NOSOLOGICAL ENTITIES?

The late whiplash syndrome: a biopsychosocial approach

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Abstract
Physicians and other therapists continue to grapple in daily practice with the controversies of the late whiplash syndrome. For decades much of the debate and the approach to this controversial syndrome has centred on the natural history of and progression to chronic pain after acute whiplash injury. Recognising that there is recent epidemiological data that defines the natural history of the acute whiplash injury outside of many of the confounding factors occurring in many western countries, and the lack of evidence for a "chronic whiplash injury", this article will thus introduce the biopsychosocial model, its elements, its advantages over the traditional model, and the practical application of this model. The biopsychosocial model recognises physical and psychological souces of somatic symptoms, but fundamentally recognises that the late whiplash syndrome is not the result of a "chronic injury".
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The authors of the 1995 monograph of the Quebec Task Force on Whiplash-Associated Disorders,[1] after reviewing over 10 000 publications, could identify no acceptable study as a suitable source for understanding the natural history and prognosis of the acute whiplash injury. The only studies available at the time were those using highly selected patient populations, without control groups and all performed in countries where there exists a multitude of confounding factors including expectation of disability, effects of intervention by the therapeutic community, and possibilities for secondary gain. Typical examples include the Swiss study by Radanov *et al*, conducted almost a decade ago and published many times since.[2] In this study, patients with whiplash were recruited after a car collision, but only if they first sought contact with their primary care physician, and were then recruited as a non-consecutive cohort selectively achieved through general advertisement in the *Swiss Medical Journal*. The number of subjects and characteristics of subjects who chose not to enter the study were not known.[3] No control population was utilised, and no consideration was given for the fact that the Swiss system encourages payments for reporting disability and time lost from work, and even if a patient returns to work, they can be compensated for not returning to full time work or having the potential for long term economic loss. The Swiss-type system (no fault system) may be less harmful to outcome than a tort system, as a tort system has been recently shown to delay recovery,[4] but even a no fault system is associated with compensation factors, and is not the best setting for prognostic or other studies of the natural history of the acute whiplash injury. Indeed, no controlled study outside the medicolegal context or studies avoiding other confounding factors in countries were the late whiplash syndrome is epidemic were identified by the Quebec Task Force, nor was the obvious necessity for such studies mentioned. The authors recommended, however, that prognostic studies be performed to determine the risk factors and the influence of compensation incentives such as that seen in Switzerland and other western countries. Since the Quebec Task Force, prognostic studies have been performed in Lithuania, Greece, and Germany.[5–10] These studies were largely free of the problems involved in the Swiss efforts and other limited evidence previously available for understanding the natural history of the acute whiplash injury.

Lithuania

Lithuania is a country in which there is no or little awareness or experience among the general population of the notion that a whiplash injury may cause chronic pain and disability. Collision victims view this as a benign injury not requiring any medical attention. Possibilities for secondary gains are minimal. In a controlled historical inception cohort study published in 1996,[5] none of the 202 subjects involved in a rear end car collision 1–3 years earlier had persistent and disabling complaints that could conceivably be linked to the collision. There were no significant differences between the collision victims and controls concerning prevalence of symptoms including

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neck pain, headache, and subjective cognitive dysfunction. In a later prospective controlled inception cohort study,[6] 47% of 210 victims of rear end car collision consecutively identified from the daily records of the traffic police had initial pain. The symptoms disappeared in most cases after a few days. No subject reported collision induced pain later than 3 weeks. After 1 year, there were no significant differences between the collision victim group and the control group concerning frequency and intensity of both neck pain and headache. In the historical cohort study,[5] 31 collision victims recalled having had acute or subacute neck pain. This symptom lasted in most cases less than a week and only two subjects had neck pain for more than 1 month. Due to recall problems, the true incidence of collision victims with acute symptoms such as neck pain or headache was unknown. The study has later been criticised for having insufficient power.[11–13] The authors of the criticism, however, confused the incidence of an acute whiplash injury—that is, the at risk population for chronic symptoms, with the number of collision victims who remembered having had neck pain shortly after the collision. According to the prospective study performed in a comparable inception cohort, the 95% confidence limits for the true incidence of acute symptoms were 40% and 54% giving an estimated minimum of altogether about 180 subjects with acute whiplash injury in both studies. As none of the collision victims seemed to have developed persistent and disabling symptoms due to the collision, the studies either evaluated alone or together have sufficient power to reject estimates of the incidence of the so-called late whiplash syndrome in previous, methodologically inferior studies and to question the validity of the condition as a chronic physical injury.

Greece

The late whiplash syndrome seems to also be a rare event in Greece. Of 130 consecutive collision victims, all had acute whiplash injury, 91% recovered in 4 weeks, the remainder having substantial improvement to the point where their frequency of neck pain was similar to the general population, and indeed recovering within 3 months.[7] Extending this data to 180 patients confirmed this result, not only for recovery from neck pain, but from the other symptoms commonly reported as part of the acute injury syndrome.[8]

Germany

The prognosis of acute whiplash injury is also remarkably good in Germany, a country where there is widespread awareness of the possibilities of acute symptoms after whiplash in the general population, but little expectation of chronic disability. In a study of physiotherapy treatment, by 6 weeks the active treatment group and control (healthy) groups were equal in their symptom reporting. Even the group given only a collar for 3 weeks and no other therapy recovered by 12 weeks. That is, the acute whiplash injury does not seem to confer a greater risk of reporting chronic symptoms than found in the general, uninjured population.[9] A prospective outcome study by Keidel *et al* of 103 subjects in another locale in Germany found the same good prognosis, recovery often within 3 weeks, and virtually all within 6 weeks.[10]

Experimental and other voluntary collisions

Experiences from experimental collisions, fairground bumper car driving, and car crashing contests also question the validity of a "chronic injury" model of the late whiplash syndrome. Despite being able to readily produce acute symptoms thousands of experimental collisions with volunteers have failed to produce a patient with chronic symtoms. This is despite the use of various vehicles, impact directions and speeds, restraint systems, with or without head rests, with varying head inclinations and rotation, with or without tensed neck muscles, and more recently with a wide range of young and old, both sexes, non-military volunteers (for a comprehensive review see Ferrari[14]). The collisions experienced in the fairground bumper cars have been shown to be of similar velocity changes to many apparently symptom-provoking rear end collisions with automobiles. Yet chronic symptoms are not reported.[15] In studies of drivers in car crash contests or demolition derbys, which bests replicate accidental whiplash injury, none of the drivers reported chronic disabling symptoms despite the fact that the drivers had an average career total of many hundred collisions.[16 17]

Facet joint studies

Zygapophysial joint pain has been claimed to be the most common basis for chronic neck pain as a result of whiplash injury.[18] In one study, 39 people with chronic neck pain were investigated. Five of the 39 had not been in motor vehicle accidents, but apparently had had neck injuries in other types of accidents. Two of 39 claimed that their chronic pain began 3 months after an accident. Some of the accidents took place 44, 27, and 21 years before entering the study. Those in motor vehicle accidents are reported to have experienced high speed collisions, far higher than most whiplash victims.[19] Looking for a cause of current neck pain in these subjects, the investigators found that the facet joint or nearby structures could be a source for current neck pain in some members of this highly select, heterogeneous, non-representative group of what they arbitrarily called "whiplash patients". This indicates only that neck pain in some cases may have a current physical cause. The results do not confirm that the current cause is also a past cause of the neck pain, or has been for, say, the last 44 years. The results tell us nothing about the injury (if there was one) in these subjects and nothing about whether an acute injury can develop into a chronic physical source of pain. It is difficult to exclude that a very small proportion of subjects could have chronic structural damage in countries such as Lithuania, and that current studies with background prevalence of chronic neck pain in the control

population of about 10% are not large enough to distinguish an additional 2%-3%. Yet these additional patients are not the group of greatest concern. It is the high percentage of patients with chronic accident-attributed pain (50% in Canada[4] and 58% in Norway[20]) that provide the greatest health care and economic burden, and facet joint studies are irrelevant to this larger group.

The facet joint studies illustrate that patients, and the researchers, are prepared to carry or place the label of "whiplash patient" on anyone who wants to attribute their chronic neck pain to an accident. What cultural factors promote this non-scientific decision to make such an attribution? Why can it be assumed that a current cause of neck pain has any relation to an accident 44 years ago? That such assumptions were made is the greatest revelation of the facet joint research. Physical sources of pain can and do exist, but it is how people interpret the significance of that pain in relation to other events that creates the problem. If these subjects attribute their neck pain to an accident, then they are "whiplash patients". If they choose to dismiss the attribution, then they are not "whiplash patients"; the label has such a limited and flimsy a basis that it can be, on a mere whim, dismissed or clung to passionately.

Thus, less research is needed at trying to pinpoint an anatomical source for pain, and more research at trying to find the cultural source for behaviour in response to an acute pain—a simple neck sprain.

The need for a biopsychosocial model

The need for the biopsychosocial model arises primarily out of the epidemiology of the late whiplash syndrome. By showing that the prevalence of chronic symptoms after whiplash is of the same order of magnitude as in the general population, and indeed also after whiplash in other countries,[21] the studies in Lithuania, Greece, and Germany were the first to effectively document that both acute and chronic symptoms are genuine; although, the issue arises as to the extent that chronic symptoms may be related to the accident injury. The issue thus remains how the acute injury is perceived to evolve into chronic pain in some countries and not in others. In view of the above evidence, it cannot simply be assumed that the progression to chronic pain is a result of malingering or psychosomatic disorder in most patients (although exaggeration of symptoms, underperformance in neuropsychological testing, and underreporting of pre-accident symptoms may of course occur[22–24]). There is until now no convincing evidence of a specific neck injury that can be expected to commonly cause chronic damage in the neck and continue to generate chronic pain or other chronic symptoms of the late whiplash syndrome.[14 22 25 26] Notwithstanding these observations, given that in Lithuania, Greece, and Germany the acute whiplash injury commonly occurs with pain resolving within 6 weeks, the exact pathology of most acute whiplash injuries may be largely irrelevant. From engineering studies, radiological studies, and traumatological principles, the acute injury is in most cases a muscle or ligament sprain.[14 22 26] This may well vary, but it is less relevant than the fact that the outcome of the acute injury is invariably benign in some countries, and invariably leads to epidemic proportions of chronic pain beyond 6 weeks in other countries. For the better understanding of the epidemic of the late whiplash syndrome in western countries, it seems neccessary to consider biological, psychological, and social factors together by a biopsychosocial approach.

Elements in a biopsychosocial model

Whereas it can be accepted that some aspect of the symptoms these patients report arise as the somatic component of depression or anxiety disorder, it is equally reasonable that many of the symptoms have physical sources. The fact is that many of these same symptoms of patients with whiplash, with often unidentifiable causes, often occur in normal people.[27] That being the case, there is a substrate (symptom pool) immediately available, on which psychosocial factors may act, and this leads to further behaviours that become "the illness". Thus the first factor of the biopsychosocial model is that there is a general symptom pool that includes headache, neck pain, back pain, numbness, fatigue, dizziness, joint or limb aches and pains, limb stiffness, poor concentration, poor hearing, and sleep disturbance.[27] Yet the cause of these symptoms, even though at least some would be presumed to have a physical basis in the healthy person, is largely unknown.

We have the strong possibility that the symptoms of the late whiplash syndrome arise from multiple sources (including physical ones), and the more relevant aspect of the psychosocial factors (or psychological distress) is that they act on this substrate.

The first question is then how are these symptoms perceived and acted on differently in patients with whiplash than in healthy people? The second question is how does this maladaptive behaviour create new sources of symptoms? This brings us to the other factors operative in the biopsychosocial model—symptom expectation, amplification, and attribution.

Expectation, amplification, and attribution

In North America, as in many other countries including Norway, there is overwhelming information on the potential for chronic pain outcomes after whiplash injury, with widespread knowledge of the expected symptoms even among people with no personal experience of having a collision.[28 29] This expectation will in turn lead the person to become hypervigilant for symptoms, to register normal bodily sensations as abnormal, and to react to bodily sensations with affect and cognitions that intensify them and make them more alarming, ominous, and disturbing—symptom amplification. It is noteworthy that in countries such as Lithuania, Germany, and Greece, where again the late whiplash syndrome is rare,

recent studies using the methodology of Aubrey *et al*[28] and Mittenberg *et al*[29] in those countries found a lack of expectation of chronic symptoms—the whiplash injury is viewed as benign (R Ferrari, unpublished data, 2000).

The circumstances of the collision immediately create an impression that the minor injury is not benign. The patient's fear may start when paramedics take him out of the car on a special stretcher, apply a hard collar, and warn him not to move. Symptoms are intensified when they are attributed to a serious disease than to more benign causes such as lack of sleep, lack of exercise, or overwork. This is not to say that it is the psychological trauma of the accident event that is operative, but rather of the perceived nature of the injury. In Lithuania, Greece, and Germany the accident itself, as a threat to existence in general, would be expected to, even there, have a certain degree of psychological impact, as it would in any country; yet despite this, there is a lack of chronic pain as a result in Lithuania, Greece, and Germany. This suggests that psychological trauma is not likely an independent or substantial factor in the progression from acute to chronic pain.

Another aspect of symptom amplification occurs when others have the collision victim repeatedly draw attention to the symptoms (every time the patient sees a therapist, or is asked to keep a diary of symptoms, etc). Attention to a symptom amplifies it, whereas distractions diminish it. Thus the more often patients are asked to rate their pain, the more intense they rate it.

This symptom expectation and amplification may cooperate to alter a collision victim's behaviour in a detrimental way. Feeling severe pain and fearing future disability, they develop the cognitions and behaviours that lead to withdrawal from activities after minor injury, and, for example, develop maladaptive postures. Yet it is known, for example, that postural abnormalities, if induced in healthy subjects, cause pain.[14] The patients with whiplash, in response to their heightened pain and their anxiety have just created a new source of pain—and a physical source at that. This new source forms a further part of the substrate on which symptom amplification can act—the patients not realising that they have a new source of pain, but instead they feel they have a "chronic injury"—such was their expectation. Psychosocial factors ultimately generate, in this example, a physical source for pain. Another example of what this behaviour does includes the use of medications. The patient, experiencing amplified and fearful symptoms, seeks medications. Yet, the medications commonly used for pain have as their adverse effects dizziness, cognitive disturbance, etc, a new physiological source for symptoms that the patient will be informed (from what they have read or from the input of their therapist) is part and parcel of their injury effects. This new source of symptoms is there to be amplified, there to be attributed to a "chronic injury", and arises because of the initial behaviour of the collision victim and those in their environment.

The final factor of this triad is thus symptom attribution. As a collision victim becomes hypervigilant for symptoms, and as the victim may expect chronic symptoms, the problem of symptom attribution is a natural result. In the setting of amplification, previously unintrusive symptoms, largely ignored in daily life, become far more intrusive after the collision. The patient regards them as new (they are now being registered), and attributes them to the collision. The symptom pool for new symptoms is drawn on while the acute injury resolves. The pool arises from life's aches and pains, occupational sources, symptoms from medication use, and potentially the symptoms that arise from maladaptive postures and changes in physical fitness that arise as patients withdraw from normal activities. It is true that it is expected that these various benign, physical sources would not be capable of causing severe or significant pain (and they likely did not in the past for the patient), but that is the effect of symptom amplification, to alter the naturally benign appearance of the symptoms. A biopsychosocial model is therefore not a "psychogenic model"—that is, a model which assumes that the chronic pain has no physical basis, but is merely the somatic expression of psychological disorder. The biopsychosocial model instead suggests that what the patient expects, how they perceive symptoms, and how they focus and attribute symptoms will in turn alter the character of those symptoms and the patient's behaviour, and that the symptoms have various physical sources in some cases.. Following this, entirely new physical problems may arise to contribute to the symptom pool. Add whatever further contribution is made by anxiety, depression, and compensation systems, and the late whiplash syndrome evolves.

Summary

The late whiplash syndrome is not merely psychosomatic. At the same time, it is not the result of a "chronic injury. The biopsychosocial model that considers an effect of cultural expectation, cultural factors that generate symptom amplification and attribution, as well as the possibility that physical and psychological causes for symptoms coexist seems more helpful. It negates the concept of "chronic injury", but at the same time takes away the stigmata of the psychiatric label, while explaining that people's behaviour in response to their injury may generate much of the illness, and therefore the illness is not an incurable injury.

The psychosocial elements, which may amplify otherwise benign bodily symptoms, or transform a minor injury into one that is viewed as serious and generate anxiety, may set in motion the phenomenon of symptom expectation and amplification. These processes eventually lead a person to attribute new and even previous symptoms to a "chronic injury". This reattribution then further amplifies the symptoms themselves, as they now take on a different significance, and become more intense, noxious, and worrisome. The concern that a person is seriously injured, together with medical scrutiny, and media induced attention

to the latest syndrome, may corroborate that person's fears. Changes in behaviour because of these fears and the influences of others, may in turn generate whole new physical problems, generating more symptoms, and a self validating and self perpetuating cycle of symptom amplification and disease conviction ensues.[27] Physicians and other therapists, if they hope to assist patients in altering that behaviour need to be compassionate, recognise the validity of the symptoms, recognise that they may have various physical causes, and be able to communicate to patients that the various chronic symptoms often arise out of the steps the patients take in response to the initial problem. The next steps in research are best directed at identifying the biopsychosocial elements and how they account for the variance in outcomes, so that we can then inform the whiplash cultures plagued by the late whiplash syndrome from whence their suffering arises. We can also use the biopsychosocial model to develop education and treatment approaches that address the psychosocial factors causing the adverse outcomes.[27–30]

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