patient without further treatment of his or her acromegaly is safe. If the concentration of growth hormone is >5 mU/l and symptoms remain, with an increased concentration of insulin-like growth factor 1, external beam irradiation should be considered. Until growth hormone concentrations fall to ≤ 5 mU/l either bromocriptine or octreotide can be given and withdrawn at yearly intervals to assess the effects of radiotherapy on growth hormone secretion.

Symptomatic elderly patients, particularly those in whom surgery is contraindicated, are more susceptible to the beneficial effects of octreotide than younger patients.¹⁹ In patients with macroadenomas (>1 cm in diameter) transphenoidal surgery is the best treatment. Again, external beam radiotherapy and bromocriptine or octreotide may be used until growth hormone concentrations fall below 5 mU/l.

We need further large scale studies to decide whether reducing growth hormone concentrations to <5 mU/l is safe; a nationwide register of patients with acromegaly would help. We need too to ascertain the effects of abnormally low growth hormone concentrations after treatment—hypopituitarism has been associated with an increased mortality and the development of premature arterial degeneration.²⁰

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Genetic testing and insurance

Emphasises the irreconcilable dilemma in underwriting

The question of whether companies that issue policies for health insurance, disability insurance, and life insurance should have access to the results of genetic tests should be addressed as part of a more fundamental issue: the irreconcilable dilemma in underwriting. Traditionally, an insurance policy affords protection against very large costs resulting from the occurrence of an undesirable event whose probability is small. If the probability of loss is the same for each person then each will pay the same premium. But if the insurance company has information about the relative risk to each person it might charge premiums proportional to the risk. In the extreme case that there is sufficient knowledge to predict definitively to whom events will occur, the traditional concept of insurance breaks down as the person's premium minus his or her share of the company's expenses and profits will exactly equal his or her loss.

No solution to this dilemma exists. If insurance premiums are set to be equal for all people then the phenomenon of adverse selection may arise. Those people with a high risk may tend to purchase a great deal of insurance; those with a low risk may tend to purchase less. A vicious circle ensues as insurance companies are forced to raise their premiums to cover their expenses. On the other hand, if a company charges rates that depend on the risk those at high risk, who often form a small proportion of the total population and are most in need of insurance, will not be able to afford policies.

This discussion raises two questions. Firstly, should information be used in setting insurance rates, and, secondly, is genetic information any different from any other medical information? To answer these questions some people argue that it is useful to separate health insurance from disability

insurance and life insurance. To an increasing extent, health insurance is considered to be a necessity that should be available to all. Even in the United States, the last Western country not to have universal health care, there is a consensus that a system guaranteeing a basic level of health care to all is essential.

Although it can be argued strongly that disability insurance and life insurance are also necessities and that such cover should be universal, this position is controversial. Consequently, companies offering these types of insurance will continue to engage in underwriting. Whether these companies should have access to the results of genetic tests depends on whether genetic tests are different from other types of medical tests.

Genetic tests for single gene disorders that manifest in all environments are most easily distinguished from non-genetic medical tests. Unlike tests for most non-genetic conditions, genetic tests for such monogenic conditions as Huntington's disease, cystic fibrosis, and haemochromatosis can be used to diagnose a disease before clinical symptoms appear. On the basis of information obtained from these types of genetic tests insurance companies and employers have discriminated against asymptomatic carriers and presymptomatic people.^{1,2} We believe that discrimination against these classes of people is unjustified as the asymptomatic carriers will never become ill and some of the presymptomatic people may develop only mild symptoms of their disease.

In addition, genetic information is different from other medical information in that it can often provide information about the health of other members of the family of the person tested. These people, too, can be victims of unfair discrimina-

tion in insurance. Finally, the use of genetic tests exacerbates the dilemma of underwriting described above as the tests increase the amount of information available regarding a person's risk of becoming ill. Because of these distinctions between monogenic and non-genetic tests a strong case can be made for forbidding the use of tests for monogenic conditions.

Given the rapid technical advances in medical genetics, testing for a predisposition to many common disorders—such as heart disease, diabetes, and various forms of cancer—may soon be possible. For these diseases both genetic and environmental factors are usually implicated in the pathogenesis. As these diseases, like most diseases, are neither exclusively genetic nor exclusively environmental, tests for them cannot be readily distinguished from other medical tests.

The blurring of the distinction between genetic and non-genetic medical conditions does not mean that the use of tests that provide genetic information about multifactorial conditions will be no more problematic than non-genetic medical tests. Typically, genetic tests, especially those for multifactorial conditions, provide less predictive information about the course of a disease than is commonly believed. Moreover, stigmatisation and discrimination will not disappear so long as genes are believed to play a dominant part in determining our lives. The Insurance Task Force of the Human Genome Project proposed a moratorium on the use of genetic tests in

health insurance until a national health care programme was in place.³ We would argue that, given the problems raised by genetic tests, a moratorium similar to that suggested by the Nuffield Council on Bioethics should be extended to all forms of insurance in which underwriting is based on medical risks.^{4,5}

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Benign paroxysmal positional vertigo

Usually self limiting

Benign paroxysmal positional vertigo is characterised by brief but severe episodes of rotational vertigo that last less than a minute and occur on sudden changes of head position—for example, looking up or turning towards the affected ear while lying in bed.

Although the condition was first described by Bárány in 1921,¹ Dix and Hallpike defined the classic diagnostic test,² which should be performed after a suggestive history has been elicited. With the patient supine, the head is lowered some 30° below the level of the couch and turned some 30° to 40° to one side. If the test is positive the patient experiences vertigo and a rotatory nystagmus towards the undermost ear after a latent period of a few seconds. This diminishes within 50 seconds of onset—a phenomenon known as adaptation. On sitting up, the patient experiences a milder bout of vertigo, and nystagmus may occur. Any nystagmus induced by this test which does not fit this description may be positional nystagmus of central origin and warrants further neurological investigation. Occasionally, benign paroxysmal positional vertigo may occur bilaterally.

In most cases the cause is unknown and may be spontaneous degeneration within the labyrinth.³ Benign paroxysmal positional vertigo is, however, the most common vestibular disorder after head injury⁴ (when the symptoms and clinical course tend to be more severe) and has also been described after viral illness,⁵ stapes surgery,³ and chronic middle ear disease.²

The pathological basis for the condition is believed to be displacement of the otoconia from the maculae (the sensory receptors for linear acceleration situated in the utricle and saccule) either by degeneration or by trauma. The otoconia subsequently settle on the most dependent part of the labyrinth—the cupula of the posterior semicircular canal (hence the alternative term “cupulolithiasis”).³ The evidence

comes from two postmortem studies of known sufferers in whom basophilic deposits, thought to be macular otoconia, were found in the cupula of the posterior semicircular canal.

The displaced otoconia are believed to alter the function of the cupula by making it sensitive to linear gravitational forces. The characteristic rotatory nystagmus is due to ampullofugal stimulation of the posterior semicircular canal which causes contraction of the ipsilateral superior oblique and contralateral inferior rectus muscles.⁶ The latency of onset and fatigability of the nystagmus may be explained by the delay in the onset of the ampullofugal forces and the dispersal of the otoconia in the posterior semicircular canal respectively.

Idiopathic cases tend to occur from the fifth decade onwards.⁷ The average recovery period is six months but, in a few patients, the symptoms persist much longer and are sufficiently troublesome to result in hospital referral. Electro-nystagmography may show a positional nystagmus, and caloric responses may be normal, increased, decreased, or absent, so these investigations are not routinely performed in this condition. As the condition is generally self limiting, management is expectant, based on an explanation of the condition, reassurance of its benign nature, and the prescription of labyrinthine sedatives when indicated.

The Cooksey Cawthorne exercises, described in 1945, were designed to rehabilitate patients with disabling vertigo after head injury or inner ear surgery. These consist of a series of graduated head and body exercises that aim to restore balance and confidence.⁸ A canalith repositioning procedure has been described by Epley; this aims to disperse the otoconia from the posterior semicircular canal into the utricle by specific, supervised, head manoeuvres. This technique, however, requires premedication, needs to be accurately performed, and may require repeated hospital visits until the symptoms