annual level in 1999, but this was largely due to an increase in heterosexually acquired infection, and there was little evidence of a change in rates of diagnoses of HIV infection in homosexual men.[12]

There are at least two reasons why an increase in unprotected anal sex among gay men may not increase the incidence of HIV. Firstly, as the HIV viral load in semen is reduced to below detectable levels in most men being given combination antiretroviral therapy, treatment with these agents may decrease infectivity.[13] Secondly, the increases in unprotected anal intercourse may not put the participants at risk for new HIV infection.

Much of the unprotected anal intercourse reported by gay men is between seroconcordant partners, where there is no possibility of a new HIV infection.[14] In Australia, however, increases in unprotected anal intercourse have occurred both in HIV positive and in HIV negative men, and with both casual and regular sexual partners.[8 15] Thus it is likely that some of the increase in unprotected anal intercourse does involve sexual encounters where there is a risk of new HIV infection.

There are several challenges in preventing a resurgence of HIV in gay men. Firstly, we need better systems for the timely reporting and measurement of trends in risk behaviours and in the incidence of HIV infection. Monitoring systems for risk behaviours for HIV need to take into account the context of the sexual encounters: for example, whether the partner was casual or regular, and whether or not the HIV status of the partner was known. Secondly, there is a challenge to educationalists to design and implement behaviour change programmes that work in the new context of HIV infection, and a challenge to researchers to evaluate these programmes to ensure that the most effective interventions are broadly implemented. Thirdly, there is a challenge to gay communities around the world to recognise and respond to this threat.

In a situation where the immediate, overwhelming threat of death from AIDS is no longer present, promoting condom use is likely to be much more difficult than in the 1980s. However, if antiretroviral therapy becomes less effective because of viral resistance, then the rate of infection may well increase and current levels of unsafe sexual behaviour may lead to an increased incidence of HIV infection.

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1 Coutinho RA, van Griensven GJP, Moss A. Effects of preventive efforts among homosexual men. *AIDS* 1989;3(suppl):S53-6.
2 Crawford J, Bermingham S, Kippax S. *An analysis of trends over time in social and behavioural factors related to transmission of HIV in men who have sex with men. Technical appendix 3, Evaluation of the National HIV/AIDS strategy 1993-94 to 1995-96.* Canberra: Australian Government Publishing Service, 1996. (Catalogue No 9603026.)
3 Palella FJ, Delaney KM, Moorman AC, Loveless MO, Fuhrer J, Satten GA et al. Declining morbidity and mortality among patients with advanced human immunodeficiency virus infection. *N Engl J Med* 1998;338:853-60.
4 Van de Ven P, Kippax S, Knox S, Prestage G, Crawford J. HIV treatments optimism and sexual behaviour among gay men in Sydney and Melbourne. *AIDS* 1999;13:2289-94.
5 Dodds JP, Nardone A, Mercey DE, Johnson AM. Increasing high risk sexual behaviour among homosexual men in London 1996-8. *BMJ* 2000;320:1510-1.
6 Page-Shafer KA, McFarland W, Kohn R, Klausner J, Katz MH, Wohlfeiler D, et al. Increases in unsafe sex and rectal gonorrhea among men who have sex with men—San Francisco, California, 1994-1997. *MMWR* 1999;48:45-8.
7 National Centre in HIV Social Research, eds. *HIV/AIDS and related diseases in Australia: annual report of behaviour 1999.* www.arts.unsw.edu.au/nchsr/ (accessed 17 April 2000).
8 Prestage G, Van de Ven P, Knox S, Grulich A, Kippax S, Crawford J. *The Sydney gay community periodic surveys 1996-1999. Changes over time.* (National Centre in HIV Social Research Monograph 11/99.) www.arts.unsw.edu.au/nchsr/ (accessed 17 April 2000).
9 National Centre in HIV Epidemiology and Clinical Research, eds. *1999 HIV/AIDS, hepatitis C and sexually transmitted infections in Australia: annual surveillance report.* www.med.unsw.edu.au/nchecr/downloads/99anrpt.pdf (accessed 17 April 2000).
10 Martin IMC, Ison CA, London Gonococcal Working Group. Rise in gonorrhoea in London, UK. *Lancet* 2000;355:623.
11 Kellogg T, McFarland W, Katz M. Recent increases in HIV seroconversion among repeat anonymous tester in San Francisco. *AIDS* 1999;13:2303-4.
12 Public Health Laboratory Service AIDS and Sexually Transmissible Disease Centre, Scottish Centre for Infection and Environmental Health, Institute of Child Health, London, and Oxford Haemophilia Centre. AIDS and HIV infection in the UK: monthly report. *Commun Dis Rep CDR Wkly* 2000;10:37-40.
13 Vernazza PL, Troiani L, Flepp MJ, Cone RW, Schock J, Roth F, et al. Potent antiretroviral treatment of HIV-infection results in suppression of the seminal shedding of HIV. *AIDS* 2000;14:117-21.
14 Kippax S. A commentary on negotiated safety. *Venereology* 1996;9:96-7.
15 Van de Ven P, Prestage G, French J, Knox S, Kippax S. Increase in unprotected anal intercourse with casual partners among Sydney gay men in 1996-98. *Aust NZ J Public Health,* 1998;22:814-8.

New approaches to conversion hysteria

Functional imaging may improve understanding and reduce morbidity

Patients with hysterical conversion, now called conversion disorder by the main US psychiatric glossary,[1] often present with striking neurological symptoms such as weakness, paralysis, sensory disorders, or memory loss, in the absence of any pathology that could be responsible. Most patients will be referred to a neurologist or psychiatrist after consulting their family doctor.

As many as 4% of those attending neurology outpatient clinics in the United Kingdom have been estimated to have conversion disorders.[2] Similar rates have been reported for both in and outpatient clinics in other European countries.

Empirical research on hysterical conversion has lagged behind theoretical speculation. Recent advances in functional imaging (positron emission tomography scanning) and cognitive neuropsychology have, however, made the field more amenable to investigation.[3] Key clinical and theoretical problems remain over case definition and differential diagnosis, the psychological mechanisms underlying conversion hysteria, and how patients are best managed.

Despite attempts over the past century to abolish and reinstate the condition by using different labels, conversion hysteria continues to attract controversy.[4 5] The diagnosis is considered pejorative, and its place

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within psychiatric classification remains uncertain. There is no generally accepted explanation for how a psychological stress can convert into (often highly selective) symptoms. In this respect, conversion hysteria retains "the doubtful distinction among psychiatric diagnoses of still invoking Freudian mechanisms as an explanation."[6]

The diagnosis of conversion hysteria is typically made after excluding organic pathology and identifying a relevant psychological stressor. Problems with diagnosis include the extent to which organic disorders can and should be excluded; agreement on what constitutes relevant psychological conflict; and the criteria used to exclude malingering.

Technology reduces the risk of missing organic disease

There are few, if any, empirical data to support the diagnostic criteria given in psychiatric glossaries, and in clinical practice intuition and experience play a large part in the diagnosis. There is little evidence to determine what constitutes a relevant psychological stressor. A recent study, however, showed that the use of modern technology minimised the likelihood of missing organic disease.[7] It is likely that particular people may be at risk because of some underlying vulnerability. Evidence for this may prove difficult to find, and it remains impossible to exclude malingering as a potential cause.

For much of this century the search for the neurological systems responsible for conversion hysteria was largely ignored. The crux of the problem is to explain how abnormal psychological states can produce specific, long term neurological symptoms and disability in patients (who claim not to be consciously responsible) in the absence of detectable pathology.[4]

Recent evidence from functional imaging provides some indication of the possible brain areas involved. The functional imaging study by Marshall et al of a patient with left sided paralysis (but with no detectable lesion) found that when the patient tried to move her affected limb, considerable activity was seen in the right anterior cingulate and orbitofrontal cortex.[8] These activations were identified as the prefrontal structures responsible for inhibiting the patient's volitional movements.

To bridge the gap left by the traditional overreliance on psychodynamic theory, several neuropsychological accounts have emerged.[3] Instead of trying to explain conversion hysteria, these are more concerned with working out how impairments to normal cognitive processes such as volition, memory, and motor and sensory control may cause clinical symptoms.[9]

The conceptual link between hypnosis and hysteria has also been highlighted. Particularly in the acute stage, conversion symptoms and hypnotic phenomena share many features, to the extent that experiments on hypnosis (considered a kind of controlled hysteria) have long served as experimental analogues for the study of hysterical symptoms. The view that conversion symptoms can be usefully thought of as an autosuggestive disorder gains some support from a recent functional imaging study by Halligan et al, which showed that the areas of the brain activated by paralysis induced by hypnosis are similar to those activated in hysterical paralysis.[8 10 11]

There have been no controlled studies of treatment of patients with conversion hysteria. The uncontrolled case reports and series that exist are difficult to evaluate, as some patients improve spontaneously and the psychological benefits of any intervention may be more important that the specific intervention.

The lessons learnt in the treatment of chronic fatigue syndrome and other somatoform disorders may be applicable.[12] There is potential in using a cognitive behavioural approach, avoiding reinforcement of the abnormal illness behaviour, and facilitating more appropriate links between life situations and physical symptoms. Life events and social circumstances can dramatically change a person's prognosis, and there is emerging evidence that patients who experience a change in circumstances and life events after the onset of their symptoms have improved outcomes.[13]

Evidence is building that although conversion hysteria causes major disability, it is almost certainly not a disease with a specific pathology. Although the diagnosis carries a negative connotation for patient and doctor, its aetiology and management deserve further study.

Whether new developments in functional imaging and cognitive neuroscience can move the debate beyond disputes about how the disorder should be classified to testable hypotheses about the neuropsychological and social mechanisms involved in the disorder remains to be seen.

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1 American Psychiatric Association. *Diagnosis and statistical manual of mental disorders*, 4th ed. Washington, DC: APA, 1994
2 Perkin GD. An analysis of 7936 sucessive new outpatient referrals. *J Neurol Neurosurg Psychiatry* 1989; 52, 4478.
3 Marsden CD. Hysteria—a neurologist's view. *Psychol Med* 1986;16: 277-88.
4 Webster R. *Why Freud was wrong*. London: Fontana, 1996.
5 Ron MA. Somatisation in neurological practice. *J Neurol Neurosurg Psychiatry* 1994;57:1161-4.
6 Crimlisk HL, Bhatia K, Cope H, David A, Marsden CD, Ron MA. Slater revisited: 6 year follow up of patients with medically unexplained motor symptoms. *BMJ* 1998;316:582-6.
7 Marshall JC, Halligan PW, Fink GR, Wade DT, Frackowiak RS. The functional anatomy of a hysterical paralysis. *Cognition* 1997;64:B1-8.
8 Halligan PW, David AS, eds. *Conversion hysteria: towards a cognitive neuropsychological account*. Hove: Psychology Press, 1999.
9 Spence S. Hysterical paralyses as disorders of action. In Halligan PW, David AS, eds. *Conversion hysteria: towards a cognitive neuropsychological account*. Hove: Psychology Press, 1999:203-26.
10 Oakley DA. Hypnosis and conversion hysteria: a unifying model. *Cognitive Neuropsychiatry* 1999;4:243-65.
11 Halligan PW, Athwal BS, Oakley DA, Frackowiak RSJ. The functional anatomy of a hypnotic paralysis: implications for conversion hysteria. *Lancet* 2000;355:986-7.
12 Sharpe M, Hawton K, Simkin S, Surawy C, Hackmann A, Klimes I, et al. Cognitive behaviour therapy for the chronic fatigue syndrome: a randomized controlled trial. *BMJ* 1996;312:22-6.
13 Binzer M, Kullgren G. Conversion symptoms: what can we learn from previous studies? *Nordic J Psychiatry* 1996;50:143-52.