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Clinical Review

Whiplash injury [1]

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Contents

1. Introduction 284
2. Definition 284
3. Incidence 284
4. Chronicity 285
5. Prevalence 286
6. Demographic features 287
7. Aetiology and pathogenesis 287
   7.1. Extension 288
   7.2. Flexion 288
   7.3. Lateral flexion 288
   7.4. Shear forces 288
8. Pathology 289
   8.1. Zygapophysial joints 289
   8.2. Disc 290
   8.3. Muscles 290
   8.4. Trigger points and myofascial pain 291
   8.5. Ligaments 291
   8.6. Atlanto-axial complex 292
   8.7. Cervical vertebrae 292
   8.8. Brain 292
   8.9. Temporomandibular joint 292
   8.10. Other tissues 293
   8.11. Synopsis 293
9. Symptoms 293
   9.1. Neck pain 293
   9.2. Headache 294
   9.3. Visual disturbances 295

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9.4. Dizziness 295
9.5. Weakness 296
9.6. Paraesthesia 297
9.7. Concentration and memory disturbances 297
9.8. Psychological symptoms 298

10. Litigation neurosis 299

11. Factors influencing prognosis 299

12. A model 299

13. Treatment 300

14. Conclusion 302

References 302

1. Introduction

Whiplash is perceived to be a very common and troublesome disorder, and conflicts prevail as to whether it is a syndrome arising from neurosis and the desire for compensation or a definite organic disorder. In order to address these conflicts this review draws together the available information concerning the epidemiology, clinical features, pathophysiology and treatment of whiplash injury.

2. Definition

The very definition of whiplash injury remains controversial. The essential elements are that the injury takes place in a motor vehicle accident (MVA) and that the head is subject to acceleration forces that result in bending of the neck. Although classically described in association with movements in the sagittal plane following rear-end impact, it is clear that neck pain may also follow lateral or frontal collisions. Accordingly, the latter are included in this review. The term whiplash has been applied to the mechanism of injury, to the injury resulting from this mechanism (whiplash injury) and to the syndrome of neck pain with or without other symptoms following such an injury (whiplash syndrome). The definition of whiplash injury proposed for the current review is an injury to one or more elements of the cervical spine that arises from inertial forces being applied to the head in the course of a MVA that results in the perception of neck pain.

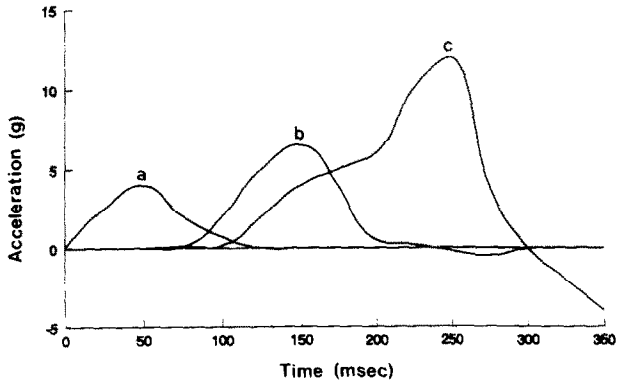

Fig. 1. Idealized acceleration curves of the struck vehicle (a), shoulders (b), and head (c), following rear-end impact (modified from Severy et al. 1955). Note that the peak acceleration of the head is considerably greater than that of the car and is followed by significant deceleration.

3. Incidence

The prevalence of whiplash injury has never been determined by a population-based study, and the actual incidence has never been measured prospectively. However, there is general agreement that the condition is common. American figures from the early 1970s indicate that there were 3.8 million rear-end impact MVAs at a time when the population of the USA was approximately 200 million. Of those exposed to a rear-end MVA, it has been suggested that approximately 20% will develop symptoms from their neck (States et al. 1970). From this data, the derived annual incidence of symptoms from whiplash is 3.8 per 1000 population. A 3-year study of disabling neck injury from MVA in female factory workers (presumably a population more at risk being daily users of cars), reported an incidence of 14.5 per 1000 women workers (Schutt and Dohan 1968). This study is unique in that the study population was defined *a priori* and the outcome measure was the presence of the condition as defined by a medical assessment rather than an insurance claim. In Switzerland, with a population of 6.6 million, 60% of all the

working population are insured with a single insurer. Between 1978 and 1981, 9983 cases of 'soft tissue injuries' to the cervical spine were recorded by the insurance company, of which 55% were sustained in MVAs, constituting an annual incidence for the entire Swiss population of 0.44 per 1000 (Dvorak et al. 1989). Norwegian figures attest to a higher incidence of 2 per 1000 (Olsnes 1989). Australian figures from 1982-1983, from the Victorian Motor Accident Board and Road Traffic Authority, report an incidence of 1 per 1000 while New Zealand statistics for the same period, obtained from the Accident Compensation Commission and The Ministry of Transport indicate a much lower incidence of 0.1 per 1000 (Mills and Horne 1986). Unpublished figures from the New South Wales Motor Accidents Authority for 1992 indicate an annual incidence of 0.8 per 1000.

Unfortunately, most of these latter estimates are derived from insurance or compensation claim statistics which have been taken to be a reliable measure of the frequency of injury. An insurance claim, however, constitutes a behaviour that arises from a complex combination of motivation, enabling circumstances, perceived benefits, perceived costs, social norms, peer and family pressure, and fear of current or future pain or disability. Hence, there are numerous factors extraneous to the injury itself that contribute to a person making an insurance claim. To regard only those patients with claims as having whiplash is an example of selection bias, and neither the consistency nor direction of this bias is known.

Notwithstanding their inaccuracy and variability, the above estimates indicate that whiplash injury constitutes a substantial problem with an approximate incidence of 1 per 1000 in western societies. However, in settings where an accurate measurement is required, and in particular where the effects of an intervention are being assessed, insurance claims are an inadequate outcome measure. In this regard the experience of the Motor Accident Board in Victoria, Australia, is most illustrative. A fall in the number of insurance claims for whiplash followed the introduction of legislation that created bureaucratic barriers, disincentives and up-front costs for people intending to file claims (Transport Accident Commission of Victoria 1990). This fall has prompted some observers to conclude that it is possible to reduce the incidence of whiplash by legislation alone and have taken this to imply that whiplash is a behaviour and not an injury (Awerbuch 1992). A more sober view of the data suggests a different conclusion: if it is harder to make a claim, less people will make one. To extrapolate beyond this is unjustifiable and potentially dangerous. In epidemiological terms, the apparent change in incidence is simply due to reporting bias; a similar 'fall' in the incidence of sexually transmitted diseases might occur if there were financial or social disincentives to notifying new cases.

Caution must also be exercised in comparing insurance claim rates between countries since there is no consistency internationally in notification of accidents, or insurance and compensation procedures. Conclusions drawn from such comparisons (Mills and Horne 1986) cannot be sustained and are subject to the 'ecologic fallacy' (Feinstein 1985). Even less robust are data derived from anecdotal reports or unstructured, non-standardised interviews of small numbers of doctors (Balla 1982). They constitute the poorest quality of data according to contemporary criteria (Sackett et al. 1985) and risk being fatally corrupted by recall bias, case-selection bias, sampling bias and expectation bias.

4. Chronicity

Not all patients who suffer a whiplash injury develop chronic symptoms. Indeed, despite its reputation, whiplash is a relatively benign condition; most patients recover. The rate of recovery after whiplash injury has been explored in three studies (Maimaris et al. 1988; Gargan and Bannister 1990; Olsson et al. 1990). All indicate that those patients destined to recover will do so in the first 2–3 months after injury. The rate of recovery then slows dramatically to become asymptotic, with no further change in symptoms after 2 years. Viewed simplistically, the outcome for an individual patient is dichotomous; either the neck pain will resolve in the first few months or it will persist indefinitely. What is unclear is what proportion of patients fail to recover.

Estimates based on personal, clinical experience are fraught with danger because an individual's recollections are invariably tainted by recall bias and case-selection bias (Sackett et al. 1985). Furthermore, the accuracy of any estimate of the proportion of patients with a given outcome is dependent upon the size of the sample. If an individual practitioner follows up 40 patients with acute whiplash injuries, and finds that 20% recovered completely, the 95% confidence intervals for this estimate are 8% and 32%. This degree of inaccuracy makes the initial estimate unhelpful. Meaningful prognoses can only be derived from formal studies, which can be judged according to explicit criteria (Department of Clinical Epidemiology and Biostatistics, McMaster University, 1981).

The most important requirement of any study of the progress of a disease is that an inception cohort is assembled at the outset. It is unacceptable to start with a group of patients who enter the study simply because they are accessible to follow-up. Furthermore, the sample should be representative of those patients with the condition of interest. Among published studies of

whiplash injuries, the most representative samples available (i.e., least affected by possible sampling bias) are derived from hospital-based studies. Applying these simple but vital criteria requires that, in determining the natural history of whiplash injury, most series should be discarded from further consideration (Gotten 1956; Pietrobono et al. 1957; Macnab 1964, 1966, 1971, 1973; DePalma and Subin 1965; Janes and Hooshmand 1965; Gates and Benjamin 1967; Bingham 1968; Schutt and Dohan 1968; States et al. 1970; Gukelberger 1972; Farbman 1973; Hohl 1974; Greenfield and Ilfeld 1977; Balla 1980, 1982; Mills and Horne 1986; Balla and Karnaghan 1987; Dvorak et al. 1987c, 1989; Pearce 1989).

The studies meeting these minimal acceptable criteria are summarised in Table I and reveal that there is a significant proportion of patients who develop chronic neck pain after whiplash injury. Differences in the definition of persistent symptoms between these studies preclude a dogmatic proclamation of the size of this group. Norris and Watt (1983) reported that 67% of their cohort had neck pain of any severity at the end of 20 months follow-up, but only 15% of those regularly required time off work. Deans et al. (1987) found that 62% of patients developed neck pain after a MVA and that 42% of these still had some neck pain after 1 year, 6% suffering continuous pain. Long-term follow-up of the cohort first studied by Norris and Watt (1983) has demonstrated that 88% of patients who were able to be followed up still had residual symptoms, 28% had 'intrusive symptoms' and 12% had 'severe' neck pain (Gargan and Bannister 1990). Applying a worst case analysis, and assuming that all those patients who were unable to be followed up recovered completely, reveals that 62% of patients were symptomatic, 20% had intrusive symptoms and 8% had severe symptoms. Together, these studies indicate that between 14 and 42% of patients with whiplash injuries develop chronic neck pain and that approximately 10% will have constant, severe pain indefinitely.

5. Prevalence

No explicit figures on the prevalence of chronic symptoms following whiplash are available, but a coarse estimate can be calculated from incidence rates, the natural history of the condition and the age of the affected population.

Given an incidence of 1 per 1000, and given that approximately 25% of patients with whiplash injury progress to chronic symptoms, with 10% suffering severe pain, one could expect 0.25 new cases per 1000 population per annum developing chronic pain, and 0.1 cases with severe pain. If the average age of a person sustaining a whiplash injury is 30 and the average life span is 70 years, the cumulative effect over 40 years yields a prevalence in the entire population of about 1% with chronic pain and 0.4% with severe pain. Notwithstanding the crudeness of this estimate, the magnitude of the problem remains significant. Even discounting the derived figure by a factor of two still leaves 0.5% of the population with chronic neck pain following whiplash injury and 0.2% with severe pain. For comparison, this latter figure is the same as the prevalence of epilepsy. The true figure will only be

TABLE I
STUDIES IN WHICH AN INCEPTION COHORT WAS ASSEMBLED TO DETERMINE PROGNOSIS FOLLOWING WHIPLASH

Reference	Type of study	Study Population	n	Follow-up rate (%)	Mean duration of follow-up (months)	Proportion with neck pain at end of follow-up (%)
Norris and Watt (1983)	Prospective	All patients presenting to a single hospital after rear-end collision	61	100	20	67 (15% severe)
Olsson et al. (1990)	Prospective	Volvo drivers with non-serious neck injury	33	100	12	36
Pennie and Agambar (1991)	Prospective	Consecutive whiplash patients at 2 hospital accident departments	144	95	5	14
Miles et al. (1988)	Prospective	Consecutive whiplash patients at a hospital who had X-rays taken	73	100	24	29
Deans et al. (1987)	Retrospective	Consecutive car accident victims who developed neck pain	85	78	18	42 (6% constant pain)
Maimaris et al. (1988)	Retrospective	Consecutive whiplash patients at a hospital accident department (included cohort of Miles et al.)	102	85	24	35
Gargan and Bannister (1990)	Retrospective	Same cohort as Norris and Watt	43	70	120	88 (28% intrusive 12% severe)
Watkinson et al. (1991)	Retrospective	Same cohort as Norris and Watt	35	57	120	86 (26% intrusive, 9% severe)

revealed by a carefully conducted, population-based study but until such work is conducted, calculated estimates from the available data confirm the clinical impression that the problem is a common entity.

6. Demographic features

The true characteristics of those people affected by whiplash injury can only be ascertained from studies in which the study population is representative of the affected population. Consequently, the majority of case series of whiplash victims can be discarded in that they are referral-based and are therefore subject to unacceptable referral bias and case-selection bias (Gay and Abbott 1953; Gotten 1956; Macnab 1966; Gates and Benjamin 1967; Schutt and Dohan 1968; Bingham 1968; Gukelberger 1972; Farbman 1973; Hohl 1974; Greenfield and Ilfeld 1977; Balla 1980; Bring and Westman 1991). Using only those studies with a hospital or community based sampling frame (States et al. 1970; Norris and Watt 1983; Deans et al. 1987; Maimaris et al. 1988), reveals that there is no particular gender bias and that the average age of the patients would appear to lie in the late fourth decade. These figures do not take account of the gender or age distribution of the motoring population and hence it is impossible to determine whether any particular group is particularly at risk. However, in one of these studies (States et al. 1970) the age distribution of those suffering whiplash injury was reported to approximate that of the motoring public, although females were over-represented.

7. Aetiology and pathogenesis

Whiplash injury has classically been attributed to rear-end impacts, and early clinical reports suggested that this resulted in forced flexion of the neck (Gay and Abbott 1953). This belief has been refuted in subsequent experimental studies (Severy et al. 1955; Clemens and Burow 1972) and computer models (McKenzie and Williams 1971; White and Panjabi 1978) that have clearly defined the sequence of events following a rear-end collision. At the time of impact, the vehicle is accelerated forward, followed after 100 msec by a similar acceleration of the patient's trunk and shoulders induced by the car seat. The head, with no force acting upon it, remains static in space, resulting in forced extension of the neck as the shoulders travel anteriorly under the head. Following extension, the inertia of the head is overcome, and it is also accelerated forward. The neck then acts as a lever to increase the forward acceleration of the head and force the neck into flexion (Fig. 1). The forces involved are considerable; at an impact speed of 20 mph (32 km/h) the human head reaches a peak acceleration of 12 g during extension (Severy et al. 1955).

All mathematical models and experimental data on rear-impact collisions have assumed that the impact force is transmitted directly along the long axis of the vehicles and that the victim's head is in the anatomical position, looking straight ahead. By implication, this would produce acceleration forces exclusively in the sagittal plane which is unlikely to be the case in most accidents. If the head is in slight rotation, a rear-end impact will force the head further into rotation before extension occurs (Dvorak et al. 1987b). This has important consequences in that cervical rotation pre-stresses various cervical structures, including the capsules of the zygapophysial joints, intervertebral discs, and the alar ligament complex (Lysell 1972; Dvorak et al. 1987b; Dvorak and Panjabi 1987), rendering them more susceptible to injury.

There is less data on the response of the neck and head complex to side or frontal impact, since these impacts are more likely to cause injuries to other structures (Forret-Bruno et al. 1990) and, therefore fail to attract attention to 'classical' whiplash injuries. However, the reported data from computer models and cadaver experiments is consistent with the predictions derived from simple physics and extrapolation from the data on rear-end accidents. Frontal impacts rapidly decelerate the motor vehicle. The body of the passenger, having momentum, continues forward until decelerated by the seat belt. The head, which has not yet had a force act upon it, will continue moving forwards until decelerated by the neck itself, with the force being applied at the atlanto-occipital joint and then at C6 (Clemens and Burow 1972). Since this force is eccentric to the direction of movement of the head, the head rotates forwards, forcibly flexing the neck. There is then a degree of recoil as the elastic properties of the posterior neck structures pull the head out of flexion, and extend the neck. Experimental and mathematical models of frontal impact have demonstrated that the head is subject to marked rotational acceleration at the occipital condyles in the first 25 msec after impact, followed by a reversal of the direction of acceleration as extension occurs (Deng 1989). The forces involved are again considerable, with models indicating that at an impact speed of 63.5 km/h resulting in a vehicle deceleration of 90 g, the head is subject to negative acceleration of 46 g. Consequently the neck dissipates force initially through shear and then torque which can easily exceed the known tolerance levels of bone and ligament, leading to neck injury even in the absence of head injury (Deng 1989).

Therefore, in MVAs, the neck is subject to forced flexion, extension and lateral flexion as well as shear forces parallel to the direction of impact. These movements are unlikely to occur around physiological axes

(Lysell 1972; Frankel 1976; Penning 1991) as the muscles that normally help control the direction and amplitude of motion do not have time to respond to the forces applied to them (Foust et al. 1973; Schneider et al. 1975). In an individual accident there is likely to be a complex interaction between different forces depending upon the speed and direction of impact and the attitude of the head and neck. In the first instance, the possible sites of injury can be determined by considering theoretically those structures at risk from each of these movements. This requires an appreciation of the anatomy of the cervical spine and how its elements are potentially affected by the various forces that might be applied to them.

7.1. Extension

Forced extension of the cervical spine will apply compressive forces to posterior structures and tensile forces to the anterior structures. The anterior structures principally at risk are the oesophagus, anterior longitudinal ligament, anterior cervical muscles, odontoid process and the intervertebral discs. The posterior structures at risk are the spinous processes and the zygapophysial joints. Although the exact centre of rotation for each individual segment during forced extension is not known, almost any shift away from the physiological axis will result in the zygapophysial joints being the first site of bone-to-bone contact during extension, and hence the fulcrum for further rotation. Forcing the neck further into extension after the cartilage at the zygapophysial joints has been fully compressed must then cause either compressive failure (crush fracture) of the articular pillar or further stretch the anterior structures, possibly beyond their elastic limit, resulting in tears of the muscles, ligaments or discs, separation of the disc from the vertebral end plate or even fracture of the vertebral body.

7.2. Flexion

Forced flexion applies compressive forces to the anterior elements and tensile forces to the posterior elements of the cervical spine. The structures resisting flexion anteriorly are the intervertebral discs and vertebral bodies, whereas the posterior structures stretched by flexion are the zygapophysial joint capsules, articular pillars, ligamentum nuchae and posterior neck muscles. Flexion at the atlanto-axial joint will stress the alar ligament complex as the atlas attempts to rotate anteriorly over the axis.

7.3. Lateral flexion

Throughout the cervical spine lateral flexion of a given segment is strictly coupled to rotation of that segment, and the degree of coupling is determined by the orientation of the cervical zygapophysial joints (Penning 1991). If an external force laterally flexes the neck, the structures at risk of injury will be determined by the extent to which coupling occurs. If the force simply reproduces physiological movements, the zygapophysial joint capsules on both sides and the intervertebral discs will be most at risk from axial torque, whereas, if there is little coupling, lateral flexion will compress the ipsilateral zygapophysial joint and distract the contralateral joint.

7.4. Shear forces

In the seated position in a motor vehicle, the long axis of the cervical spine is approximately vertical.

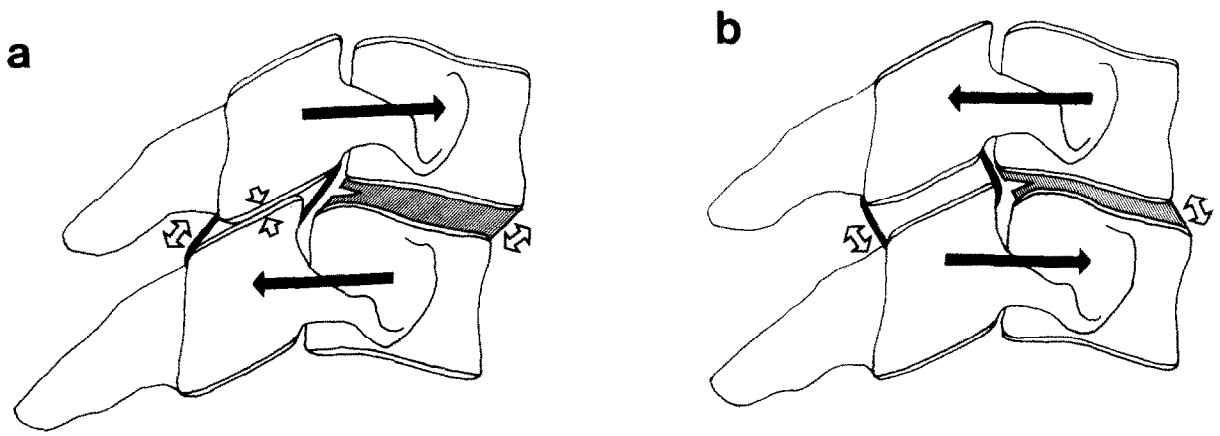

Fig. 2. Cervical spine structures at risk from horizontal shear forces applied to a typical motion segment. a: superior vertebral body translating anteriorly relative to the inferior vertebral body. The movement is resisted by the effacement of the articular surfaces of the zygapophysial joints and tension within the anterior annulus fibrosus of the intervertebral disc and the zygapophysial joint capsule (open arrows). b: superior vertebral body translating posteriorly relative to the inferior body. The movement is again resisted by the intervertebral disc and the capsule of the zygapophysial joints (open arrows).

Typically, MVAs produce horizontal forces so that most shear will be perpendicular to the long axis of the neck. Movements produced by shearing forces in this setting are of small excursion and are less likely to affect muscles which are vertically orientated; elastic structures. However, frontal impact will produce horizontal shear between cervical vertebrae, resulting in compression at the surfaces of the zygapophysial joints and stretching of the annular fibres at the anterior part of the disc (Fig. 2). The posterior disc is typically fissured as part of the normal, ageing process (Hirsch 1972; Tondury 1972; Bland and Boushey 1990; Penning 1991) and is therefore less likely to sustain significant injury from shear forces. Rear impact will have less effect on the zygapophysial joint surfaces but will tense the joint capsules and stress the anterior part of the disc (Fig. 2).

Notwithstanding theoretical considerations as to which structures are mechanically at risk, the actual likelihood of a lesion occurring cannot be extrapolated from such analysis alone. The exact distribution of force and the specific tolerances of different tissues, as well as any interactions, would need to be considered. Consequently, in the absence of such precise and comprehensive data, pathological lesions predicted from biomechanical observations need to be ratified by experimental or observational studies.

8. Pathology

Since whiplash injuries leading to chronic symptoms are non-fatal, no formal pathological studies are available from which to determine the site or nature of any lesions. Therefore, evidence for pathological entities has been obtained through indirect means, including animal experiments, cadaver experiments, post-mortem studies, clinical observations and radiographic studies. Each of these approaches has limitations, which must be borne in mind when evaluating any findings.

Animal studies are limited by the extent to which lesions produced in the animal reflect those sustained by human beings in actual accidents. Unfortunately, there is no reliable means of ascertaining the representativeness of a given animal model because of the large number of interacting variables that must be considered, including size, weight and morphology.

Cadaver experiments are accurate in terms of gross anatomic relationships but do not simulate the mechanical properties of living tissues, cadaveric matter being usually stiffer.

Post-mortem studies are available either from individuals who have whiplash injuries but die from other, unrelated causes or from victims of fatal trauma who have injured their necks. The former group is rare, amounting to only 4 cases in the literature (Abel 1975; Rauschning et al. 1989; Taylor and Kakulas 1991). It may be argued that the latter group constitute severely injured individuals whose injuries are not representative of those surviving trauma. However, MVAs typically produce increasingly severe injuries with increasing forces. The likelihood of death depends in part on qualitative factors, such as the exact part of the body injured, but is typically proportional to the forces involved (Clemens and Burow 1972). Victims of lethal accidents are unlikely to suffer a single, fatal injury. Rather, as they suffer an increasing gradient of forces they suffer cumulative injuries proportional to force experienced, culminating in the lethal injury — typically a head injury or an injury to the C1 segment. This indeed is the pattern of injuries seen at post-mortem (Jónsson et al. 1991). In these cases, if the obvious cause of death is disregarded, the other, non-lethal injuries of the neck are a reasonable indication of what might have occurred had the victim been subjected to forces just short of those that were lethal.

Clinical observations are limited to those lesions that can be detected on clinical examination or at operation. Other than bruising, bleeding or swelling, there is little that can be detected on clinical examination, and even then the findings are limited to superficial tissues. Few patients with whiplash injuries are treated by surgery, and if operations are performed it is late in the course of the disease, so that any findings may not necessarily be related to the initial trauma. Findings on plain X-rays are limited to osseous injuries and changes in soft tissue shadows, particularly the prevertebral space (Shmueli and Herold 1980; Penning 1981). Even so, several studies attest to the insensitivity of plain films for detecting significant bony injury, particularly of the articular pillars and zygapophysial joints (Abel 1958, 1975; Weir 1975; Smith et al. 1976; Binet et al. 1977; Woodring and Goldstein 1982; Yetkin et al. 1985; Clark et al. 1988; Jónsson et al. 1991).

Notwithstanding these limitations, marshalling the evidence from clinical, animal, cadaver and post-mortem studies can indicate trends and concordance to support one or more putative, pathological lesions (Fig. 3).

8.1. Zygapophysial joints

Evidence that the cervical zygapophysial joints are damaged in whiplash injury is compelling. There is striking consistency between experimental data from cadavers, radiographic findings, operative findings and post-mortem studies. Fractures of the joints themselves or the supporting articular pillar have been noted in several clinical studies (Abel 1975, 1982; Binet et al. 1977; Jeffreys 1980; Smith et al. 1976; Clark et al. 1988) and identical fractures have been produced in cadavers (Abel 1958; Clemens and Burow 1972). Moreover, post-mortem examination of a patient with a recent

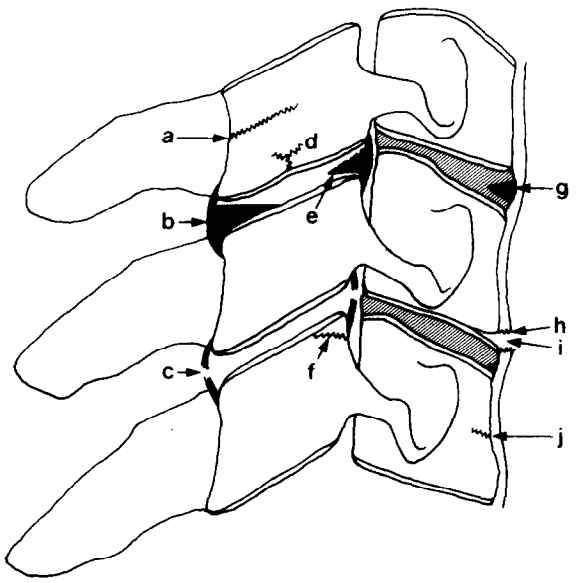

Fig. 3. A sketch of the more common lesions affecting the cervical spine following whiplash. a: articular pillar fracture; b: hemarthrosis of the zygapophysial joint; c: rupture or tear of the zygapophysial joint capsule; d: fracture of the subchondral plate; e: contusion of the intra-articular meniscus of the zygapophysial joint; f: fracture involving the articular surface; g: tear of the annulus fibrosus of the intervertebral disc; h: tear of the anterior longitudinal ligament; i: endplate avulsion/fracture; j: vertebral body fracture.

history of extension injury and neck pain, but who died of unrelated causes 4 months later, has revealed a typical, healing fracture of the articular pillar on the side of the pain (Abel 1975). Other post-mortem studies have found similar lesions (Jónsson et al. 1991).

Even using optimal imaging parameters in cadavers, the soft tissue elements of the cervical zygapophysial joints are poorly seen with plain X-ray, CT or MRI (Fletcher et al. 1990). Consequently, there are no imaging studies of pathology to these structures. However, tears of the joint capsules have been identified at operation on several occasions (Janes and Hooshmand 1965; Buonocore et al. 1966; Jeffreys 1980) and similar injuries have been found at post-mortem (Bucholz et al. 1979; McMillan and Silver 1987; Jónsson et al. 1991) and in cadaver studies (Clemens and Burow 1972). Animal experiments have produced damage and haemarthroses in the zygapophysial joints in a significant proportion of the animals examined (Wickstrom et al. 1967; Macnab 1971; La Rocca 1978).

Of interest in the setting of chronic pain after whiplash injury, is the report of a single case of severe and isolated arthritic change in a cervical zygapophysial joint found at post-mortem. The patient had severe, disabling and refractory neck pain for many years after a whiplash injury, culminating in suicide (Rauschning et al. 1989). It is an appealing hypothesis, consistent with known biological models, that injuries to the osseous or soft tissues of a joint predispose it to premature, painful, osteoarthritic change (Mankin 1989). Such an hypothesis could explain the tragic sequence of events in this and other cases.

8.2. Disc

Injuries to the intervertebral discs have repeatedly been reported from a number of sources. The typical lesions are avulsion of the disc from the vertebral end-plate and tears of the anterior annulus fibrosus of the disc. Separation of the disc from the vertebra or fracture of the vertebral end-plate have been seen on plain X-rays and MRI (Keller 1974; Davis et al. 1991), found at operation (Buonocore et al. 1966; Macnab 1966) reproduced in animal experiments (Wickstrom et al. 1967; La Rocca 1978) and found at post-mortem (Jónsson et al. 1991). Lesions in the anterior annulus of the disc have been identified on MRI scan (Davis et al. 1991). Tears at corresponding sites have been identified at operation (Buonocore et al. 1966) and have been a consistent finding in post-mortem studies which have included some patients who survived the initial injury before coming to autopsy (Jónsson et al. 1991; Taylor and Kakulas 1991; Taylor and Twomey 1993). Other studies have reported disc injury or narrowing without specifying the precise site or nature of the lesion (Billig 1956; La Rocca 1978; Bucholz et al. 1979). A carefully conducted study of whiplash injuries to cadavers has produced anterior disc lesions and noted that they were more common after hyperextension than hyperflexion (Clemens and Burow 1972). Although tears of the annulus fibrosus from direct traction would seem the most likely mechanism of injury, it has been suggested that anterior tears could result from the nucleus pulposus bursting through the anterior annulus after being compressed by the extension of the motion segment (Clemens and Burow 1972).

A further piece of evidence indicating damage to the discs or zygapophysial joints in whiplash injury is the observation that in a group of patients with significant symptoms after 10 years, all patients had developed degenerative changes on their X-rays (Watkinson et al. 1991). Furthermore, the age-adjusted prevalence of degenerative changes was significantly higher in those patients who had suffered whiplash than in control groups. These findings are consistent with initial, occult injury to the cervical spine leading to later osteoarthritic change.

8.3. Muscles

Muscle tears and sprains have been revealed by clinical examination (Frankel 1976; Jeffreys 1980) and have also been visualised on ultrasound examination

(Martino et al. 1992). Muscle damage has been seen in animal experiments (Macnab 1966; Wickstrom et al. 1967; La Rocca 1978) and post-mortem examinations (Jónsson et al. 1991). The typical pathology observed is as predicted from the forces involved, with muscles demonstrating partial and complete tears and haemorrhage. No studies have addressed the question of the presence of any chronic, painful muscle pathology following trauma in whiplash patients, and indeed there is little basis for such an entity in conventional pathology teaching. The usual expectation would be that sprains or tears of muscles would heal in a matter of weeks, forming a scar within the muscle but leaving the patient with no residual pain.

8.4. Trigger points and myofascial pain

The notion of trigger points and myofascial pain enjoys a considerable degree of popularity, particularly in North America. It provides a generic, theoretical basis for chronic pain ostensibly stemming from muscles in many regions of the body (Travell and Simons 1983). The neck is one such region and many practitioners seem convinced that trigger points and myofascial pain can develop after whiplash (Evans 1992; Fricton 1993; Teasell 1993). It is not the purpose nor the intention of this review to contest the theory of myofascial pain in general, but there is a duty to explore its pertinence in the context of whiplash.

In the first instance, there are absolutely no epidemiological data on the prevalence of myofascial pain in patients with whiplash. Even a most recent review provides no such data (Fricton 1993). It reiterates the theory of trigger points and explains how they can be diagnosed and treated; it opens with a statement that "myofascial pain ... is one of the most common causes of persistent pain following flexion-extension injuries" (Fricton 1993), but cites no data in support of this statement. No studies using an appropriate inception cohort, and using specific, diagnostic criteria have been conducted to verify the impression that myofascial pain is common after whiplash. Yet there are reasons to doubt that it is so common.

Formal studies (Wolfe et al. 1992) have shown that myofascial experts have difficulty in agreeing as to the very presence of a trigger point — the cardinal feature of regional myofascial pain syndromes. Although they might agree on the presence of tenderness, they could not agree on the presence of the other diagnostic features of trigger points. The theory of trigger points, therefore, lacks demonstrated internal validity. Less expert practitioners may well be finding tenderness in the neck muscles of patients with whiplash injuries, but there is no evidence that this tenderness indicates a trigger point or a primary myofascial diathesis.

More specifically, it is conspicuous that several of the classical trigger points of the neck muscles lack the statutory diagnostic features of a trigger point (Bogduk and Simons 1993). Tenderness is present but not the palpable band nor the twitch response. Since they do not satisfy the formal definition of a trigger point these sites cannot be held to be trigger points. It is furthermore conspicuous that, topographically, the so-called trigger points of the neck overlie the cervical zygapophysial joints and that the reported pain patterns of cervical trigger points are identical to those of referred pain from the zygapophysial joints (Bogduk and Simons 1993). There are grounds, therefore, to believe that what may have been misrepresented as cervical trigger points after whiplash actually represent painful, tender zygapophysial joints.

Whatever might be written or believed about myofascial pain in general, there is no explicit, reliable data on its occurrence after whiplash, yet there is data that casts doubt on the reliability of diagnosis of trigger points in general, and of trigger points in the neck in particular.

8.5. Ligaments

Ligamentous injuries of the neck cannot be diagnosed clinically. However, tears of the anterior longitudinal ligament have been consistently reported in major series of animal experiments (Macnab 1966; Wickstrom et al. 1967), found at operation (Buonocore et al. 1966; Howcroft and Jenkins 1977), identified at post-mortem (Bucholz et al. 1979; McMillan and Silver 1987) and found in cadaver experiments (Clemens and Burow 1972). Magnetic resonance imaging has also confirmed the presence of such lesions in patients not subjected to surgery (Davis et al. 1991). Anatomical studies have indicated that the anterior longitudinal ligament merges imperceptibly with the anterior annulus of the intervertebral disc (Rauschning 1986), indicating that ligamentous injuries may be frequently associated with disc injuries. Injuries to the interspinous ligament have also been identified on MRI (Davis et al. 1991), at operation (Janes and Hooshmand 1965; Jeffreys 1980), at post-mortem and in animal experiments (Wickstrom et al. 1967). However, the significance of any injury to this ligament is questionable since in normal humans it constitutes a delicate, thin, fascial sheet, separating the muscular compartments of the left and right of the posterior neck (Rauschning, 1986). Damage to the posterior longitudinal ligament and the ligamentum flavum from whiplash injury has never been reported at operation or from any imaging studies, but has been seen in animal experiments (Wickstrom et al. 1967), cadaver experiments (Clemens and Burow 1972) and at post-mortem (Bucholz et al. 1979; Jónsson et al. 1991). These are both highly elastic structures and injury to them would reflect severe trauma involving large, destructive, and probably lethal excursions of the cervical vertebrae.

8.6. Atlanto-axial complex

Fractures of the atlas or axis may be dramatic events resulting in death or serious neurological injury (Levine and Edwards 1989), and it is therefore not surprising to find such injuries in post-mortem studies (Bucholz et al. 1979; Jónsson et al. 1991). However, more subtle, occult injuries may occur in the setting of whiplash. Fractures of the odontoid peg have been detected clinically (Seletz 1963; Signoret et al. 1986), and produced in animal experiments (Wickstrom et al. 1965). Evidence of bony injury to other parts of C2, including the laminae and superior articular process, have been obtained from radiographic (Seletz 1958; Signoret et al. 1986; Craig and Hodgson 1991) and operative (Signoret et al. 1986; Craig and Hodgson 1991) assessments. Atlas injuries are reported less frequently, but have been found on plain X-rays and reproduced in cadaver experiments (Abel 1958, 1975).

The atlanto-axial joints permit a wide range of axial rotation (Dvorak et al. 1987b) and their integrity is maintained by ligaments, particularly the alar and transverse ligaments (Dvorak and Panjabi 1987; Dvorak et al. 1988; Saldinger et al. 1990). These structures would appear to be susceptible to injury on the basis of post-mortem studies (Jónsson et al. 1991) but demonstration of injuries in vivo is difficult. However, a recent, controlled series involving the elegant application of functional CT scanning has permitted the detection of pathological hypermobility due to disruption of the alar ligaments in patients with pain after whiplash injury (Dvorak et al. 1987a).

8.7. Cervical vertebrae

Patients with overt fractures of the cervical vertebrae below C2 can be readily classified, and conventional management algorithms applied. However, in patients with whiplash injuries, fractures may be missed either because it is not recognised that flexion-extension alone, without direct head impact, can cause vertebral fractures or because some fractures, particularly of the posterior elements, are impossible to see on conventional radiographs. Experimental studies in animals and cadavers (Wickstrom et al. 1967; Clemens and Burow 1972; Abel 1975) as well as post-mortem observations (Bucholz et al. 1979; Jónsson et al. 1991) have confirmed that vertebral fractures can occur from whiplash-type injuries. When carefully sought with specialised views, fractures of the pedicles and laminae have been seen in patients (Abel 1975). There are also isolated reports of transverse process fractures (Norris and Watt 1983; Jónsson et al. 1991) and compression fractures of the vertebral bodies themselves (Cammack 1957; Norris and Watt 1983). Fracture of the spinous processes appears to be a rare event, but has been noted on plain radiographs and produced in a cadaver experiment (Gershon-Cohen et al. 1954).

8.8. Brain

Careful animal experiments have demonstrated haemorrhage in and around the brain from acceleration injuries without direct trauma to the head (Wickstrom et al. 1967; Ommaya et al. 1968; Sano et al. 1972; La Rocca 1978). Sub-dural haematoma has also been noted following whiplash injury in a human (Ommaya and Yarnell 1969). It may be that cerebral injuries from whiplash are under-reported as the presence of a significant head injury, irrespective of how it was acquired, would distract attention from any neck symptoms and therefore any association between brain injury and whiplash injury will be unapparent. In addition, subtle injuries to the brain may be beyond resolution using conventional imaging.

8.9. Temporo-mandibular joint

Injuries to the temporo-mandibular joint from whiplash have been suspected on clinical grounds for many years (Frankel 1965; Roydhouse 1973), and two recent reviews attest to the considerable support for the view that the temporo-mandibular joint can be injured in whiplash accidents (Epstein 1992; Brooke et al. 1993). However, much of the supporting evidence stems from clinics specialising in temporo-mandibular joint pain, but the data is retrospective, not prospective. Many patients with temporo-mandibular pain report a history of cervical trauma, but these studies do not indicate the prevalence of temporo-mandibular problems after whiplash.

The study of Weinberg and Lapointe (1987) reported an uncontrolled, referral-based series of 28 patients in which internal derangements were detected in 22 of 25 patients investigated with arthrography, and amongst whom pathology was confirmed in 10 patients who proceeded to surgery. This sample, however, lacked a control group and is not likely to be representative; an important consideration when evaluating such a common condition (Kupperman 1988).

On the other hand, other investigators have brought contrary evidence to bear. Heise et al. (1992) followed an inception cohort of patients with whiplash, seen at a surgical trauma, emergency department, and found the incidence of temporo-mandibular joint symptoms to be very low. Of 155 patients, 22 reported masticatory and temporo-mandibular pain when first seen, and none had persistent symptoms at follow-up after 1 year.

The evidence is, therefore, clearly divided. Doubtless, specialists in temporo-mandibular pain do see patients with a history of cervical trauma but for a controversial condition that otherwise has variously be ascribed to causes as diverse as depression, myofascial pain and trauma (Dworkin et al. 1990), a causative link to whiplash has still to be demonstrated.

8.10. Other tissues

Injuries to various other tissues have been reported following whiplash injury. Horner's syndrome, indicating damage to the cervical sympathetic nerves, has been noted (Jeffreys 1980). Avulsion of part of the occipital bone, a lesion consistent with observed injuries to the ligamentum nuchae (Janes and Hooshmand 1965), has been detected radiographically (Cammack 1957). Perforation of the cervical oesophagus is a rare complication of whiplash injury (Spenler and Benfield 1976), but may be more likely in those patients with pre-morbid, intervertebral, osteoarthritic changes and anterior osteophyte formation. Prevertebral haematomas compromising the airway have been reported (Howcroft and Jenkins 1977; Biby and Santora 1990), as well as damage to the recurrent laryngeal nerves leading to vocal cord paralysis (Helliwell et al. 1984).

Two studies have reported finding perilymph fistulae at operation in patients with vestibular symptoms following whiplash injury (Grimm et al. 1989; Chester 1991). However, these observations are uncontrolled and relate to a referral-based population, so that it is unclear how representative they are.

Devastating spinal cord injury can occur from pure acceleration-deceleration injuries of the cervical spine without obvious bony injury (McMillan and Silver 1987). Any symptoms stemming from the cervical structures are usually overwhelmed by the severity and consequences of the neurological damage. There have been isolated case reports of anterior spinal artery syndrome complicating 'cervical sprain' (Grinker and Guy 1927; Foo et al. 1984).

8.11. Synopsis

On balance, given the extent to which experiments of different natures have converged to the same conclusion, the leading contenders for explaining chronic neck pain following whiplash injury are injuries to the zygapophysial joints, the intervertebral discs and the upper cervical ligaments.

Damage to other cervical structures can occur, but the available evidence suggests that these are less frequent sources of chronic pain. Included in this list are various components of the cervical vertebrae, the anterior longitudinal ligament, cervical musculature and temporo-mandibular joint. Symptoms other than pain may occur through damage to the sympathetic trunk, brain, inner ear and oesophagus.

9. Symptoms

Authorities differ as to what they consider to be the symptoms of whiplash injury. Moreover, none of the prospective, hospital-based reports concerning whiplash has explicitly sought to describe the clinical features stemming from the injury (Norris and Watt 1983; Deans et al. 1987; McNamara et al. 1988; Olsson et al. 1990; Pennie and Agambar 1991). The following represents a list of those symptoms most commonly reported in these and other, retrospective, referral-based studies.

9.1. Neck pain

The cardinal manifestation of whiplash injury is neck pain. When the features of the pain have been described, there has been reasonable consistency between reports (Gay and Abbott 1953; Cammack 1957; Janes and Hooshmand 1965; Gates and Benjamin 1967; Schutt and Dohan 1968; Hohl 1974; Balla 1980; Norris and Watt 1983; Maimaris et al. 1988; Pearce 1989; Olsson et al. 1990; Bring and Westman 1991; Pennie and Agambar 1991). Typically, the pain is perceived over the back of the neck and is either dull and aching with exacerbations on movement, or a sharp pain related to movement, or any combination of the two. Frequently, there is associated neck stiffness or restricted movement. Pain may radiate to the head, shoulder, arm or interscapular region. These patterns of somatic referred pain do not necessarily indicate which structure is the primary source of pain, but rather, suggest the segmental level mediating the nociception (Bogduk 1988). None of the reported series discriminate between the features of early and late neck pain. Conspicuously absent from the literature are any studies correlating neck pain and pathology. The relationship between most of the pathological entities described above and the patients' symptoms remains circumstantial. The investigation of whiplash injuries has typically consisted of increasingly elaborate imaging techniques – plain radiographs being followed by specialised views, tomography, computerised tomography and even MRI. However, none of these investigations has ever been calibrated against a gold standard of known, painful pathology and hence their utility in determining the cause of neck pain in whiplash injury remains unknown.

The most logical approach to investigating neck pain following whiplash injury is to provoke or eliminate the pain by stimulating or anaesthetising structures suspected of being symptomatic. This approach dispenses with concerns about whether or not abnormalities observed on imaging studies are the cause of the patient's pain, by addressing the pain itself. Techniques that anaesthetise the cervical zygapophysial joints have been developed (Bogduk and Marsland 1988). These involve local anaesthetic blocks of either the joint itself or the nerves that supply it, and allow a joint to be implicated as a source of pain. Zygapophysial joint pain can therefore be confirmed or refuted by testing each putatively painful joint in turn. Application of these techniques to a large cohort of patients, many of whom had

sustained whiplash injuries, revealed that between 25% and 62% were suffering from cervical zygapophysial joint pain (Aprill and Bogduk 1992). These observations were made on the basis of single, uncontrolled blocks in an heterogenous group of neck pain patients. Subsequent studies have demonstrated the specificity of the technique of medial branch blocks of the cervical dorsal rami for the diagnosis of cervical zygapophysial joint pain, and have found these blocks to be reliable investigations when applied under double-blind, controlled conditions using different local anaesthetics (Barnsley and Bogduk 1993; Barnsley et al. 1993a). Applying this stringent investigative protocol to a cohort of referred patients with chronic whiplash has revealed that 54% have pain arising from at least one cervical zygapophysial joint (Barnsley et al. 1993b). No other potential source of pain in the neck has been so clearly defined, or found to be so common.

Anaesthetic blocks of other structures, such as the greater occipital nerve and ventral rami of the spinal nerves, can occasionally be useful in confirming or eliminating structures in their territories as causes of pain but their reliability and specificity are not known and their true utility in the routine assessment of the whiplash patient remains unclear.

The only regularly used provocative test in the neck is provocation discography, in which a disc is punctured by a needle and distended by injecting contrast medium. A positive discogram occurs when the procedure reproduces the patient's usual pain and implicates that disc as the source of pain. The response can occasionally be confirmed by injecting local anaesthetic in an attempt to abolish the pain (Simmons and Segil 1975; Roth 1976). In practice, the procedure itself can be quite painful and it is often difficult for the patient to judge whether it is their usual pain that is being reproduced (Holt 1964; Klafta and Collis 1969). The reliability of discography has been called into question by the observation that, in a significant proportion of patients, pain reproduced by discography can be completely eliminated by subsequent zygapophysial joint blocks at that level (Aprill and Bogduk 1992). Since zygapophysial joint blocks do not have any effect on pain perception from the disc, these observations must indicate that discograms are liable to false-positive interpretations, wrongly incriminating the disc as a cause of pain, when the true source lies in the zygapophysial joint. It seems that in some patients, provocation discography appears positive when other structures innervated by the same segmental nerves sensitise that segment to noxious stimulation (Aprill and Bogduk 1992). These concerns do not pertain to anaesthetic block procedures because the segment is not mechanically stressed, and only those structures affected by the anaesthetic are incriminated by a positive response. On the basis of current evidence, discograms should only be considered as truly positive if zygapophysial joint blocks at that level are negative (i.e., no pain relief).

9.2. Headache

After neck pain, headache is the most frequently reported complaint following whiplash injury (Pietrobono et al. 1957; Schutt and Dohan 1968; Bingham 1968; Farbman 1973; Hohl 1974; Balla 1980; Maimaris et al. 1988) and in some series constitutes the principal symptom (Gates and Benjamin 1967; Bring and Westman 1991). The pain is typically reported to be sub-occipital or occipital, radiating anteriorly into the temporal or orbital regions. Some authors have suggested that the headache results from concussion (Gay and Abbott 1953; Cammack 1957), but provide no convincing evidence in support of this proposition. In some circles, the headache is assumed to be cervical in origin. In neuroanatomical terms, afferents of the upper three cervical nerves (C1–C3) terminate in the cervical portion of the trigeminal nucleus (forming the trigemino-cervical nucleus), so that any pain arising in the distribution of these spinal nerves can be referred to the territory of the trigeminal nerve (Bogduk 1986b). Since the ophthalmic division of the trigeminal nucleus projects most caudally, pain originating from the upper cervical structures is likely to be referred to the orbital and temporal regions. Therefore, it is possible to explain the presence of sub-occipital headache through injury to one of the upper cervical structures with orbital or temporal radiation indicating referral via the trigemino-cervical nucleus.

There are no non-invasive means of accurately diagnosing the structure involved in the production of cervical headache. Careful clinical studies of patients investigated with third occipital nerve blocks for the diagnosis of C2–3 zygapophysial joint pain were unable to identify any clinical features, on history or examination, that could be used to predict correctly the response to subsequent blocks (Bogduk and Marsland 1986). Pain from the lateral atlanto-axial joint is also perceived in the occipital or sub-occipital region (McCormick 1987) and would appear to be clinically indistinguishable from C2–3 zygapophysial joint pain.

A recent study (Lord et al. 1994) employing double-blind, controlled, diagnostic blocks of the C2–3 zygapophysial joints found that referred pain from this joint occurred in 27% of 100 consecutive patients with chronic pain after whiplash; and amongst those patients in whom headache was the dominant pain complaint, referred pain from the C2–3 joint was the basis of their headache in 53% of cases. Similar studies have not yet been conducted to determine the prevalence of pain from the lateral atlanto-axial joints after whiplash injury.

Other candidate structures that may be responsible

for cervical headache following whiplash injury are the transverse and alar ligaments and the median atlanto-axial joint. However, as no safe and reliable techniques exist to anaesthetise or provoke pain from these structures, it is currently impossible to confirm that they are a source of pain. Consequently, the relative frequency of different painful lesions in the aetiology of headache in whiplash injuries is not fully known.

In addition to cervical causes of headache, intracranial causes such as haemorrhage or other concurrent injury should be considered, but in the chronic setting it seems likely that the majority of chronic whiplash headache will be cervical in origin.

9.3. Visual disturbances

Patients often complain of visual disturbances following whiplash injury but only rarely has any attempt been made to characterise the abnormalities. A single, retrospective series ascribed the visual disturbances to accommodative errors but failed to provide objective evidence in support of this (Gates and Benjamin 1967). Accommodative power is reported to be decreased in whiplash patients (Horwich and Kasner 1962) but this study did not provide details of the study population or the reliability of the techniques used. However, a powerful, controlled study has been able to demonstrate objective abnormalities of oculomotor function in patients with chronic neck pain following whiplash injury (Hildingsson et al. 1989). Velocity, accuracy and pattern of eye movements were objectively measured in three groups of patients. The first group had chronic neck pain and stiffness following whiplash injury, the second group comprised individuals who were asymptomatic following whiplash and the third were healthy volunteers. The results showed no differences between the healthy and asymptomatic groups, but significant oculomotor impairment in the chronically symptomatic patients. Despite being the only study of its type, the strength of the research design endows this work with significant weight.

Pathophysiological explanations for these visual disturbances have been only tentative or speculative, and include concepts such as impaction of the ventral aspect of the midbrain against the clivus (Horwich and Kasner 1962), damage to the vertebral artery (Macnab 1966) or damage to the cervical sympathetic trunk (DePalma and Subin 1965; Macnab 1966).

Although injuries to the vertebral artery have been described in animal (Wickstrom et al. 1965) and post-mortem studies (Taylor and Twomey 1992), it is extremely unlikely that such damage is the mechanism underlying visual disturbances. The only means by which vertebral artery damage can cause neurological deficit is through ischaemic injury to the brain stem or cerebellum. However, neurological examination of patients with objective evidence of oculomotor defects and complaints of visual disturbance failed to reveal any abnormalities (Hildingsson et al. 1989). The likelihood that a single, discrete ischaemic lesion could affect only those brain-stem nuclei responsible for accommodation and focussing, without affecting long tracts, cerebellar connections or other cranial nerve nuclei to produce neurological signs, is very small.

Being an anterior structure in the neck, the cervical sympathetic trunk would certainly be liable to stretch or rupture by hyperextension of the neck, and indeed such damage has been produced experimentally in monkeys (Macnab 1966). Interruption of the sympathetic trunk, however, would manifest as Horner's syndrome, a condition described far less frequently than visual disturbance alone (Seletz 1963; Jeffreys 1980). Blurred vision, on the other hand, without signs of sympathoplegia, cannot be explained on the basis of traumatic sympathectomy, and therefore demands another explanation.

A plausible mechanism that has been advanced to link neck pain and eye symptoms involves the cilio-spinal reflex (Bogduk 1986a). In this reflex, a noxious stimulus to the skin of the face or neck evokes dilation of the pupil (Walton 1977). In more general terms, a painful stimulus delivered to the cutaneous territory of the upper cervical nerves evokes an efferent sympathetic discharge to the eye. Given this background, blurred vision could be explained if two assumptions hold true. The first is that, like cutaneous afferents, deep nociceptive cervical afferents can evoke sympathetic reflexes to the eye. The second is that sympathetic stimulation can flatten the ocular lens (Middleton 1956). Hypothetically, therefore, blurred vision could be construed as due to inappropriate accommodative power produced by sympathetic activity evoked by cervical pain. Alternatively, since the cilio-spinal reflex manifests in the eye ipsilateral to the stimulus, in patients with lateralization of their pain, discordance in the accommodative power between the eyes may result, and produce blurring and difficulty focussing.

9.4. Dizziness

Sensations of disequilibrium or dizziness, often in association with other auditory or vestibular symptoms, have been reported in many series of whiplash injuries (Gay and Abbott 1953; Cammack 1957; Norris and Watt 1983; Pearce 1989; Dvorak et al. 1989; Olsson et al. 1990; Bring and Westman 1991; Pennie and Agambar 1991). Typically, these complaints have occurred in the absence of clinically apparent vestibular or neurological dysfunction. Interest in this phenomenon has stimulated a number of reports using electronystagmography (ENG) as a means of objectively verifying patients' complaints (Compere 1968; Toglia et al. 1969, 1970; Pang 1971; Toglia 1972, 1976; Rubin 1973;

Chester 1991). These studies have all been of patients referred to specialised ENT units because of vestibular symptoms, and have all lacked clearly defined control groups. Nevertheless, they have demonstrated that between 54 and 67% of patients complaining of dizziness after whiplash injury have abnormal ENG studies, most commonly on rotatory testing. Canal paresis and other caloric test abnormalities were also noted in many of these patients (Compere 1968; Toglia et al. 1970; Pang 1971; Toglia 1972, 1976). Control observations are mentioned in only two studies. Oosterveld et al. (1991) reported that ENG studies were significantly abnormal in whiplash patients when compared to a control group of normal individuals. No details of the controls are provided. In contrast, when testing only for neck torsion nystagmus, Calseyde et al. (1977) found no difference in the frequency of abnormalities between 916 consecutive medico-legal cases and 137 healthy asymptomatic controls undergoing routine, clinical assessment for pilot training. The frequency of nystagmus following neck torsion was 11% in both groups. The characteristics and mode of injury of the cases was not described and it is not clear whether the cases were even symptomatic. Furthermore, the frequency of ENG abnormalities is considerably lower than that reported by any of the above studies. This may reflect a peculiarity of the test used but is more likely due to the population studied. On balance, the evidence indicates that symptomatic patients frequently have objective evidence of vestibular dysfunction on ENG testing, suggesting either central or peripheral injury.

One study has reassessed patients with proven ENG abnormalities after 12 months (Oosterveld et al. 1991). Only a subset of the original cohort was examined and the selection criteria for this group were not given. Nevertheless, less than 5% of the patients demonstrated any improvement over 12 months.

The exact mechanism by which dizziness occurs following whiplash injury remains speculative. It has been argued that vertebral artery injury or irritation may compromise vertebral artery flow, but as discussed above, such a mechanism would be expected to produce distinctive neurological signs and symptoms related to ischaemia or infarction of the brain stem or cerebellum (Kistler et al. 1991). More subtle disturbances of balance and equilibrium may result from interference with postural reflexes that have cervical afferents (De Jong and Bles 1986). Anaesthetising the neck muscles of animals and humans results in ataxia and/or nystagmus (Biemond and De Jong 1969; Igarashi et al. 1969, 1972; De Jong et al. 1977; Bogduk 1981), indicating that important proprioceptive information arises from these structures. It is conceivable that disturbance of this output may result from pain or spasm following damage to these muscles, or related structures. Such a proposition is further supported by experimental work on 44 patients with whiplash injury (Hinoki and Niki 1975). In this study, deep cervical muscle tone, as measured by EMG, was increased by the administration of isoproterenol, a β-sympathetic agonist. Vestibular function deteriorated and symptoms worsened in 8 of 13 patients. Conversely, when propranolol, a β-sympathetic blocker, was administered, cervical muscle tone decreased, vestibular function and symptoms of vertigo improved. Blocking or stimulating α-sympathetic receptors made no difference to either muscle tone or vestibular signs and symptoms.

Direct damage to the vestibular apparatus has also been proposed as a cause of dizziness following whiplash injuries. Perilymph fistulas of both the round and oval windows have been found in patients with vertigo and disequilibrium (Grimm et al. 1989; Chester 1991). A detailed analysis of patients with perilymph fistulae has indicated that such patients may experience a wide range of vestibular and even cognitive symptoms including poor concentration, poor visual tracking, disorientation in visually complex situations and clumsiness (Grimm et al. 1989). To the casual observer, such symptoms may easily be dismissed as inexplicable or may even be attributed to neuroticism or malingering. However, a high index of suspicion and careful assessment may reveal an important and potentially curable lesion.

9.5. Weakness

Where weakness occurs in recognised myotomal distributions, and is accompanied by consistent reflex and sensory signs, a diagnosis of nerve root involvement can be made, and the patient investigated and treated accordingly. Far more puzzling, and more common, are subjective sensations of weakness, heaviness or fatigue in the upper limbs that are unaccompanied by clear-cut abnormalities on clinical examination. The inconsistency between symptoms and signs has been attributed to malingering or hysteria (Berry 1976), but there is evidence that sensations of heaviness in the limbs have an organic, neurophysiological basis and can be caused by pain.

The sensation of heaviness has been assessed in patients with both pathological and experimentally induced muscle weakness. The pathologically-weak group constituted stroke patients with pure, unilateral motor defects. The experimentally weakened group had one arm partially curarized. Both of these groups perceived a given weight as heavier with the weak arm compared to the normal, contralateral arm. Sensation was normal in both arms so, the impression of heaviness must have arisen through an appreciation of the amount of effort required to lift the weight (Gandevia and McCloskey 1977). Further, elegant experiments have shown that painful cutaneous stimulation can reduce the maximum

effort that can be applied by a muscle group through reflex inhibition of muscle contraction, a phenomenon that is independent of voluntary control (Aniss et al. 1988). Together, these studies show that cutaneous stimulation can inhibit motor power, and the resultant need for increased 'central effort' for a given task is perceived as heaviness or weakness.

In clinical settings, reflex inhibition of the quadriceps muscle has been noted in patients with joint or muscle pain, and also in patients with no pain but with previous joint injury (Rutherford et al. 1986). Furthermore, treatment of chronic knee pain in patients with objectively verified quadriceps inhibition has been shown to reduce inhibition (Stokes and Young 1984).

In the context of whiplash injury, those patients with chronic neck pain may well develop reflex inhibition of those muscle systems that act on, or in conjunction with, the neck. This would include those muscles of the upper limb which are contracted during lifting. To overcome this inhibition, more central effort is required, resulting in a sensation of increased heaviness or weakness. In addition, where arm pain is present, either through nerve root involvement or as somatic referred pain from the neck, the arm muscles may be inhibited directly, again producing a sensation of heaviness.

9.6. Paraesthesia

Sensations of tingling and numbness in the hands, particularly of the ulnar two fingers, have been reported in both prospective and retrospective series (Gay and Abbott 1953; Schutt and Dohan 1968; Hohl 1974; Norris and Watt 1983; Bring and Westman 1991; Pennie and Agambar 1991). In the presence of muscle weakness, reflex changes and objective abnormalities on sensory testing, these symptoms can be attributed to nerve root compression and may be appropriately investigated using well established algorithms (Nakano 1989). More commonly, however, symptoms are intermittent and are not associated with overt neurological signs (Norris and Watt 1983; Pennie and Agambar 1991). It is this latter group that constitute a diagnostic problem and whose symptoms demand an adequate explanation.

One of the most plausible theories is that the paraesthesiae may be due to thoracic outlet syndrome arising from compression of the lower cords of the brachial plexus as they pass between the scalenus anterior muscle and the scalenus medius muscle, and under the clavicle. In a referral-based series of 35 patients with post-traumatic neck pain and arm symptoms, 30 had objective evidence of slowed nerve conduction across the thoracic outlet on nerve conduction studies (Capistrant 1977). The mean conduction velocity in this group was 55 m/sec compared with 72 m/sec in an asymptomatic control group. Where the symptoms were unilateral, conduction velocities were slower in the symptomatic arm. No formal statistical analysis of this data was performed. In a later study by the same author, clinical tests in conjunction with electrodiagnostic studies suggested a diagnosis of thoracic outlet syndrome in 31% of patients referred to a private neurology practice for evaluation of symptoms following whiplash injury (Capistrant 1986). Notwithstanding any reservations about the electrodiagnostic technique, the former study, by virtue of providing control observations, provides evidence in support of a real abnormality of ulnar nerve conduction in patients with arm symptoms following whiplash injury. The latter study is limited in that it is a retrospective review of referred patients, but the mere fact that a substantial proportion of patients had features suggestive of thoracic outlet syndrome would indicate that this is not a rare phenomenon and is worthy of further, more formal study. Exactly how this syndrome develops remains speculative, but reflex spasm of the scalenus muscles, due to pain from other structures in the neck, might compress the lower cords of the brachial plexus and account for an intermittent and at times sub-clinical impairment of ulnar nerve function.

There is evidence that other pathological processes may affect the brachial plexus after whiplash injury. Recent anecdotal reports of 2 patients with persistent arm and hand symptoms describe the operative finding of 'massive fibrosis' in and around the brachial plexus (Bring and Westman 1991). These observations invite further research and provide hypotheses for consideration in individual patients.

9.7. Concentration and memory disturbances

The development of cognitive impairment following minor head injury or whiplash is not widely appreciated in the general community (Aubrey et al. 1989). Patients are therefore unlikely to disclose symptoms reflecting cognitive difficulties, fearing that they may be ascribed to neurotic anxiety, exaggeration or malingering. Formal, psychometric assessments of patients with chronic symptoms after whiplash injury have confirmed the presence of objective cognitive impairments, and raised intriguing possibilities to explain their aetiology. A Swiss study of 18 patients with neck pain and cognitive disturbances after whiplash injury, assessed their performance on an extensive battery of neuropsychological tests (Kischka et al. 1991). The results were compared to those of a carefully matched control group of healthy volunteers without a history of whiplash injury. The symptomatic group displayed deficits in the areas of attention, concentration and memory. On the other hand, in a compatible group of patients, a Norwegian study failed to find any differences in neuropsychological test outcomes between whiplash patients,

and those with similar somatic symptoms and no history of whiplash (Olsnes 1989). At first glance these results may seem contradictory, but closer inspection reveals that the control groups were quite different. Considering these studies together, it is possible to draw the conclusion that whiplash injured patients have impairment of memory and concentration compared to healthy subjects, but are no more impaired than patients with similar somatic complaints and no history of trauma. The possibility raised by these studies is that cognitive impairments in whiplash patients arise not from direct injury to the brain itself, but are somehow related to chronic neck and head pain (Anderson et al. 1990; Radanov et al. 1992a).

Whether reported abnormalities of cognition in whiplash patients represent primary, organic, brain injury remains unclear. Studies using CT scans and MRI in patients after whiplash, have disclosed only non-specific abnormalities in only 5–7% of cases (Yarnell and Rossie 1988; Ettlin 1992); but it may be that mild diffuse injury may not be radiologically apparent.

Electroencephalographic studies have revealed conflicting results. Torres and Shapiro (1961) studied a group of whiplash patients and a group who had suffered direct head injuries, and found that between 40% and 50% of patients in both groups exhibited abnormalities in their electroencephalograms (EEG), compared to only 1% of their control group. A more recent, uncontrolled study failed to corroborate this result (Jacome 1987). On the other hand, reports of studies on a group of whiplash patients (n = 35) and a group of direct head-injury patients using megimide-activated EEGs found that 57% of whiplash patients, and 73% of head injury patients, had abnormal activated EEGs following injection of megimide. It was argued that the threshold dose of megimide required to produce EEG activation was lower in whiplash and head injured patients than historical controls (Koshino et al. 1972). Thus, in so far as EEG abnormalities can imply brain damage or dysfunction, it would appear that whiplash is just as capable of producing such damage as direct injuries to the head.

The issue of mild brain injury and EEG, however, has been addressed in a recent, thorough review (Shapiro et al. 1993). From that review, it is clear that the studies purporting to show a relationship between impaired cognitive functioning and whiplash, and between impaired function and EEG were inadequately controlled. What has not been excluded is that deficits in cognitive function are due to the impact of chronic pain, depression, and anxiety, or the effects of medication (Shapiro et al. 1993; Merskey 1993). Indeed, what has emerged from two studies is that the presence specifically of headache, rather than any other feature, correlates with impaired attention (Radanov et al. 1992b,c).

9.8. Psychological symptoms

Many series have noted 'psychological factors', 'psychoneurotic reaction', 'emotional factors' or 'functional overlay' in whiplash patients (Gay and Abbott 1953; Gotten 1956; Cammack 1957; Macnab 1966; Farbman 1973; Balla 1980; Pearce 1989). These terms have often been used without meaningful definitions, and do not reflect the use of published, standardised, psychiatric diagnostic criteria (American Psychiatric Association 1987). If one does accept that whiplash patients may display identifiable psychopathology, the fundamental question is the relationship between psychological disturbance and the symptoms and the whiplash injury. Some authors have maintained that pre-existing personality traits or psychiatric problems create, or contribute to somatic symptoms after whiplash injury (Hodge 1971; Gorman 1974, 1979; Blinder 1978). It has even been advanced that 'traumatic neurosis' occurs in neurotics who have 'been looking for a trauma and have now found one' (Gorman 1979). However, these hypotheses have been refuted by a recent Swiss study (Radanov et al. 1991; Editorial 1991). This prospective, observational study involved 78 consecutive whiplash patients. All were assessed, using standard tests, for psychosocial stress, negative affectivity and personality traits soon after suffering a whiplash injury. None of these factors were found to predict the persistence of symptoms at 6 months. Furthermore, illness behaviour after whiplash does not appear to be related to the patient's perception of the severity of the accident or their concern over illness or disability (Radanov et al. 1992b). Nor do patients with neck pain after whiplash score differently from patients with non-traumatic neck pain on scales for neuroticism, and indeed, both types of patient score within normal ranges (Radanov et al. 1992c).

An alternative viewpoint is that patients suffering injury followed by persistent pain may develop psychological symptoms as a secondary phenomenon (Merskey 1984). Indeed, this interpretation has been reiterated in a recent, formal review (Merskey 1993). Some patients may exhibit an overt, post-traumatic stress disorder, but there is no formal evidence that the pain of whiplash is due to psychological factors. The principal reason for psychological illness in association with cervical sprain injuries is the injury itself (Merskey 1993).

It would be hardly surprising if some patients, faced with refractory pain, suspicious doctors, potential loss of employment and the stress of a legal dispute, became depressed or anxious. Conversely, there is no reason to suggest that those patients with psychopathology are not injured in MVAs, so that some patients with psychological distress and whiplash injury simply have two, common, independent conditions.

On balance, there is general recognition that patients with whiplash injury do exhibit abnormal psycho-

logical distress. However, the currently available data indicate that psychological factors do not predict chronicity of symptoms, and that any excess psychological symptomatology is a consequence of the injury and its profound physical, social, legal and vocational effects.

10. Litigation neurosis

In vivid contrast to the abundant evidence in support of painful, organic lesions in whiplash patients there is little more than speculation and pejorative anecdote to suggest that the symptoms are due to 'litigation neurosis'. Nonetheless, some authors maintain that exaggerated complaints of pain and injury are made in order to secure financial gain (Hodge 1971; Gorman 1974; Berry 1976; Balla 1982; Mills and Horne 1986). However, competent follow-up studies of whiplash patients report that the likelihood of chronicity of symptoms following whiplash injury is independent of litigation (Norris and Watt 1983; Maimaris et al. 1988; Pennie and Agambar 1991). Formal study of litigation or compensation neurosis unearths little evidence in support of the concept, but reveals a plethora of reports demonstrating that compensation patients are no different to non-compensation patients (Mendelson 1982, 1984, 1992; Shapiro and Roth 1993).

Close scrutiny of the early reports proffered in support of the concept of malingering reveals disturbing methodological flaws, unacceptable by contemporary standards. Miller's original report (Miller 1961) of patients with 'accident neurosis' comprised descriptive data on 50 patients assessed for head injury at a major referral centre. After settlement, 41 of 45 employed patients returned to work. However, these patients were a small subset who displayed 'gross neurotic symptoms' and who had been selected from more than 4000 patients. It is impossible to draw any generally applicable, externally valid conclusions from such biased sampling. Gotten's study of whiplash patients after settlement of compensation reports that 88% of patients showed recovery after settlement and over one-half had no residual symptoms (Gotten 1956). The study concluded that there was great difficulty in evaluating whiplash patients due to the complicating factor of monetary compensation, and that the injury was being used as a 'lever for personal gain'. However, this study was conducted with a unvalidated questionnaire administered by a single individual. Only 100 of 219 potential subjects were able to be contacted, a response rate of only 45%. No control group was included so that the effect of the natural tendency of many patients to improve spontaneously in the first few months after injury was not considered. Finally, many of the conclusions are based on the anecdotes and opinions of the 'interrogator' administering the questionnaire. In the light of these methodological defects, the conclusions drawn from the study cannot be sustained.

Therefore, there is no real evidence that malingering for financial gain, contributes in any significant way to the natural history of whiplash injury. The unavoidable conclusion is that the majority of whiplash injuries result in real, organic lesions in genuine patients.

11. Factors influencing prognosis

Several studies have sought to identify factors that influence the prognosis of whiplash injury. However, any study aiming to determine those factors which predict the outlook for an individual patient should include sufficient numbers of patients to allow powerful statistical techniques, such as regression analysis, to be applied and therefore enable the 'risk of chronicity' to be calculated. The only study meeting these requirements in the context of whiplash has shown that increasing age, injury related cognitive impairment and the severity of the initial neck pain were predictive of persistent symptoms at 6 months (Radanov et al. 1991). However, this study was primarily concerned with psychosocial factors and it is unclear whether or not other potential predictors were included in the analysis.

In other studies, objective neurological signs, degenerative changes on X-ray, and thoracolumbar pain have been found to be associated with, but not necessarily predictive of, a poor prognosis (Norris and Watt 1983; Maimaris et al. 1988; Miles et al. 1988; Watkinson et al. 1991). Since degenerative changes occur more frequently with increasing age (Friedenberg and Miller 1963), it is possible that age is a confounding variable in the relationship between degenerative changes and a poor prognosis following whiplash. In other words, older people do worse after whiplash injury and coincidentally have degenerative changes; alternatively, the converse may hold true — older people fare worse because incidentally they also had pre-existing degenerative changes. The analyses performed in studies to date do not yet allow the independent effects of these variables to be separated.

12. A model

The data collated into this review suggest a model that is distinct from that implied by past and contemporary opinions. Admittedly, no studies have yet explicitly demonstrated the pathology underlying either the acute or chronic pain of whiplash. However, the anatomical, biomechanical and experimental data demonstrate that, in whiplash injuries, pre-vertebral

and post-vertebral muscles may be torn and zygapophysial joints and intervertebral discs can be damaged. Possibly but less commonly, the sympathetic trunk, brain, inner ear and oesophagus may be damaged as well.

Tears of muscles and ligaments are acceptable, possible causes of pain. Analogous with injuries to these tissues elsewhere in the body, and being vascular structures, muscles and ligaments would be expected to heal over several weeks with scar formation and loss of pain. Such a pattern would be consistent with the observation that the majority of patients quickly recover after whiplash injury. Minor, occult fractures would also follow this pattern with painless function following healing after 6–8 weeks. On the other hand, injuries to the zygapophysial joints or intervertebral discs would be expected to have a different prognosis.

Discs are avascular, and tears to the annulus fibrosus or separation of the disc from the adjacent vertebral body are unlikely to heal, yet these structures are innervated and therefore constitute an anatomical substrate for pain (Bogduk et al. 1988). However, although there is circumstantial evidence of injuries to discs after whiplash from experimental studies (Clemens and Burow 1972), post-mortem studies (Jónsson et al. 1991; Taylor and Kakulas 1991; Taylor and Twomey 1993) and imaging studies (Davis et al. 1991), there is no clinical evidence. No studies have yet shown that these apparent disc injuries occur only in patients with pain or that these lesions are at all painful. The data on zygapophysial joints stands in contrast.

Injuries to the cervical zygapophysial joints have been produced experimentally (Abel 1958; Clemens and Burrow 1972), found at post-mortem (Jónsson et al. 1991) and noted in several clinical studies (Abel 1975, 1982; Binet et al. 1977; Jeffreys 1980; Smith et al. 1976; Clark et al. 1988). Injuries to the zygapophysial joint or to the underlying bone may disrupt the congruity of the joint surfaces, producing a painful post-traumatic osteoarthritis. Alternatively, haemarthrosis or injury to the intra-articular structures may lead to a chronic, post-traumatic synovitis with ongoing pain and joint damage. Therefore, patients with injuries to the discs or joints may be expected to have prolonged pain with little chance of healing or spontaneous recovery.

Injuries of the zygapophysial joints, however, are difficult, if not impossible, to detect in vivo. Lesions of the capsules or meniscoids are invisible to X-rays. Even fractures of the joints are not apparent on plain films, and require special techniques or high resolution CT to be demonstrated. Consequently, it has not been possible to compare the incidence of these injuries in symptomatic and asymptomatic individuals. However, it has been possible to detect painful zygapophysial joints using local anaesthetic blocks.

In an initial study, Aprill and Bogduk (1992) administered diagnostic blocks of the zygapophysial joints to a consecutive series of 318 patients with post-traumatic neck pain. In a worst-case analysis they found that 25% of patients suffered zygapophysial joint pain, and that amongst patients who underwent blocks the prevalence of zygapophysial joint pain was 65%. A subsequent study, using double-blind, controlled diagnostic blocks found that, amongst 50 consecutive patients with chronic neck pain after whiplash, the prevalence of zygapophysial joint pain was 54% (Barnsley et al. 1993b). Thus, despite the absence of morphological evidence of injury, there is strong physiological evidence that painful injuries to the zygapophysial joints do occur, and are common.

In essence, this model embraces two types of injury: acute muscle tears and sprains, which probably affect the majority of victims of whiplash, and which resolve favourably with the passage of time; but as well, injuries of the discs or zygapophysial joints which affect a minority of patients, and which do not resolve and become a source of chronic pain.

In terms of this model the futility of previous studies and the enigma of whiplash can be understood. In the acute phase most patients exhibit features of muscular pain, but are destined to recover. Amongst them, however, are patients with disc and zygapophysial joint injuries that cannot be seen on plain radiographs and are elusive even on CT or MRI. These injuries do not cause neurological signs and exhibit no known, pathognomonic clinical features. Consequently, they are not diagnosed or recognised. Meanwhile, these latter patients continue to suffer pain but their complaint is disbelieved, and they are even accused of malingering. As a result, they develop disease conviction, hostility, anxiety and depression.

In the case of zygapophysial joint pain, the diagnosis can be revealed if controlled, diagnostic blocks are implemented. In the case of discogenic pain, the source may be revealed by discography, but serious reservations about the reliability of cervical discography have been raised (Bogduk and Aprill 1993). Discogenic pain and any other putative cause of chronic neck pain still await the development of reliable diagnostic techniques. Until that is achieved, the comprehensive evaluation of every patient with chronic neck pain may not be possible.

13. Treatment

It should not be surprising that such a poorly understood condition as whiplash has attracted a plethora of therapeutic options. What is disappointing is the dearth of controlled trials.

The only randomised, controlled trials of treatment for whiplash injury concern the acute phase of the

injury. These trials addressed the relative roles of rest and different physiotherapy modalities. In one study comparing mobilising physiotherapy with 'standard' treatment of rest and a cervical collar, significant improvements in cervical movement and pain were noted 8 weeks after the accident in the group receiving mobilisation (Mealy et al. 1986). Subsequent studies have shown no benefit from out-patient physiotherapy when compared to a home exercise program (McKinney et al. 1989). On the other hand, a randomised trial comparing physiotherapy and traction to a cervical collar and analgesics found no difference in outcome (Pennie and Agambar 1990). The use of short-wave diathermy has been subjected to a randomised, controlled trial, which demonstrated a faster resolution of pain in the treated group, but no difference between the groups at 12 weeks (Foley-Nolan et al. 1992). Other proposed treatment modalities have included sterile water injections into alleged trigger points (Byrn et al. 1991), transcutaneous electrical nerve stimulation (Richardson and Siqueira 1981), and 'subarachnoidal injection' (Tsumura and Hoshiga 1971). Notwithstanding the absence of a clear physiological rationale for many of these treatments, there is no support for their empirical use from appropriate, randomised, controlled trials.

In light of the natural history of whiplash injury, the real value of any early treatment is unclear. The expectation would be that approximately 75% of patients will spontaneously improve in the first few months following injury. Any therapy that merely sped up this process would be of questionable efficiency. On the other hand, an intervention that prevented the development of chronic symptoms would be of exceptional value. No treatment yet assessed has demonstrated this capability.

The treatment options for the chronic whiplash patient are even less satisfactory. A recent review outlines a variety of options (Teasell et al. 1993) but none is endorsed by any form of controlled trial. Moreover, several therapies are without rational foundation, despite their apparent popularity.

Analgesics and tricyclic antidepressants may be used in a palliative sense to reduce pain, but their effect is not specific; they do not address any specific or reversible cause of pain.

There are no data to vindicate the use of exercises, physical modalities, traction, massage or manipulation for chronic pain after whiplash. Occipital nerve blocks, as conventionally performed, are neither diagnostic nor therapeutic. They are based on the mythology that somehow the greater occipital nerve can be damaged or trapped where it pierces the trapezius (Bogduk 1989). The blocks lack any target specificity if more than 0.5 ml of local anaesthetic is used, and a temporary response to local anaesthetic blocks is not an invitation for greater occipital neurectomy (Bogduk 1989). Although once popular, this operation carries the risk of anaesthesia dolorosa and painful, neuroma formation (Bogduk 1989; Teasell et al. 1993)

There is no rational basis for cervical epidural steroid injections, even though these are said to be commonly performed in whiplash patients (Teasell et al. 1993). Moreover, despite assertions to the contrary (Teasell et al. 1993) they are not without hazard (Catchlove and Braha 1984; Purkis 1986; Shulman 1986; Cicala et al. 1989; Williams et al. 1990; Tuel et al. 1990), and some studies attest to response rates less than what would be expected from placebo alone (Shulman 1986).

Soft collars do not immobilise the cervical spine (Colachis et al. 1973) and there is no evidence that collars achieve anything more than a placebo effect or a reminder to the patient not to move their neck much (Huston 1988).

For patients where an anatomical source of pain is determined, the options are only slightly better. Treatments for specific injuries to the upper cervical ligaments (Dvorak et al. 1987a), have not yet been reported. Trials of therapy for discogenic pain are hamstrung by the significant false-positive rate of cervical discography as a diagnostic test to determine entry into surgery (Bogduk and Aprill 1993). Furthermore, there are no randomised, controlled trials of surgery for cervical disc pain. The standard of reporting has been limited to proclamations by surgeons that the use of discography improves operative success rates (Kikuchi et al. 1981; Whitecloud and Seago 1987). None of the extant studies meets the editorial standards for trials of spinal surgery for the journal *Spine* (Nachemson and La Rocca 1987).

For cervical zygapophysial joint pain several investigators, on the basis of open and uncontrolled observations in small studies, have advocated intra-articular injections of corticosteroids (Dory 1983; Wedel and Wilson 1985; Dussault and Nicolet 1985; Roy et al. 1988; Hove and Gyldensted 1990). A randomised, double-blind, controlled trial, however, has shown that steroids offer no therapeutic benefit over a diagnostic block with local anaesthetic alone (Barnsley et al. 1994).

Radiofrequency denervation of painful cervical zygapophysial joints has also been advocated, but, yet again, data are limited to uncontrolled, open series (Schaerer 1978; Sluijter and Koetsveld-Baart 1980; Schaerer 1980; Sluijter and Mehta 1981; Hildebrandt and Argyrakis 1986; Schaerer 1988; Vervest and Stolker 1991; Bogduk and Barnsley 1992). However, the present authors are currently conducting a randomised, double-blind, controlled trial of percutaneous radiofrequency neurotomy of the medial branches of the cervical dorsal rami for cervical zygapophysial joint pain, the results of which should be available in 1995.

Given the lack of any grounds for the pain of whiplash to be of primary psychological origin, there is

no legitimate place for behavioural therapy as a primary modality. However, that is not to deny a plausible role for psychological therapy to address, in parallel, the psychological sequelae of chronic pain, or simply to help the patient in pain while they wait for validated therapies to be developed and implemented.

The authors might well be accused of engendering a sense of therapeutic nihilism in the context of chronic neck pain after whiplash; but there is no evidence that anything works and every likelihood that what is being used does not work. Under those circumstances it is not surprising that patients do not get better. The tragedy is that for a condition such as whiplash that is so costly in terms of personal suffering, demand for health care, litigation and the eventual impact on insurance premiums, the therapeutic armamentarium is in such a primitive state.

14. Conclusion

In motor vehicle accidents, whiplash injuries occur when the head accelerates relative to the body, resulting in excessive torque and shear being applied to the structures of the neck. This causes damage through both compression and distraction of tissues. Clinical, animal, cadaver and post-mortem studies have demonstrated that the cervical zygapophysial joints, intervertebral discs, muscles and ligaments can be seriously injured in such accidents without necessarily producing clinical or radiological signs. The majority of victims will improve spontaneously over the first few months after injury, and have probably sustained minor injury to muscles and ligaments. However, a significant proportion will have chronic and unremitting symptoms reflecting serious damage to structures such as the zygapophysial joints or intervertebral discs. These patients are likely to be older, to have more severe pain immediately after the injury and to have injury-related cognitive impairment, but there is no sound evidence to sustain the belief that psychological factors or desire for monetary gain adversely affect the outlook for whiplash patients.

In addition to neck pain, many patients report additional symptoms including headache, visual disturbances, dizziness, weakness, paraesthesiae and cognitive deficits. Although unapparent on routine clinical examination, careful, focussed investigation of these symptoms often reveals objective evidence of pathology. Furthermore, pain may be an aetiological or exacerbating factor for many of these complaints so that a single, painful lesion may account for a range of seemingly diverse symptoms. Investigation of chronically symptomatic patients should aim to determine the site of pain production using techniques such as cervical zygapophysial joint blocks and, possibly, discography. The treatment of whiplash injury is not well developed. In the early phases of the injury, mobilisation is favoured over rest. Other modalities, such as short wave diathermy, may help decrease pain. However, there is no evidence of long-term benefit from these interventions and there are no, proven, efficacious treatments for the patient with chronic neck pain after whiplash. Future studies should focus on the identification and treatment of specific anatomical lesions in this unfortunate and misunderstood group of patients.

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