Written, designed and compiled by;
Dr Nigel Peek
Chiropractor
MZCA MTech: CHIRO (SA)

Migraine - a Patient Guide

What is Migraine?

Migraine is a common, disabling nervous system disorder defined by neurological symptoms that usually include headache, aura and heightened sensory arousal. It is usually moderately to severely disabling. Those affected by a migraine attack are often unable to function as a result of its symptoms. Up to 1 in 5 people experience migraine. It is one of the most disabling neurological conditions worldwide and is responsible for significant loss of working productivity and working absenteeism (1-4).

Quick Facts:

Migraine is not a headache. Migraine is a complex neurological disorder, whilst headache is the most common feature, the disease is not solely defined by headache.

Migraine sufferers often report other features of the disorder without the headache. Other common symptoms associated with migraine include nausea, vomiting, and sensory disturbances including light and noise sensitivity (3).

Certain more severe types of migraine are characterised by reversible loss of movement, dizziness and loss of sensation, these symptoms can mimic a stroke (hemiplegic migraine and vestibular migraine) (3).

Migraine headache is usually on one side of the head and is characterised by a pulsating sensation usually around the eye. Neck pain often accompanies migraine and is sometimes mistaken for the cause of the headache (3, 10).

What Happens in a Migraine Attack?

Whilst our understanding of migraine has advanced considerably, there is still controversy over the exact mechanisms that lead to a migraine attack.

The phases of migraine attack are associated with a sequence of mechanisms in the brain. Brain imaging studies show us that the premonitory phase has been linked to the activation of areas in the brainstem responsible for control of the body's autonomic and pain systems (mostly the hypothalamus). Increased activation of these areas likely leads to the discomfort associated with this phase (including nausea, fatigue, irritability)(10).

The aura phase of an attack has been extensively studied in recent years, and it is now thought that this phase is associated with a rapid depression or depletion of activity on the surface of the brain. In effect this change in activity disrupts higher brain functions like thinking, reasoning or remembering (cognitive function)(3, 9, 10). This disruption makes the brain more susceptible to sensory overload and pain.

The pain of migraine has been linked to an activation of a network of sensory nerves that supply the head, face, linings and blood supply to the brain. Hence, during the pain phase of a migraine, these nerves transmit painful information from this network resulting in headache (6).

Research and treatment trends have focused on the role of certain chemical triggers that activate the sensory pathway in migraine sufferers. Studies show an increase in these chemicals during the pain phase of migraine. Research also indicates that migraineurs are more susceptible to the actions of these chemicals (1).

Following the pain phase of migraine, patients are often left with symptoms including general feeling of discomfort, illness, or unease, and other symptoms such as mental fogginess, mood changes, sensitivity of the head and scalp and gut symptoms (9). This is called the postdrome or "hangover" effect.

What Causes and What Triggers a Migraine Attack?

Migraine has a strong genetic and familial link. Whilst there is no "one gene" that is linked to migraine, researchers have found numerous genetic codes unique to migraineurs (3). It is important to note that whilst genetics are clearly associated with migraine, we know that migraine is only expressed when an individual is exposed to environmental factors (3). Some migraineurs may have only a few attacks in a lifetime, whilst others may have numerous attacks in a week.

Typically, migraineurs tend to report that certain triggers usually cause the onset of an attack. This is an interesting area of discussion as many migraineurs report triggers such as intense light or sound exposure, strong odours, changes in weather and food and alcohol. Whilst this may seem likely to be the case for those suffering an attack, we also know that many symptoms precede a migraine for up to 72 hours, including hunger, fatigue, restlessness and sleep changes. Therefore, intense stimuli and other so-called "triggers" may purely be indicators of a migraine in progress and retrospectively implicated as causative. Research studies have yet to demonstrate unequivocally that certain stimuli provoke or trigger an attack (16).

Possible internal and external factors contributing to an attack include hormonal changes in women, changes of day-night rhythm, strong sensory stimuli, hunger, stress or intense physical activity (3).

Migraine is linked to other Disorders and Pain Conditions

Research has demonstrated that migraine is in fact a multisystem disorder. This is likely because the migraine nervous system is hypersensitive and therefore linked to many disorders that reflect this sensitivity.

⇒ Neck, and back pain
⇒ Irritable bowel syndrome
⇒ Balance disorders
⇒ Dysautonomia (poor regulation of blood pressure, temperature control, digestion)
⇒ Hormonal and reproductive disorders (endometriosis and menstrual disorders)
⇒ Pelvic pain and vulvodynia
⇒ Hypermobility and collagen vascular disorders (Ehlers Danlos Syndrome)
⇒ Fibromyalgia and widespread pain
⇒ Chronic headache (tension type headache)
⇒ Attention-deficit-hyperactivity disorder (ADHD)
⇒ Anxiety and depression
⇒ Restless leg syndrome
⇒ Sleep disorders
⇒ Epilepsy
⇒ Stroke (14, 15, 17-21)

Migraine - a Disorder of Sensitivity

Migraine is a processing disorder, one could liken this to a "software" problem, rather than a "hardware" failure.

In the case of pain, we usually associate pain with tissue damage, however we now know that pain can exist in the absence of damage. Pain is a protective mechanism that is produced by the brain in order to avoid or protect us from harm. However, when pain is produced in the absence of harm the benefit is no longer useful. Migraine sufferers may in fact over process and convert normal stimuli into a protective response (i.e. pain).

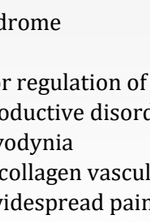
Migraine sufferers experience a range of heightened stimuli or over excitation. Normally the central nervous system regulates through a process called inhibition. Migraineurs tend to over process stimuli, and therefore experience a world of heightened excitability (this applies not only to pain but also to a range of increased sensory and emotional sensitivity) (1,13).

"The excitation - inhibition tug of war"

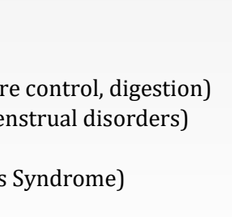
1. Normal interplay between excitation and inhibition.

Red - Excitation

Blue - Inhibition



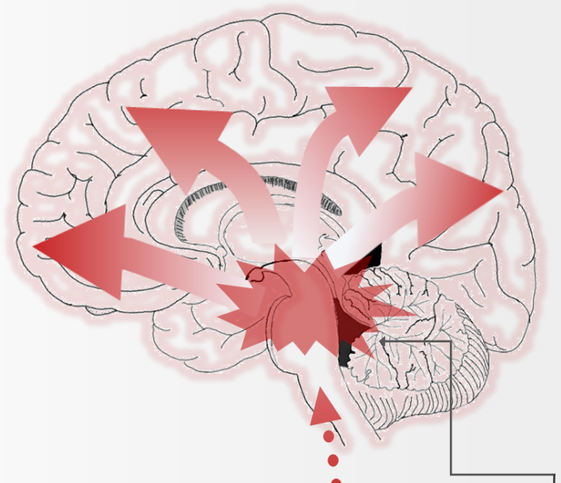
2. Migraine Nervous system —Excitation dominates inhibition



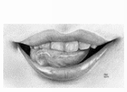
The Premonitory Phase
(Pre-attack)

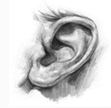
Brainstem Activation

- Fatigue
- Irritability
- Concentration difficulties
- Mood Changes
- Yawning
- Neck stiffness
- Digestive discomfort



Stimuli—mechanical, chemical, hormonal, sensory, emotional, stress, sleep disturbance



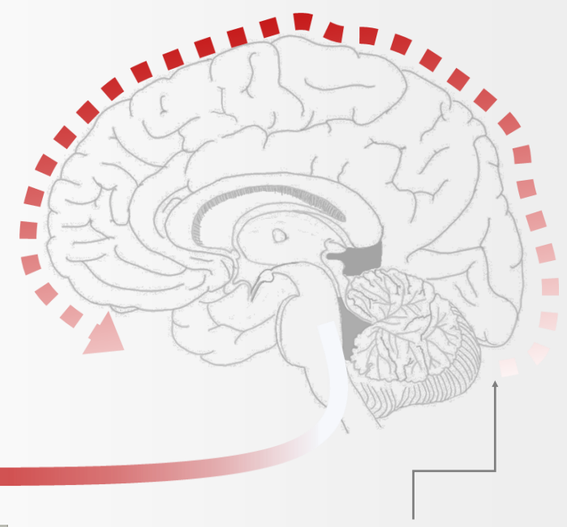


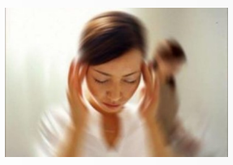
Abnormal activation of the brainstem disrupts the body's internal regulation including temperature control, appetite, emotion, sleep and digestion.

Aura Phase
(Cortical depression)

Sensory Distortion

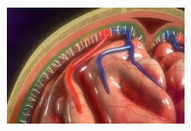
- Visual disturbance
- Movement disturbance
- Dizziness
- Language and speech difficulties





Cortical depression is a wave of rapid excitation followed by depression of nerve impulses at the surface of the brain. This results in a mass depletion of brain activity for up to 30 minutes

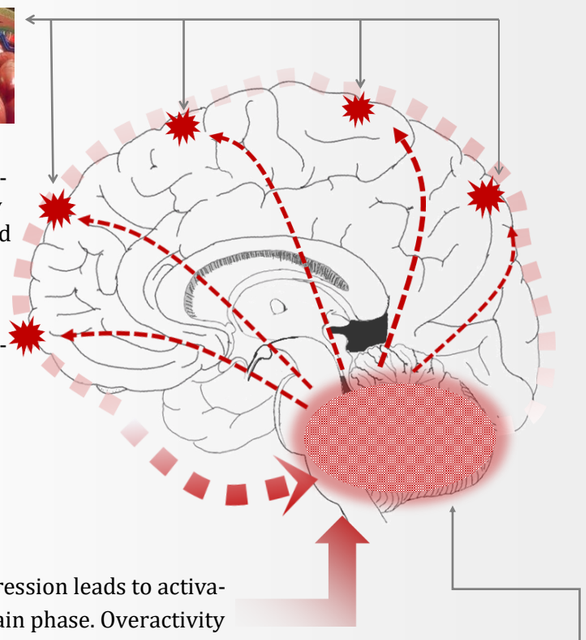
The Pain Phase
(Headache and neck pain)



Pain network activation

- Usually one sided throbbing, moderate to intense, lasting 4-72 hours.
- Neck pain is often a feature of the pain phase. It is sometimes mistaken for the cause of migraine.

Migraine pain arises from sensitivity of brain linings and blood vessels (resulting in the throbbing, pulsating nature of headache)



Cortical depression leads to activation of the pain phase. Overactivity of the brainstem and fatigue of the surface of the brain result in a state of hypersensitivity and pain.

Network activation centre and release of pain sensitising chemicals

** the phases and symptoms of migraine do not always unfold in sequence, and pain is not always a feature of a migraine attack (for example, some patient may experience visual aura without the onset of pain)*

Management and Treatment of Migraine

Whilst there have been significant advances in the management of migraine, there is no one treatment for migraine. This is because migraine is a complex disorder that is triggered by a combination of a number of different individual environmental and genetic factors. Like many persistent pain conditions, research supports and multidimensional approach to management.

Medical	Behavioural and Psychological	Nutritional	Movement Therapies and Exercise	Complementary and Alternative
• *Anti-inflammatories* (e.g. naproxen, diclofenac, ibuprofen)(22) • *Triptans* (e.g. sumatriptan, rizatriptan) (22) • *CGRP antagonists* (e.g. erenumab)(23) • *Anticonvulsants* (e.g. valproate, gabapentin)(22) • *Betablockers* (e.g. propanolol, metoprolol) (22) • *Antidepressants* (amitriptyline, venlafaxine)(22)	• *Cognitive behavioural therapy* (24, 25) • *Educational and mindfulness practices* (25) • *Sleep behavioural management* (3)	• *Magnesium* (500 mg daily)(26) • *Melatonin* (10 mg daily) (27) • *Riboflavin* (20 mg daily) (22) • *Co-enzyme Q10* (75mg twice daily) (22) • *Butterbur* (50-75 mg twice daily) (22) • *Ketogenic diet* (28) • *Caffeine* (3)	• *Yoga and Tai Chi* (29) • *Moderate exercise* (30)	• *Manual Therapy* Including spinal manipulative therapy (31), massage, mobilisations (chiropractic, osteopathy, physiotherapy, massage therapy)(25) • *Acupuncture* (25)

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