitated a period of peritoneal dialysis treatment. Renal recovery was delayed and she was subsequently transferred to our unit for further management. Investigations, including a radio-labelled renal 1st circulation study, were compatible with a diagnosis of acute tubular necrosis, and there was also evidence of underlying narrowing of the left renal artery. The patient is now well but she still has moderate impairment of renal function.

Allergic side effects of streptokinase are well recognized and may include hypotension and a serum sickness type reaction which has been observed in association with transient impairment of renal function. Although significant hypotension (systolic blood pressure <80 mmHg) is observed in approximately 10% of patients with acute myocardial infarction who receive streptokinase at standard infusion rate (1.5 million IU/h), the effect is usually short-lived (<10 minutes) and inotropic support rarely required. However, hypotension which is refractory to catecholamines may complicate the administration of streptokinase to patients with severe left ventricular dysfunction, and it has been recommended that such haemodynamically unstable subjects should receive the infusion at a much slower rate (200 IU/kg/min) – this perhaps should also be the case when treating some elderly patients. As similar hypotensive effects may complicate therapy with other thrombolytic agents, it is unlikely that a specific action of streptokinase is aetiologically important.

We have shown that significant renovascular narrowing is a common finding in middle-aged and elderly patients who have evidence of generalized atheroma. Such renal artery pathology may be associated with hypertension or may remain clinically occult, particularly in elderly patients. These should be identified as the patients most likely to have kidneys that will be vulnerable to ischaemic damage if systemic hypotension supervenes. Efforts to minimize renal hyperperfusion in such patients are necessary if acute renal failure is to be avoided, and a slower rate of streptokinase infusion may need to be considered in the at-risk (especially elderly) population requiring coronary thrombolysis.

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