Welsh trial with plastic cards of a simpler type[3]), and the technology has functioned well.

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Drug Points

Oramorph in diabetes

Dr K COWCHER, Mr E MCELLIGOTT, and Dr G W HANKS (Royal Marsden Hospital, Sutton, Surrey SM2 5PT) write: Recent experience with a patient with advanced cancer and insulin dependent diabetes has brought to our attention the sugar content of Oramorph, a recently introduced formulation of oral morphine sulphate solution. Oramorph is manufactured in two strengths, 10 mg in 5 ml and 100 mg in 5 ml, and the low strength version has a taste mask containing sucrose and corn syrup; 10 ml of this solution of Oramorph contains about 3·2 g sugar (sucrose, glucose, and maltose). The concentrated solution does not contain sugar.

Morphine sulphate in solution is used when patients are first starting to take oral morphine and the dose is titrated to control their pain. Initially a low strength solution is used, and in this hospital we use the 10 mg in 5 ml strength of Oramorph. Patients may take up to 20 ml every four hours and occasionally more before being prescribed a more concentrated solution. The sugar content of a total daily dose of 120 ml is 38·4 g. This dosage may result in a small but appreciable rise in blood sugar concentration in a diabetic patient. Most patients need at some stage to change to a more concentrated solution of morphine, and neither the in house formulation used at the Royal Marsden Hospital nor the high strength Oramorph solution contain sugar.

It is important that the relative sugar content of these solutions is known because a change from one to the other may affect patients with cancer who also have diabetes. We understand from the manufacturers of Oramorph that the sugar content of the solution will be clearly labelled in future.

Ischaemic chest pain after abuse of a topical nasal vasoconstrictor

Messrs H B WHITTET (Radcliffe Infirmary, Oxford) and D VEITCH (Royal National Throat, Nose, and Ear Hospital, London WC1X 8DA) write: The excessive use of nasal decongestants may result in disabling local complications such as rhinitis medicamentosa.[1] Drug interactions from systemic absorption have also been described,[2] and fatal myocardial infarction has been caused through the use of phenylephrine eyedrops.[3] We highlight here a similar systemic complication resulting from the overuse of sympathomimetic nasal drops obtained "over the counter" and taken inappropriately for nasal obstruction.

A 50 year old woman was admitted with acute onset of palpitations, breathlessness, and central chest pain radiating to the neck and left arm. She had a tachycardia of 140 beats/min and was mildly hypertensive with a blood pressure of 150/100 mm Hg. An electrocardiogram confirmed a sinus tachycardia with evidence of ST depression. There was no history of cardiovascular disease and she was taking no medication. She had, however, been suffering from troublesome nasal obstructive symptoms from which initial transient relief had been obtained with proprietary nasal decongestants, notably 1% ephedrine and 0·1% xylometazoline (Otrivine) nose drops. At the time of admission she was using the drops every half hour, and examination confirmed nasal appearances of rhinitis medicamentosa. There was no evidence of myocardial infarction, and the tachycardia, blood pressure, and electrocardiographic changes reverted to normal when the drops were stopped. A subsequent exercise electrocardiogram showed no evidence of underlying coronary arterial disease, and the nasal symptoms were controlled by the use of nasal corticosteroids and surgical trimming of both inferior turbinates.

There are two groups of nasal vasoconstrictors in current use: the sympathomimetic agents ephedrine and phenylephrine and their imidazoline derivatives oxymetazoline and xylometazoline.[4] The latter are longer lasting, but both may cause rebound nasal obstruction and rhinitis medicamentosa when abused. This case illustrates that potentially fatal myocardial systemic side effects may also be produced in an otherwise healthy patient when used to excess.

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Flumazenil causing convulsions and ventricular tachycardia

Drs BRADLEY MARCHANT, RICHARD WRAY (St Helen's Hospital, Hastings), ANDREW LEACH, and MOHAN NAMA (Royal East Sussex Hospital, Hastings) write: We report a case of flumazenil induced convulsion and ventricular tachycardia in a patient who had taken overdoses of a tricyclic antidepressant and a benzodiazepine.

A 57 year old woman was found unrousable in bed by her husband. She had been well apart from depression, for which she had been receiving lorazepam 2 mg/day and amitriptyline 75 mg/day, but no empty bottles were found at home. On admission she was deeply unconscious with no response to pain and absent tendon reflexes. Pupils were mid-dilated and non-reacting. Peripheries were cool and systolic blood pressure was 55 mm Hg. She was bradypnoeic with a poor tidal volume and a core temperature of 34·4°C. The cardiac monitor showed a nodal rhythm and frequent multifocal ventricular premature beats, ventricular couplets, triplets, and salvos of ventricular tachycardia. Supportive treatment was started and in view of the depth of the coma, probable hypoventilation, and history of presumptive benzodiazepine ingestion a trial of flumenazil was made. It was given intravenously, more slowly than indicated in the datasheet, to reduce the likelihood of unmasking the central nervous system stimulatory effects of the tricyclic antidepressant. Two 50 μg boluses were given, each in 30 seconds two minutes apart, followed by 100 μg every 3-4 minutes to a total dose of 500 μg. Respiratory rate increased to 20/min accompanied by increased depth of respiration but with no change in conscious level. Five minutes later she developed grand mal convulsions, followed in 15 seconds by ventricular tachycardia, with no cardiac output, which reverted to sinus rhythm with direct current cardioversion. The convulsion ceased spontaneously after 30 seconds but recurred within two minutes, again followed by sustained ventricular tachycardia. This pattern of events recurred, a total of nine cardioversions being made. Convulsions were eventually terminated with intravenous diazepam and thiopentone. Supportive treatment was continued in the intensive care unit and the patient recovered over 48 hours. A history of lorazepam and amitriptyline overdose was later obtained.

Flumazenil is a specific benzodiazepine antagonist used to reverse the central sedative effects of benzodiazepines. It can cause generalised convulsions in patients whose epilepsy is controlled by benzodiazepines. Caution is also advised in intentional overdose because of the risk of precipitating the toxic effects of other psychotropic drugs,[1] but reports of this actually occurring are rare.[2] Among 45 patients treated with flumazenil for self poisoning,[3 4] only one (who had taken clobazam and maprotiline) had a generalised convulsion.[4]

Self limiting ventricular tachycardia has been reported in a patient who had taken an overdose of oxazepam and chloral hydrate.[5] In our case the ventricular tachycardia was sustained and associated with cardiac arrest. The proximity of the convulsion to the arrhythmia and the repetition of the sequence suggest that the ventricular tachycardia was causally linked to the convulsion. Grand mal convulsions produce raised concentrations of circulating catecholamines and possible hypercapnia and hypoxia,[6] and these, in the presence of cardiac sensitisation by the tricyclic antidepressant, were the most likely precipitants of the arrhythmia. Another factor might have been the loss of the sympathetic damping effect of the lorazepam after flumazenil.

When a combined benzodiazepine and tricyclic antidepressant overdose is suspected the advantages of benzodiazepine reversal are outweighed by the risks, and consequently flumazenil is contraindicated.

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Manic depression induced by dapsone in patient with dermatitis herpetiformis

Dr DAVID GAWKRODGER (Royal Hallamshire Hospital, Sheffield S10 2JF) writes: Following the observation of Carmichael and Paul[1] and the comments of Daneshmend[2] on the idiosyncratic reaction to dapsone I report a further case.

About six months after starting dapsone 50 mg daily a 35 year old man with a three year history of dermatitis herpetiformis (proved by biopsy) developed symptoms clearly related to ingestion of dapsone. Within two to three hours of taking dapsone by mouth he developed headaches, had feelings of depression and anger, and became argumentative and upset. Without the dapsone he did not have these symptoms, but the eruption returned within two days despite his gluten free diet. When he changed to sulphapyridine 500 mg four times a day these symptoms disappeared almost immediately, and after six months of sulphapyridine treatment he had had no recurrence. He had no history of psychiatric disorder. This patient's symptoms were similar to those reported by others.[1 3] They occurred with a low dose of dapsone, and, unlike those described by Fine *et al*,[3] were not also present with sulphapyridine.

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