Comment

The toxic shock syndrome was first reported in seven children in 1978[2]; in five of these *Staphylococcus aureus* was isolated from various sites. The occurrence of toxic shock syndrome in menstruating women was first reported in 1980[3]; since then 299 cases have been reported in the United States with 25 deaths.[4] About 95% of these cases have occurred in menstruating women, all of whom had been using tampons. *Staphylococcus aureus* has been isolated from vaginal swabs in more than 90% of cases; blood cultures are always sterile. Our patient had all the features of the toxic shock syndrome, and phage group 1 *Staph aureus* was isolated from her vagina and from the tampon she was using.

The occurrence of scarlatiniform rashes in staphylococcal infections is well known and many of the features of toxic shock syndrome occur in staphylococcal septicaemia; in toxic shock syndrome, however, the organisms have not been found in the blood stream. It has been postulated that staphylococci in the genital tract produce a toxin which causes the syndrome[4]; the nature of this toxin has yet to be elucidated. Because we saw her before the toxic shock syndrome had been associated with tampons, we did not attempt to see if the staphylococcus produced a toxin.

The risk of toxic shock syndrome may be reduced by using tampons intermittently rather than continuously during a menstrual period. The condition should be managed by intensive fluid replacement, and antistaphylococcal antibiotics should be administered after appropriate cultures have been obtained. It is recommended that women who have had an episode of toxic shock syndrome should not use tampons for several menstrual cycles.

I thank Dr H Pullen for permission to report this case admitted under his care.

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Department of Infectious Diseases, Seacroft Hospital, Leeds LS14 6UH
PAUL HOLT, MRCP, senior registrar

Theophylline and depression

The cerebral stimulant effects of the bronchodilator theophylline are well known. Nevertheless, we have recently seen two cases of severe depression with this drug, a paradoxical and unreported reaction.

Case reports

(1) A 19-year-old asthmatic girl (weight 48 kg) had theophylline 225 mg twice daily added to her standard regimen of inhaled salbutamol and beclomethasone because of increasing bronchospasm. Over a period of one month she unaccountably became depressed and irritable having previously had a stable personality. Her symptoms disappeared promptly on withdrawal of theophylline alone, but its reintroduction two weeks later for an exacerbation of bronchospasm precipitated a further episode of profound depression. Stopping the drug again relieved the symptoms.

(2) An 11-year-old asthmatic girl (weight 31 kg) with eczema and dyslexia was admitted to hospital. Since treatment with salbutamol, beclomethasone, and disodium cromoglycate inhalation was insufficient she was started on theophylline 225 mg twice daily. She was discharged one week later. Shortly afterwards she became acutely depressed, had frequent episodes of crying, and on one occasion admitted to "wanting to take all the tablets and finish everything." Again, no exogenous cause could be identified and after theophylline was stopped her depression disappeared immediately.

Comment

These two cases suggest a paradoxical depressive reaction induced by theophylline. The absence of premorbid depression, the temporal relationship of the onset of symptoms to starting theophylline, their prompt disappearance when it was stopped, and (in the first patient) their return with the reintroduction of theophylline suggest a cause-and-effect relationship. Paradoxical reactions to central nervous depressants are well known and are attributed to selective depression of inhibitory neuronal systems or initial transient release of excitatory transmitters,[1] but we found only one allusion to stimulant-induced depression. This was an authoritative but unreferenced statement that the "CNS excitation produced by large amounts of caffeine is followed by depression."[2] There is considerable experimental evidence that xanthines promote the release of catecholamines from the adrenal medulla[3] and peripheral adrenergic nerve endings.[4] The occurrence of such neurotransmitter depletion in the central nervous system of susceptible individuals could explain the theophylline-induced depression in our patients. Neural catecholamine exhaustion is the basis of Jacobsen's widely accepted hypothesis[5] on the aetiology of depression.

The paradoxical reaction seen in our two patients is obviously uncommon, but clinicians should be aware of it as a possibility in unexpected depression in a patient taking theophylline.

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Medical Professorial Unit, University College Dublin, St Vincent's Hospital, Dublin 4
MICHAEL B MURPHY, MB, MRCPI, medical registrar
AILEEN DILLON, MB, medical intern
M X FITZGERALD, MD, FRCP, professor of medicine

Controlled trial of bladder drill for detrusor instability

We report a controlled trial of inpatient bladder drill for detrusor instability. Sixty women participated and results were assessed both subjectively and by repeat urodynamic studies.

Patients, methods and results

Sixty women aged 27-79 with urinary incontinence due to idiopathic detrusor instability diagnosed by pressure-flow studies entered a clinical trial of bladder drill. None was taking a drug known to affect urinary tract function or had coexisting genuine stress incontinence. Cystoscopy and urethral dilatation were performed under general anaesthesia to exclude local disease and measure bladder capacity. Each patient was then allocated at random either to inpatient bladder drill or to serve as a control; controls were advised that they should now be able to hold their urine for four hours, be continent, and allowed home. All patients were reassessed clinically and by repeat pressure-flow studies after three and six months.

Symptoms of patients before and six months after treatment

Symptoms	Bladder-drill group (n = 30)		Control group (n = 30)	
	Before	After	Before	After
Diurnal frequency	30	5	30	23
Nocturnal frequency	27	3	25	20
Urgency	30	4	30	23
Urge incontinence	30	3	30	23
Stress incontinence	21	3	20	16

We use the following bladder drill in our unit. (1) The rationale is explained. (2) The patient is instructed to pass urine at specific intervals