and has made explicit the criteria it will use in deciding which regulatory actions to take. Because the guidance has only just been issued there is no experience with its use. Much of its success will depend on how much the NHS Executive can separate out its task as market manager from the many other tasks it undertakes. If it cannot, regulatory rules may be downplayed or ignored to ensure that other goals are met.

The executive's guidance deals less with the second type of regulatory activity—controlling monopoly. Arguably, this is the more important task facing an NHS regulator. One reason for the lack of guidance is that the NHS Executive already regulates the behaviour of providers through trusts' financial arrangements and the behaviour of district health authority purchasers through its line management of purchasers. But these forms of regulation are questionable. Under trusts' financial arrangements the "price equals cost" form of price regulation used by the executive is difficult to monitor and gives poor incentives for efficiency. Trusts are not allowed to have formal saving mechanisms, and in the long run this makes decentralised decision making inefficient: those who work harder than the average are offered no reward for improving the quality and quantity of services delivered.

What is urgently needed is a review of these arrangements. Other regulatory tools are used elsewhere in the British economy and in other health care systems. The executive should assess where such regulation would be useful and then design tools to regulate simply those activities. Regulation of all prices in all circumstances is not likely to be necessary or helpful.

The importance of the purchasing role means that poor incentives for purchasers to respond to the needs of their users may pose the largest threat to the beneficial workings of the internal market. More needs to be done to ensure that purchasers respond. Ways to compare purchasers and to simulate the effects of competition where no competition exists should be developed. League tables are a partial example of such measures. To simulate competition fully, however, those organisations that are not up to a suitable standard must face some penalty. Franchising (competition

for the right to act as the purchaser) is another possible route. Another, more radical, option is to increase competition in the market by allowing greater competition among general practitioners for patients, perhaps by changing the rules on where general practitioners can set up new practices. All these mechanisms have their associated pros and cons. What is important is that the executive identifies where the problem lies, limits regulation to those areas, and uses mechanisms that give purchasers positive incentives to improve their behaviour where it is lacking.

Theoretically, the 1980 Competition Act could be interpreted as implying that procompetition policy is the responsibility of the Monopolies and Mergers Commission (the body responsible for procompetition policy in the rest of the economy).² The NHS Executive has currently taken that role on itself. In the long run, "regulator capture" (where the regulator acts to protect the regulated and not the general public interest) may occur. It may then be sensible to have a three tier system with the Monopolies and Mergers Commission responsible for competition policy; a separate regulatory body—an Ofhealth—to set prices and monitor customers' complaints; and the executive to manage providers and purchasers to ensure that central policy goals are met within the framework of the internal market. Given the mixed experience of regulation of the utilities in Britain, it may be best to give the present arrangements time to prove themselves while pressing the Department of Health to review its price regulation of trusts and its policy to ensure that purchasers meet the needs of their patients and populations.

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Hepatitis C and haemophilia

Coinfection with HIV is common and will demand great resources

The haemophilic community in Britain, already hit by HIV infection, is now facing the problem of chronic hepatitis C. The high incidence of hepatitis after treatment with clotting factor concentrate from a large pool was first identified by Kasper and Kipnis in 1972.¹ They found that infection was more common in young patients having their first treatment. It is now known that there was virtually a 100% rate of transmission of hepatitis C virus to previously untreated patients with haemophilia until effective procedures to inactivate the virus were introduced in 1985–6.^{2,3} Transmission of hepatitis C occurred with plasma from NHS and commercial sources. Studies of the hepatitis C virus genotype found in British patients with haemophilia who have been treated with concentrates have shown types 1, 2, and 3, reflecting contamination from the British and North American donor pool. Type 4 (reported in the Middle East and Zaire) and type 5 (reported in South Africa) have also, however, been found in a few patients, leading to speculation about the source of some commercial concentrates.⁴

The scale of the problem is large: 232 out of 241 patients at

the Royal Free Hospital in London who had received unsterilised clotting factor concentrate from large donor pool were positive for antibodies to hepatitis C.⁵ The date of first exposure was documented in 183 patients, and from these data it was calculated that over 11% could be expected to have liver failure at 20 years. Furthermore, we know that these patients were infected from 1965 and on average 17 years ago. In Britain there are 3122 haemophilic patients who have been treated with unsterilised blood products since 1977 (when the United Kingdom Haemophilia Directors Organisation started its registry). All of these patients must be infected with hepatitis C.

Two large studies in haemophilic patients have shown that coinfection with HIV could accelerate the development of liver failure.^{5,6} Viral replication of hepatitis C is increased in the presence of HIV (probably because of immune deficiency). From 1978 to 1993 concentrations of hepatitis C RNA in co-infected haemophilic patients increased 58 times compared with a trebling in those infected with HCV alone.⁷ The relative risk of developing liver failure in hepatitis C infection

increased 21-fold after HIV infection.⁵ All patients who are infected with HIV from clotting factor concentrate must, therefore, also be infected with hepatitis C. In our centre 10 out of 11 patients who died of liver failure were infected with HIV. This can cause formidable management problems. The failing liver can cause other clotting factor deficiencies that are superimposed on the existing deficiency of factor VIII or IX. A severe haemorrhagic state may develop in the final stages of the illness.

Patients with liver cirrhosis are at an increased risk of developing hepatocellular carcinoma, and the same could be true of patients with haemophilia. A worldwide questionnaire sent to 11 801 patients with haemophilia identified 10 cases of hepatocellular carcinoma—a risk 30 times higher than normal.⁸

The sexual transmission of hepatitis C virus is an important issue for patients with haemophilia, their families, and those involved in their health care. Studies indicate that transmission is rare.⁹ HIV infection has been reported as a cofactor and could reflect the higher viral load of hepatitis C virus in coinfecting people.¹⁰ The use of barrier contraception (condoms) is likely to increase safety.

What about treatment? Interferon alfa remains the most promising treatment for hepatitis C. For the patients without haemophilia, liver biopsy is essential in deciding who will benefit from treatment, but this is a hazardous procedure for a patient with haemophilia. Knowledge of other variables, such as hepatitis C virus genotype and viral load, may be helpful as patients with virus types 2 and 3 and with lower viral loads have the greatest chances of responding.¹¹

Patients treated with multiple batches of concentrate will have been exposed to a large amount of virus as well as to many viral genotypes.^{4,12} Since patients are infected with multiple species, a change in genotype is likely to be due to a change in dominance. The clinical importance of the change in dominance brought about by treatment with interferon is unclear.¹² The ultimate treatment for liver failure is liver transplantation, and this has successfully been performed in patients with haemophilia, curing not only the liver failure but also the haemophilia. The liver, however, may be

reinfecting with hepatitis C virus, and transplantation is difficult for patients infected with HIV.

The progression of hepatitis C and coinfection with HIV in haemophilic patients will demand tremendous resources over the next decade. It is important to appreciate the achievements that have been made in treating haemophilia despite their devastating side effects. In 1937 Birch wrote a descriptive monograph on haemophilia; she reported that 82 out of 113 patients died before their 15th year and only 6 out of 113 lived beyond the age of 40.¹³ We now have recombinant factor VIII (and soon factor IX), which cannot transmit bloodborne viruses. We need the resources to provide these factors for the children with haemophilia who were born after 1985. They are free of the scourges of hepatitis C virus and HIV, and prophylaxis with a safe clotting factor concentrate could offer them a full life without disability.

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Integrating pharmacy fully into the primary care team

Much to recommend it

Since teamwork, especially in small, cohesive task specific groups,¹ is the central plank on which primary care is now being built, greater interaction between pharmacists and general practitioners could be fruitful. Nevertheless, personal communications from ministers and civil servants reiterate the traditional view that "the separation of prescribing and dispensing ensures that the skills of doctors and pharmacists are used to best effect and that the public has access to both professions." It is time that these attitudes received the same scrutiny that the rest of primary care has received, especially as the cost of distributing medicines through pharmacies accounts for 30-40% of the total medicines bill.²

Changes in dispensing are under way in other countries,³ involving, for example, mail order. But these fail to build on potential professional collaboration, the desirability of which was implied in a Nuffield report of 1986: "closer relations between GPs and community pharmacists would be in the interests of patients and . . . more efficient use of resources

within the NHS."⁴ An opportunity exists for Britain to pioneer the integration of pharmacy into primary care.

If community dispensing were to become a central function of the primary health care team, with both geographic and functional integration of pharmacy, wide ranging and large scale benefits to patients, health care professionals, and the exchequer could accrue. Linked consultations between patient, doctor, pharmacist and other team members could be a radical new force in primary care.

Exhortations to closer cooperation between community pharmacy and general practice are no substitute for genuine integration, with the professions on the same team sharing work space, hours, databases, and budgets. Furthermore, commercial pressures would be likely to limit the scope of such closer cooperation, given current arrangements. Professional advice is difficult to submit to quality assurance but integration, with constructive mutual scrutiny and criticism by team members sharing the same values and