not seen in club scramblers. In this small series the incidence of injuries around the right knee was high, and further work is being done to define injury patterns, mechanisms, and possible preventive measures in scrambling accidents in all ages.

Our purpose is to highlight these injuries to children, which do not appear in official road accident statistics. We have shown that they may be of a substantial nature and warrant further study.

We thank the orthopaedic consultant staff for permission to report on their patients.

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Nocturnal deaths among patients with chronic bronchitis and emphysema

Patients with chronic bronchitis and emphysema may develop episodes of severe hypoxaemia with associated ventricular arrhythmias during sleep.[1 2] We examined the possibility that such nocturnal abnormalities predispose such patients to sudden death.

Patients, methods, and results

We studied patients who died in this hospital from chronic bronchitis and emphysema during 1977-81, excluding those with coexisting lung cancer. There were 54 such patients (42 men, 12 women), whose mean (SD) age was 71 (8·7) years. We also examined two control groups matched for age and sex, consisting of 54 patients who died from non-respiratory neoplasms and 54 who died from cerebrovascular disease.

For analysis the day was divided into three periods of eight hours each: 11 pm to 7 am (night), 7 am to 3 pm (day), and 3 pm to 11 pm (evening). In this hospital patients are settled down to bed between 10 30 and 11 pm and woken up around 7 am. Thus the night period approximated to the expected period of sleep.

The table shows that there was no significant difference in the pattern of mortality over time between the two control groups, whereas most of the patients with chronic bronchitis and emphysema died at night ($p < 0·05$, χ^2 test). The highest mortality in these patients was in the first hour of the night (seven deaths). Furthermore, 11 patients died between 4 and 7 am, whereas only two died between 7 and 10 am. Age and sex had no influence on the incidence of nocturnal deaths.

Times of death of patients with chronic bronchitis and emphysema compared with two control groups. Figures are numbers of patients dying

Diagnoses	7 am-3 pm (day)	3 pm-11 pm (evening)	11 pm-7 am (night)
Control groups:			
Non-respiratory neoplasms (n = 54)	19	21	14
Cerebrovascular disease (n = 54)	16	21	17
Bronchitis and emphysema (n = 54)	11	17	26*
Type 1 respiratory failure (n = 17)	3	8	6
Type 2 respiratory failure (n = 24)	6	3	15**

Significance of difference in numbers of deaths from other time periods: *$p < 0·05$; **$p < 0·01$.

Patients with type 2 respiratory failure (hypoxaemia with hypercapnia) showed a highly significant preponderance of nocturnal deaths ($p < 0·01$), whereas there was no such increase in patients with type 1 respiratory failure (hypoxaemia without hypercapnia). Patients in whom the partial pressure of oxygen was maintained above 8 kPa (60 mm Hg) with controlled low flow oxygen treatment showed no increase in nocturnal mortality. More patients who received hypnotics and narcotic analgesics, however, died at night (10 (63%) of 16 patients who received such medication *v* 16 (42%) of 38 who did not), although the difference was not significant.

Comment

Our findings are a logical extension of previous reports of serious hypoxaemia and cardiac arrhythmias in "blue bloater" patients with chronic bronchitis and emphysema but less severe respiratory and cardiac problems in "pink puffer" patients.[1 2] The findings also complement other reports of increased nocturnal mortality in patients with asthma.[3] The lack of increased nocturnal mortality in patients whose nocturnal hypoxaemia was corrected by treatment with low flow oxygen supports previous reports that treatment of such patients with oxygen at night is associated with a reduction in ventricular arrhythmias[1] but not with serious carbon dioxide retention.[2] In addition, treatment with oxygen has been shown to improve long term survival in patients with severe chronic bronchitis and emphysema.[4]

The fact that the greatest number of nocturnal deaths occurred in the first hour of the night supports the notion that many of our patients were asleep at the time of death, as the onset of sleep is normally associated with slight alveolar hypoventilation and carbon dioxide retention,[5] which might prove critical in some patients with severe respiratory failure.

The finding of maximum numbers of nocturnal deaths in patients with type 2 respiratory failure also accords with current concepts of respiratory physiology related to sleep, as respiration during sleep is believed to be critically dependent on the metabolic respiratory control system, which may be defective in patients with chronic hypercapnia.[5]

In conclusion, our findings indicate that patients admitted to hospital with an acute exacerbation of chronic bronchitis and emphysema should be carefully monitored at night, particularly if they have hypercapnia. The findings also emphasise the importance of controlled low flow oxygen in such patients.

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Dexamethasone and high dose metoclopramide: efficacy in controlling cisplatin induced nausea and vomiting

Cisplatin is one of the most active chemotherapeutic agents in a wide variety of malignant tumours but is among the most emetogenic drugs used in clinical practice. Improved control of platinum induced vomiting is intrinsically desirable and in addition may give a lead to ways of reducing emesis due to other cytotoxic agents. Several workers[1 2] have shown that metoclopramide in high doses may dramatically reduce cisplatin induced nausea and vomiting and that dexamethasone[3] may offer additional protection. We report the results of the first double blind cross over trial of high dose metoclopramide given with and without dexamethasone as an antiemetic in patients receiving cisplatin.

Patients, methods, and results

Forty five patients had received a total of 133 courses (median 2, range 1-11) cisplatin alone or in combination with other cytotoxic agents for a range of tumours. The dose of cisplatin was either 30 mg/m^2 (73 courses) or 100 mg/m^2 (60). The median age of the patients was 45 (range 30-68).

Patients were randomised to receive high dose metoclopramide (5 mg/kg) with either placebo (4 ml isotonic saline) or dexamethasone (20 mg in 4 ml). Metoclopramide was made up in 500 ml isotonic saline and infused in 100 ml aliquots beginning half an hour before administration of cisplatin (one hour infusion) and every two hours thereafter. Dexamethasone or placebo was administered by intravenous push half an hour before the cisplatin. The total number of high dose metoclopramide plus placebo courses was 68 and of high dose metoclopramide plus dexamethasone 65. The results were compiled using a standard questionnaire and the patient interviewed by one of us (SGA) about 16 hours after administration of cisplatin. (In the past major inconsistencies have occurred between recollections of nurses and patients, especially as regards retching.) Nausea was recorded on a visual analogue scale; episodes of dry retching were counted separately from episodes of vomiting. The duration of nausea and vomiting was recorded and any side effects attributable to the antiemetics carefully noted. The incidence of platinum induced diarrhoea was also recorded. The table gives the results expressed as percentages of courses.

The addition of dexamethasone to high dose metoclopramide clearly enhanced antiemetic control. Complete control of emesis was recorded in 43% compared with 26% of courses of high dose metoclopramide plus placebo and major control of emesis (0-2 episodes) in 65% compared with 46% of courses. The improvement in nausea after adding dexamethasone did not reach statistical significance but the duration of nausea after chemotherapy was significantly reduced (table). The number of patients who had no nausea, retching, or vomiting on at least one occasion was 11 in the dexamethasone arm compared with four in the placebo arm. The number of patients who had at least one episode of more than six spasms of retching or vomiting was 20 in the dexamethasone arm and 19 in the placebo arm. There was no significant difference in the number of meals eaten by patients during their chemotherapy (43% of patients taking high dose metoclopramide plus dexamethasone *v* 34% taking high dose metoclopramide plus placebo ate all their meals). The incidence of diarrhoea after cisplatin was dramatically reduced during the high dose metoclopramide plus dexamethasone courses (6%) as compared with during the high dose metoclopramide plus placebo courses (21%; $p=0.05$).

Effect of high dose metoclopramide with and without dexamethasone

Effect analysed	% Of total courses	
	Dexamethasone + metoclopramide	Placebo + metoclopramide
Complete control of all nausea, retching, vomiting	23	9
Complete control of retching and vomiting	43*	26
Major control of retching and vomiting (<3 episodes total)	65*	46
Complete control of nausea	32†	24
Major control of nausea (<33% visual analogue scale)	62†	47
Duration of nausea <24 hours	43	14
Duration of nausea >72 hours	19	39

All except † significant at $p=0.05$. *Significant at $p=0.01$.

Extrapyramidal reactions occurred in only three out of 133 courses in two patients and were easily controlled using the anticholinergic agent procyclidine hydrochloride. Tremulousness occurred in 30% of courses in both groups but was short lived and well tolerated. Drowsiness occurred in 40% of both groups, was always mild, and was of symptomatic benefit to many patients.

Comment

Both high dose metoclopramide[1 2] and dexamethasone[4] are effective against cisplatin induced emesis and each has been used for other cytotoxic agents such as doxorubicin with some success.[2 5] Our study shows the improved control of vomiting and retching, the enhanced recovery from nausea, and the diminished diarrhoea when dexamethasone is added to high dose metoclopramide during cisplatin chemotherapy. The added benefit of high dose metoclopramide plus dexamethasone was seen at both dosages of cisplatin. Side effects of antiemetic treatment were not altered by dexamethasone and no specific side effects were attributable to dexamethasone. In terms of altered antitumour activity, Aapro and Alberts showed that dexamethasone did not inhibit the antitumour properties of cisplatin in experimental systems.[4]

We conclude that adding dexamethasone to high dose metoclopramide enhances the protection against cisplatin induced nausea and vomiting with good control of nausea, dry retching, and vomiting in nearly two thirds of courses.

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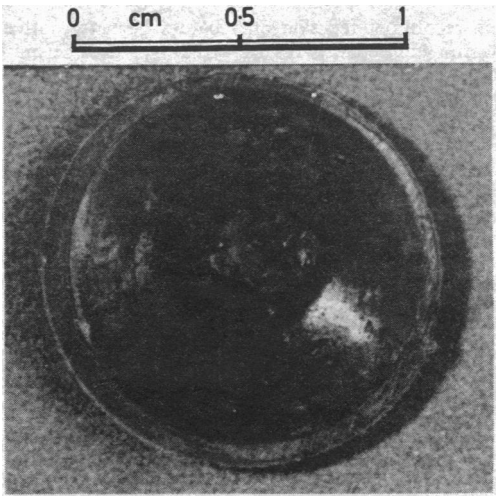
Impaction of a foreign body in the palate

Various foreign bodies from the prosaic to the exotic find their way into the body's orifices and cavities. In most cases this is accidental, in some it is iatrogenic, and in the more bizarre it is usually deliberate. Children commonly put foreign bodies into their mouths; subsequent ingestion of the foreign body into the alimentary tract, or aspiration into the tracheobronchial tree, may result in dire emergencies.[1] Foreign bodies in the mouth are often related to dental procedures and appliances and may become lodged in the tongue, tonsils, or pharynx. Impaction of a foreign body in the palate is rarely reported,[2] but we recently saw two almost identical cases.

Case reports

Case 1—An 11 month old boy presented to the paediatric department with a four week history of an abnormal appearance of his palate, which had been noted incidentally and was apparently symptomless. A whitish brown area at the centre of the arch of the palate was initially thought to be an infective lesion, but bacteriological cultures yielded negative results. The infant was referred to the cleft palate clinic, where a dental probe was used to remove a brown plastic cap that fits over the head of a screw (screwhead cap; figure) from the palatal mucosa, which was slightly inflamed.



Plastic screwhead cap removed from palate (diameter 12 mm).

Case 2—An 18 month old girl presented to the casualty department having had something stuck in the roof of the mouth for four weeks; her parents thought it was toffee. On examination a brown, hard area in the centre of the arch of the palate was noted. There were signs of an acute chest infection (fever of 38·3°C), but this was considered to be unrelated to the palatal abnormality. Ulceration of the hard palate was diagnosed and a course of antibiotic treatment started. She was referred to the cleft palate clinic, where