Epikeratophakia is safe. If inaccurate refraction or other complications occur, then the lathed lens can be removed leaving the patient's cornea more or less undamaged. This is necessary in between 4% and 10% of cases.[7,8]

Epikeratophakia is suitable for patients with aphakia in whom lens implantation is contraindicated and a trial of contact lens wear has failed. It is particularly suitable for patients with monocular aphakia, who might have intolerable diplopia with glasses. The use of epikeratophakia for patients with congenital cataract is still experimental, and contact lenses seem better, although operations have been performed on children under 1 year.[9] Patients with severe myopia experience image distortion with glasses, and should genuine contact lens intolerance occur then the patient may benefit from epikeratophakia.[8]

There are likely to be important changes in refractive surgery. Development of the excimer laser, which can vaporise corneal tissue precisely with minimal effect on surrounding tissue, may make radial keratotomy obsolete in the next decade. Laboratory manufactured lenses for epikeratophakia may overcome the problems of using biologically variable donor tissue.

Refractive surgery is major surgery. Patients must understand that an operation will not improve their best corrected visual acuity and that complications may occur.

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Changing the law on children in cars

People would not carry eggs, it has been said, as they so often carry children in the rear seats of cars—unrestrained and much less secure than their parents in the front. Yet children have been found to suffer head and face injuries far more commonly than unrestrained adults; in particular, young children with their light weight become high velocity missiles and may be thrown on to the road, a rare occurrence in those who are restrained.

In 1986, says the Transport and Road Research Laboratory, 8560 children under 14 were injured in the rear of cars and light vans, 89% of the child casualties in these vehicles; 67 died and 940 were seriously injured. According to the laboratory, using restraints correctly reduces deaths by around three quarters in children under 5; and American studies suggest reductions of 80-90% in fatal and serious injury to children. But only 37% of children use restraints, and only 31% of children in the rear—55% of babies and progressively fewer older children.[1] Children aged 5-13 have 65% of the child casualties but only 17% use restraints. Improvement here would pay particular dividends, but, as with front seat belts, progress is slow without legislation.

The government has so far refused to introduce legislation on the retrospective fitting of rear belts in cars with anchorage points only, manufactured between October 1981 and October 1986. Now as a first step a private member's bill, with all party support, is to be introduced by Mr Stephen Day. This requires the use of restraints, if seat belts are fitted in the rear, by children under 14. This may not seem much; but, although only three million cars at most have rear belts, the proportion will increase (nearly 10% of the 19 million cars were new last year). Even this bill, according to cautious estimates, could prevent about 50 serious and fatal injuries a year now, rising to around 350.

Compulsory restraint of children in cars has been successfully introduced in The Netherlands; Australia, New Zealand, Canada, and the United States. According to information gathered by the Parliamentary Advisory Council for Transport Safety, injuries have fallen by up to half after legislation. Belatedly Britain must follow their example. In a recent Gallup poll 91% of drivers supported the compulsory use of restraints for children in the rear of cars. Doctors could support the bill by writing to their members of parliament and to the press, for many can attest to the terrible and unnecessary injuries that children still suffer.

Staff editor, *BMJ* DAPHNE GLOAG

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Child abuse and osteogenesis imperfecta

One diagnosis in children, particularly babies, who suffer unexplained fractures is osteogenesis imperfecta—"brittle bones disease." Babies suspected of having been non-accidentally injured may be claimed to have "brittle bones." How great is the risk of confusion?

Osteogenesis imperfecta is not a homogeneous condition but comprises at least four main varieties subdivided into various subtypes.[1,2] All appear to result from different genetically determined abnormalities of connective tissue, particularly type I precollagen.[3-5] Thus tissues other than bone may be affected. Controversy continues over the genetics of osteogenesis imperfecta, but the commonest form, accounting for about 80% of cases,[6] is an autosomal dominant disorder (type I)[7] almost invariably associated with blue sclerae.[8] There are two probably autosomal recessive forms of the disease: the first (type II osteogenesis imperfecta) is extremely severe with multiple fractures at birth and early death; and type III is similar to type II but less severe. A fourth and rare form of the disorder (type IV) is autosomal dominant with occasional spontaneous mutations. It tends to be intermediate in severity, but most cases appear to be severe enough to be difficult to distinguish from type III.

Thirty seven of 40 cases in one series combining types III and IV showed striking radiological femoral changes.[9]

Estimates of the incidence of osteogenesis imperfecta range from 1 in 15 000 births to 1 in 60 000. One in 20 000 is the figure I will use,[10] and type I could thus be assumed to occur in about 1 in 30 000 births.[2]

Neither type II osteogenesis imperfecta nor type III should be confused with non-accidental injury. In both there are recurrent multiple fractures with severe skeletal deformity. Nor should type I pose any serious problems as blue sclerae are almost universal: in one study they were noted in 370 out of 392 subjects in a pedigree of 60 families.[11] Osteogenesis imperfecta type IV is not, however, associated with blue sclerae.[12]

Other clinical and radiological features would be expected in both types I and IV of osteogenesis imperfecta. These include radiological evidence of wormian bones and osteopenia in the child and hyermobility of the joints, deafness, and dentinogenesis imperfecta in relatives. Not all these features will always be present, but their absence reduces the possibility of the diagnosis. For example, dentinogenesis imperfecta was seen in two thirds of families with type IV osteogenesis imperfecta,[12] and more than 10 wormian bones arranged in a mosaic pattern were found in all patients with osteogenesis imperfecta after the neonatal period.[13] In doubtful cases of suspected non-accidental fractures careful radiological examination of the skull with both lateral and Townes views may be advisable with repeat examination of neonates in later infancy. The absence of wormian bones is strong evidence against osteogenesis imperfecta.

All children with fractures and their parents should be examined for blue sclerae. A glance at a parent's bulbar conjunctivae may save an awkward moment in the witness box. Other clinical stigmata of osteogenesis imperfecta—such as joint laxity and a triangular facial appearance—should be noted but are in themselves too non-specific to allow a diagnosis of osteogenesis imperfecta. Detailed family histories should be obtained, and when they suggest osteogenesis imperfecta they should be verified by checking past medical records—it is all too easy to claim a history of multiple fractures. Radiographs should be scrutinised by a radiologist experienced in examining the bones of young children.

The problem that most often leads to doubt and medicolegal confusion is a young infant with one or more fractures. How likely is it that such a child could have osteogenesis imperfecta if neither child nor parent has blue sclerae, radiographs are normal, fractures do not recur after the child is removed from home, and there is no family history of deafness, osteogenesis imperfecta, or dentinogenesis imperfecta? Only sporadic cases of the very uncommon type IV should give rise to real difficulty.[14]

An estimate of the likelihood of encountering such a case can be made from a large population study.[15] Of 180 patients with osteogenesis imperfecta nine were classified as having type IV, and only one had no family history. Thus at most 5% of cases of osteogenesis imperfecta would have neither blue sclerae nor progressive deformity, and only 0·6% would in addition have no family history. If we accept an overall prevalence of 1 in 20 000 for osteogenesis imperfecta, the concurrence of this disease with the absence of blue sclerae, progressive deformity, and family history would occur in about 1 in 3 000 000 births. Since, however, at least some cases of type IV osteogenesis imperfecta do have deforming disease this is an overestimate. In another study the ratio of type I osteogenesis imperfecta to IV was between 1 in 4 and 1 in 5.[12] If the incidence of type I osteogenesis imperfecta is 1 in 30 000 the incidence of type IV would be 1 in 120 000. The absence of a family history of either osteogenesis or dentinogenesis imperfecta would increase these odds to about 1 in 1 000 000. The additional absence of appreciable wormian bones after the neonatal period would probably exclude the diagnosis.

Thus in a city of 500 000 people with 6000 births a year the chance of encountering a child under 1 year old with osteogenesis imperfecta who shows no other features or family findings of the disease would be between 1 in 1 000 000 and 1 in 3 000 000. This would produce an incidence of 1 case every 100 to 300 years. The annual incidence of fractures caused by non-accidental injury would be about 15 cases.

Medical witnesses need to formulate their opinion in the light of such odds. Provided care is taken osteogenesis imperfecta does not provide a satisfactory reason for unexplained fractures in otherwise healthy babies.

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Community acquired pneumonia

Pneumonia is the commonest fatal community acquired infection in Britain. Although not all deaths can be prevented, any guidance on the best immediate treatment and on those features which indicate a poor prognosis is welcome. A recent multicentre study coordinated by the British Thoracic Society has shed a new and authoritative light on these problems of clinical management.[1]

In a prospective study of 453 adults (aged 15-74) admitted to 25 British hospitals with pneumonia strenuous efforts were made to reach an aetiological diagnosis by microbiological and serological means (including tests for pneumococcal antigen in sputum, blood, and urine). For each case the relation of the causative organism and other features to the outcome were subjected to exhaustive multivariate analysis, which had been designed to avoid any preconceived bias.

Reassuringly the results endorse the principles that have