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

strangulated hernia and non-occlusive infarction.[2] Thirty were further excluded because the diagnosis was reached only at necropsy or because no attempt was made at surgical rescue. The remaining 62 patients were subdivided into those treated during the first 14 years of the study and those treated in the second 12 years. In the first period the policy was resection of apparently dead bowel followed by primary anastomosis, which carried a mortality of 82% (14/17). In the second period it became possible to revascularise the bowel. The mortality fell to 21% (9/45), and on the basis of this the authors recommend a more aggressive policy, including emergency aortography and a "second look" operation in every doubtful case. Others have proposed the same approach[16] while making little distinction between arterial and venous obstruction. Although clinically indistinguishable, cases of arterial and venous obstruction differ widely in cause and outlook. A vocal and articulate group argues strongly in favour of the selective injection of vasodilators into the superior mesenteric artery in all suspected cases in which the arteriogram suggests the presence of vasospasm.[17]

The difficulty is to reconcile the advances in our knowledge of the role of the intestinal circulation in all manner of critical illnesses with the problems that confront the surgeon in an ordinary hospital. Intestinal ischaemia is comparatively rare, and most surgeons will not have a special interest in it. They do, however, see many elderly people with chronic or acute abdominal pain. Most chronic abdominal pain is not connected with the intestinal circulation, but some of it is, and the relief of intestinal angina is worth while, although the diagnosis is not easy.[18] These patients are in danger of a fatal infarct, and eliminating this risk factor would certainly improve results.[19]

Clearly an aortogram in a "suspected case" of acute intestinal ischaemia would be useful, and some advocate this as a routine procedure. But what its proponents have never reported is the number of negative investigations that their policy has thrown up. We need to learn the size of the iceberg before commenting on the importance of its tip—in other words, the specificity of an expensive and invasive test.

Given the limitations of aortography and the relative uselessness of laboratory investigations in acute intestinal ischaemia[20 21] we have to accept that in practice the diagnosis is often made by a surgeon who has opened an abdomen because of peritonitis of unknown origin. What should he or she do?

Obviously, conservation of bowel length is important, and a young patient with a mesenteric embolus should have it removed, provided that the gut is viable. Although few surgeons will have experience of doing this operation, most know how it should be done and will do it reasonably well. A second look operation on the next day is essential, when any dead bowel can be resected. This condition is sufficiently rare to make little demand on resources.

The elderly patient who is discovered at surgery to have ischaemically compromised bowel is in another category. If the entire tract from stomach to colon is infarcted it is inoperable, but lesser degrees of damage deserve a more optimistic and aggressive approach. People can exist normally on less than 25 cm of jejunum, so if the remaining body systems are in reasonable shape it makes sense to resect massive lengths of compromised small bowel. Primary anastomosis must be avoided[22]: it is far better to bring out the ends of the normal bowel to the abdominal wall and restore continuity later, when the patient has regained metabolic normality.

To revascularise or resect? The first is the more glamorous choice and hence the one which has achieved attention in the surgical journals. But although the visceral arteries can be reconstructed by bypass, reimplantation, or endarterectomy, they are inaccessible and unfamiliar territory for most general and vascular surgeons—any of whom, however, can resect the bowel. The deleterious effects of reperfusion injury in the ischaemic bowel are only now becoming apparent, as are the capabilities for hypertrophy of viable segments. Arguably, therefore, the time has come for a strategic retreat from the position of "vascularise at all costs" to one of "remove the damage and hope for the best."

Given our new found appreciation of the role of the intestinal circulation, it is important to identify patients at risk and to devise measures to help them. Risk factors include pre-existing occlusion of the superior mesenteric artery, immunosuppression, digitalis overdose,[23] major sepsis,[24] non-pulsatile cardiac bypass,[25] and several others.[26 27] Protective measures (apart from the maintenance of normal homoeostasis, which has become routine in intensive care units) include vasodilator drugs such as papaverine injected directly into the superior mesenteric artery; antibiotics (polymyxin B may have a specific effect against lipopolysaccharide endotoxin[28]); and anticoagulants. From the laboratory evidence it would seem logical to use antioxidants such as allopurinol (which inhibits xanthine oxidase),[29] superoxide dismutase (which scavenges the superoxide radical),[30] and dimethyl sulphoxide (which mops up peroxide). The timing of such treatment, however, is difficult to determine. Ideally, it should be administered before the ischaemic injury occurs, or at least before reperfusion is attempted, but this does not often fit with clinical realities. Knowledge of these treatments derives entirely from the laboratory, and as yet there is no clinical information on their uses or dangers.

Given the nature of the illness, controlled trials in the management of acute intestinal ischaemia will never be practicable. None the less, it would seem sensible to offer such measures to people with abdominal pain and a history of intestinal vascular disease, particularly if a reconstructive operation is planned.

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Vitamin A, liver consumption, and risk of birth defects

Liver is a cheap source of many nutrients

An excessive intake of vitamin A immediately before or during pregnancy substantially increases the risk of birth defects.[1] The Teratology Society has therefore recommended that vitamin A supplementation (as retinol or retinyl esters) should be limited to 8000 IU (2400 μg) a day.[2] The chief medical officer has reiterated this advice in even stronger terms by cautioning women who are, or who may become, pregnant "against taking any dietary supplements—including tablets and fish-liver oil drops—containing vitamin A, except on the advice of a doctor or antenatal clinic."[3] He goes on to say that as a matter of prudence such women should not eat liver and liver products because of their high retinol content. Is he being overcautious and sowing the seeds of yet another food scare?

A recent epidemiological study of birth defects diagnosable within the first three days of life showed that among 11 293 non-chromosomal cases of birth defect and 11 193 controls, 16 cases and 14 controls were exposed to vitamin A intakes over 10 000 IU/day (odds ratio 1·1; 95% confidence interval 0·5 to 2·5). The use of vitamin A alone rather than multivitamin supplements conferred a much higher risk (9·9; 1·4 to 430·1), probably because of the higher doses received (63 636 IU *v* 20 263 IU). When a dose-response relation was examined intakes over 40 000 IU were associated with an odds ratio of 2·7 (0·8 to 11·7), and the risk of birth defects was highest when supplements were taken in the first two months of pregnancy. None of the mothers had a clear medical reason for taking vitamin A supplements. The authors concluded that exposures below about 10 000 IU/day were unlikely to be teratogenic.

Typical dietary intakes of retinol from food sources among women of childbearing age in Britain were recently reported to be about 1400 IU/day.[5] The 97·5 centile was around 20 000 IU/day over the one week survey. This does not imply that 2·5% of women were consuming more than 20 000 IU/day week in, week out but rather that in the week of the survey they consumed high amounts; their average intake, measured over several weeks, would probably have been substantially less. There is no evidence, therefore, that most women are in danger of exceeding an average intake of 10 000 IU/day.

The average retinol content of liver has increased over the past decade to reach values typically between 66 000 IU and 130 000 IU/100 g. These high levels might be of concern for women who regularly eat more than 100 g of liver a week, but there is only one reported case of birth defects in a child born to a woman who ate large amounts of liver daily. Given that the placental barrier to vitamin A is incomplete between the fourth and tenth weeks of gestation,[6] there might be concern about a bolus effect, but there has been only one reported case to substantiate that concern: a woman who accidentally ingested 500 000 IU in the second month of pregnancy gave birth to an infant with multiple birth defects.[7] All other reports of birth defects have been associated with a daily consumption of 25 000 IU or more over several weeks or months.[1] The recommendation that a large segment of the population should exclude liver entirely could thus be seen as an overinterpretation of very limited evidence.

It is important to remember the other end of the nutritional spectrum: those women whose diets may be lacking in nutrients. Doyle and colleagues reported that among women on low incomes low birth weight was significantly associated with poor intakes of thiamin, riboflavin, niacin, zinc and iron,[8] all of which (together with other nutrients such as folate and vitamin B-12) liver provides in generous amounts at low cost.[9] Fetal nutritional insufficiency, low birth weight, and the associated risk of defects is thus likely to be a far greater problem on a population basis than the risk of defect associated with vitamin A toxicity.

Most women do not need to take vitamin A supplements either before or during pregnancy, and those who do should be under medical supervision. There is justifiable concern about the excessive concentrations of vitamin A in liver, but it is equally important to remember that liver is a source of many nutrients and a desirable adjunct to the diets of many women, particularly those on low incomes. It might therefore be sensible to be less alarmist and to advise women who are pregnant or who may become pregnant not to consume more than 50 g of liver a week until the amount of vitamin A in liver is reduced through appropriate action by the meat producers. Consumption of liver sausage or paté, in which the liver is diluted by the inclusion of other ingredients, should similarly be limited to about 100 g a week.

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