Figure 1 MRI of the spinal cord (sagittal plane). (A) T2 weighted images, (B) T1 weighted images with gadolinium enhancement. MRI disclosed C2-7 cord swelling. The intramedullary region of the swelling cord was low on T1 weighted images and high signal intensity on T2 weighted images. Gadolinium-DTPA images showed contrast enhancement in the C4/5 intramedullary region which extended longitudinally through the surface of the spinal cord.

670 ml (normal 200 ml–600 ml), indicative of impaired bladder sensation. A detrusor hyperreflexia was noted at the end of bladder filling. When asked to void after the detrusor hyperreflexia, her detrusor pressure increased slightly with poor urinary flow although the rectal catheter was pulled off at the end of voiding. There was no detrusor-sphincter dyssynergia. In the pressure-flow analysis² the point of a detrusor pressure (Pdet) at a maximum urinary flow rate (Qmax)—that is, PdetQmax—indicated equivocal obstruction (Abrams-Griffiths' nomogram) and normal detrusor contraction (Schäfer's nomogram). Above results indicated the presence of neurogenic bladder dysfunction. To avoid bladder distension, she was taught clean intermittent self catheterisation and started taking 2 mg/day oral prazosin hydrochloride, a selective α 1-antagonist. After starting these treatments, her autonomic dysreflexia successfully ameliorated together with voiding difficulty, and her residual urine volume was lessened to 100 ml.

Autonomic dysreflexia is a syndrome of paroxysmal hypertension, headache, sweating, vasodilatation, and bradycardia. This syndrome consists of sympathetic discharge below the level of the cord lesion in response to afferent stimuli that would be innocuous in the normal person. Reflexive parasympathetic discharge occurs above the level of the lesion. It affects 66%–90% of all patients with complete spinal cord injury at the T5 dermatome or above.^{3,4} It may occur among patients with incomplete spinal cord injuries, although the reaction seems to be milder than

in those with complete lesions. Causes of spinal cord lesions other than traumatic have also been reported to give rise to autonomic dysreflexia—for example, intramedullary haemorrhage caused by a spinal haemangioma or a spinal astrocytoma mimicking a pheochromocytoma. This is the first report showing a spinal cord sarcoidosis to be a cause of autonomic dysreflexia. As in the patients with spinal cord injury,^{3,4} attacks of autonomic dysreflexia in our patient were most commonly precipitated by neurogenic bladder dysfunction, although it occurred with only slight bladder sensation. Urodynamic studies showed the presence of detrusor hyperreflexia. Detrusor hyperreflexia may reflect lesions in the lateral columns of the spinal cord,⁵ which contain centrifugal pathways from the pontine micturition centre to the sacral preganglionic cells innervating the detrusor. Urodynamic studies also showed voiding difficulty with large postmicturition residuals without presence of detrusor-sphincter dyssynergia, although we did not assess bladder neck function in the patient. Studies of autonomic dysreflexia in patients with spinal cord injury showed that the rise in blood pressure is closely associated with not only the voiding phase but also the urinary filling phase, in which detrusor hyperreflexia and bladder distension are the common features.³ Furthermore, our patient had impaired bladder sensation, most probably reflecting lesions in the dorsal column of the spinal cord.⁵ Impaired bladder sensation may allow the bladder to distend silently, easily leading to autonomic dysreflexia. The patient

was taught clean intermittent self catheterisation and started taking oral prazosin hydrochloride, a selective α 1-antagonist, with benefit. During autonomic dysreflexia, concentrations of plasma noradrenaline (norepinephrine) increase and other substances—such as neuropeptide Y—may also increase in certain regions.⁴ The bladder neck (internal urethral sphincter) is innervated by sympathetic nerve and abundant with α 1A/D-adrenergic receptors. The α 1-antagonists relax both vascular (mainly α 1B) and urethral (mainly α 1A/D) smooth muscles, which may account for the amelioration of both autonomic dysreflexia and neurogenic bladder dysfunction in the patient.

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CORRESPONDENCE

The late whiplash syndrome: a biopsychosocial approach

In their admirable review, Ferrari and Schrader¹ re-introduce² the biopsychosocial model which recognises that the late whiplash syndrome is not the result of a chronic injury. They note the influence of compensation incentives such as that seen in Switzerland and other western countries. It is the high percentage of patients with chronic pain attributed to accidents that provide the greatest health care and economic burden.

The biopsychosocial model considers an effect of cultural expectation, cultural factors that generate symptom amplification and attribution. I agree with their conclusion that it negates the concept of "chronic injury",³ but at the same time takes away the stigma of the psychiatric label, while explaining that people's behaviour in response to their injury may generate much of the illness. The authors surprisingly neglect the final mechanism of the symptoms so often claimed in medicolegal practice, but so seldom encountered in the hospital clinic. They seem to blame cultural expectations and society at large, but they fail to consider the "victim",

who stands to gain sums of money, often larger than he or she has ever handled before the accident. The social and cultural factors they so well describe are real; but the production of symptoms⁴ ultimately depends on the conscious will of the claimant in providing a description of their severity, duration, and the consequent disabilities and loss of earnings. In litigation practice, deliberate exaggeration is common.^{5,6} It is misleading to inculcate society alone, and insulting to the patients or claimants to deny their exercise of free choice.

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Ferrari replies:

Pearce¹ completes the biopsychosocial model(s) of whiplash by recognising factors that we could not, for lack of space, include, although they have been discussed in detail elsewhere.² Illness behaviour is the end result of many factors, including pathophysiological processes, experience biased interpretations of abnormal versus normal bodily sensations, and environmentally influenced attributions affecting further expressions of illness behaviour. In most cases, the sum effects of these factors is often considered to be normal illness behaviour, as it is the norm in most illnesses that these factors come into play in most people.

Less normal, or frankly abnormal, illness behaviour is relevant in some illnesses,³ although there is a paucity of research in this area concerning collision victims. It is clear that there are at least three subgroups of patients with whiplash: (1) those who recover in days to weeks; (2) those who recover in weeks to months as they choose to resume their normal existence once more and free themselves of the environment that disables them; and (3) those who report a chronic, disabling illness that lasts years. Our article dealt primarily with the first two groups, but there are formulations and constructs that have evolved to understand subjects whose illnesses are characterised by grossly abnormal and extreme illness behaviour, the economy of secondary gain, adoption of the sick role through conscious and preconscious motivations, and so forth.^{4,5} Whereas this group may be the smallest group of patients in number, they consume disproportionately greater health care costs.

Collision victims make choices, but they need cultural support to enact those choices. Perhaps what makes us tend to underestimate the conscious choices of the patients is that they are blameless in the way lawyers, therapists, the media, and others, knowingly or not, encourage an illness behaviour that is at best maladaptive, and at the worst, greed driven. It is unfortunately all too easy, within

the whiplash megaindustry, for those engaged in the pursuit of tertiary gain (gain from the illness of others) to act as permissive agents for those in society willing to succumb to the their characterologic flaws of misguided righteousness, deservedness, and greed.⁶

Thus, we gather most whiplash patients are victims, not so much of the collision, but of a system that endangers their health. It is further clear that the day has come to view the whole beast that is whiplash. Researchers that assert that psychosocial factors do not primarily determine the outcome of illness behaviour in patients with whiplash, or that the primary determinants are to be found in the microscopic nature of the cervical spine are simply too wrong to be given any further credence or consideration.⁷⁻¹⁰ There are so many "facets" to whiplash, that the whiplash injury itself becomes of less importance when we desire to understand the larger range of illness behaviour, and why it evolves.¹¹

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- 1 Pearce JMS. The late whiplash syndrome: a biopsychosocial approach. *J Neurol Neurosurg Psychiatry* 2001;71:000-000.
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CORRECTION

Kalamangalam GP, Ellis SJ. Migrainous brain stem disturbance in Norrie disease: case report. *J Neurol Neurosurg Psychiatry* 2001;70:815-6.

Chen ZY, Denney RM, Breakefield XO. Norrie disease and MAO genes: nearest neighbours. *Hum Med Genet* 1995;4:1729-37 was misquoted in the second sentence of paragraph 8 of the above letter. The sentence should read "The gene is flanked by the MAO-A and MAO-B loci".

NOTICE

British Neuropsychiatry Association 2002 Annual Meeting

21/22 February 2002

The British Neuropsychiatry Association 2002 Annual Meeting will be held at the Institute of Child Health, central London on 21/22 February 2002.

The meeting will cover four topics:

- "Clinical and Neurobiological aspects of new variant CJD"
- "The Mind's Ear"
- "Pervasive Developmental Disorders"
- "New Drugs for Neuropsychiatry"

The meeting includes keynote addresses from prominent international and United Kingdom speakers, along with a session for members' contributions.

For further information please contact: Gwen Cutmore, BNPA Conference Secretary, Landbreach Boatyard, Chelmer Terrace, Maldon, Essex. CM9 5HT, (tel/fax: 01621 843334; email: gwen.cutmore@lineone.net, website: www.bnpa.fsnet.co.uk).

For details of membership to the BNPA, open to medical practitioners in psychiatry, neurology, and related clinical neurosciences, please contact: The Secretary, Professor A S David, Department of Psychological Medicine, Institute of Psychiatry, De Crespigny Park, Denmark Hill, London SE5 8AF.