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Cyclists need helmets

Cycling has many attractions. It provides independence, needs no licence, has an extremely high mechanical efficiency, produces no pollution, and seldom causes injury to others. Injuries to riders are, however, all too common—about 300 deaths and over 5000 serious injuries in Britain annually. Pedal cycling is second only to motorcycling as the most dangerous form of travel per mile.

The Transport and Road Research Laboratory has shown that many of the cycle accidents that include a second vehicle occur at T junctions, and often the cyclist is travelling straight ahead and therefore should have priority.[1] As the report states, "In over half of these accidents the other vehicle was turning, suggesting that a significant number of these vehicles either did not see the cycle, saw it but misjudged its speed, or saw it but unreasonably expected it to give way."

Many injuries to cyclists could be prevented if other road users gave them a better opportunity to ride safely. For instance, stones and other rubbish tend to find their way into the sides of the road—but it is here that cyclists are often forced to ride by the bulk of other vehicles. The bicycle wheel is particularly vulnerable to irregular surfaces, and the rider may easily be thrown off and injured. The speed of the cycle may contribute little to the impact, but the height of the fall from the riding position is important. Injuries to the head are an important cause of death and are present in about three quarters of seriously injured cyclists. In many accidents the cycle is struck by the front of a car, and experiments show that modifying the front of cars to reduce their "aggressivity" to pedestrians also helps prevent serious injuries to cyclists.

A recent study from a large accident and emergency department provides some striking comparisons between cyclists' and motorcyclists' injuries[2]: the cases of 506 injured pedal cyclists and 456 injured motorcyclists were reviewed together with necropsy findings on fatal cases from the same catchment area. The study showed that pedal cyclists "were more likely to suffer a head injury than motorcyclists and that those dying suffered, on average, more severe head injuries than motorcyclists who died." Motorcyclists who died with head injuries usually suffered other major injuries, which is consistent with their higher impact velocities and the protection afforded by helmets. A study from Oxford published today (p 1161) also compared injuries to cyclists and motorcyclists and showed that head injuries were significantly more common in the cyclist. The authors also produce data to show that cycle tracks reduced the number of accidents.

The medical evidence for cyclists to wear head protection is strong. The comparative efficacy of different designs of helmet has been studied in Australia in a follow up of accidents to cycling club members: the best protection was from helmets with a hard shell and firm energy absorbing linings.[3]

There is now a British Standard specification for such helmets (BS 6863:1987); this tests for performance on impact, strength of strap, extent of permitted vision, and ear clearance. Many helmets now available are imported, which should be a challenge to British manufacturers to meet the British standard and add refinements such as better ventilation and a buckle that can easily be fastened with the gloved hand.

The medical case for helmets has been restated by an American doctor in family medicine.[4] He concludes: "We have a tremendous opportunity to diminish the death toll from childhood bicycling accidents. Simple educational measures are likely to be effective. All we have to do is to act."

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What sort of "health checks" for older people?

The recent white paper *Promoting Better Health* tantalisingly suggested that the government might be prepared to encourage "health checks" for older patients within primary care.[1] "Regular and frequent health checks... for some elderly people" are to be discussed with relevant professions. There might even be special payments for such procedures: "The government shall, through changes in the remuneration system, encourage doctors to provide comprehensive regular care for elderly people."

Though this encouragement of prevention for older patients is welcome and long overdue, we must be quite clear what is meant by "health checks." Medical screening, especially that using multiple laboratory tests and measurements ("multiphasic screening"), has been shown by controlled trials not to reduce morbidity and mortality and not to improve use of services.[2,3] Enthusiasm for screening for precursors of disease in older people was severely restricted by these negative results, and in the past few years it has become clear that the emphasis in prevention for this age group should be not on earlier detection of disease but rather on assessing loss of function.[4]

The intervention should aim at helping older people to avoid the adverse effects of established disabilities by planned programmes of case finding. Functional assessment should encompass physical, mental, and social function, and since the well being of many older people depends on the morale and competence of informal carers the welfare needs of the carer also need to be assessed. The broad scope of such case finding means that it is best carried out as a multidisciplinary activity within primary care.

Many older people are fit and well, and it is wasteful for all those over 65 to be visited and subjected to a detailed assessment. Thus programmes of case finding need two stages: the first stage simply to identify those who are at high risk and likely to benefit from the second stage, the detailed assessment of functional capabilities.

One approach to selecting those at high risk of disability is to identify the very old (85 or older), those recently discharged from hospital, the recently bereaved, and those taking multiple medications. Taylor claimed that only the first two of these categories were likely to be valid indicators of high vulnerability.[5] Another approach has been using postal questionnaires,[6 7] and a recent initiative has used volunteers to visit all older people; those identified to be in need are then followed up by general practitioners, health visitors, and district nurses.[4]

Freer has shown the feasibility of opportunistic case finding, which takes advantage of the fact that three quarters of old people contact their general practitioners at least once a year. This contact may be used to ask questions and make observations aimed at identifying those at high risk, who may then be offered further assessment.[8] Several studies have shown the benefits of planned programmes of case finding that concentrate on functional assessment.[9 10] The time has come to implement the programmes in primary care to improve the quality of life for vulnerable old people and reduce the need for continuing institutional care.

It would be unfortunate, however, if the recent government interest in prevention for older people was to be channelled into medical screening. We suggest rather that case finding should be encouraged (by financial inducements and otherwise). Evaluation should be an integral part of the case finding programmes and should consider "life satisfaction," which case finding seems to improve.[11]

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DNA topoisomerases in cancer treatment

DNA topoisomerases have emerged in the past three years into the clinical limelight.[1] They were first identified over 15 years ago as important enzymes in bacterial systems, but recent work has shown that they may be unique targets for anticancer drugs.

There are two enzymes—topoisomerase 1 and topoisomerase 2. Their name arises from their crucial function in catalysing the conversion between topological isomers of DNA. They assist in relaxing and supercoiling DNA, intertwining DNA into rings, and tying knots and untying them again, and they may act as swivels to reduce the torsional stress when DNA is transcribed by RNA polymerase. DNA topoisomerases stop the double helix of DNA tying itself into an impossible tangle when it has to divide into two single strands. They can create a break in either one strand (topoisomerase 1) or in both strands (topoisomerase 2), allowing one strand or both strands to pass through the gap. They then catalyse the resealing of the gateway.

Topoisomerase 1 seems not to be essential for bacteria or cells, whereas topoisomerase 2 is. Most attention has therefore been paid to possible drug interactions with topoisomerase 2, and it has become clear that it is the target for several DNA intercalating agents, such as doxorubicin, elliptocines, amsacrine, and the epipodophyllotoxins—teniposide and etoposide. Teniposide binds to topoisomerase 2, thus stabilising the cleavage complex formed between topoisomerase 2 and DNA strands. This complex is associated with cell death. The precise reason for cell death is not known, but several elegant experiments have suggested that the cytotoxicity of the drugs is related to the production of double strand breaks (but not single strand breaks). Topoisomerases seem to mediate drug induced cytotoxicity independently of free radical production.

The selectivity of cytotoxic drugs acting through topoisomerase may be partly explained by the enzyme being present in low concentrations in resting cells and increasing in concentration in tissues proliferating in response to growth factors. The concentration increases in human leukaemic cells when they enter the cell cycle and is high in solid tumours such as adenocarcinoma of the prostate. A further interesting finding is that erythroleukaemia cells in the mouse have a high concentration of topoisomerase, which falls dramatically after differentiation is induced by adding hexamethylene bisacetamide. It may also be important that topoisomerase 2 sequences have been reported in certain genes, including the proto-oncogene c-fos, and that etoposide and amsacrine both stimulate breaks in exon 1 of the c-myc proto-oncogene. It may be therefore that some activated oncogenes are the targets for drugs that work through topoisomerase 2.

Resistance of tumour cells to cytotoxic drugs has obvious clinical interest, and tumour cell lines in tissue culture have shown two sorts of resistance to inhibitors of topoisomerase 2: cell lines from patients with chronic lymphatic leukaemia that are resistant to adriamycin have low concentrations of human topoisomerase 2, while Chinese hamster cells resistant to etoposide show a mutant form of the enzyme. Other factors may be important in resistance—for instance, drug uptake and alterations of the catabolism of the topoisomerase cleavable complex. A prospective study is now underway in patients with myeloid leukaemia to try to predict the likelihood of response to amsacrine by screening for low topoisomerase concentrations, mutant enzymes, or both.

The clinical importance of unravelling this novel mechanism of action may be limited, but predictive testing is an exciting and logical consequence of the basic work. Furthermore, the interaction between epipodophyllotoxins and topoisomerase 2 explains the importance of giving teniposide and etoposide at the right time and in the right relation. This new information may also give us more clues about the