plasma volume ("relative polycythaemia"). Some case reports have suggested that chronic alcohol abuse may cause a sustained increase in the packed cell volume by this mechanism,[17,18] but drinkers are usually smokers and the relative influence of the two factors in such cases may be hard to establish.[19]

The evidence that chronic abuse of alcohol might lead to increased erythropoiesis and absolute polycythaemia rather than just a fall in plasma volume is circumstantial. Studies in animals with liver damage have shown that they have higher concentrations of erythropoietin in response to anaemia[20] and hypoxia[21] than animals with normal livers. An outbreak of hepatitis B in a renal unit led to dramatic rises in haemoglobin and fall in transfusion requirements in affected patients during their period of liver dysfunction.[22] These data suggest a link between liver disease and erythropoietin production or metabolism, and high concentrations of erythropoietin have been found in patients with hepatocellular carcinoma.[23] Possibly alcoholics with liver damage might have increased erythropoiesis and a tendency to absolute polycythaemia independent of any relative polycythaemia due to haemoconcentration, but the evidence is lacking.

A further possible mechanism deserves attention: intermittent hypoxia causes polycythaemia in animals,[24] and many authorities believe that hypoxia during sleep may be an important determinant of the degree of secondary polycythaemia developing in patients with chronic obstructive airways disease.[25-27] Alcohol depresses respiration[28]; in patients with obstructive sleep apnoea it increases the frequency and duration of apnoea and worsens desaturation, and it precipitates overt apnoea in snorers.[29] Heavy drinkers might have more pronounced nocturnal hypoxia than abstainers—with the consequent stimulation of erythropoiesis and a tendency to an absolute polycythaemia.

The message is clear regarding smoking in patients with polycythaemia, and measurement of carboxyhaemoglobin concentrations should be part of their investigation. Although polycythaemia is less certainly linked with alcohol, an association seems possible. The smoking and drinking habits of the patient should be borne in mind when a high packed cell volume—or absolute polycythaemia—is either inexplicable or seems to be out of proportion to the degree of any hypoxia present.

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Faecal incontinence is not inevitable

Faecal incontinence is one of the more unpleasant consequences of failing health in old age—distressing for the sufferer, and unpleasant for his or her carers. Despite the lack of published evidence geriatricians and the nurses who work with them know from experience that faecal incontinence is not inevitable. Why then is it not prevented?

The main obstacle is ignorance. Tobin and Brocklehurst have recently shown in a controlled trial the effectiveness of simple measures available to any primary care team.[1] From 30 residential homes in Manchester they chose 82 residents with regular faecal incontinence, a 10% prevalence in line with previous studies.[2,3] Those with diarrhoea were excluded. Fifty two residents were randomly selected for treatment and 30 as controls. The two groups were comparable in terms of age, duration of incontinence, mobility, and mental function. Three quarters were mentally impaired. So low were expectations that only two residents had been referred to their general practitioners—yet three quarters had been incontinent for over a year.

Faecal impaction was considered to be the cause of the incontinence when there was a history of continuous faecal soiling in the presence of a loaded rectum and a lax anal sphincter. Neurogenic incontinence was diagnosed when formed stools were passed and rectal examination gave normal results.

With the collaboration of the primary care team patients with faecal impaction were treated with daily enemas until there was no response. They were then given lactulose twice daily and a weekly enema to prevent recurrence. Those with neurogenic incontinence were treated with codeine phosphate to produce constipation. They were then given two enemas a week to produce a bowel movement at a predictable time.

After two months two thirds of the study patients were no longer incontinent compared with only a third of the controls. Those with faecal impaction did better than those with neurogenic incontinence and some patients did not comply fully with the prescribed regimen. In those in whom full compliance was achieved the results were even better,

87% regaining continence. Initially the staff had accepted the incontinence as inevitable and a nihilistic attitude had prevailed. But with increased awareness and appropriate medical and nursing support attitudes were revolutionised.

The authors estimate that as many as 10 000 old people in homes in Britain might be relieved of faecal incontinence if the lessons of this research were heeded. This well conducted study deserves wide publicity and highlights the need for better education in geriatric medicine among both doctors and those concerned with residential care.

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Spoiled soft contact lenses

A patient who damages or loses his or her own expensive hydrophilic contact lenses will be annoyed—but the complaint will be much louder if the damage is caused by drugs prescribed without appropriate warning.

Rifampicin may cause reddish discoloration of the urine, sputum, and tears. It may produce permanent orange discoloration of soft contact lenses, and data sheets advise that "the patient should be forewarned of these possible effects."[1] Similar discoloration has been reported with sulphasalazine.[2] Presumably other drugs which discolour tears or body fluids may also affect soft contact lenses.

It has long been known by both optical and medical contact lens practitioners, but not necessarily by all doctors, that fluorescein stains soft lenses and may permanently spoil them. A high molecular weight dimer of fluorescein (Fluorexan) does not concentrate as rapidly in soft lenses and may be used for fitting.[3] Alternative techniques avoiding the need for dyes are more usually employed. Rose Bengal is similarly contraindicated—but the concentrated dye may be removed by repeated leaching processes.

Topical preparations may also spoil lenses. Adrenochrome staining of the contact lenses of patients using adrenaline drops for treating glaucoma was reported in 1974[4] and 1976.[5] Melanin deposits may also appear in the conjunctiva of patients using adrenaline; when the drug is absorbed by these lenses ocular enzymes cause oxidation and polymerisation to a type of melanin. Though the melanin is locked into the lens, it may be cleared with hydrogen peroxide, which breaks the molecule into water soluble elements. In practice, such lenses are usually discarded.[4] The prodrug dipivalyl epinephrine (dipivefrine hydrochloride) has been used successfully without staining soft lenses,[6] but the inclusion of benzalkonium chloride as a preservative in some preparations contraindicates its use.

Soft contact lenses are spoiled in many other ways: some are "natural causes" such as the deposition of calcium on the surface or the build up of organic matter from the tear film and shed surface cells of the cornea and conjunctival sac. Such spoilation is of interest to both wearers and practitioners. The iatrogenic spoiling of soft lenses is much less well known, but of greater importance to the medical profession in general.

The number of contact lens wearers in this country is not known with any certainty, but 6% to 7% of people requiring refractive corrections are thought to wear contact lenses (in the United States the figure is double). There may be up to two and a half million contact lens wearers, and 60% of all contact lenses sold are soft. This figure does not reflect the proportion of wearers using soft lenses because these require more frequent replacement. Clearly a million or more people may be at risk of having their lenses spoiled by inadvertent use of drugs. The use of hard (polymethylmethacrylate) contact lenses, which may be worn by many patients throughout the waking day, avoids all the above disadvantages. Furthermore, patients will then have better vision through more durable, cheaper lenses which are easier to handle, sterilise, and store, and subject to none of the iatrogenic problems that will become commoner if the number of people taking drugs and wearing soft lenses increases. Soft lenses are ideal for the patient who cannot tolerate hard ones. Many wearers, however, have been fitted with soft lenses without a full and frank explanation of the options available. Soft lenses are certainly more comfortable at the initial fitting and in the early period of acclimatisation. This perpetuates the commercial and media pressure to sell this type of lens. The longer term problems may outweigh the short term gain, and iatrogenic spoiling of lenses is one factor to be put in the balance sheet.

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A senseless sacrifice: the fate of intercalated degrees

Among the many current threats to academic medicine is one that will be more harmful than it might seem. Each year about 10% of medical students decide to add an extra 12 months to their course and work for an "intercalated" honours degree in science (or occasionally subjects such as psychology or medical history). At Oxford, Cambridge, and Nottingham universities an honours degree year is built into the course and applies to all students. At the other British universities the intercalated year is optional, and for that reason education authorities rarely agree to pay the fees or give a grant for the extra time. Most students in England and Wales have been supported by the Medical Research Council, while in Scotland they have been funded by the Scottish Education Department.

Now it seems that the Medical Research Council is being pressed to stop the funding—not because anybody disputes the value of intercalated degrees but simply because of the page in the government's account book from which the money comes.