on the incidence, aetiology, and prognosis of the condition are still premature. As regards management, most authorities now recommend prophylactic antibiotic cover against bacterial endocarditis when there is a murmur, but not when a click is the only sign[23]; anticoagulant treatment is not justified unless there has been evidence of systemic emboli. Above all the physician must steer the difficult course between engendering anxiety by overconcern and investigation and treating unsympathetically symptoms such as atypical chest pain, which may seem neurotic but which are likely to have a physical basis.

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Understanding hepatic regeneration

The liver has a considerable capacity to regenerate after a surgical or toxic insult. Studies in rats have shown rapid cell division in the first four days after a two-thirds partial hepatectomy, with the size of the liver restored within two to three weeks.[1] The rate of liver regeneration in man is slower: serial liver scans, liver biopsy specimens, and repeat laparotomy have shown that though regeneration begins within three days of surgical resection, the process is not complete for about six months.[2]

When resection of an otherwise healthy liver is needed to control bleeding after trauma or to remove a localised tumour the mortality is low (once past the immediate postoperative period), with a complete return to normal function. In contrast, when liver resection has been attempted in patients with cirrhosis there is little regeneration; such patients often go into liver failure, leading to coma and death. The clinical circumstances in which liver regeneration is most obviously essential to survival is in fulminant hepatic failure, where there is massive necrosis of liver cells. Liver regeneration may be inhibited in some of these patients because of circulating toxins[3] or an unfavourable hormonal balance, but the explanation is far from well defined.

Indeed, despite much research into the factors that control regeneration of the liver many of the mysteries remain. A distinction needs to be drawn between those substances that initiate cell division and those that promote regeneration once it has begun. Most hypotheses have centred on blood-borne factors that might control regeneration. There may be stimulatory substances which are normally removed by the intact liver or else hepatotrophic substances which are released by the damaged liver or another organ in response to liver damage. Alternatively, the liver may continuously produce inhibitory substances, whose concentrations fall with liver damage.[4 5]

For some time the altered blood flow within the hepatic remnant was considered to be the main stimulus for regrowth, but careful studies in animals showed that regeneration still occurred after portal ligation or portacaval shunt. Hormones —particularly those from the gut—are now thought likely to play an important part in liver regeneration.[6] Infusion of both insulin and glucagon into eviscerated rats promotes regeneration of the liver and will increase survival rates in mice with liver failure due to murine hepatitis virus. Thyroxine, parathyroid hormone, calcitonin, growth hormone, and epidermal growth factor (an insulin-like peptide chemically identical with urogastrone) will increase liver regeneration, whereas adrenal hormones suppress the response.

Some recent studies, particularly those of Terblanche *et al*[7] and Goldberg *et al*,[8] have focused attention on a hepatic regenerative stimulator substance which can be extracted from the regenerating liver. This factor has been prepared from both dogs and rats after partial hepatectomy and will stimulate synthesis of DNA and mitosis in both normal animals and those that have had a partial hepatectomy. Further work is needed to characterise this substance. The part possibly played by prostaglandins in the early stages of liver regeneration is now being investigated. Increased concentrations of prostaglandin E_1 have been found in portal blood. Inhibitors of prostaglandin synthesis inhibit synthesis of DNA in the hepatic remnant[9] and their contribution should be evaluated further.

So while there is as yet no clear understanding of the factors controlling liver regeneration, it seems likely to be a multifactorial process with hormone changes playing a major part. Purification of the hepatic regenerative stimulator substance could possibly be of clinical value for increasing the regenerative response in some diseases.

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Patient participation: more pipedream than practice?

Are those enthusiasts who want to spread the idea of patient participation groups in general practice getting their message across to the right people? To judge by a recent conference at the King's Fund Centre it seems not. Representatives of community health councils, area health authorities, the press, and students and lecturers were more in evidence than general practitioners and patients. Moreover, no good evidence was produced that the groups are beneficial, and some people had unrealistic expectations about what they might achieve.

It is now eight years since the first patient participation group was started, and there are still only 32—a drop in the ocean when there are nearly 11 000 practices in Britain. Most have been started not by patients but by doctors, and one reason there are so few may be that many general practitioners have not heard of the idea: thus 10 of 15 general practitioners in the north west of England who were responsible for training groups of doctors had not heard of patient participation groups.[1] Groups have started up to meet the needs of their particular practices and do not conform to any particular pattern. Health centres seem to have the right conditions, but nobody knows why none flourish in single-handed practices. Perhaps a doctor practising alone is more likely to build up close relationships with his patients, and only those who work in and attend large health centres feel the need for a structured group to encourage communication. Nor does anyone know what patient participation groups achieve or even try to achieve. The aims of some are very broad: their accomplishments include helping doctors; looking at how the services are used—for example, whether it is practicable to have an antenatal clinic at 1 pm for working mothers; organising car services to surgeries in areas with poor public transport; and planning health education activities. But they seem unlikely to achieve anything as ambitious as changing the way that doctors behave in individual consultations. The conference heard several speakers argue for better communication between doctors and individual patients, but nobody made it clear what patient participation groups can do about this.

Understandably community health councils are interested in what patient participation groups are doing and want to work more closely with them nationally and locally. But the health councils cover large geographical areas and are concerned with hundreds of thousands of patients, while the main virtue of patient participation groups is that they work locally. Patients work together with general practitioners and other health staff to improve the services for their own "community" in their own practice. It is surely human nature to want to focus on narrow personal interests, but if the idea of patient participation really caught on in general practice the benefit to the larger "community" might be great. The health councils must not expect too much too soon.

So should every practice have a patient participation group? Those who have successful groups seem to enjoy them and be convinced of their worth, but some groups have faded away and some practices have found it impossible to start them. Also no one has produced convincing evidence of benefit. Nevertheless, Dr John Horder, president of the Royal College of General Practitioners, was convinced of their worth, advocating at the conference that all trainees in general practice should learn about them and going on to say that this should be added to the college's priorities of prevention and audit.

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Death of a quango

Quangos are not popular, so ministers run little risk of disapproval when they kill one off. If the quango is a minor one—a mere subcommittee—its demise is unlikely to be newsworthy. Yet one such subcommittee, which has recently been eliminated by the health and agriculture ministers, deserves at least a decent obituary and perhaps even an attempt to find a way of performing the functions intended for it but never made possible.

The quango in question was the Joint Sub-Committee on Anti-microbial Substances. It was set up as a result of recommendations made by the Swann Committee,[1] which was created because of concern that the use of antibiotics for promoting growth in animal husbandry without proper veterinary supervision might produce a great reservoir of antibiotic-resistant bacteria which would prejudice the use of antibiotics as therapeutic agents for man and animals. The relevant recommendations were ". . . that one committee should have overall responsibility for the whole field of use of antibiotics and related substances whether in man, animals, food preservation or for other purposes . . . and that this committee should be empowered to demand, on a basis of confidentiality, such returns as it considers to be necessary." The Swann Report and its recommendations were blessed by the Labour Government in 1969 and by the Conservative Government elected