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Neck sprains after car accidents

A common cause of long term disability

Sprains of the neck occur in 15-30% of car occupants examined soon after their accidents,[1 2] but in the longer term about 60% experience neck sprain.[2] Deans *et al* reported pain starting at least 12 hours after injury in 22% of patients,[2] and States found that symptoms were delayed for up to 24 hours in nearly half of those who injured their necks.[3] The incidence of neck injuries rose in Britain after it became compulsory to wear seat belts in January 1983,[4] although there were reductions in deaths and serious injuries among car occupants of around a quarter.[5]

Pain in the neck among car occupants may follow an impact from any direction, though rear impacts cause it almost twice as often as frontal collisions.[1 2] Those wearing seat belts have more neck sprains than those not wearing them.[1 2] The incidence of neck injury to front seat occupants is far higher (16%) than that for rear seat passengers (10%) in rear end collisions, suggesting that the use of seat belts is a factor, but interpretation is difficult as 40% of rear seat occupants are less than 14 years of age. Front seat passengers (19%) have a marginally higher incidence of neck sprain than drivers (15%),[1] which may be explained by preimpact bracing and the position of the steering wheel.

The likelihood of receiving a neck injury as a restrained front seat occupant in a frontal impact is unaffected by the presence of a head restraint.[1] Head restraints do, however, reduce the incidence of neck injury in rear end impacts by 10% in cars with adjustable head restraints and by 17% in cars with fixed restraints.[6] Neck pain after road traffic accidents was significantly more common in women in some studies,[1 2] but this may not mean that the female neck is more vulnerable than the male neck because most drivers are men and most front seat passengers are women. Age does not seem to affect the incidence of neck sprains.[1]

Acute sprains of the cervical spine may occur because sudden motion bends the spine.[3] The severity of these injuries depends on the relative movements of the head and the neck on the torso and the forces of acceleration.[7] Rear end, front end, or side impacts may result in sudden movements producing injuries, which doctors often indiscriminately refer to as "whiplash" injuries. In the more precise work of biomechanics the term whiplash is restricted to hyperextension motion of the neck followed by forward flexion. This type of injury usually results from rear end impacts, and only 8% of cars in collisions are struck in the rear.[8] Whiplash should not be used as a diagnosis. Rather doctors should diagnose acute neck sprain or soft tissue injury, particularly as the general public associates whiplash injuries with lasting disability and litigation.

Poor prognostic signs after injury include objective neurological signs, sharp reversal of the cervical lordosis on a radiograph,[9 10] restricted motion at one level on radiographs taken in flexion and extension, the use of a cervical collar for more than 12 weeks, and relapse necessitating a further course of physiotherapy.[9] Pre-existing degenerate changes may also worsen the prognosis.

Permanent disability occurs commonly after acute neck sprain. Deans *et al* reported that 26% of patients experienced intermittent pain in the neck one year after injury and that 4% had continuous pain.[2] Hohl reported that 43% of 146 patients followed for five years or more had important permanent disability.[9] Larder *et al* found that 59% of patients report that their injury caused some interference in their daily lives, especially with work and driving,[1] and Juhl and Seerup reported that 39% of patients took time off work or were unemployed because of their cervical injury.[11] In 1965 in a report entitled "Whiplash injuries of the neck," McNab reported to the American Association of Automobile Medicine that 45% of patients had important symptoms even two years after impact, and Norris and Watt suggested that litigation has little influence on symptoms.[10]

Pain, suffering, and disability after acute neck sprains may be reduced by doctors recognising that these injuries, especially those that occur after rear end impacts, may cause long term disability. Many patients are seen by their general practitioners or by a casualty officer who orders an *x* ray examination, prescribes a soft collar, and discharges the patient, often with the label of a whiplash injury. I prefer to prescribe a non-steroidal anti-inflammatory drug, supply a cervical collar, and review all patients in the next outpatient clinic, where many will be referred for immediate physiotherapy. Follow up is essential, and there must be good communication with general practitioners, particularly about patients returning to work. The collar may be worn day and night if symptoms are severe, though it is quickly changed to a soft collar for night wear. The patient should be weaned from a collar under the supervision of the physiotherapist.

Car design and safety development may reduce the incidence of neck sprains. Between 75% and 90% of head restraints are not adjusted correctly, nearly all being in the fully down position. This makes a strong case for fixed head restraints, but as only 8% of impacts are to the rear and 55% are frontal, head restraints more often cause injury to the faces

of rear seat occupants in frontal impacts than prevent whiplash in front seat occupants struck from behind.[12] The compulsory wearing of rear seat belts with rear seat head restraints would lessen the incidence of this and other injuries.

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High density lipoprotein and coronary heart disease

How it protects is still a mystery

In 1975 Miller and Miller emphasised the previously described, but largely neglected, inverse relation between plasma high density lipoprotein (HDL) cholesterol concentration and coronary heart disease.[1] Since then prospective studies in several countries have confirmed this relation and found it independent of other risk factors (p 998).[2-5] Although much is now known of the role of HDL in lipid transport,[6-8] how it protects against atherogenesis is not understood.

An attractive explanation depends on the participation of HDL in reverse cholesterol transport, whereby cholesterol returns from peripheral tissues to the liver, the major site of cholesterol excretion.[9] In this process HDL may accept cellular unesterified cholesterol by interacting with specific cell surface receptors, which reversibly bind HDL and regulate cholesterol transport out of the cell.[10] Low plasma HDL concentrations might therefore mean inefficient reverse cholesterol transport, explaining the inverse relation between plasma HDL concentration and atherosclerosis. Other explanations do not entail HDL in such a direct role; for example, high plasma HDL concentrations may reflect efficient catabolism of triglyceride rich lipoproteins (including remnant particles), which are atherogenic.[8] The relation of HDL to atherogenesis might be mediated through effects on thrombotic factors such as platelet aggregation, prostacyclin production, and fibrinolysis.[11-13]

As well as providing an additional assessment of the risk of vascular disease, measuring the plasma HDL concentration permits the calculation of plasma low density lipoprotein (LDL) cholesterol concentration, the major atherogenic particle, by the Friedewald formula (plasma LDL cholesterol concentration (mmol/l)=plasma total cholesterol concentration−plasma HDL cholesterol concentration−plasma triglyceride concentration/2·19).[14]

Some studies have emphasised the predictive power of the ratio of the plasma concentration of LDL to HDL in risk assessment.[2] Knowledge of the plasma HDL concentration may affect clinical management, subjects with a slightly increased plasma total cholesterol concentration due solely to an increased plasma HDL cholesterol concentration do not need treatment. Although the relation of hypertriglyceridaemia to coronary heart disease is not clear, hypertriglyceridaemia in the presence of low plasma HDL cholesterol concentration is possibly associated with an increased risk of vascular disease.[15] (In subjects with a pronounced family history of vascular disease without an appreciably increased plasma total cholesterol concentration the plasma HDL cholesterol concentration may be low (<0·9 mmol/l), suggesting familial hypo-α-lipoproteinaemia.[16]

The main aim of treating hyperlipoproteinaemia is to reduce the LDL cholesterol concentration.[17,18] Current evidence is insufficient to argue for a causal role for a low plasma HDL cholesterol concentration, although the Helsinki heart study suggested an independent benefit of increasing plasma HDL cholesterol concentration by drug treatment in preventing coronary heart disease.[19] In addition, a substantial increase in the concentration of serum HDL cholesterol (together with a reduction in that of LDL cholesterol) followed pharmacological intervention in the cholesterol lowering atherosclerosis study, which resulted in reduced progression of atherosclerotic plaques and their regression in an appreciable number of patients.[20]

What then is a reasonable clinical approach to take? Plasma HDL cholesterol concentration tends to be low in overweight subjects, cigarette smokers, and sedentary subjects[21] and correcting these factors will tend to increase plasma HDL concentration. Drugs, particularly thiazide diuretics and non-selective β adrenergic blocking agents, may lower plasma HDL cholesterol concentrations, and alternatives should be considered for patients with hyperlipoproteinaemia.[22] It seems reasonable to adopt a more aggressive approach to lowering plasma LDL cholesterol concentration in the presence of a low plasma HDL cholesterol concentration and to attach more importance to moderate degrees of hypertriglyceridaemia when this is associated with a low plasma HDL cholesterol concentration. Much, however, remains unknown; for example, will increasing the plasma concentration of HDL cholesterol alone protect against coronary heart disease? Some hypolipidaemic drugs, particularly the fibrates and nicotinic acid, increase plasma HDL cholesterol concentration, but so too does alcohol. Given the complicated metabolism of HDL, it seems unlikely that all interventions that increase the concentration of plasma HDL cholesterol will affect the process or processes by which it protects against atherosclerosis.

Until we know more, measuring the concentration of plasma HDL cholesterol as part of the fasting lipid profile provides a more comprehensive assessment of the risk of vascular disease and a sounder basis for making therapeutic