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Seat belts and risk compensation

The introduction of the compulsory use of front seat belts in January 1983 led to big reductions in death and injuries to front seat car occupants. The trend line has shifted downward by about 25% at the same time as the use of belts rose from about 40% in 1982 to over 90% after the law came in.[1] The seat belt law is saving about 400 lives a year and perhaps 5000 casualties being admitted to hospital. This law—and the surprisingly high level of acceptance of it by the driving public—must be one of the most successful pieces of public health legislation ever. We might, therefore, expect strong pressures to extend the law to occupants of rear seats, coaches, and trucks, with politicians lining up to introduce such lifesaving measures. Instead, some doubts have been cast on the measure because of the risk compensation hypothesis.

In its general form the risk compensation hypothesis suggests that the introduction of any safety measure results in changes of the behaviour of road users which may negate the supposed benefits of the measure. Indeed, risk compensation is a well established, observable phenomenon when there is direct feedback between the driver and the road system. If cars are made with better brakes, better handling and stability, improved tyres, and more powerful lights, some of these extra abilities will be used up by drivers leaving their braking later, cornering more severely, or going faster at night. That may or may not result in a changed collision rate, but it does not automatically follow that the accidents will become more frequent. Drivers using studded tyres in Scandinavia drive round corners on snowy roads faster than drivers using ordinary tyres, but they also have greater margins of safety because of the studded tyres' superior performance.[2] Where direct feedback exists risk compensation is at least a credible if complex phenomenon, but does it apply to passive safety measures such as energy absorbing steering systems, laminated glass windscreens, head restraints, or indeed to seat belt legislation? Peltzman proposed this in relation to crash protective standards for vehicles introduced in 1969 in the United States, but his analysis was shown to be flawed.[3] Wilde proposed a risk homoeostasis theory, which in turn has been shown not to be applicable to road accidents data.[4] And recently Adams has suggested that seat belt legislation actually increases road deaths by its effects on driving behaviour causing more accidents to cyclists and pedestrians; and, though his methods are demonstrably wrong, the risk compensation theory is still being quoted as an argument for removing the seat belt legislation.[5]

Research—as opposed to speculation—shows that when risk taking behaviour by drivers is studied those who wear seat belts (under either voluntary or mandatory regimens) in fact take fewer risks than those who do not wear belts. Measurements of speeds, headways, and gap acceptance (the time interval between cars in the approaching traffic stream which you accept or reject when you are turning right across it) all show that, if anything, use of belts is related to lower levels of risk taking; risk compensation applied to seat belt use cannot be detected in actual traffic studies.[6,7]

On the other hand, a superficial look at the numbers of casualties after the law had been passed purports to give some credence to risk compensation. The raw numbers of pedestrians and bicycle riders who were killed and injured in 1983 and 1984 have increased in comparison with the 1981 and 1982 numbers. Road traffic has increased substantially (7%), however, and there has been a particular increase in the use of bicycles by the under 15 year olds, perhaps because of the popularity of BMX machines. Within normal confidence limits, the changes in the past two years in the numbers of casualties among other road users—cyclists, pedestrians, motorcyclists, and rear occupants of cars—are within the normal seasonal and annual fluctuations when changes and uncertainties in traffic volumes and traffic patterns are taken into account.[8] Risk compensation does not appear to be a tenable theory when applied to the seat belt law.

Moreover, the best justification for the seat belt legislation is contained in a recent study by Rutherford *et al.*[9] This project, based on 14 hospitals, has documented the changing patterns of injuries and their frequencies before and after the seat belt law for over 13 000 patients. Admissions of front seat occupants were reduced by 30%, brain injuries were down by 39%, facial wounds by 53%, and injuries to the lungs by 40%. The multiplicity in injury in the severely injured is reduced, an important factor in their response to treatment. With the exception of minor neck strains and fractures of the sternum all body regions benefit from use of seat belts.

For the future it is important that the current provisional legislation for front seat belt use is made permanent when it is

brought before parliament in early 1986. The next and obvious stage is to extend the requirement to rear seat occupants. Britain is one of the last of the motorised nations which does not yet require fitting rear seat belts. In depth studies of crashes have shown that one of the limitations to the protection of front seat occupants occurs in frontal collisions when correctly belted front occupants are injured by unrestrained rear seat passengers. If rear seat occupants used seat belts as frequently as front seat occupants do now there would be two benefits: rear seat occupant deaths and injuries would be reduced by some 70%, and there would be a further reduction of some 6% in front seat casualties.[10]

Beyond this obvious measure many technical improvements to restraint systems and car interiors can and should be made. By design, drivers wearing restraints suffer face contacts with the steering wheel in most cars in a crash of more than some 25 mph. The answer lies in better padding or supplementary airbags in the steering wheel together with preloading of seat belts. Anchorage points mounted on the seat improve the lap belt geometry and diminish abdominal injuries due to submarining, which occurs when the pelvis rotates out from under the lap belt section in a frontal collision. But what about the obese, the aged, the pregnant woman, and the child? Have we really provided adequate protection for all those who actually use cars? What about a truly "friendly" interior, in which rational crash protective design has been applied effectively?

In each case the introduction of such protective measures as seat belts, laminated windscreens, head restraints, anti-burst door latches, and airbags represents a potential advance comparable with the introduction of a new drug. In epidemiological terms the benefits and side effects of such measures when used by the population at risk can be profound and often unexpected. And yet these measures do not receive the attention and evaluation from the medical community which they deserve. Perhaps the Rutherford study on the effectiveness of seat belts will generate some new interest in traffic injury research and its prevention. Road accidents cost Britain about £2·5 billion annually. Research into traffic injury reduction has an annual budget of less than one tenth of 1% of that figure. Most other industrialised countries devote far greater resources to the problem. Cannot the success of the seat belt legislation be used as a spur to more effort in tackling the general problems of trauma in our motorised society?

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1 Mackay GM. Two years' experience with the seat belt law in Britain. *Proceedings of annual conference of Society of Automotive Engineers Inc.* Warrendale, Pa: Society of Automotive Engineers Inc, 1985. (Paper No 851234.)
2 Rumar K, Berggrund U, Jernberg O, Ytterborn U. Driver reaction to a technical safety measure. *Hum Factors* 1978;**18**:443-54.
3 Peltzman S. Effects of automobile regulation. *Journal of Political Economy* 1979;**83**:677-723.
4 Wilde GJS. The theory of risk homeostasis, implications for safety and health. *Risk Analysis* 1982;2: 209-55.
5 Adams J. The efficiency of seat belt legislation. London: University College, 1981. *(Occasional Paper.)*
6 Evans L, Wasielewski P, vonBuseck CR. Compulsory seat belt use and driver risk-taking behavior. *Hum Factors* 1982;**24**:41-8.
7 Ashton SJ, Mackay GM, Camm S. Seat belt use under voluntary and mandatory conditions. *Proceedings of the American Association for Automotive Medicine.* Arlington Heights, Ill: American Association for Automotive Medicine, 1983:65-77.
8 Ashton SJ, Thomas PD, Harms P, Mackay GM, Galer MD. Effects of mandatory seat belt use in Great Britain. *Proceedings of Conference on Experimental Safety Vehicles, July, 1985.* Washington, DC: National Highway and Traffic Safety Administration (in press).
9 Rutherford WH, Greenfield AA, Hayes HRM, Nelson JK. *The medical effects of seat belt legislation.* London: HMSO, 1985.
10 House of Commons Transport Committee. *First report on road safety.* London: HMSO, 1985: 103-I:51.

Prevention and treatment of brain ischaemia

Cerebral ischaemia is a common and frustrating clinical problem, and a recent symposium issue of the *British Journal of Anaesthesia* on the topic should interest clinicians from many disciplines.[1] Despite a wealth of experimental data, only limited, often anecdotal information seems to be available on the efficacy of alternative treatments for patients with brain ischaemia. Consequently clinicians still feel relatively powerless to influence the outcome after ischaemic insults and emphasise the importance of preventive measures; yet episodes of cerebral ischaemia may often neither be predicted nor be prevented. What interventions, then, might ameliorate ischaemic brain damage by modifying events after the insult?

The pathological events which may culminate in ischaemic brain damage include cardiorespiratory arrest, stroke, and severe head injury as well as some operative procedures such as carotid endarterectomy, cardiopulmonary bypass, and induced hypotension. The ensuing lesion may be focal or global and may be exacerbated by complicating factors such as pre-existing cerebrovascular disease or hypertension, impaired autoregulation, hypoxaemia, or seizures. Thus, in contrast with many of the techniques used in studies on animals, accidental ischaemic insults are often complex. Moreover, the results of animal studies are influenced by both the nature of the ischaemia (global or focal, complete or incomplete, permanent or transient) and the timing of treatment in relation to the insult (before, immediately after, or delayed). Hence we need to be careful in extrapolating the results of laboratory investigations to clinical practice.

The rapid depletion of cellular energy stores after sudden, complete ischaemia[2] leads to failure of the ionic pump, membrane depolarisation,[3] and cellular swelling.[4] There is also a dramatic increase in calcium ions in the cytosol,[3 5] which may be associated with "burst firing" in selectively vulnerable neurones induced by excitatory amino acid neurotransmitters.[6] This intracellular accumulation of calcium may initiate several harmful reactions (including the release of free fatty acids, particularly arachidonic acid, and the production of free radicals of oxygen[5]) and may be the "final common pathway" leading to cell death.

When ischaemia is incomplete these events are modified by the residual flow, which increases the formation of oedema and also provides glucose for anaerobic glycolysis, thereby enhancing lactic acidosis and exacerbating neuronal damage. Similarly, hyperglycaemia, either preceding complete ischaemia or during an episode of incomplete ischaemia, increases the severity of the acidosis and further augments brain damage.[7]

If the decrease in cerebral blood flow is progressive threshold values can be defined for alterations in cerebral electrical activity, electrical silence,[8 9] and membrane failure.[8] Possibly alterations in calcium ion homoeostasis may have the lowest threshold of all.[10] Furthermore, in experimental focal ischaemia infarction is ultimately confined to those areas in which flow is reduced below the threshold for membrane failure, even though in the acute stage neuronal function may be disturbed over a larger region—the "ischaemic penumbra."[11]

It is the identification of these thresholds—together with the observation that events occurring during recirculation may initiate or exacerbate cell damage[2]—that suggests that