BMJ
1840-1990
LONDON, SATURDAY 1 SEPTEMBER 1990

Whiplash injury

Long term prospective studies are needed and, meanwhile, pragmatic treatment

In regretting having coined the term "whiplash injury" in 1928, Harold Crowe later emphasised that whiplash "describes only the manner in which a head was moved suddenly to produce a sprain in the neck."[1] The accepted usage, which is deplored by many, refers to the cluster of symptoms that follow this mechanical event. Bony damage and other serious injury are usually excluded in this definition. The acute symptoms are a common presentation in casualty departments,[2,3] with many more patients seen in general practice. The later complications are only too familiar to a variety of hospital specialists.

Whiplash injury occurs typically in a rear end motor collision but may also result from front or side impacts or other types of accident. Seat belts prevent serious injury but may make neck sprain more likely[4]; head restraints protect the neck but only if designed and fitted correctly.[5,6] The injury causes stretching and bruising of the muscles and supporting ligaments of the neck. These lead to cervical pain and stiffness, often extending into the shoulders and interscapular region and associated with occipital pain or more generalised headache. Cervical root symptoms are less common but occur even in the absence of pre-existing spondylosis. In some cases the symptoms may not develop until several hours after the accident.[2] In contrast with motor accidents generally, whiplash injuries are more common in women.[7-9]

There is no clear consensus on the treatment of acute neck sprain, which ranges from prescribing a rolled up newspaper for neck support to a carefully supervised programme of intensive treatment. A randomised study contrasted early mobilisation and modified Maitland exercises with treatment with a soft collar and rest and showed a significantly greater reduction in pain and cervical stiffness in the actively treated group at four and eight weeks.[10] Another randomised study showed a worse outcome in patients treated with only a collar and analgesia than in those receiving outpatient physiotherapy, but mobilisation at home after a single session of instruction by a physiotherapist was equally effective.[3]

A pragmatic approach is to prescribe analgesic or anti-inflammatory drugs with a soft collar and to advise removal of the collar every few hours to allow gentle self mobilisation of the neck; patients should be given an explanatory leaflet to inform and reassure them. When reviewed a few days later many patients will be well on the road to recovery, and only those with stubborn pain and neck stiffness need then to be seen promptly by the physiotherapist, who will assist cautious mobilisation with measures for pain relief.[11,12] The use of traction is illogical. Abnormal findings on cervical radiology and symptoms of the arm are recognised as risk factors for prolonged disability, and such patients may need closer attention.[7,13,14] Nevertheless, the prognosis is usually good, and most patients will return to work within a month. As the late Henry Miller pithily observed, doctors should "encourage a robust attitude to minor injury."[15]

The thorny problem of the late whiplash syndrome is seen in those whose disability persists for six months or more, estimated at just over a quarter in one large retrospective series.[8] In some, psychological factors and expectations of successful claims for compensation intermingle with the persisting symptoms of the neck injury. Conscious or unconscious fabrication of symptoms undoubtedly occurs, which can be recognised in the medicolegal consultation by such signs as "the groaning and quivering which ensues when forward spinal flexion is tested,"[16] but experience shows that it is usually the persistence of symptoms that leads a patient to litigation, and not vice versa. After settlement of a claim many patients remain symptomatic, and the chronic syndrome is seen in patients not involved in litigation.[7] Treating the late whiplash syndrome requires strong reassurance, the judicious use of physiotherapy, other timely interventions, and advice on self help.[17] Encouragement to settle claims early may help in some cases, but most important is the recognition that time is the best healer.

Most published work on whiplash injury has been retrospective or dealt with selected groups of patients, often those seen in medicolegal practice. The few prospective studies have been short. A large prospective study is needed, taking as its starting point the first assessment of the acute neck sprain in the casualty department, following patients through randomised treatment protocols, and, finally, defining accurately the genesis and outcomes of the chronic syndrome.

P K NEWMAN

Consultant Neurologist,
Middlesbrough General Hospital,
Middlesbrough,
Cleveland TS5 5AZ

1 Crowe H, cited in Breck LW, Van Norman RW. Medicolegal aspects of cervical spine sprains. *Clin Orthop* 1971;**74**:124-8.
2 Maimaris C, Barnes MR, Allen MJ. Whiplash injuries of the neck: a retrospective study. *Injury* 1988;**19**:393-6.
3 McKinney LA, Dornan JO, Ryan M. The role of physiotherapy in the management of acute neck sprains following road-traffic accidents. *Arch Emerg Med* 1989;**6**:27-33.
4 Deans GT, Magalliard JN, Kerr M, Rutherford WH. Neck sprain—a major cause of disability following car accidents. *Injury* 1987;**18**:10-12.
5 Morris F. Do head-restraints protect the neck from whiplash injuries? *Arch Emerg Med* 1989;**6**: 17-21.
6 Olney DB, Marsden AK. The effect of head restraints and seat belts on the incidence of neck injury in car accidents. *Injury* 1986;**17**:365-7.
7 Hohl M. Soft-tissue injuries of the neck in automobile accidents: factors influencing prognosis. *J Bone Joint Surg* 1974;**56A**:1675-82.
8 Balla J, Iansek R. Headache arising from disorders of the cervical spine. In: Hopkins A, ed. *Headache: problems in diagnosis and management*. London: Saunders, 1988:249-55.
9 Pearce JMS. Whiplash injury: a reappraisal. *J Neurol Neurosurg Psychiatry* 1989;**52**:1329-31.
10 Mealy K, Brennan H, Fenelon GCC. Early mobilisation of acute whiplash injuries. *Br Med J* 1986;**292**:656-7.
11 Porter KM. Neck sprains after car accidents. *Br Med J* 1989;**298**:973-4.
12 Hashemi K. Early mobilisation of acute whiplash injuries. *Br Med J* 1986;**292**:1079.
13 Norris SH, Watt I. The prognosis of neck injuries resulting from rear-end vehicle collisions. *J Bone Joint Surg* 1983;**65B**:608-11.
14 Miles KA, Maimaris C, Finlay D, Barnes MR. The incidence and prognostic significance of radiological abnormalities in soft tissue injuries to the cervical spine. *Skeletal Radiol* 1988;**17**: 493-6.
15 Miller H. Accident neurosis. *Br Med J* 1961;i:919-25.
16 Miller H. Accident neurosis. *Br Med J* 1961;i:992-8.
17 McKenzie R. *Treat your own neck*. New Zealand: Spinal Publications, 1983.

Progression of Parkinson's disease

Drug treatment may slow the disease process

The prospects of the patient with Parkinson's disease have improved substantially since the advent of the dopaminergic drugs, dopamine agonists, and subcutaneous apomorphine.[1] More recently the promise, as yet unproved, of fetal cell implantation has raised further hopes.[2] But the bleak fact is that the disease shows inexorable progression despite treatment, and the patient suffers the on/off fluctuations, dyskinesias and dystonias, and the psychiatric morbidity induced by the drugs.

Attempts have recently been made to find ways of slowing the onset or progression of early Parkinson's disease with a view to improving the prognosis. At the onset of symptoms about 80% of dopaminergic neurones have degenerated and a proportionate loss of striatal dopamine has occurred. Research with MPTP (1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine), the chemical discovered by chance as a contaminant of illicit drugs, has shown that environmental toxins can cause selective nigrostriatal damage in primates and humans. Experimental work in primates has indicated that in the case of MPTP this can be completely blocked by the prior administration of monoamine oxidase B inhibitors (for example, selegiline), which prevent the metabolism of MPTP (the protoxin) to MPP^+, the active, highly selective dopaminergic poison.[3] If environmental toxins analogous to MPTP are at work in Parkinson's disease then in theory early detection in the preclinical stage would be enormously beneficial—if a drug could be found to block this neurotoxin before neuronal damage had occurred or to arrest or retard progression.

The search for diagnostic markers is now on and has several aims: firstly, to confirm the diagnosis; secondly, to find a measure of disease activity that could be used to monitor both progression and the effects of treatment; and, finally, to detect preclinical disease, which might in turn be responsive to treatment. A vast array of biochemical and metabolic indices has been scrutinised, but at present none has proved to be clinically useful.

The recently published DATATOP (deprenyl and tocopherol antioxidant therapy of parkinsonism) trial attempted to confront these theoretically inviting concepts in early Parkinson's disease.[4] It was based on the concept that free radicals, superoxides, and hydrogen peroxide released from degenerating neuronal proteins and neuromelanin produce toxic lipid peroxides. These cause dysfunction of cell membranes and ultimately damage the nigrostriatal pathway. In theory, therefore, the inhibition of monoamine oxidase B and of oxidative pathways might retard such toxic damage and improve the prognosis. The trial comprised 800 patients with early disease (stages 1 and 2, Hoehn and Yahr scale) aged 30 to 70 (mean age 61). Patients were not taking antiparkinsonian drugs or were able to stop taking such drugs without deterioration. Patients with depression and dementia were excluded.

Selegiline (deprenyl) 10 mg a day or tocopherol 2000 IU a day, or both, and placebo were used in a double blind design. The end points were the time interval from randomisation until the "blinded" investigator judged levodopa necessary, or two years of treatment. At one year 176 controls but only 97 patients treated with selegiline had been deemed in need of levodopa, a 57% reduction. Projected times for each group to reach the end point were 15 months for controls and 26 months for those receiving deprenyl (I Shoulson, symposium on the recent advances in the treatment of Parkinson's disease, London, 1990). No decline was noted during one month when treatment with selegiline was stopped. The data on tocopherol have not yet been analysed. Similar results were obtained by Tetrud and Langston in 27 patients with early disease given selegiline 10 mg a day and 27 matched patients given placebo in a randomised double blind trial.[5] The time before levodopa was deemed necessary was increased from 312 days in those receiving placebo to 549 days in those receiving selegiline.

There are several possible explanations for these findings. Possibly, selegiline may simply improve symptoms and so seems to bridle progression over two years; or it may exert a protective effect against some persistent unknown environmental toxin that is damaging the nigral cells; or it may prevent the expected decline in the established disease process.

These results have yet to be confirmed. The end point is to some extent subjective. The half life of selegiline is more than 24 hours, but side effects are inconspicuous—with no "cheese reactions" as seen with monoamine oxidase A inhibitors. The drug has an additional advantage in that it smooths out the on/off swings, particularly reducing the off periods in patients with more advanced disease. These promising but preliminary results justify the prescription of selegiline 10 mg each morning to patients with early disease, especially the younger ones, in the hope of impeding progression.

J M S PEARCE

Consultant Neurologist,
Hull Royal Infirmary,
Hull HU3 2JZ

1 Pearce JMS. Modern treatment of Parkinson's disease. *Br J Hosp Med* 1987;**37**:59-66.
2 Pearce JMS. Adrenal and nigral transplants for Parkinson's disease. *Br Med J* 1989;**296**:1211-2.
3 Langston JW, Ballard P, Tetrud JW, *et al*. Chronic parkinsonism in humans due to a product of meperidine-analog synthesis. *Science* 1983;**219**:970-80.
4 Shoulson I, Parkinson study group. DATATOP: a multicentre controlled clinical trial in early Parkinson's disease. *Arch Neurol* 1989;**46**:1052-60.
5 Tetrud JW, Langston JW. The effect of deprenyl (selegiline) on the natural history of Parkinson's disease. *Science* 1989;**245**:519-22.