urological investigation and monitored uneventfully after a non-invasive nephrological assessment. After a mean follow up of 3·5 years only two had developed non-glomerular haematuria after repeated urine microscopy, both of whom were later found to have underlying urothelial disease.

Despite its initial promise this technique has not been widely adopted in the United Kingdom,[21] probably because of concern about interobserver variations in reporting microscopy results.[23] Interest has instead been directed towards using automated haematological analysers to assess urinary erythrocyte morphology,[24,25] but the inaccuracy of this technique at low red cell counts may limit its practical application.[26] In our experience variations in reporting microscopy findings may be reduced with a more precise classification of urinary red cell variants (unpublished observations) and the accuracy of automated analysers substantially enhanced by using simple modifications.[27]

Over the next few years these newer techniques may well achieve acceptable standards of accuracy and reproducibility for routine use and, with the adoption of widespread urinary screening by general practitioners, are likely to have an impact on the practice of both nephrologists and urologists. Until then further investigation of patients with asymptomatic dipstick haematuria, even in the absence of confirmatory microscopy or other markers of underlying disease, is recommended. We also suggest a standard non-invasive nephrological assessment and long term follow up to detect potentially progressive renal disease in those patients with urologically unexplained blood loss.

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First line treatment in hypertension

Still β blockers and diuretics

The vigorous debate in the *BMJ* on first line treatment for hypertension[1,2] indicates the fragile nature of the agreed recommendations of the British Hypertension Society.[3] I had suspected at the time that the recommendations of the working party, which I had the dubious privilege of chairing, had all the durability of a peace settlement in Beirut, and so it has proved as members of our working party pull out their artillery. Nevertheless, I believe that the conclusions that we reached still stand and are, indeed, if anything reinforced by more recent evidence.

We concluded that diuretics and β blockers were still the preferred first line treatment in patients with uncomplicated hypertension. The reasons are straightforward. These drugs have been shown beyond dispute to reduce the risk of stroke in hypertensive patients. At the time our report was written a pooled analysis of the large trials of treatment had suggested, however, a disappointingly non-significant impact of treatment on coronary artery disease.[4] A more recent analysis with a slightly different mix of trials (and authors) concluded that the incidence of myocardial infarction was reduced by antihypertensive treatment, although the confidence intervals of this analysis were so wide that the results were also compatible with virtually no effect and complete reversal of hypertensive risk.[5]

We have no evidence from trials of the effect of the newer classes of drugs—that is, the angiotensin converting enzyme inhibitors, calcium antagonists, and α blockers—on either strokes or heart attacks. There is a further consideration that is of growing importance to us all. A year's course of bendrofluazide costs £2-£4; for calcium antagonists or angiotensin converting enzyme inhibitors the figure is £100-£200. Between 20% and 30% of the adult population are candidates for lifelong antihypertensive treatment, and the new contractual arrangements will inevitably encourage doctors to identify more of these patients. The newer drugs may or may not be better at reducing the risk from myocardial infarction. There are no data to guide us. The indications are hardly propitious. Trials of calcium antagonists after myocardial infarction, justified by theoretical arguments, far from showing efficacy, showed a slight worsening of prognosis.[6]

In the absence of clinical evidence the pharmaceutical industry has promoted a series of scientific hypotheses which predict that the newer drugs might be better at preventing myocardial infarction. Thus we have been told that the calcium antagonists reverse a specific abnormality of smooth muscle calcium handling and that angiotensin converting enzyme inhibitors correct angiotensin II receptor modulation, cardiovascular structural remodelling, and (most recently)

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,