cefoxitin agar (Oxoid) selective for *Cl difficile*. Methods of identification of isolates and tests for *Cl difficile* cytotoxin in stools are described elsewhere.⁴ Typing was attempted by testing all strains against 20 antibiotics using a conventional disc sensitivity method.

Over eight months *Cl difficile* was recovered from 10 patients, seven of whom had undergone antibiotic gut decontamination (see figure). All had

Clinical details of 10 patients from whom *Clostridium difficile* was isolated from August 1980 to March 1981. Boxed areas indicate period on unit and hatched sections within these areas indicate duration of antibiotic gut decontamination and protective isolation.

been nursed in cubicles except for two: one (case 1) had also spent some time in the ward and the other (case 9) had been nursed entirely in the ward. The organism first appeared during decontamination and protective isolation in four patients, while in the other three it was isolated after these procedures had been stopped. At this time few commensal organisms were grown from the stools; enterobacteria were found in only one patient and non-sporing anaerobes in three. Three further patients were colonised, one of whom was a general medical case. Toxin was present in the stools of four of the six patients tested. One of these (Case 2) had abdominal cramps and bloody diarrhoea, and though sigmoidoscopy was not done to confirm the presence of pseudomembranous colitis her symptoms rapidly resolved after treatment with vancomycin 10 mg/kg/day for seven days. In the two other patients given vancomycin the organism reappeared when the drug was stopped. The index case (case 1) continued to excrete *Cl difficile* intermittently throughout the period of the study. The other patients were unfortunately not followed up for persistent carriage after discharge. Two distinct antibiograms were generated by the isolated strains, which differed in sensitivity to penicillin and tetracycline. They were recovered from seven and four patients respectively, one patient being sequentially colonised by both. Our strains were distinguishable from 10 strains obtained from other centres in Britain.

Epidemiological studies—The cubicles, patients' bedpans, and the unit's bedpan-washing machine were regularly screened for *Cl difficile*. It was repeatedly recovered from within the machine and on one bedpan used by a known case both before and after the washing cycle. It was not found in a further screen of bedpans and washing machines on other wards in the hospital. Impression cultures of nurses' hands and cultures of stools from all staff and other patients on the unit were negative when performed on two separate occasions.

Comment

Both our findings and epidemiological studies in an animal model⁵ support the evidence that *Cl difficile* can be transmitted to hospital patients by cross-infection. Removal of patients' commensal flora by antibiotic gut decontamination probably made them particularly susceptible to colonisation. Although members of staff were not found to carry *Cl difficile* they remain the most likely vehicles for transmission between patients. The importance of the bedpan-washing machine as a reservoir of *Cl difficile* is not clear, but we were able to show that the washing cycle failed to kill the organism and conclude that contaminated bedpans should be regarded as a potential source for colonisation of nurses' hands and clothing.

In an attempt to prevent any further spread of *Cl difficile* we have recently instituted strict barrier nursing of known cases on the unit and have continued protective isolation of susceptible patients until their discharge.

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Cephaloridine encephalopathy

We report a case in which cephaloridine infusion resulted in the development of a severe encephalopathy.

Case report

A 62-year-old woman was admitted to a cottage hospital with a 48-hour history of severe vomiting and bloody diarrhoea. Rebound tenderness in the right iliac fossa was observed. At laparotomy the ascending colon was found to be inflamed and a caecostomy was fashioned. After the operation she received three litres of intravenous fluid daily, each containing 2 g cephaloridine, and metronidazole 500 g thrice daily. No urine was passed before operation, and 24-hour urine volumes on the three subsequent days were 150, 500, and 150 ml respectively. By the evening of the fourth day after operation she had become drowsy and muscle twitching was observed. Renal failure was suspected and she was transferred to this hospital.

On arrival she was severely dehydrated and unresponsive to questioning. Generalised myoclonic jerks, exacerbated by any noise or movement, were observed. Reflexes were symmetrically brisk and the plantar responses extensor. Antibiotics were stopped and rehydration started. Serum creatinine concentration rose from 607 to 720 $\mu\text{mol/l}$ (6.8 to 8.1 mg/100 ml) over the next 24 hours. Two grand mal seizures occurred and treatment with diazepam and phenytoin was started. Serum cephaloridine concentration was 48 mg/l 24 hours after the drug was withdrawn and the cerebrospinal fluid concentration 1.4 mg/l four days later. Regular haemodialysis was undertaken, complete anuria having become established. Her conscious level continued to decline, endotracheal intubation and later tracheostomy becoming necessary. Over the next four weeks spontaneous respiration and eye, head, and some limb movements returned. She remained unable to speak, swallow, or respond to commands. Maintenance haemodialysis was continued for five weeks, after which renal function was sufficient to maintain the serum creatinine concentration around 900 $\mu\text{mol/l}$ (10.1 mg/100 ml). She developed bronchopneumonia and died six weeks after the original admission without showing signs of further neurological improvement. At necropsy the brain was macroscopically normal but histological examination showed diffuse severe neuronal degeneration. In the kidneys the typical appearance of acute tubular necrosis was observed.

Comment

It is well recognised that benzylpenicillin in high dosage may cause a distinctive encephalopathy. Myoclonic jerks exacerbated by any stimulus, convulsions, and coma are characteristic.^{1,2} Like penicillin, cephaloridine has been reported to cause convulsions after intrathecal administration.³ Although cephalosporin concentrations in the cerebrospinal fluid tend to be lower than those in the serum,⁴ accumulation in brain tissue occurs.⁵ A single case of cephaloridine-induced encephalopathy has been reported in which the drug was also

administered by continuous intravenous infusion in the presence of impaired renal function.²

We suggest that our patient developed a severe irreversible encephalopathy secondary to cephaloridine administration in the presence of acute renal failure, which may itself have been related to the drug. Recognition of the encephalopathic potential of cephaloridine is important, especially in the presence of impaired renal function.

We thank Dr M K Ward for permission to report this case.

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Death during play: a study of playground and recreation deaths in children

Childhood injuries caused by playground equipment may be severe,¹ but few studies have been undertaken to place this problem in perspective. Playground deaths among children (excluding motor-vehicle accidents and drownings) have not previously been studied, and only one large series of injuries not resulting in death has been reported.²

Recently adventure playgrounds have become popular, with a corresponding proliferation of equipment designed to develop physical confidence, balance, curiosity, and co-ordination. In general, such new playgrounds tend to be well supervised and are safe and exciting places for children; casualty officers, however, see a constant flow of children who have been injured on slippery slides,¹ swings, see-saws, and other equipment. We were concerned at the number of children injured in playground accidents attending casualty departments and report here a study of deaths due to this cause.

Methods and results

Children (age range 0-15 years) were included in the study if (a) they had died as a result of an injury received while playing and (b) their death had occurred at school or in a private playground, public park, recreation area, or their own garden. Children were excluded if a motor vehicle or bicycle had played a part in the accident or if they had drowned. Cases were identified and details obtained from the coroner's records and forensic records of the Institute for Forensic Pathology and the Laboratory of Microbiology and Pathology, Brisbane. Every death of a child (from whatever cause) during 1976-80 (inclusive) was initially reviewed, and cases satisfying the above criteria were selected for study. All such cases were checked individually for an association between accidental injury during play and recreation.

Brisbane is a modern, urban metropolis of one million people, of whom 266 000 were children aged under 15 years at the mid-survey point. Thus the survey was a total population study, without selection, of fatal recreational accidents to children in a modern Western society.

Fourteen children (nine boys, five girls) died during play over the five-year period (table). This gives an annual population incidence of 0.3/100 000 and an annual age-specific child population incidence (0-15 years) of 1.1/100 000. No specific modal age was particularly at risk. Three children died while playing with ropes: two were strangled while playing in trees and one while swinging from a rope attached to a brick chimney at a derelict site. Only two children were killed in accidents caused by playground equipment: in both cases the equipment was not attached firmly to the ground and fell and fatally crushed the child. Electrocution is now rare in children. In one of the two

cases in this series a model aircraft became entangled in overhead electric wires and a 15-year-old boy was electrocuted. Only one child died from injuries received in a school playground, this being the result of him colliding with another child while both were running.

Summary of 14 deaths among children at play, 1976-80

Type of accident	No of cases	Age of victims (years)	Traumatic agents
Crush injury	5	12, 9, 9, 9, 4	Rope, chimney; hay cart; slippery slide; monkey bars; amusement-arcade machine
Strangulation	3	12, 5, 2	Rope, tree (two cases); laundry trolley
Electrocution	2	15, 1	Overhead power lines, model aeroplane; electrical wire, wet grass
Fall	2	14, 1	Height; horse-riding
Impact	1	7	Collision between two boys in playground
Kick	1	1	Horse-riding

Comment

Current legislation concerning playground equipment, and current practices with respect to the safety of children during recreational activities, are obviously effective since no child died as the result of an accident in an adventure playground, in a city of one million people, over the five-year survey period.

In providing an enriched, adventurous environment for children a balance has to be struck between safety on the one hand and offering the child a stimulating and exciting play environment (supervised) on the other. Three-quarters of the injuries reported in the large Sheffield study² were trivial; 10%, however, were serious enough to warrant admission to hospital. Our study indicates that it is not primarily problems with equipment or its design that causes serious trauma during play but rather inadequate supervision while children are experimenting and being adventurous in what is largely an unmodifiable environment.

Attention to design and further tightening of safety legislation regarding the installation of recreational equipment, child adventure playgrounds, and so on are obviously important and an essential baseline for child safety; but this will not reduce the age-specific incidence rate below about one per 100 000 per year.

As in all types of accident prevention there are three possibilities—namely, education, improvement in ergonomic features and design, and safety legislation. In child recreational safety these three approaches are mutually inclusive.³ With major community resources being directed against fatal motor-vehicle accidents and drownings in children, increasing attention must be paid to other accidents ranking as important causes of serious child trauma, such as suffocation, burns, and falls (P O'Connor, for the Child Accident Prevention Foundation of Australia, unpublished data).

Non-accidental injury (the syndrome of child abuse and neglect) is often attributed by parents to the collapse of or a fall from some type of child's play equipment.⁴

This study has shown that the widespread provision of adventure playgrounds for young children does not result in any appreciable increase in fatal accidents to children at play if they are properly planned and properly supervised.

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