colder solution. Warming lignocaine may reduce the latent period as a consequence of temperature related changes in the pKa of lignocaine,⁹ resulting in a faster onset of neuronal blockade, inhibiting impulse conduction before the noxious stimulus is fully appreciated.

A similar theory has been proposed for the reduction in the pain of injection after the alkalisation of local anaesthetic solutions.¹⁰ Indeed, it has been suggested that bicarbonate may be added to reduce the discomfort associated with injection of local anaesthetic agents.¹¹ However, unlike the relatively simple process of warming, this entails mixing the two solutions, resulting in a reduction of solubility and possibly a reduction in stability.¹²

Although the technique of warming lignocaine to 37°C before injection has been adopted by dental practitioners, it has unfortunately not received widespread acceptance by the medical profession. We strongly recommend that all practitioners should adopt this technique to minimise patients' discomfort.

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Purging with paracetamol: report of four cases

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The drive to lose weight is paramount in patients with eating disorders and overrides any concerns about the damaging physical consequences that may ensue. The commonest methods used to control weight are vomiting and abuse of laxatives; the dangers of these methods have been well documented. Often medication is abused in the pursuit of weight loss: insulin may be omitted¹ and metabolic stimulants such as thyroxine² taken in large quantities. We describe titrated self poisoning with paracetamol in order to induce nausea and vomiting.

Case reports

Case 1—A 22 year old traffic warden with bulimia nervosa (body mass index 30.1 (weight in kilograms divided by square of height in metres)) as defined by the *Diagnostic and Statistical Manual of Mental Disorders, Third Edition, Revised* (DSM III-R) gave a history of habitually taking between 10 and 20 tablets of paracetamol on a weekly basis to induce vomiting after large binges. She described taking the tablets before going to bed and awakening about six hours later with severe nausea followed by vomiting.

Case 2—A 19 year old secretary fulfilled DSM-III-R criteria for bulimia nervosa (body mass index 21.3). She reported taking around 30 tablets of paracetamol at least once a week, which led to immediate vomiting. She described taking paracetamol as the most reliable method of inducing vomiting known to her and one that she reserved for unusually large binges.

Case 3—An 18 year old cleaner (body mass index 19.5) had excessive weight concerns but no objective binges. When feeling very overweight, which she estimated was once every two weeks, she took eight tablets of paracetamol to induce vomiting. This also stopped her eating as she then felt nauseous and generally unwell.

Case 4—A 21 year old hospital orderly met DSM-III-R criteria for bulimia nervosa (body mass index 19.2). She abused paracetamol fortnightly taking up to

14 tablets. Vomiting occurred three to four hours later. She valued the associated symptoms of nausea and anorexia as they reduced the likelihood of her bingeing next day.

Comment

These cases highlight a previously unreported form of paracetamol abuse—persistent titrated self poisoning. Deliberate self harm with paracetamol is common and produces a range of toxic effects which lead to at least 100 deaths a year in the United Kingdom.³

Our first two patients discovered the emetic properties of paracetamol after an overdose, but none of the patients presented acutely with paracetamol poisoning. In case 2 subsequent use of paracetamol produced vomiting immediately after ingestion. This may have been due to a conditioned taste aversion to paracetamol after the earlier overdose as appreciable absorption was unlikely. In the other cases vomiting occurred later, possibly due to acute gastric irritation, which produces vomiting within a few hours of overdosage.⁴ In serious self poisoning vomiting as a result of hepatotoxicity can occur up to 24 hours later.⁴

Paracetamol is excreted largely as its conjugate, glucuronide and sulphate. In chronic use glucuronidation dominates, proportional to the dose of paracetamol. This adaptive mechanism allows efficient metabolism of chronic doses of up to 12 g/24 h⁵ and may account for the apparent lack of serious medical consequences despite the potentially lethal doses used. Cognitive behavioural treatment of the eating disorder, supplemented with information about the medical consequences of paracetamol abuse and behavioural goals to limit consumption, proved successful in modifying this self poisoning behaviour in these patients. We suggest that patients with an eating disorder should be questioned about analgesic abuse during their initial assessment.

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