Ontario

Headache

Discussion paper prepared for

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Dr. John Edmeads graduated from the Faculty of Medicine of the University of Toronto in 1959. He pursued post-graduate studies in neurology and became a Fellow of the Royal College of Physicians and Surgeons in 1965. He served the College as Chief Examiner in neurology from 1981 to 1983. He joined the University of Toronto faculty in 1967 and became Professor of Medicine in 1984. He led research in cerebral blood flow, chronic headache and medical education. He received numerous awards for his work and teaching in these areas. Dr. Edmeads was Head of Neurology at Sunnybrook Health Science Center from 1969 to 1993 and Head of the Department of Medicine and Physician in Chief at Sunnybrook Health Science Center from 1994 to 2001. Dr. Edmeads died in April 2007.

WSIAT literature search reviewed by Dr. D. Rowed in 2010, who is of the opinion that this paper still provides a balanced overview of the medical knowledge in this area.

This medical discussion paper will be useful to those seeking general information about the medical issue involved. It is intended to provide a broad and general overview of a medical topic that is frequently considered in Tribunal appeals.

Each medical discussion paper is written by a recognized expert in the field, who has been recommended by the Tribunal's medical counsellors. Each author is asked to present a balanced view of the current medical knowledge on the topic. Discussion papers are not peer reviewed. They are written to be understood by lay individuals.

Discussion papers do not necessarily represent the views of the Tribunal. A vice-chair or panel may consider and rely on the medical information provided in the discussion paper, but the Tribunal is not bound by an opinion expressed in a discussion paper in any particular case. Every Tribunal decision must be based on the facts of the particular appeal. Tribunal adjudicators recognize that it is always open to the parties to an appeal to rely on or to distinguish a medical discussion paper, and to challenge it with alternative evidence: see *Kamara v. Ontario (Workplace Safety and Insurance Appeals Tribunal)* [2009] O.J. No. 2080 (Ont Div Court).

HEADACHE

Headache is not a disease. It is a symptom, with many causes, mostly benign (like migraine) and rarely ominous (like brain tumor). This discussion will focus on headaches encountered in the setting of the workplace - headaches associated with traumatic injury to the head and/or neck, and headaches related to exposure to toxic substances.

As prologue, it is important to become familiar with the causes of headaches in general, and with the benign dysfunctional headaches (such as migraine and tension-type headaches) that so many people have as a result of hereditary, stress, or idiopathic (ie unknown) factors.

In general, what causes headaches?

Headaches occur when pain-sensitive structures in the head (and sometimes neck) are disturbed by malfunction or disease. The pain-sensitive structures of the head and neck, and the things that may irritate them, are shown in the table. Note that the brain tissue itself is *not* sensitive to pain, but its coverings (the meninges) and the blood vessels that supply it, are. The coverings of the skull (scalp tissue, muscle, nerves and blood vessels), the eyes, the sinuses, the teeth, and the jaw joints, are all sensitive to pain.

Structure	Stimulus	Type of Headache (h/a)
Scalp & neck muscles Scalp nerves	Idiopathic* contraction Traumatic injury Traumatic injury	**Tension-type headaches** *Post-traumatic tension h/a's* *Site of injury head pains*
Joints & ligaments of neck	Traumatic injury	*"Whiplash headaches"*
Blood vessels of scalp	Idiopathic* dilatation	**Migraine headaches**
Blood vessels inside head	Idiopathic* dilatation Chemicals → dilatation Trauma → dilatation Displacement/stretching	**Migraine headaches** *Toxic headaches* *Post-traumatic migraine* Brain tumor, hematoma
Coverings of brain (meninges)	Infection Inflammation (by blood)	Meningitis Bleeding into brain
Nasal sinuses	Infection, inflammation	Headaches of sinusitis
Jaw joints and muscles	Trauma, "wear & tear"	"TMJ syndrome"

Headache types in **bold** are the common **benign dysfunctional headaches**. These are *idiopathic - ie no one really knows what causes them, but a complex interplay of hereditary, situational and stress factors seems to be involved in most people. Headache types shown in *italics* are the *post-traumatic* or *toxic* headaches that are the major focus of this review.

Headache “entities” enclosed in “quotation marks” are controversial, with dispute about their frequency, their cause, and sometimes their existence (see text).

Migraine, though usually a **benign dysfunctional** entity, may be aggravated or precipitated by *trauma* to the head and perhaps the neck.

The benign dysfunctional headaches

These are discussed first because they are so common that there may be difficulty sorting them out from occupationally related headaches.

Why are they termed “benign dysfunctional” headaches? “Benign” speaks to the fact that the great majority of these headaches are nuisances rather than disabilities; they hardly ever produce long term inability to function; and they are never fatal. “Dysfunctional” means that the headaches are caused by intermittent malfunctioning of structurally normal tissues (such as, for example, the blood vessels of the head in migraine), and not by disease.

“Prevalence” denotes the number of people in a population who have a condition at any designated point in time. General population surveys have shown the prevalence of **tension-type headaches** in Canada to be 37% of females and 21% of males; in the U.S.A it is 42% of females and 36% of males, and in the U.K it is 35% of females and 29% of males. The differences reflect different techniques of data gathering and are inconsequential. The important thing is that these headaches are very common. Similarly, the prevalence of **migraine** in Canada is 23% of females and 9% of males; and in the U.S.A is 19% of females and 8% of males.

Tension headaches were given that name because at one time it was widely believed that they were caused by increased tension (contraction) of the muscles of the scalp and neck, which in turn was produced by increased emotional tension. An equivalent term was “muscle contraction headaches”. The concept has fallen into disfavor because recordings of scalp and neck muscle activity in people with these headaches have not always shown increased muscle contraction. Few experienced clinicians would dispute that emotional tension (worry, anxiety, depression) plays a major role in producing these headaches, and the current hypothesis is that the head pain receiving centre of the brain (the nucleus caudalis trigeminalis) has had its sensitivity “turned up high” by increased activity of those higher centers of the brain which mediate stress, so that ordinarily innocuous stimuli are perceived as uncomfortable or painful. To accommodate the controversy, the International Headache Society coined the noncommittal term “tension-*type* headaches”.

Tension-type headaches are dull, low grade aches which involve both sides of the head and may spread down into the neck. They may be mostly in the front, or mostly in the back of the head, or may invest the whole scalp, like a tight cap. The headaches are worse with anxiety but not usually with activity; indeed, many people with tension-type headaches will take a walk or do some other activity to divert themselves. Over-the-counter pain killers such as aspirin, Tylenol®, etc, usually bring some relief. Unlike migraine, there are no accompaniments to the headache such as nausea, vomiting, visual disturbance, or dizziness. The headaches typically last a half-hour to a day or two, and while most people have them only occasionally, a few may have them every day or almost every day; these people (whether as cause or effect) usually have significant psychological disturbances, such as depression.

Tension-type headaches are pretty nondescript. Having the 'flu, or an adverse reaction to a medication, or a brain tumor, can all produce a headache with similar features. Therefore, while often the entire picture (age, patient's circumstances and behavior, the general medical history, and the findings of a careful physical examination) makes this diagnosis evident, occasionally some special laboratory or neuroimaging tests (for example, CT scan) will be necessary before the prudent physician feels confident in diagnosing tension-type headaches.

Migraine, in contrast, exhibits a very specific pattern. The International Headache Society has formulated, validated and published clinical diagnostic criteria for migraine which are now the gold standard. These criteria stipulate that migraine is a recurrent headache, lasting a few hours to a few days, which has at least two of the following four features:

- involves one side (left or right) of the head
- is throbbing
- is moderate to severe in intensity
- is worse with usual types of activity

and which has at least one of the following two accompaniments:

- nausea and/or vomiting
- aversion to noise and light (phonophotophobia)

and occurs in a patient who has nothing on history or examination to suggest another cause for the headaches.

If a headache fulfills these criteria, then it is migraine, and special tests are unnecessary for diagnosis (though they are frequently done for reassurance).

About one migraine patient in seven has an aura with some or all headaches, This aura, typically, is a visual disturbance that precedes the headache, lasts 20 to 30 minutes, and then clears as the headache appears. Sometimes there may be, as part of the aura, numbness or tingling of one side of the face and of the limbs on one side of the body; very rarely there may be weakness of the limbs. Many patients (and a few uninformed physicians) think that if there isn't an aura, then the headache can't be migraine; this is incorrect.

Migraine is a genetic disorder. More often than not, there is a history of migraine in a "first order relative" (parent, sibling, child). The headaches arise from episodic dilatation of the blood vessels of the head, both in the scalp and on the surface of the brain. The brain is believed to be the major driver of these vessel changes. Some triggers of migraine such as emotional stress, fatigue, exposure to certain frequencies of light, and likely the hormonal changes of the menstrual cycle, appear to work through the brain. Other migraine triggers, such as exposure to some substances in foods or in the environment, likely work through a blood-borne effect on the cranial blood vessels.

Migraine is most frequently treated with analgesics (pain killers), which is unfortunate for two reasons. First, these often aren't very effective and so the overuse of analgesics (particularly analgesics containing caffeine and/or codeine) resulting from vain attempts to get relief with them can itself produce chronic "rebound" headaches. Second, there are specific medications (the triptans) available which are much more effective in terminating the acute attack of migraine, and other medications are available for reducing the frequency of migraine attacks (migraine prophylactics). The expense of the triptans prevents many from using them, and the education of physicians regarding the wider use of migraine prophylaxis is a work in progress.

Migraine and tension-type headaches in the workplace

Many people complain to their physicians that things in their workplaces trigger, intensify, or generally aggravate their pre-existent headaches. Women are more likely to make this observation because, as noted above, they are more likely than men to have these headaches in the first place.

Emotional stress in the workplace can worsen both migraine and tension-type headaches, making them more frequent and sometimes more intense. Ergonomic factors, such as seating, desk height, cradling a telephone, etc can produce muscular stresses and strains that, in an individual subject to tension-type headaches, may trigger them. Some migraine patients feel that

hours spent in front of a video display terminal precipitate and aggravate their attacks. Some migraine patients complain bitterly that the after-shave or perfume worn by their immediate co-workers triggers attacks. Some migraine sufferers implicate fumes, smoke, or just “poor ventilation” in the workplace. There is some scientific basis for all of these attributions.

Once triggered by an environmental factor, the migraine or tension-type headache will run its usual course, lasting anywhere from ½ hour (in the case of a tension-type headache) or a few hours (in the case of a migraine) to a few days, just like those not triggered by the environment. Identification and removal of the environmental trigger should result in the cessation of occurrence of the headaches. If the headaches do not stop occurring in the workplace once those factors blamed by the headache sufferer have been adequately addressed, then it becomes overwhelmingly probable that the headache sufferer was wrong, and that those factors never did play a part in causing the headaches.

Post-traumatic headaches

The headaches following trauma, in contrast, tend to be much more refractory, and are a frequent source of disability and compensation claims. Head injury itself is remarkably common, being reported in about two million people in the USA every year (about 1% of the population). Forty-five percent of these injuries are due to motor vehicle accidents, thirty percent to falls, twenty percent to occupational and recreational accidents, and the remainder to miscellaneous causes including violence. Eighty percent of these head injuries are deemed mild, ten percent moderate, and ten percent severe, using as an index of severity the conventional criterion of duration of post-traumatic amnesia (the length of time after the injury for which the person has no memory). The incidence (the number of people in a population who *develop* a condition within a given time span) of post-traumatic headaches is highly variable, depending on the make-up of the population studied, and ranges from 30 to 70%. Older people are more likely to develop post-traumatic headaches, as are people who have had previous benign dysfunctional headaches such as migraine or tension-type headaches, and women. There is a paradoxical and controversial (see page 10) *inverse* relationship between the severity of the head injury and the likelihood of developing post-traumatic headaches. Some studies have shown that in societies (for example, a former Iron Curtain country) which do not have traditions of compensation for headaches or of private litigation, there is a much lower incidence of post-traumatic headaches than in neighboring NATO countries or in North America; these studies have provoked much emotionally-charged debate (see page 8 for a discussion of the controversy about physical versus psychological factors).

Injury to the neck, especially the so-called “whiplash” injury in which, as a result usually of a rear end collision in a motor vehicle, the head suddenly is propelled backward and forward, has been implicated as a cause of headache. Both head injury and whiplash-type neck injuries may be followed not only by headache, but by other symptoms including dizziness, imbalance, vertigo (a form of spinning dizziness, often precipitated by changes in posture), blurred vision, ringing in the ears, sensitivity to noise and bright light, fatigue, difficulty with concentration and memory, sexual dysfunction and personality changes such as irritability and depression. This concatenation of symptoms is called the “post-traumatic syndrome” generically, the “post-concussion syndrome” when it follows head injury, and the “whiplash syndrome” when it follows a neck injury.

These are the kinds of headaches that have been described following head or neck injury:

1. **Headaches resembling *tension-type headaches.*** These are the most common kind of post-traumatic headaches. They can come on after trauma to either head or neck. Like the common benign dysfunctional tension-type headaches experienced by many non-injured people, these are dull, not very intense, heavy, pressure-like, aching discomforts involving both sides of the head and often the back of the neck. Unlike most benign dysfunctional tension-type headaches, however, the post-traumatic headaches are persistent and often constant; they are worsened by activity and exertion; and they are often accompanied by some or all of the other symptoms of the post-traumatic syndrome (eg. dizziness, fatigue, difficulty with concentration, etc.). These headaches, and the accompanying symptoms, are usually resistant to therapy. Simple analgesics seldom take the discomfort completely away, as they do in benign dysfunctional headaches, and over-use of analgesics may complicate and perpetuate the problem by causing rebound headaches. Benign dysfunctional tension-type headaches, if frequent, often respond well to amitriptyline - a medication originally introduced as an antidepressant and subsequently found to be useful, even in those not depressed, for chronic headaches when taken every day. Post-traumatic tension-type headaches also respond to amitriptyline, but not usually as well, and usually only if started early. On the premise that scalp and neck muscle contraction might be contributing to these headaches, muscle relaxant medications such as carisprodol, tizanidine, baclofen, dantrolene etc have been prescribed, but not with great success; physical measures aimed at relaxing muscle such as massage, stretching, heat, etc usually provide some transient benefit. These post-traumatic tension-type headaches (and the closely related cervicogenic headaches) are the ones that are associated with the most disability.

2. ***Cervicogenic headaches*** are so called because of the notion that they come from the cervical spine (neck). They are almost identical to the post-traumatic tension-type headaches described above, except that the discomfort is perceived as starting in the neck and being maximal in the back of the head, and that the headache may be aggravated by neck movements. The term is more likely to be applied if the preceding event was a whiplash injury rather than a blow to the head. Likely, these two headaches are the same. Certainly, both are chronic and often constant, both tend to be associated with a host of other symptoms, and both are usually refractory to treatment. In addition to the therapy noted above for tension-type headaches, people with cervicogenic headaches sometimes receive manipulation of the neck, facet injections of the cervical vertebrae, local anesthetic blocks of the cervical nerves, and sometimes injections of corticosteroids in the neck. There is no evidence from randomized controlled trials that these measures are more effective than placebo, and they may be harmful. Even more invasive measures such as surgical section (cutting) of nerves and nerve roots are equally unsupported by evidence

3. ***TMJ (temporomandibular*** [or jaw] **joint**) injuries seldom result in frank headaches though they may cause pain in the face and jaw. The typical presentation is a person who has been struck in the face, or sometimes, has had a whiplash injury. Shortly afterwards, pain begins in the jaw or face, often on one side, and may spread up into the head. There may be crepitus (grating) and pain in the jaw joint when talking or eating, and tenderness when feeling over the jaw joint. Most of the pain is believed to come, not from the jaw joint itself, but from the muscles and ligaments surrounding the joint. Thus, the term "*myofascial pain dysfunction syndrome*" is to be preferred. With this more accurate term comes the clear implication that tooth grinding to realign the bite, and surgery or injections within the jaw joint are to be discouraged unless there is unequivocal objective evidence of damage to the joint itself. Conservative treatment, including nonsteroidal anti-inflammatory drugs (NSAID's), massage, and local heat or cold, frequently brings relief, unless the situation has been contaminated by post-traumatic tension-type headaches with their associated symptoms.

4. ***Post-traumatic migraine*** is more common than many physicians suspect; they are not clued in to seeking out the characteristic features of this headache. It is important to do so, because of all the post-traumatic headaches, migraine is the one most responsive to appropriate treatment. Typically, the person has had migraine before, or has a strong family history of migraine. Migraine can, however, arise de novo following a head or, perhaps, a neck injury. The headaches will fulfill the diagnostic criteria outlined on page 4. Fortunately, they are not

often associated with other symptoms of the “post-traumatic syndrome” - though they may be, in which event the response to treatment is not as good. The treatment is that of “regular” benign dysfunctional migraine - carefully supervised non-habituating analgesics or NSAID’s for mild attacks, triptans (sumatriptan or Imitrex®, zolmitriptan or Zomig®, rizatriptan or Maxalt®, naratriptan or Amerge®) for moderate or severe attacks, and a migraine prophylactic medication (a beta-blocker, tricyclic, or anticonvulsant) to reduce the frequency of attacks. The majority of people with post-traumatic migraine respond well to this standard therapy.

5. ***Site of injury*** headaches are the most straightforward of the post-traumatic headaches, though the least frequent. It is believed that a blow to the head may damage, usually temporarily, one of the scalp nerves, producing an area of tenderness, hypersensitivity to stimuli such as the touch of a hat or a hairbrush or a cold wind, and spontaneous pain which may be described as headache but more often is called a head pain. Complete severance of the nerve will not produce this picture - instead there will be a non-painful area of numbness. Incomplete damage, such as bruising, upsets the balance of pain-transmitting and pain-inhibiting fibers in the nerve, making it irritable and painful. The physician, by physical examination, can map out and confirm the area of nerve damage. Usually, this type of head pain will resolve spontaneously, or respond to appropriate local therapy by an anesthesiologist or a neurosurgeon.

6. ***Subdural hematoma*** headaches are equally straightforward. Head injury may produce leaking from blood vessels on the surface of the brain, leading to an accumulation of blood over the brain (subdural hematoma) which creates headache by increasing the pressure on the brain, displacing pain-sensitive structures. Diagnosis is clear-cut (CT scans show the hematomas), and headaches caused by the hematomas clear when the larger hematomas are neurosurgically removed or the smaller ones are allowed to spontaneously resolve. These headaches are seldom chronic.

Much of the controversy over post-traumatic symptoms has to do with the common tension-type and the so-called cervicogenic headaches. In these situations, despite the individual’s bitter complaints of nonspecific chronic headaches and of multiple other symptoms, there is usually no abnormality on physical examination or on neuroimaging (X-rays, CT scans, MRI, etc). Financial compensation is usually an issue. Controversy arises because there are no objective findings to corroborate these people’s complaints or to substantiate their claims. As a result of this situation, two diametrically opposed positions on the genesis of post-traumatic headaches have emerged, and can be described as follows:

1. These headaches and other symptoms are largely psychological. Transient headaches and dizziness and other symptoms do occur regularly after head and/or neck injury, but outside of a compensation or insurance situation usually clear completely within a couple of days. For example, athletic head injuries hardly ever result in long term headaches; the sports culture emphasizes “getting back into the game” and being “up” for the next event. A fall at home causing a minor head injury very seldom leads to long term symptoms, whereas a similar event in the workplace frequently does. The explanation given for these epidemiological observations is that if an individual operates in a culture (see page 6) where there is an expectation that headaches can persist long term after even a trivial blow to the head or strain on the neck, then under the reinforcing influence of friends, family, and health professionals, expectations become reality. The symptoms which in other circumstances would clear within a day or two, persist and are perpetuated by the individual’s largely unconscious needs for legitimization and vindication of his position by recognition of his disability (ie compensation) and by the imposition of a “sick role” by health professionals who unnecessarily restrict activities and who give unnecessarily prolonged treatment. Frank malingering (the conscious manufacture of symptoms for gain) is rare, but does occur. In this model, eventual removal of compensation issues is unlikely to terminate the symptoms, even though they were largely behaviorally induced, because the individual is now trapped in the sick role that he has been cast in. Also, the individual may sense that losing his symptoms after settlement of compensation might create the impression that his symptoms never were physical. In this school of thought, appropriate treatment consists of attempting to distance the patient from the illness model by encouraging “wellness behavior”, by improving the general state of well-being with physical therapy and exercise, by medical treatment of anxiety and/or depression and of headache (amitriptyline, etc), by keeping the patient away from overuse of analgesics which could complicate the situation by causing “analgesic rebound” headaches, and by emphasizing an early return to full activities including occupational.

2. The other, and opposite, school of thought believes that these tension-type headaches and other symptoms are entirely physical. Supporters of this position point to studies in which animals have been struck on the head (which they believe approximates mild head injury in humans) and later, killed, with autopsies showing areas of brain damage which, because microscopic, cannot be visualized by CT or MRI

scans. They further postulate that neck injury can cause brain injury by causing the head to jerk back and forth, so that long delicate processes (axons) of nerve cells in the brain and spinal cord may be stretched and torn, again causing microscopic injuries which cannot be seen on CT or MRI scans. They argue that the part of the neck where some of the movement of the whiplash injury takes place is the part that contains the nerve fibers which transmit head pain, which is why headache may be a prominent part of the “whiplash syndrome”. Other fibers that they postulate are stretched and damaged mediate brain functions such as memory, emotions, balance, etc., accounting for the other symptoms of the “post-concussion” or “whiplash” syndromes. According to this school of thought, these microscopic (“diffuse axonal”) injuries may or may not recover. Appropriate treatment consists of physiotherapy and exercise to maintain the patient in reasonable condition until such time as the damaged parts of the brain can reconstitute themselves or have their functions assumed by undamaged areas, medical treatment of anxiety and depression and of headache, avoidance of overly frequent use of analgesics, and by resting the patient and not returning him to full activities, including occupational, until his putatively damaged brain is able to support such a return.

These are polar positions, presented as such for clarity. The dispute is not yet resolved, and is unlikely to be in the near future. Despite the very large number of people involved, the evidence which would settle the argument - autopsy confirmation or denial of microscopic damage in the brains of a series of people with persistent post-concussive symptoms and normal CT and MRI scans - is not at hand. For practical purposes, most physicians tend to approach their head-injured or “whiplash” patients on the basis that, in most cases, both psychological and physical factors may be involved and may necessitate treatment. Treatment is an empirical amalgam of physical and psychological measures. Note that in the two polar positions described above, the major difference in treatment is the vigor with which attempts are made to return the individual early to full activities. Note also that it is the tension-type headache and the often accompanying post-concussive/post-whiplash symptoms that are at the centre of the controversy; post-traumatic migraine, in contrast, has a generally agreed genesis and when diagnosed and treated appropriately, a generally good outcome.

There is an emerging consensus on how to diagnose post-traumatic headaches. The International Headache Society (IHS) has published diagnostic criteria for post-traumatic headaches. The type of headache is first established by how well it confirms to the IHS criteria for tension-type headache, migraine, etc. The issue of how likely that headache is to be causally related to head trauma is then assessed by the post-traumatic criteria, which are paraphrased as follows:

1. Headache follows *significant* head trauma , the significance being demonstrated by at least one of:
   - Loss of consciousness occurred
   - Post-traumatic amnesia occurred, and lasted more than 10 minutes
   - At least *two* of the following tests showed relevant abnormalities:

     - clinical neurological examination, skull X-ray, CT scan, MRI, evoked potentials, spinal fluid examination, vestibular function studies, neuropsychological testing

2. Headache begins within 14 days of trauma

3. If the headache disappears within 8 weeks following the trauma, it is termed “acute”; if it does not, it is termed “chronic”.

A headache that begins more than 14 days after the trauma is deemed unrelated to the trauma. A head trauma that does not meet the criteria for significance (“significant” does not necessarily equate with “severe”, but rather with “not trivial”) may still be followed by headache and other symptoms, but the causal relationship is then less definite.

The prognosis of post-traumatic headaches has been studied extensively, but the results of these studies have not been uniform because of:

- Different definitions of what constitutes a post-traumatic headache (the IHS criteria were not published until 1988)
- Different mixes of post-traumatic headache types in the populations studied. Post-traumatic migraine does much better, and site of injury headaches do better, than post-traumatic tension-type headaches and whiplash syndromes, but the various types were not always separated in the studies
- Inadequate stratification of the patients in terms of significance of injury, presence or absence of prior headaches, etc. in many studies
- Inadequate population controls - ie. failure to compare the prevalence of headaches and other symptoms in the study population with the prevalence of headaches and other symptoms in similar (matched for age and gender) populations who have *not* been injured.

Despite the shortcomings of the studies, a few factors have gained wide acceptance as being *useful* in predicting the outcome of post-traumatic headaches and other symptoms:

- The older the patient, the more likely are the headaches and other symptoms to persist
- Post-traumatic migraine, promptly and properly treated with appropriate medications, does better than the other types of post-traumatic headaches
- For the other types of post-traumatic headaches, people who receive prompt and vigorous physiotherapy do better than those who do not
- The longer the interval between the trauma and the onset of symptoms, the more likely are the symptoms to persist (the inference being that delayed onset symptoms are more likely to be psychological and therefore less likely to respond to physical treatment or to clear spontaneously).
- In general, the longer the symptoms have lasted, the more likely they are to be permanent. About one-half of people with post-traumatic headaches will be free of symptoms by two months after the trauma. After six months (assuming that appropriate treatment has been tried and failed), it is more likely than not that the symptoms will not clear - though there is still a possibility that they may. Chances of improvement become remote at one year post-trauma, and by four years after the trauma the symptoms will be permanent.

Other features are now widely regarded as *not* affecting prognosis:

- Once a topic of hot controversy, the presence or absence of litigation or compensation has now been shown in a preponderance of studies, not to affect the prognosis of post-traumatic headaches and other symptoms, once established and chronic. (Whether or not they influence the *development* of such chronic symptoms is an unresolved issue; see discussion on pages 6 and 9).

Some prognostic features, even though extensively studied, remain *controversial*:

- The presence of recurrent headaches of any type prior to the trauma has been found to adversely affect outcome in some studies, but not in others
- The relationship between the severity of the trauma and the development and persistence of symptoms is identified as inverse in some studies (ie, the less severe the trauma, the more likely are headaches and other symptoms to develop and persist) and as direct (ie the more severe the injury, the more likely are headaches and other symptoms to develop and persist) in other studies.

Headaches due to environmental toxins

These headaches occur with use of, or exposure to, chemicals in the workplace or other environment. It is believed that most of the substances that produce headache do so by causing the pain-sensitive blood vessels of the head to dilate. A few substances (for example, the insecticides chlordecone [Kepone] and benzene hexachloride [Lindane]) may cause swelling (“edema”) of the brain, which causes headache by increasing the pressure on the brain. Toxic headaches are diffuse, often throbbing, worse with activity, and of variable severity. It is believed, but not proven, that people who already have benign dysfunctional headaches such as migraine are more sensitive to some of these substances and are therefore more likely to develop headaches on exposure.

Carbon monoxide is probably the most common cause of environmental headaches (malfunctioning heating systems, poorly-ventilated auto shops), but there are many other substances which have been identified as causing headaches. The following list is far from exhaustive:

- long chain alcohols, aniline dyes, arsenic, balsam, borate, bromate, camphor, carbon disulphide, carbon tetrachloride, chlorates, chlordecone, copper salts, EDTA, heptachlor, hydrogen sulphide, iodine, kerosene, lead, lithium, methyl alcohol, methyl bromide, methyl chloride, methyl iodine, mercury, napthalene, nitrates, nitrites, and organophosphates.

The headaches clear once the individual is removed from the environment, and typically this clearing occurs quickly, usually within a day or two, as the dilated cranial blood vessels return to normal. It may be delayed for weeks, however, if the toxic substance has accumulated in the body (eg lead) or if the headache is mediated by brain edema, which may take some time to subside.

Certain factors assist in the diagnosis of these toxic headaches:

- a toxic substance has been identified in the immediate environment
- other people in proximity to that toxin may be similarly affected
- analysis of body fluids such as blood and urine may show the presence of the toxin
- the headaches clear within a day or two of removal of the person or the toxin from the environment, unless there has been demonstrable accumulation of the toxin within the tissues of the individual, or unless the headache can be demonstrated (usually by CT or MRI scan) to be mediated by cerebral edema.

A vexatious problem is the case of the individual who claims headaches to be occurring on the basis of sensitivity or allergy to a non-toxic substance within the environment, or hypersensitivity to substances present in acceptable levels (for example, dust). There is no question that some forms of allergy like serum sickness, anaphylaxis, urticaria, and rhinitis complicated by sinusitis may cause headaches among other prominent symptoms (such as wheezing, hives, collapse of blood pressure, etc). What is extremely controversial is whether or not hypersensitivity or allergy can cause headaches alone. In cases such as this, resolution may be aided by the finding in blood cells, plasma, cerebrospinal fluid and/or urine of chemical or cellular evidence of an acute allergic reaction during a headache; such evidence can be provided by an immunologist or allergist. A positive skin test is not enough to link the probable presence of an allergy (which is what the skin test signifies) with the headache; both allergy and headaches are extremely common as independent entities in the general population.

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   The above two papers provide background on what migraine and tension-type headaches are, and how they affect the lives of those who have them.

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   The IHS classification and criteria, above, are the gold standard for diagnosing headaches.

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The above seven papers are a sampling of views about post-traumatic symptoms and how societal factors such as expectations and compensation influence, or do not influence, their development and persistence. The editorial by Deyo (ref. 8) is a current, brief, and well-balanced overview.