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(Accepted 13 May 1981)

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Adoption of Glasgow Coma Scale in the British Isles

One million head-injured patients attend British hospitals each year. Deciding who to admit and who to refer for specialised management depends on a reliable means of estimating conscious level—that is, an index of both the initial severity of injury and, if this changes, the development of intracranial complications. The Glasgow Coma Scale is a reliable and practical method of assessing conscious level in terms of three independently measured features—namely, eye opening, motor response, and verbal response.¹ It has been widely recommended² and adopted in many parts of the world as a guide to the management of patients with various forms of acute brain damage. Little information exists, however, on how widely the scale has been adopted by the two different kinds of unit—primary surgical units and neurosurgical units—that care for head injuries in Britain or about the factors that led to its adoption.

Methods and results

We sought information from all 31 Scottish hospitals that treat at least 10 head-injured patients a week and admit patients to their own wards for observation³ and requested similar data from the 42 neurosurgical units in the British Isles other than our own. We asked each unit about the method used to assess conscious level, its origin, and the reasons for its adoption and requested a specimen of the head-injury observation chart in local use.

Methods used to assess head-injured patients in neurosurgical units in British Isles and Scottish primary surgical hospitals

	Glasgow Coma Scale	Other graded systems	Entries in case notes
Neurosurgical units	21	9	10
Scottish hospitals* served by neurosurgeons who use:			
Glasgow Coma Scale..	21		2
Another method ..		6	2

*Two regional neurosurgical units used Glasgow Coma Scale and two another method.

Two-thirds of British neurosurgical units with a formal grading system used the Glasgow Coma Scale (table); no two of the nine other units used the same system. Of the six primary Scottish hospitals that used an alternative system, only two used the same one. Only where the regional neurosurgeons used the Glasgow system did the primary hospitals do so.

Comment

The adoption of the Glasgow Coma Scale by primary surgical and neurosurgical units is continuing. No alternative method is in common use, and no hospital that has introduced the Glasgow scale has later replaced it by an alternative system. Half the neurosurgical units in

the British Isles have adopted the Glasgow Coma Scale, in response either to personal recommendation by other neurosurgeons or to the publicity that the scale has received at meetings in the medical⁴ and nursing press.⁵ No geographical pattern is discernible—for example, seven of the units in London use the scale but the five others do not; two units in Scotland use it and two do not.

By contrast, Scottish primary hospitals are influenced by the practice of the regional neurosurgical unit; where the neurosurgeons use the Glasgow scale the primary hospitals tend to follow their example. This reflects partly the direct influence of the neurosurgeons but also the stimulus of the relatively mobile population of junior hospital doctors and nurses, who carry word of the scale from hospital to hospital. Several units commented on the improved communication about patients after the adoption of the scale.

Where the regional neurosurgical unit did not use the Glasgow Coma Scale the Scottish primary surgical hospitals had no uniform method of recording conscious level; we even found different systems in different departments of the same hospital. If this pattern of practice is typical of that in Britain as a whole then many thousands of acutely brain-damaged patients are being assessed by a potentially bewildering range of techniques. The danger is that communication about a patient's state may be faulty, particularly when primary physicians and surgeons have to discuss management with neurosurgeons on the telephone. Hospitals would do well to review their practice with a view to agreeing on the use of a common system, in collaboration with their regional neurosurgeons.

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(Accepted 19 May 1981)

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Spread of *Clostridium difficile* among patients receiving non-absorbable antibiotics for gut decontamination

Although the pathogenic role of *Clostridium difficile* in pseudomembranous colitis is well established,¹ the epidemiology of infection with this organism is still poorly understood. Reports of clustering of cases of pseudomembranous colitis²⁻³ suggest, however, that *C. difficile* is readily transmissible among hospital patients.

We report on the spread of this organism among children receiving oral non-absorbable antibiotics to suppress their commensal bowel flora in preparation for bone marrow transplantation or during chemotherapy for acute leukaemia. The large number of antibiotics prescribed and the occasional occurrence of symptoms resembling those of pseudomembranous colitis prompted us to screen for *C. difficile* in this group.

Patients, methods, and results

The transplant unit is comprised of cubicles permitting strict protective isolation and an adjacent open ward containing both patients convalescing after transplants and general medical patients. Those undergoing antibiotic gut decontamination received neomycin sulphate 20 mg/kg/day, colistin sulphate 40 000 units/kg/day, and amphotericin 15 mg/kg/day. After admission a weekly bacteriological screen, which included culture of stools, was performed to monitor the patients' bacterial flora. Stools were cultured on 7% horse-blood agar and MacConkey agar (Oxoid) aerobically; anaerobic cultures were performed on kanamycin blood agar and on cycloserine

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.

We acknowledge the contribution of other members of the Westminster Hospital transplant team to the management of the patients discussed.

SPB thanks Upjohn International for their support.

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(Accepted 27 May 1981)

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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