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 sources of somatic symptoms, but fundamentally recognises that the late whiplash syndrome is not the result of a “chronic injury”. (J Neurol Neurosurg Psychiatry 2001;70:722–726)

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The authors of the 1995 monograph of the Quebec Task Force on Whiplash-Associated Disorders, 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. 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. 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, 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 medi-colegal 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. 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, 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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alone or together have su-
due to the collision, the studies either evaluated
developed persistent and disabling symptoms
of the collision victims seemed to have
minimum of altogether about 180 subjects with
inception cohort, the 95% confidence lim-
the prospective study performed in a compara-
pain shortly after the collision. According to
chronic symptoms, with the number of colli-
lash injury—that is, the at risk population for
ever, confused the incidence of an acute whip-
a greater risk of reporting chronic symptoms
therapy recovered by 12 weeks. That is, the
given only a collar for 3 weeks and no other
treatment, by 6 weeks the active treatment
in their symptom reporting. Even the group
control (healthy) groups were equal
general population, but little expectation of
remarkably good in Germany, a country where
The prognosis of acute whiplash injury is also
rare event in Greece. Of 130 consecutive colli-
sion 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. 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.

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. 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 vehi-
cle accidents are reported to have experienced
high speed collisions, far higher than most
whiplash victims. Looking for a cause of cur-
tent neck pain in these subjects, the investiga-
tors found that the facet joint or nearby struc-
tures could be a source for current neck pain in
some members of this highly select, heteroge-
neous, 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 Lithua-
nia, 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 and 58% in Norway) 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.

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. 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. 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. 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. and Mittenberg et al. 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. 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 seem more helpful. It negates the concept of “chronic injury”, but at the same time takes away the stigma of the psychiatric label, while explaining that people’s behaviour in response to their injury may generate much of the illness, 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 they may have 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.”

12 Freeman MD, Croft AC. Late whiplash syndrome. Lancet 1996;348:125.
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