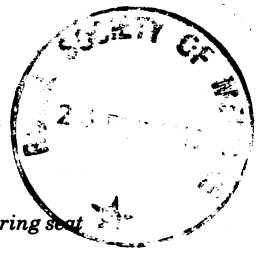
Editorials



Seat belts-six years on

It is now six years since the Road Traffic Act came into effect on 31 January 1983 making it compulsory for front seat occupants of most motor vehicles to wear seat belts. It was estimated that some 1000 lives and 10 000 serious injuries would be saved in the United Kingdom per annum[1,2]. A recent report estimates that the use of seat belts would reduce fatalities among previously unbelted front seat occupants by 43%[3]. The report by Taylor *et al.* (p. 170) in this issue prompts a timely review of the impact of seat belt legislation in the United Kingdom and the changing patterns of injury since its introduction. The efficacy of seat belt legislation may be assessed by examination of (a) compliance, (b) reductions in fatality and serious injury figures, and (c) the deaths and injuries attributable to wearing seat belts; the seat belt syndrome.

Compliance with seat belt legislation has been good. The use of front seat belts increased immediately from under 40% to over 90% and has remained at about that level although there has been a drop in use in vans to about 70%[2,4]. Reductions in fatal and serious injury rates have been significant, but not as great as predicted. In the year following legislation the numbers of deaths in front seat occupants in motor vehicle accidents fell by 457 (21%) and serious injuries by 6753 (24%). It is not clear why these reductions fall short of the estimated fall of some 40-45%[1-3]. There are several possible explanations for this shortfall: (1) those who do not comply with seat belt legislation are most likely to be involved in accidents[2,3]; (2) the estimates failed to take into account the relatively high usage of seat belts (around 40%) before introduction of legislation: clearly the smaller the increase in seat belt usage the smaller the observed benefit[5]; (3) seat belts provide less protection in lateral impact collisions than in frontal collisions[6] and (4) the Risk Compensation hypothesis which indicates that the compulsory wearing of seat belts encourages drivers to take more risks[4,7], i.e. protecting motorists from the consequences of bad driving encourages bad driving.

Finally there has been a notable change in the pattern of injuries sustained in motor vehicle accidents following seat belt legislation[5,6,9,10]. There has been a marked reduction in brain and facial injuries, intra-abdominal solid organ injuries and long bone fractures. On the debit side there has been in increase in the incidence of whiplash neck injuries, thoracic injuries, and intra-abdominal hollow viscus injuries. These injuries, caused by deceleration, contusion, shear or crushing in seat belt wearers, constitute the seat belt syndrome[11-13] and include some unusual injuries with which the surgeon may be unfamiliar (see list). One such injury is a traumatic abdominal wall hernia as described by Taylor. Apart from well-described traumatic hernia of the diaphragm, there are few reports of abdominal wall injuries[13-18]. The diagnosis may be made by a high index of suspicion and the presence of local contusion, swelling or tenderness. However, obesity or associated injuries may make clinical diagnosis difficult and in such cases radiology, ultrasound or CT scanning may be useful and help to differentiate hernia from haematoma[19]. Failure to make a prompt diagnosis may result in delayed and possibly complicated presentation.

Some injuries sustained by front seat occupants wearing seat belts in motor vehicle accidents

Injury	*Reference*
Cervical and other spinal injuries	5,6,9,13,20,21
Rib fractures	6,9,22
Sternal fractures	5
Pulmonary injury	6,9
Cardiac injury	6,22
Diaphragmatic hernia	6,9,15
Abdominal wall hernia	13,14,15,16,17,18
Abdominal wall haematoma	13
Intraabdominal injuries gastro-	
intestinal contusion or perforation	6,9,13,15,21
mesenteric tear or haematoma	9,13,15
liver	6,9,22
spleen	6,9,22,23
pancreas	9,24
Aortic and major arterial injury	6,22,25,26
Retroperitoneal haematoma	9
Renal injury	9
Urinary bladder injury	9
Breast or prosthetic injury	27,28

J N L Simson
St Richards Hospital, Chichester

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Malignant mesothelioma

Knowledge about some aspects of malignant mesothelioma has increased in recent years, but sadly effective treatment still eludes us and the prognosis remains very poor.

The tumour arises from mesothelial cells or possibly from more primitive submesothelial cells. It occurs most commonly in the pleura or peritoneum but, rarely, may arise in the pericardium or tunica vaginalis testis.

The existence of primary mesothelial tumours was not generally accepted until the late 1950s although reports of pleural and peritoneal tumours can be recognized much earlier. In 1960 Wagner and colleagues described 33 cases of diffuse pleural mesothelioma and all but one had experienced probable exposure to crocidolite (blue asbestos)[1]. Subsequently it came to be recognized that occupational asbestos exposure is responsible for most cases. Among subjects without exposure the annual incidence is probably around 1 to 2 per million[2]. A few childhood cases, apparently unrelated to exposure to asbestos or other fibrous minerals have occurred[3].

Deaths from mesothelioma, currently more than 600 per year in the UK, are still rising here and in other industrial countries. The increase is likely to continue until the turn of the century, reflecting the increasing use of asbestos, with inadequate or non-existent respiratory protection, until about 1970. In heavily exposed populations more than 10% of subjects may die of mesothelioma[4]. The tumour is commoner in males, reflecting the greater frequency with which they have been exposed to asbestos.

Naturally occurring mineral fibres also cause mesothelioma. Endemic pleural mesothelioma in Karain, a remote village in central Turkey was reported in 1978[5]. Mesothelioma accounted for the majority of adult deaths and the youngest patient was aged 12[6]. The materials responsible were found to be a fibrous zeolite called erionite and possibly other environmental asbestos minerals present in the volcanic tuff which is quarried and used for building. Naturally occurring tremolite, and perhaps chrysotile, cause mesothelioma in Cyprus[7] and Greece[8].

Glass fibres can cause mesothelioma in animals[9] and concern arose that asbestos substitutes might lead to mesothelioma in man. Fortunately, commercially used man-made mineral fibres are mostly of much larger dimensions than those used experimentally and a study of 25 000 workers engaged in their manufacture found only one of 1505 deaths to be due to mesothelioma[10].

There are no ideal studies that report the dose-specific mesothelioma risk based on individual exposure estimates but several studies have shown that the risk of mesothelioma increases with cumulative exposure[11-13]. Animal studies confirm this relation[14]. Cases of mesothelioma attributable to home or neighbourhood exposure have been identified[15] and this had led to the widely held belief that even trivial exposure to asbestos is associated with a substantial risk of mesothelioma. However, such cases occurred among a huge population of persons exposed in this way and the risk associated with low level or brief exposure is very small[16-17].

The incidence of mesothelioma increases with time elapsed since first exposure to asbestos in proportion the third or fourth power of the time elapsed[18]. The relative risk is not related to age at first exposure, although the absolute risk is greater with earlier exposure because there is more time for mesothelioma to develop. The risk of mesothelioma is not affected by smoking.

In a UK study of cases from various sources, in which 85% of those for whom information was available had definite or possible asbestos exposure, the mean interval from onset of exposure to death was 38 years with a range of 3.5-53 years[19]. Intervals of less than 20 years were uncommon in this, as in other series. The long interval between exposure and death

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