

the first few months then gradually reaches a plateau.⁵ The total amount of new bone formed is limited by the initial rate of bone formation. This limit of 5-10% can be termed the remodelling barrier.⁶ Only drugs that independently stimulate bone formation by osteoblasts (such as fluoride) can overcome this barrier. Thus, encouraging results of brief studies do not imply continuing increases in bone mass.

Even without technical error, assessing the effects of changes in bone mass on bone strength is difficult. In ordinary populations it is reasonable to assume that lifestyle risk factors (such as smoking), which reduce the bone mass by 5%,⁷ should increase the fracture rate by 50%. But pharmacological interventions that increase bone mass by 5% do not necessarily decrease fracture risk by 50% because the structure of trabecular bone is damaged with bone loss, especially that associated with the menopause. Entire trabecular struts are lost.⁸ With the antiresorbing drugs the disconnected trabeculae are not reconnected but the existing trabeculae widen. Also, some of the increase in density occurs without any increase in bone volume because lowering bone turnover eventually results in a larger proportion of old bone, which is denser than newly formed bone. Fluoride causes a disparity between bone mass, which increases dramatically, and

strength, which decreases.⁹ Thus the relation between the relative risk of fracture and bone mass cannot yet be used to predict whether a change in bone mass will change the risk of fracture.

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Preventing injuries from bar glasses

Temper the nonik

The Home Office has estimated that each year in Britain between 3400 and 5400 offences occur in which glass is used as a weapon. Two surveys of victims of assault who attended accident and emergency departments in Bristol and south London found that the most commonly used sharp weapons were bar glasses.^{1,2} Another study found that three quarters of such injuries had arisen through assaults with straight sided bar glasses of one pint (0.57 l) capacity ("noniks"—or no nicks).³

Contrary to expectation, three quarters of the glasses were intact until they were thrown or thrust at someone and then broke on impact. Almost all injuries were to the face, and doctors working in accident and emergency departments predicted that deformity at six months would be "noticeable" or "very noticeable" in three quarters of the victims. The British Association of Hand Surgeons identified more than 200 accidental hand injuries due to bar glasses in three months in 1987.⁴ These surveys highlight the morbidity produced by bar glass—mostly in young people, in whom initiatives aimed at preventing accidents have been given priority in the *Health of the Nation*.

Glasses' resistance to impact varies according to manufacturer and degree of wear and tear. A laboratory investigation of new and worn glasses of one pint capacity (nonik and tankard designs) available in Britain found that noniks from one manufacturer were more than six times more resistant to impact than all the others, and when they were worn they were twice as resistant to impact as similarly worn noniks from other manufacturers. These noniks were also three times more resistant to impact than tankards. Heavy wear and tear, however, substantially weakened all the designs.⁵

When the glasses that were comparatively resistant to impact failed they disintegrated into cuboid fragments with angles that tended towards 90°. This particularly applied to the thicker bases. In contrast, other glasses disintegrated into

larger, jagged pieces, and the base of the glass generally remained intact and usable as a weapon.

In the search for an explanation for these differences it became apparent that the glassware that was resistant to impact had been tempered (toughened) during manufacture while all the other glassware had been annealed. Although tempering has long been applied to the manufacture of car windscreens, plate glass, and cooking containers, only two manufacturers currently temper bar glasses. This process involves rapid cooling of the glass after its initial formation so that a compressive outer skin is produced. This holds together the outer layer of glass and particularly the microflaws and cracks that are common to all glassware after manufacture. This explains both their increased resistance to impact and the different pattern of fragmentation.

As well as its increased safety, tempered glassware has several other advantages, including durability and longevity. For example, in a large office complex tempered glasses lasted up to 25 times longer than annealed glasses of the same design.⁶ Manufacturers that do not produce tempered bar glassware claim that injury may follow explosive disintegration of tempered glassware and that such glassware discolours more than annealed glassware,⁷ though no evidence for this has been published.

Following these findings, a search elicited no safety guidelines or codes of practice in relation to bar glasses in Britain or internationally. Nothing therefore prevents manufacturers from producing glasses of thinner and thinner material; indeed, a commercial incentive exists to do so, particularly as the British market for bar glasses is worth about £100m a year.

A survey of bar workers found that 40% had been injured by bar glasses—mostly while stacking and washing noniks of one pint capacity.⁸ Although most injuries were to the hand and produced only minor inconvenience, about one in 10

substantially disrupted work and one in three required treatment in an accident and emergency department. Clearly, this level of injury from sharp objects would be unacceptable in a laboratory or other working environment.

What should be done? Firstly, safety standards need to be developed to protect consumers and bar workers. Glassware in many countries bears a government stamp to signify that the capacity of the glass conforms to certain standards of volume. This should also signify adherence to standards of safety. In Britain an obvious objective is that every half pint or one pint beer glass should bear the British Standards Institution's "kite mark." Secondly, a code of practice should be developed regarding the type of glass that should be used in particular environments—for example, in pub gardens and urban pubs. Thirdly, the safety of disposable and reusable plastic containers should be acknowledged. Although levels of injury do not warrant wholesale replacement of glass, plastic has obvious advantages in some environments—for example, at beer festivals and sporting occasions. And lastly, licensees should ensure that empty bottles and glasses are

regularly collected and that worn glasses are replaced before they break in use.⁹

Jonathan Shepherd's group received a research grant to study risk factors for urban violence from the glass manufacturers, J G Durand. This followed the publication of the results of a study by Shepherd and colleagues reporting that tempered glasses—as made by Durand—were more resistant to impact than annealed glasses (*BMJ* 1991;303:1330).

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Fibrin glue

Topical haemostasis for areas of bleeding large and small

Fibrin glue consists of two main components: fibrinogen and thrombin.^{1,2} These are loaded into two syringes with tips forming a common port. When injected the two components meet in equal volumes at the point of delivery. The thrombin converts the fibrinogen to fibrin by enzymatic action at a rate determined by the concentration of thrombin. The more concentrated thrombin solution produces a fibrin clot in about 10 seconds, and the more dilute thrombin solution forms the clot about 60 seconds after the glue is applied to the surgical field. Both the extrinsic and the intrinsic mechanisms of blood coagulation are bypassed, but the physiological final common pathway of coagulation is faithfully replicated. Factor XIII (present in the fibrinogen component of the glue) cross links and stabilises the clot's fibrin monomers. Some preparations of fibrin glue contain aprotinin to delay the fibrinolytic action of plasmin.³

Modern surgical techniques are efficient at securing haemostasis, but oozing of blood from multiple pinpoint on a large raw surface remains difficult to control. The use of electrocautery, sutures, or metal clips and staples may not be appropriate in delicate surgery on small yet functionally vital tissues and structures. In these settings fibrin glue is ideal for topical application to secure haemostasis. In cardiovascular and thoracic surgery, for example, fibrin glue may be sprayed with use of pressurised gas or compressed air to arrest bleeding from the surfaces of the heart, pericardium, mediastinum, and pleura.^{4,5} It may be used in a similar way to form a thin film on the liver or the liver bed, or both,⁶ and in neurosurgical,⁷ ophthalmological,⁸ and some otolaryngological operations⁹; in vitro fertilisation¹⁰; and other microsurgery.¹¹ A further obvious indication for fibrin glue is external oozing of blood in patients with haemophilia.¹² Fibrin glue is not, however, a substitute for good surgical technique, and it is of little value in arresting arterial bleeds.

Fibrin glue has other uses besides haemostasis. It can seal leaks of air or fluid¹³ and secure anastomoses,¹⁴ and it is used in orthopaedic¹⁵ and plastic surgery.¹⁶ In all these indications the biologically interactive fibrin sealant is absorbed after it has done its job. Mixtures of fibrin glue and antibiotics are being used for local delivery of antimicrobial activity¹⁷; when

applied to contaminated surgical wounds the combination may discourage the formation of adhesions.^{18,19}

Fibrin glue is a blood product obtained from either commercial sources or some regional blood transfusion centres. The commercial products are produced from pools of plasma, and the final products usually contain high yields of fibrinogen and, as a result, produce firm coagulums. Fibrin glue derived from individual volunteer donations may have a low concentration of fibrinogen, but newer methods for small scale production are claimed to produce fibrinogen yields and coagulum tensile strengths similar to those of the commercial products.^{5,9} Until recently the thrombin used in preparations of fibrin glue was of bovine origin and caused a few serious systemic reactions including anaphylaxis and coagulopathy owing to the development of antibodies to thrombin.^{5,20} The problem has been solved by the use of human thrombin.

Non-commercial fibrin glue can be prepared from either homologous or autologous plasma; the autologous source avoids any possible risk of viral transmission. Homologous fibrin glue is prepared from donations screened in the standard, rigorous way and is as safe as other tested blood products. Most viruses can be inactivated by solvent detergent treatment, but this is ineffective against some viruses such as parvovirus B19²¹ and hepatitis A virus.²² Fibrin glue prepared from virally inactivated plasma is being assessed for safety and efficacy.²³ Another approach is to prepare fibrin glue from homologous fresh frozen plasma from donors in whom current tests for viral markers at least six months after the donation yield negative results. This simple retrospective accreditation measure excludes the theoretical possibility of the donors having been in the "window period" when they gave blood or plasma.

Despite the established value of fibrin glue for a range of surgical indications its use is still restricted to a few surgical specialties and on a named patient basis. It is widely used in most European countries. Concern about safety and unawareness of potential applications are the main factors limiting the availability and use of fibrin glue in Britain and the United States.⁵ Yet uncontrolled bleeding usually requires further blood transfusions with their comparable (but small) risk of