they thought would be delivering only average compression at the ankle.

A need clearly exists to ensure that prescribable bandages meet acceptable standards of manufacture and specified performance in terms of elasticity, elastic range, elastic modulus, and durability. Would it not be simpler to lay down these criteria and classify bandages according to some measure of elasticity and elastic modulus? The manufacturer could mark the bandage to ensure consistent application and could supply a chart with each bandage indicating the compression that would be achieved with a two layer or three layer overlap for different sizes of ankle. This would allow the doctor to build up a multilayer bandage to meet the needs of each patient. Such a bandage would be safer than a single layer of high compression, as errors in the application of a weaker bandage would average out in multiple layers—thereby reducing the risk of pressure necrosis. Simpler prescribing would be possible as a doctor need prescribe only a bandage capable of applying a given pressure to a measured ankle, and the pharmacist could dispense the most economical product meeting that specification.

Many surgical appliances, dressings, and bandages have been recognised in the drug tariff and have achieved a lucrative place in the market without having been subjected to adequate clinical trials.[3] This would be unacceptable in the pharmaceutical industry, where drug regulators insist on clinical efficacy. Surely this must be the goal for dressings and bandages and might be achieved if an advisory committee similar to the Committee on Safety of Medicines was set up.

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The fast of Ramadan

No problems for the well: the sick should avoid fasting

Healthy adult Muslims are required to abstain from food and drink from dawn to sunset daily during the month of Ramadan, which this year runs from 5 March to 3 April. Dispensation from fasting is allowed during sickness, menstruation, pregnancy, breast feeding, and travel. Recently, medical studies have focused on the effect of fasting on healthy people and its risks to patients with systemic disease.

In people who are well normal homoeostatic mechanisms seem to cope: urinary volume, electrolytes, pH, and nitrogen excretion remain within physiological limits.[1] Some studies have reported substantial weight loss and increased plasma concentrations of urea and uric acid[2] consistent with catabolism of body mass, but these findings have not been confirmed.[3] Some of the variations may be attributable to local traditions and food quality.

A trial of high carbohydrate intake (consumed after sunset) during the first fortnight of Ramadan was associated with a fall in blood urea concentration; a change to a high fat diet over the next fortnight was associated with a fall in glucose concentration, which the authors believed was due to impaired glucose homoeostasis.[4] With a normal diet hypoglycaemia does not occur. Those who consume high energy foods after sunset, unsurprisingly, gain weight.[5] Some studies of blood lipids have reported raised concentrations of cholesterol and triglycerides with changes in plasma apolipoproteins,[6] although this is not a universal finding.[7] Platelet function (assessed by aggregation), blood coagulation, and the fibrinolytic systems seem unaffected by fasting.[8] Although thyroid function does not alter, the diurnal rhythm of cortisol secretion is lost because of the change in sleeping habit. According to Ali and Amir, fasting is likely to reduce cognitive function because of the physical fatigue it induces in some people.[9] Dehydration and fasting should be avoided by people with renal colic and peptic ulcer disease.

The metabolic consequences of fasting during pregnancy have been studied in 11 women, who experienced a significant fall in concentrations of glucose, insulin, lactate, and carnitine and a rise in concentrations of triglycerides and hydroxybutyrate at the end of the fasting day.[10] This pattern of accelerated starvation was noticed by others only among women who fasted in late pregnancy.[11] A study of birth weights of more than 13 000 babies showed no effect of maternal fasting at any stage of pregnancy.[12] In another study lactating women lost 7·6% of their total body water during the hours of fasting, and their plasma osmolality and concentrations of sodium and uric acid rose more than in control subjects. Fasting changed the osmolality and concentrations of lactose, sodium, and potassium in breast milk.[13]

Patients who need to take drugs regularly throughout the day should seek their doctors' advice. One study found that more than half of patients could not keep to their prescribed drugs during Ramadan. Patients in need of frequent doses of drugs during the day should therefore avoid fasting. Hospital inpatients are not allowed to fast during Ramadan.

Insulin dependent diabetic patients are usually excused from fasting as their management becomes very difficult otherwise. In practice, non-insulin dependent diabetic patients run into very little trouble.[15,16] Asthmatic patients whose disease is stable may use inhalers, slow release drugs, and suppositories without breaking their fast. For patients with epilepsy, hypertension, endocrinal disorders, and psychiatric problems and those treated with long term oral anticoagulants a single night time dose of drugs may suffice. Patients receiving long term haemodialysis who insist on fasting may experience rises in potassium concentrations and body weight and fluid overload between dialysis sessions because of the tendency for increased food consumption at night. Renal transplant recipients with normal allograft function and intact renal concentrating ability should experience no harmful effects from fasting.[17]

Little is known with any certainty about the clinical problems during the fast of Ramadan. As perhaps 400 million people will be fasting this year during Ramadan the topic deserves more thorough scientific attention.

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Noise induced hearing loss

Could easily be prevented

People who suffer from noise induced hearing loss may be greatly handicapped, unable to participate fully in normal social affairs, and depressed and lonely. As noisy environments increase ever more people are at risk of such loss. Recently the new National Institute of Deafness and Other Communication Disorders in the United States sponsored a consensus conference on noise and hearing loss. Its final statement, drafted by the usual multidisciplinary panel after expert presentations and an open debate, shows how little we know about the causes and consequences of noise induced hearing loss.[1]

In the United States a little over 1 in 10 of the population suffer hearing loss, over one third of which is partially or wholly attributable to noise. Moreover, about 20 million Americans (1 in 13 of the population) are regularly exposed to hazardous sound level. Occupational causes are the most ubiquitous, but the additional effect of social and recreational exposure is important. Noise is thus a major public health problem, producing hearing loss at any age. Usually long exposure to excessive sound levels produces a gradual deterioration in hearing. The hearing loss is of variable severity and may be accompanied by tinnitus.

The consensus group agreed that a sound environment of below 75 dB was not harmful. Above that the amount of damage, the speed at which damage occurs, and the proportion of the population affected vary according to the sound level, the length of exposure, and individual susceptibility. For most industrial sound it takes daily exposure for several years to produce measurable damage. Above a certain level, however—140 dB or so—a single sound may produce permanent damage. In spite of intensive study the biology of noise induced hearing loss is ill understood. Clearly there is a critical sound level above which mechanical damage occurs in the cochlea and below which other factors, including "biochemical and metabolic exhaustion," are critical. Temporary damage imperceptibly gives way to permanent damage as hair cells die. There is also some degeneration of cochlear nerve fibres and changes in the central nervous system.[2]

Clinical assessment of noise induced hearing loss may be difficult. Characteristically an audiogram shows an audiometric notch, with the hearing loss greatest somewhere between 3 and 6 kHz. Further exposure and aging, however, lead to disappearance of the notch, and the audiogram may be indistinguishable from that produced by many other causes. Although noise induced hearing loss is usually bilateral, some asymmetry is not unusual. Great individual variation is found, in both humans and experimental animals. More men than women are affected, which is assumed to be due to their being exposed to more noise rather than to their being more susceptible. In diagnosing hearing loss much emphasis is placed on pure tone hearing loss, but in fact the inability to discriminate speech, particularly against a background of noise, produces the greatest impairment. The measurement of ability to communicate is inexact, and better scales are urgently needed.

Controversy remains about how to equate risk to hearing with different sound levels and exposure times. Is it simply the total sound energy to which the ear is exposed, as enshrined in the International Standard ISO 1999e? Or should the less aggressive American paradigm be used, which takes into account rest periods between exposure? No consensus was reached. Ironically, the conference veered towards the international standard, at a time when great doubt is being cast on the equal energy concept in Europe (RRA Coles, personal communication). None the less, all sound above a certain level can be harmful, whether occupational or recreational, and the two are additive.

Prevention of noise induced hearing loss begins with education. Recreational devices such as power tools should be labelled as hazardous, the use of personal hearing protectors should be encouraged at play as well as at work, and parents should educate their children about proper sound levels for listening to audio devices through personal headsets.[3] Comfortable hearing protectors should be more widely available. In the workplace greater educational efforts are required, government regulations should be monitored and implemented, and hearing conservation programmes should be given a higher profile. Enforcement of regulations governing workers' compensation might provide an incentive for better practices.

The consensus statement recommended further research, ranging from epidemiological studies to noise control engineering, from work on hearing protectors to basic biological research, including the genetic basis for susceptibility. After a century of research it is still not clear how noise damages the ear, but it is equally clear that this most common cause of acquired hearing loss in adults is totally preventable and that much human misery would be prevented if existing knowledge was implemented.

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