insulin resistance. Like most other clinicians concerned with hypertension, I have been to countless meetings devoted to these hypotheses, ending inevitably with the conclusion that the effects might be clinically important if relevant clinical evidence could be found. It never is. One hypothesis is succeeded by another. Like Omar Khayyam, we came out by the same door as in we went. Industry cannot be blamed, although the theories have proved profitable. As Poulter *et al* say, we would need at least 10 years to establish the clinical effects of newer drugs, and industry has inevitably taken the path of short term gains at the expense of a long term investment.[1] The probable returns from such an investment are dubious and in any case likely to be evident only long after patents have died. The biggest concern is that more than 10 years have come and gone since the newer drugs entered the market and we are still no further forward in assessing their impact. Instead, the field is befogged with inadequately tested hypotheses.

The therapeutic lessons are straightforward. We cannot predict outcome in such a complex clinical condition as ischaemic heart disease in a hypertensive patient by simplistic scientific hypotheses. Atheroma is not simply a metabolic disturbance: it is also a response to local mechanical factors. Patterns of turbulence reflecting different haemodynamic profiles produced by different classes of drug are likely to have different effects quite independently of the extent of blood pressure lowering.[7] These effects may outweigh any putative metabolic consequences of the drugs. This is certainly not a case in which outcome can be predicted from first principles. Newer drugs may or may not be better. None the less it would be an expensive speculation that multiplies the cost of treating hypertension more than 50-fold, particularly when a substantial proportion of the adult population is affected.

There is little hard evidence to guide us in the choice between β blockers and diuretics—as our working party pointed out. The balance has, however, tended to move in favour of β blockers despite their greater cost. Treating patients during or after myocardial infarction undoubtedly reduces the risk of death and subsequent myocardial infarction,[8] and many hypertensive patients in clinical practice will have established heart disease—in contrast to patients in most large trials. In addition, the overall infarction rate—that is, clinical and electrocardiographic infarctions—in the Medical Research Council trial was reduced in the β blocker group compared with the diuretic and placebo groups.[9] These are not the strongest arguments, but for many of us, faute de mieux, they tip the balance in favour of β blockers as first line treatment. Angiotensin converting enzyme inhibitors or the more recent calcium channel blockers or α blockers have a substantial if still unproved role when these drugs fail or are poorly tolerated. I find it difficult to see how this conclusion can change in the next decade in the absence of harder clinical evidence. If the position does change in favour of newer drugs in a cash constrained health service this can only be at the expense of a considerable impact on the care of patients with other conditions.

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Selling tobacco to children

Tobacconists selling single cigarettes help to get children hooked

People do not take up smoking in middle or old age. Most start smoking in their teens—when the long term health risks mean little and most believe that smoking is a mere habit, which can be dropped as easily as it is taken up.[1] Unfortunately this is not true. Once hooked it is difficult to stop, so most will continue to smoke as adults, until the first signs of ill health or other changes start making their mortality seem real. Then they will try to stop and discover what the United States Surgeon General announced officially in 1988—that smoking is an addiction.[2]

The most recent figures for England show that almost a quarter of boys and almost a third of girls aged 15 smoke,[3] and the rates are probably even higher in Scotland.[4] Altogether over 500 000 11-15 year olds in Britain smoke, and most of these will be hooked by the time they are 18. As about one quarter of all smokers will die prematurely through smoking[5] this means that about 100 000 of today's children will eventually be killed by their addiction. And the reduction in life expectancy is not inconsiderable, averaging 15 years among those who die early.[6]

Despite these disturbing figures relatively little has been done in Britain to make smoking unappealing to children. Tobacco advertising is attractive even to very young children[7 8] and reinforces underage smoking.[9] The government claims to want to protect young people,[10] yet the controls introduced through voluntary agreements with the tobacco industry have had little impact. They have been repeatedly breached,[11] and their limited scope ensures that children are regularly exposed to advertising and promotion through posters,[11] magazines,[12] and television.[13]

Price affects consumption powerfully in adults, and work in the United States has shown that consumption in children is even more sensitive to price.[14] Yet successive governments have failed to use this simple and effective mechanism to put tobacco beyond children's reach. Excellent school health education programmes have been developed, but their implementation is patchy. A recent survey found that under a third of first year secondary school children could recall having a lesson on smoking in the previous year.[3]

The Protection of Children (Tobacco) Act 1986 makes it illegal to sell any tobacco product to children aged under 16. The ease with which young children can buy cigarettes,

however, is well known. Research published last month in the *British Journal of Addiction* shows not just the extent of illegal tobacco sales to children but how cynically many shops ignore the law. The authors found that, in a large sample of 14-15 year olds over half the regular smokers reported having bought cigarettes one at a time from a shop.[15]

It is hard to appreciate what this really means. Many children cannot afford to buy whole packets of cigarettes. In effect shops sell them a dose of the drug they can afford until they are addicted. Then, when they can spend more, they will graduate to a larger, more regular dose. If this were cocaine or glue for sniffing, government action would be swift and decisive. Instead a drug that will eventually kill 100 000 of today's 11-15 year olds does not seem to matter.

Comprehensive government action is needed urgently. The major initiative on teenage smoking launched in England last year by the Health Education Authority is certainly a step in the right direction, although disappointingly the government has not extended it to the rest of the United Kingdom. Health education is crucial and has not been given anything like the resources justified by the enormity of the problem. By itself education is not enough, however, and government action should include a ban on all forms of tobacco promotion. We should follow the examples of Australia, Canada, and New Zealand, which have recognised that partial bans and voluntary agreements do not work.[16,17] Taxation on tobacco should be regularly increased above inflation and a proportion earmarked for research and health promotion, a move that has proved popular in Australia.[17] Finally, the law on sales to children should be enforced. Shopkeepers are rarely prosecuted, and current fines are too low to act as an effective disincentive. These two criticisms might be answered if a bill sponsored by Parents Against Tobacco is successful in the ballot for private members' bills. The Home Office supports the group's demand that the maximum fine for illegal sales of tobacco to children should be increased to £2000. The bill also calls for local authorities to enforce the law on sales to children, to outlaw the sale of single cigarettes, and to prohibit advertising of tobacco on shop fronts.

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Acute intestinal ischaemia

Resection rather than revascularisation

Vascular accidents are common in the heart, brain, and limbs, but until now the intestinal circulation has seemed fairly immune from trouble. When it does occur, however, infarction in the intestine is devastating because of the toxic and infective nature of the bowel contents, and the reported mortality of 80-90% has hardly shifted over the years.[1] A recent study from Israel suggests that aggressive treatment can reduce the mortality considerably,[2] but this may be a counsel of perfection.

In young and healthy people the mesenteric circulation practically never goes wrong. Age, however, brings with it a high incidence of atherosclerotic lesions in the visceral arteries.[3] Immunosuppression, systemic sepsis, major trauma, and the effects of cardiopulmonary bypass are all states that can diminish the mucosal blood flow and hence the efficiency of the intestinal mucosal barrier. Once oxygenation has fallen below a critical point a cascade of events is triggered off that is almost impossible to reverse and leads to multiorgan and system failure.

The precipitating factor seems to be the conversion of the enzyme xanthine dehydrogenase (present in high concentrations in the gut mucosa) to xanthine oxidase, resulting in the release of oxygen derived free radicals.[4] The released free radicals disrupt cell membranes and enzyme systems and still further damage the mucosal barrier, allowing several toxic substances to be absorbed through the portal blood and peritoneum. The most familiar of these is lipopolysaccharide endotoxin, which is derived from Gram negative organisms, but others include pseudomonas exotoxin (which is 10 000 times more potent than lipopolysaccharide[5]) myocardial depressant factors,[6] histamine, and free potassium ions.[7] Moreover, we now know that, even in normal circumstances, bacteria can translocate from the gut lumen into other areas (probably conveyed by the intact macrophage),[8] and this process will obviously be enhanced in ischaemia. As a result of these combined effects splanchnic ischaemia may injure the liver,[9] pancreas,[10] myocardium,[11] and lungs,[12] and may also cause down regulation of the immune system.[13] All in all it is not surprising that an acute reduction in the blood supply to the gut should be lethal. Moreover, the generation of free radicals seems to be accentuated by restoration of blood supply to the ischaemic tissue (reperfusion injury),[14] which explains the classic observation that release of an experimentally applied ligature on the superior mesenteric artery leads to circulatory collapse.

To apply this knowledge clinically, however, is not so easy. Ischaemic bowel disease is usually diagnosed late on the basis of clinical impression and the symptoms and signs are variable. Abdominal pain that is disproportionate to the physical findings, early leucocytosis, and metabolic acidosis are all suggestive, but no specific test distinguishes intestinal ischaemia from other acute abdominal conditions.[15]

In the recent series reported from Israel 92 patients treated between 1952 and 1987 were reviewed, excluding those with