SHORT REPORTS

Failure of child safety seat to prevent death

Seat belts have increased the protection for the occupants of front seats of cars, and the next, obvious stage is to extend this protection to rear seat passengers and children.^{1,2} We report a road traffic accident in which the car driver, restrained by a seat belt, survived but a child in a safety seat died.

Case report

A hatchback car collided with a lorry, the forces during the impact causing the car occupants to move directly forward. There was intrusion into both front footwells, but otherwise the passenger compartment remained intact. The driver, who was restrained, sustained a fracture of the femoral shaft as the only major injury. An 18 month old child, weighing 10.5 kg, was strapped into a BS 3254 approved child seat, which was mounted centrally on the rear seat of the hatchback. This seat back was split into two parts (in a ratio of one:two), and the child seat was mounted on the larger section. The child seat was designed for four point mounting (figure) and had not been installed in accordance with the fitting instructions; instead, the two upper restraining straps led down vertically behind the back of the car seat and linked into the lower anchorage points (position 3, figure).

Position of rear anchorage points showing ideal, compromise, and incorrect positions.

During the impact luggage weighing about 9 kg moved forward and loaded the back of the car's rear seat. The single, side mounted catch retaining the nearside section of the seat back failed, and the seat back and child seat rotated forward violently. The webbing of the five point harness within the child seat failed at one of its lap section adjusters, and the child was ejected from the seat and found after the accident on the floor of the car, behind the back of the front seat. The very high loads on the lap belt probably resulted from the lack of restraint available from the upper half of the harness owing to the rotation of the car seat back.

On admission to hospital the child was in cardiopulmonary arrest, and attempted resuscitation was unsuccessful. Postmortem examination showed bilateral pulmonary contusion, atlantoaxial dissociation that had caused contusion of the cervical cord, and massive intra-abdominal injuries. The abdominal injuries may have resulted from loading from the lap section of the child harness; when this failed the child was ejected, sustaining a hyperextension neck injury (a bruise was observed under the chin). The bilateral pulmonary contusions may have been caused by the harness or by the child striking the back of the front seat.

Comment

Had this seat been correctly installed the child should have survived, as BS 3254 seats have proved effective in many accidents. The possibility of failure of a split, folding rear seat has been publicised by the Consumers' Association,³ as has the difficulty in fitting a child safety seat.⁴ One study found that only one third of children were correctly restrained and that 13% of the children surveyed were in incorrectly anchored restraints.⁵ The correct fitting of this type of child seat is vital but was made difficult by the lack of suitable anchorage points in the car for the upper mounting straps. The implications of simply looping the upper straps over the back of a folding rear seat are dire.

Child seats that do not require extra upper mounting points are now available and may be easier to install in hatchback and estate cars. The weakness of a folding rear seat back may, however, increase the risk of injury for children in restraints that do not isolate the child from additional loading. Current legislation controlling the strength of seat backs does not allow for the predictable presence of luggage. We think that a review of this legislation is overdue, given the increasing use of restraints for children of all ages that do not protect the users from additional loads.

This paper is published with permission of the Crown Office. We thank the

many Grampian police officers who helped so much in the investigation, and the Transport and Road Research Laboratory for financial support and allowing this case to be investigated in detail. We thank Dr J Broomhall for permission to use the diagram of rear anchorage points.

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- 3 Anonymous. Could your car be safer? *Which?* 1983 September:400-5.
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- 5 Eatough R, Swage T, Willson P, Broomhall J. Children in cars: how are they being restrained? *Br Med J* 1985;291:1092.

(Accepted 25 March 1986)

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The real cost of joint replacement

The Griffiths report, published in 1983, advocated greater financial awareness within the National Health Service.¹ There are few instances of detailed or up to date assessment of the cost of most common forms of treatment, including major joint replacement. The increased proportion of elderly in the population, coupled with the increased prevalence of arthritis in this group (41% in those over 65 v 5% in those aged 15-44),² has resulted in replacement arthroplasty occupying an increasingly large part of the elective orthopaedic workload.³ We identified the total cost of performing hip, knee, and shoulder replacements during one year in an orthopaedic unit.

Methods and results

In 1983, 329 major joint replacements were performed in Gartnavel General Hospital, Glasgow. Each surgeon was interviewed, and details of routine technique and care before and after the operation were noted. Thirty one cases of revision surgery were identified and excluded from the initial analysis as this differs appreciably from the routine primary procedure. The table shows the costs calculated for each type of replacement.

Costs calculated for hip, knee, and shoulder replacement

Joint	No of operations annually	Mean cost (£)			Total cost (£)
		Implant	Operation	Stay in hospital (mean duration of stay in days)	
Hip	216	108	119	2 200 (20)	2 427
Knee	61	361	97	3 410 (31)	3 868
Shoulder	21	375	73	2 090 (19)	2 538
Total	298	53 224	33 154	727 100	813 478

The varying prices of the seven different prostheses used (three hips, three knees, one shoulder), ranging from £87 to £498, had to be taken into account when calculating the average cost for each type of joint. The type of prosthesis inserted is chosen to fit the patient's needs, as one form of each arthroplasty is not suitable for every case. Operative costs included all the items necessary to perform each type of operation—for example, disposable drapes and gowns, prophylactic antibiotics, bone cement, and dressings. The needs of different procedures vary, but even within one type of arthroplasty differences arise because of the variety of techniques used by different surgeons. A higher operative cost generally reflects an individual surgeon's more liberal use of items that are known or claimed to reduce the complications of joint replacement. The Scottish Home and Health Department calculated that the cost of one day of inpatient treatment in the hospital in 1983 was £110.⁴ This figure was multiplied by the mean duration of stay, extracted from the hospital's computer records, to give an estimate of the cost of admission for each type of arthroplasty.

It is worth noting that the duration of stay necessary after revision operations is consistently longer than that after a primary joint replacement (hips 28 ± 20 days; knees 40 ± 31 days). The cost of revision arthroplasty in our study was also higher than that of the primary operation in terms of both materials and time consumed.

Comment

The cost of the prostheses themselves made up 6.5% of the orthopaedic unit's annual expenditure for major joint replacement. A further 4.1% was spent on other items used in the operation, the prime aim of which is to reduce the possibility of both early complications and later failure of the operation. The greatest part of the annual cost (£727 100 = 89.4% of the total) was attributed to keeping the patient in a hospital bed.

The cost of treating each patient could most readily be reduced by shortening the period of inpatient treatment by, for example, improving rehabilitation and social support. Conversely, an increase in the amount spent on prostheses and other items could be justified if it could be established that the items reduced complications and increased the lifespan of the implant: the added cost of performing revision surgery should not be forgotten.

There is increasing demand for major joint replacement because of factors beyond our control. This may mean, in the absence of more finance to permit a further increase in workload, a reassessment of the criteria used to select patients for surgical treatment and rejection of patients who are currently considered to need surgery.

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- 2 Department of Health and Social Security. *A happier old age*. London: HMSO, 1978.
- 3 Department of Health and Social Security. *Orthopaedic services—waiting time for out-patient appointments and in-patient treatment*. London: DHSS, 1981.
- 4 Scottish Home and Health Department. *Charges for private resident and non-resident patients*. Edinburgh: SHHD, 1984. (Circular Gen: 84.8.)

(Accepted 19 March 1986)

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Severe hyperglucagonaemia during treatment with oxymetholone

Unexplained severe hyperglucagonaemia was found in a boy with aplastic anaemia treated with the anabolic androgen oxymetholone. We have reported hyperglucagonaemia during treatment with danazol, which is structurally similar to oxymetholone.¹ Hyperglucagonaemia was therefore sought in patients receiving oxymetholone.

Subjects, methods, and results

The 16 year old boy initially studied had received oxymetholone (50-200 mg/day) for aplastic anaemia diagnosed five years previously. During investigation of intermittent diarrhoea screening of gut peptide hormones showed grossly raised plasma glucagon concentrations (70-380 pmol/l (24.4-132.4 ng/100 ml); values exceeding 50 pmol/l (17.4 ng/100 ml) are suggestive of glucagonoma). Hepatic and renal function, fasting blood glucose concentration, and serum amylase activity were all normal. Computerised tomographic and ultrasonographic examination showed no evidence of an intra-abdominal tumour.

Five other patients treated with oxymetholone (four men and one woman, aged 26-61 years, mean 39.9 years) were also studied. The duration of aplastic anaemia ranged from 11 months to 34 years (mean 8.8 years), and duration of treatment with oxymetholone ranged from seven months to seven years (mean 3.2 years); current dosages were 100-150 mg/day. Three patients were also taking prednisolone (10-20 mg/day), of whom two were diabetic, treated with diet alone and insulin, respectively. Results of biochemical screening were otherwise normal except for increased serum aspartate transaminase activity (41-163 IU/l; normal range 20-40 IU/l) in three patients, consistent with oxymetholone treatment.² None of the patients had the necrolytic rash, stomatitis, or cachexia of the glucagonoma syndrome.³

Fasting glucagon concentrations were also measured in healthy, non-obese control subjects (10 men aged 25-43 years, mean 31.2 years, and 11 women aged 20-53 years, mean 31.1 years), none of whom were taking any drugs.

Samples of 10 ml venous blood were taken after an overnight fast into a

lithium and heparin tube containing 200 μ l aprotinin (20 000 Kallikrein inactivator units/ml) and were centrifuged immediately. Plasma was stored at -30°C . Plasma glucagon and insulin concentrations were measured by radioimmunoassay and plasma glucose concentration by an autoanalyser (glucose oxidase method).

All six subjects treated with oxymetholone had significant hyperglucagonaemia (>3 SD above the mean control value for the appropriate sex), and in five cases this exceeded 50 pmol/l (17.4 ng/100 ml) (figure).

Fasting plasma glucagon concentrations in six patients with aplastic anaemia treated with oxymetholone, compared with mean (SD) values in 10 normal men (left) and 11 normal women (right). Each symbol represents a single patient; measurements were repeated in one man ($\square$ = 3 estimations) and one woman ($\blacklozenge$ = two estimations).

Conversion: SI to traditional units—Plasma glucagon: 1 pmol/l $\approx$ 348 pg/100 ml.

Plasma insulin concentrations in the group treated with oxymetholone (excluding the diabetic patient who was treated with insulin) were 45.3 (SD 36.3) mU/l, significantly greater than in the 21 control subjects (7.1 (3.8) mU/l, $p < 0.01$; Wilcoxon test). Fasting plasma glucose concentration was 7.10 mmol/l (126-180 mg/100 ml) in the two diabetic patients and normal (5.5-2 mmol/l (90-94 mg/100 ml)) in the four other patients and the normal subjects (4.9 (0.5) mmol/l (88 (9) mg/100 ml)).

Comment

All six patients treated with oxymetholone showed pronounced hyperglucagonaemia, in most cases in excess of 50 pmol/l (17.4 ng/100 ml). Oxymetholone, like danazol,¹ is therefore a cause of "pseudoglucagonoma." The association between oxymetholone and hyperglucagonaemia is not described in the drug's data sheet or in the standard text on glucagon,⁴ although a study of carbohydrate tolerance during oxymetholone treatment briefly mentions that plasma glucagon concentrations were raised in two out of seven patients.⁵ We believe that this observation is important because hyperglucagonaemia induced by oxymetholone may be both frequent and severe. With the increasing use of screening for gut endocrine hormones causes of gut peptide hypersecretion not related to tumours must be recognised in order to avoid unnecessary and often highly invasive attempts to identify intra-abdominal tumours; patients with hyperglucagonaemia induced by danazol have been mistakenly subjected to both angiography and laparotomy.¹

Danazol, a substituted ethisterone derivative, is mildly androgenic. Hyperglucagonaemia has not been reported during treatment with other androgenic steroids, although high dose glucocorticoids may cause mild hyperglucagonaemia; glucagon secretion may be indirectly stimulated by increased plasma amino acid levels.⁶ As with danazol, hyperglucagonaemia induced by oxymetholone was not associated with the clinical features of glucagonoma, although impaired carbohydrate tolerance and hyperinsulinaemia (possibly secondary to high glucagon levels) occurred with both drugs.

We thank Dr E Gordon-Smith for permission to report on his patients and Drs T Cranfield, A Roth, and R Eeles, the nursing staff of the metabolic unit, and Mr K Mageean for their help.