heads of departments (biochemistry, haematology, histopathology, and microbiology), who are individual budget holders, and the senior chief medical laboratory scientific officer. One of the heads acts as coordinating chairman for a period of three years, and our system has worked well so far.

Hospitals throughout the country are in the throes of having new management structures and we are apprehensive about the far reaching implications on the terms and conditions of service as well as the professional independence of consultants in general, and departmental heads in pathology in particular. We have already raised this issue with the Royal College of Pathologists. We therefore urge the British Medical Association, the royal college, and other professional bodies to formulate a joint response and guidance to consultants on management structures under Griffiths.

MONICA GREGORY
J R B WILLIAMS
A F M S RAHMAN

Lister Hospital,
Stevenage, Herts SG1 4AB

Points

Tricyclic induced jitteriness—a form of akathisia?

Drs V K YERAGANI, M S KESHAVAN, and R POHL (Wayne State University of Medicine, Detroit, MI 48207, USA) write: We read with interest Dr E Szabadi's article (19 April, p 1034) discussing the pathophysiology of akathisia induced by neuroleptics, reflecting the blockade of dopamine receptors in the frontal cortex innervated by the mesocortical dopamine pathway. Some patients with panic disorder are exquisitely sensitive to imipramine, a tricyclic antidepressant, and respond with symptoms of insomnia, jitteriness, and irritability.[1] Some of the often reported symptoms are "trouble sitting still" and "shakiness inside." We have also observed these symptoms in patients with panic disorder treated with low doses of desipramine, a predominantly noradrenergic reuptake blocker. These symptoms usually subside when the dose of the tricyclic is gradually increased. In our experience the syndrome of jitteriness shares some common features with subjective akathisia. We have found that the syndrome of jitteriness responded to low doses of perphenazine, a neuroleptic, in two patients with panic disorder.[2] Reuptake inhibition of monoamines by tricyclic agents may have been responsible for the jitteriness syndrome in these patients and perphenazine may have relieved these symptoms by means of its dopamine or adrenergic blocking effects, or both. Adler *et al* have recently reported that both propranolol and clonidine were beneficial in the treatment of akathisia.[3] This probably reflects noradrenergic involvement in the pathophysiology of akathisia. Thus the tricyclic induced jitteriness may throw some new light on future research dealing with the pathophysiology of akathisia.

1 Zitrin CM, Klein OF, Woerner MG. Treatment of agoraphobia with group exposure in vivo and imipramine. *Arch Gen Psychiatry* 1980;37:63-72.
2 Pohl R, Yeragani VK, Ortiz A, *et al*. Response of tricyclic-induced jitteriness to a phenothiazine in two patients. *J Clin Psychiatry* (in press).
3 Adler LA, Angrist B, Peselow E, *et al*. Noradrenergic mechanisms in akathisia. Abstracts of the 139th annual meeting of the American Psychiatric Association, Washington DC, USA, 10-16 May, 1986, p 74.

HTLV-III: echoes of the past?

Dr P D WOOLLEY (Nether Edge Hospital, Sheffield S11 9EL) writes: Controversy reigns over routine testing for human lymphotropic virus type III. Dr David Miller and others (5 April, p 941) contend that routine testing is unnecessary, while Dr G R Kinghorn (3 May, p 1202) advocates that isolation of the asymptomatic carrier is of crucial importance in controlling the spread of the disease. Early this century syphilis, with no reliable method of detection or treatment beyond the primary stage, was viewed with similar public and medical concern as HTLV-III is today. Our results of performing routine syphilitic serology on 1820 patients showed 42 (2·3%) with positive results, indicating previous contact with syphilis. Of these, 32 (1·8%), with a mean age of 82·1 years, had no knowledge of the infection. Over 2% of patients tested had had contact with the spirochete, the majority of which were undiagnosed. Clearly the number of people affected by syphilis in the early part of this century was far greater than appreciated at the time. Fortunately with the widespread availability of penicillin and related antibiotics after the second world war most of these will have been treated with antibiotics for unrelated conditions. Let us hope that those who argue against routine HTLV-III testing do not add to the problem by failing to isolate asymptomatic carriers. Perhaps we should learn from the past.

Dr SUSAN BEWLEY (Department of Obstetrics and Gynaecology, Dulwich Hospital, London SE22 8PT) writes: The medical profession must encourage (not force) testing for HTLV-III antibodies, provided there is adequate counselling, despite the limitations of the test. There is an epidemic of AIDS, AIDS related diseases, and asymptomatic infections, for which we have no treatment at all. Prevention of spread is the only intervention available. Even if the problem of spread is tackled head on now, the effects will not be noticed for several years. In particular, there is an objection to the suggestion of Dr David Miller and others (5 April, p 941) of routinely screening women in high risk groups who are contemplating becoming pregnant. Increasingly, women are going to be identified as high risk because they have an antibody positive partner. Obstetricians may be able to identify female intravenous drug abusers, but the idea that they can identify the antibody positive drug abusing partners who do not attend the antenatal clinic or that they can recognise that a woman has a bisexual partner, who is unlikely to announce the fact, is absurd. They are going to need HTLV-III antibody results to be aware that their pregnant patients and their babies, and the midwifery staff, are at risk.

Risk of AIDS to health care workers

Dr MARGARET HUGHES (University Hospital of Wales, Cardiff CF4 4XW) writes: With regard to the penultimate and last paragraphs of Dr P H Wright's letter (3 May, p 1202), I am sure that the provisions of the little known, and perhaps inappropriately named, National Health Service injury benefit scheme would, to a considerable extent, compensate any health care worker who was unfortunate enough to acquire infection as a result of occupational exposure. Briefly, an employee's salary would be made up to 85% of normal in the event of his acquiring such an infection occupationally; a claim for such occupational illness is not restricted to the prescribed diseases and may be extended to a much wider interpretation.

Early mobilisation of acute whiplash injuries

MR GRAEME D RICHARDSON (Wagga Wagga, NSW, Australia) writes: Four weeks ago I was the victim of a severe rear end collision on my way to an afternoon operating list. Two hours later I operated for one hour, but the next morning I knew what a "whiplash" injury meant. For one week I had to steady my head to lift it from a pillow, such was the pain from all flexor, strap, and deep extensor muscles. The weight of my head has since caused severe extensor and trapezius muscle spasm, necessitating bed rest after two hours in the second week and four hours in the third week. A soft collar not only supported my head but has prevented me from placing added strain on my neck in my effort to return to the operating theatre. A severe soft tissue injury to my normal 40 year old neck demanded initial rest and surely requires different treatment from a minor injury to an older person with cervical spondylosis. Having examined many necks for insurance reports, I was a little alarmed to find I also had strange transient paraesthesia of areas of an arm and a leg and still have grating noises in my neck and severe headaches. While I agree with gentle stretching and mobilisation where possible (8 March, p 656), let us not discard soft collars and add to muscle trauma by overenthusiastic exercises for a condition which causes genuine symptoms and signs.

Treatment of type II diabetes

Dr E H MCLAREN (Diabetic Clinic, Stobhill General Hospital, Glasgow G21 3UW) writes: It is disappointing in an article entitled "Treatment of type II diabetes" to see that dietary manipulation is accorded two words, while the rest of the article is devoted to a consideration of sulphonylureas and insulin (19 April, p 1033). As Hadden *et al* have so convincingly shown, metabolic control in type II diabetes can be achieved by dietary means alone in most patients,[1] and the prescription of oral hypoglycaemic agents should be regarded as an admission of failure of either the medical team in providing dietary advice or the patient in following it.

1 Hadden DR, Montgomery DAD, Skelly RJ, *et al*. Maturity onset diabetes mellitus: response to intensive dietary management. *Br Med J* 1975;iii:276-80.

Terminal care at home: perspective from general practice

Dr VAL WASS (Chislehurst, Kent BR7 5AX) writes: The paper by Dr Andrew Haines and Ms Angela Booroff is to be welcomed (19 April, p 1051), but training schemes in terminal care and available facilities must be broadened to achieve the optimum service. In south east London we have an excellent hospice and community team of terminal care nurses but they are trained specifically to care for patients dying of cancer, and, except for a few patients with motor neurone disease, hospice beds are limited to patients with cancer. Because of these restrictions I recently failed to obtain support for a 48 year old woman dying at home from an undiagnosed form of presenile dementia. Help was obtained from local psychogeriatric services but this was not ideal for such a young patient or for her husband and their three adolescent children. This case highlights an important deficit in our terminal care service and the importance in planning services of providing comprehensive training to meet all expected demands. A limited service is unacceptable as no dying patient should be refused support.

Biosynthesis of thromboxane in patients with systemic sclerosis and Raynaud's phenomenon

Dr DAVID F HORROBIN (Efamol Research Institute, Kentville, Nova Scotia, Canada B4N 4H8) writes: I was disappointed to find no reference in the paper by Dr I A S Reilly and colleagues (19 April, p 1037) to what I believe was the first report of increased production of thromboxane and prostacyclin in such patients, published in the *Lancet* some two and a half years ago.[1] A possible theoretical basis for the increased eicosanoid production in systemic sclerosis has been proposed and discussed.[2]

1 Horrobin DF, Jenkins K, Manku MS. Raynaud's phenomenon, histamine and prostaglandins. *Lancet* 1983;ii:747-8.
2 Horrobin DF. Essential fatty acid metabolism in diseases of connective tissue with special reference to scleroderma and to Sjogren's syndrome. *Med Hypotheses* 1984;14:233-47.

Confidentiality

Dr G M JONES (Cardiff CF4 8DD) writes: Some weeks ago press and television in Wales reported on cases of young carriers of the acquired immune deficiency syndrome antigen, naming their schools and ages. It was mentioned in some cases that they were haemophiliacs—so no problem over identification. How and by whom such highly confidential medical information is leaked to the media I do not know, but as medicine is now largely teamwork and as the head of the team should be a doctor then final responsibility rests with our profession, members of which still swear an ancient but meaningful oath. Should there not now be a reappraisal of the care of and disposal of medical information?