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A BOOK ON "MIGRAINE WITH HOMOEOPATHIC THERAPUETICS"

JV'n Dr. Archana Verma

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INTRODUCTION

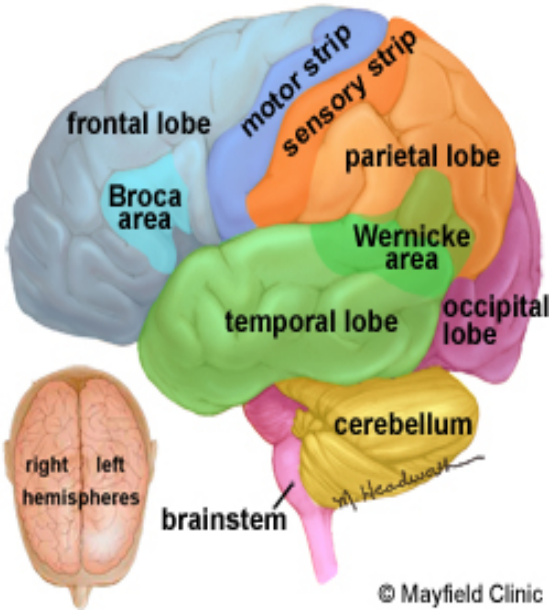
Migraine is a chronic neurological disease characterized by recurrent moderate to severe headaches often in association with a number of autonomic nervous system symptoms.

Typically the headache affects one half of the head, is pulsating in nature, and lasts from 2 to 72 hours. Associated symptoms may include nausea, vomiting, and sensitivity to light, sound, or smell. The pain is generally made worse by physical activity. Up to one-third of people with migraine headaches perceive an aura: a transient visual, sensory, language, or motor disturbance which signals that the headache wills soon occur[1].

ANATOMY OF BRAIN

NERVOUS SYSTEM

The nervous system is divided into central and peripheral systems. The central nervous system (CNS) is composed of the brain and spinal cord. The peripheral nervous system (PNS) is composed of spinal nerves that branch from the spinal cord and cranial nerves that branch from the brain. The PNS includes the autonomic nervous system, which controls vital functions such as breathing, digestion, heart rate, and secretion of hormones.[1]



Brain

The brain is composed of the cerebrum, cerebellum, and brainstem.

The brain is composed of three parts: the brainstem, cerebellum, and cerebrum. The cerebrum is divided into four lobes: frontal, parietal, temporal,.

- The **cerebrum** is the largest part of the brain and is composed of right and left hemispheres. It performs higher functions like interpreting touch, vision and hearing, as well as speech, reasoning, emotions, learning, and fine control of movement.

- The **cerebellum** is located under the cerebrum. Its function is to coordinate muscle movements, maintain posture, and balance.

- The **brainstem** includes the midbrain, pons, and medulla. It acts as a relay center connecting the cerebrum and cerebellum to the spinal cord. It performs many automatic functions such as breathing, heart rate, body temperature, wake and sleep cycles, digestion, sneezing, coughing, vomiting, and swallowing. Ten of the twelve cranial nerves originate in the brainstem.

The surface of the cerebrum has a folded appearance called the cortex. The cortex contains about 70% of the 100 billion nerve cells. The nerve cell bodies color the cortex grey-brown giving it its name – gray matter. Beneath the cortex are long connecting fibers between neurons, called axons, which make up the white matter.[1]

The folding of the cortex increases the brain's surface area allowing more neurons to fit inside the skull and enabling higher functions. Each fold is called a gyrus, and each groove between folds is called a sulcus. There are names for the folds and grooves that help define specific brain regions.

Right brain – left brain

The right and left hemispheres of the brain are joined by a bundle of fibers called the corpus callosum that delivers messages from one side to the other. Each hemisphere controls the opposite side of the body.[2]

Not all functions of the hemispheres are shared. In general, the left hemisphere controls speech, comprehension, arithmetic, and writing. The right hemisphere controls creativity, spatial ability, artistic, and musical skills.

Lobes of the brain

The cerebral hemispheres have distinct fissures, which divide the brain into lobes. Each hemisphere has 4 lobes: frontal, temporal, parietal, and occipital. Each lobe may be divided, once again, into areas that serve very specific functions. Each lobe of the brain does not function alone. There are very complex relationships between the lobes of the brain and between the right and left hemispheres.

Frontal lobe

- Personality, behaviour, emotions
- Judgment, planning, problem solving
- Speech: speaking and writing (Broca's area)
- Body movement (motor strip)

- Intelligence, concentration, self-awareness

Parietal lobe

- Interprets language, words
- Sense of touch, pain, temperature (sensory strip)
- Interprets signals from vision, hearing, motor, sensory and memory
- Spatial and visual perception

Occipital lobe

- Interprets vision (colour, light, movement)

Temporal lobe

- Understanding language (Wernicke's area)
- Memory
- Hearing
- Sequencing and organization

Messages within the brain are carried along pathways. Messages can travel from one gyrus to another, from one lobe to another, from one side of the brain to the other, and to structures found deep in the brain (e.g. thalamus, hypothalamus).[2]

Deep structures

Hypothalamus - is located in the floor of the third ventricle and is the master control of the autonomic system. It plays a role in controlling behaviours such as hunger, thirst, sleep, and sexual response. It also regulates body temperature, blood pressure, emotions, and secretion of hormones.

Pituitary gland - lies in a small pocket of bone at the skull base called the sella turcica. The pituitary gland is connected to the hypothalamus of the brain by the pituitary stalk. Known as the "master gland," it controls other endocrine glands in the body. It secretes hormones that control sexual development, promote bone and muscle growth, respond to stress, and fight disease.

Pineal gland - is located behind the third ventricle. It helps regulate the body's internal clock and circadian rhythms by secreting melatonin. It has some role in sexual development.

Thalamus - serves as a relay station for almost all information that comes and goes to the cortex. It plays a role in pain sensation, attention, alertness and memory.

Basal ganglia - includes the caudate, putamen and globus pallidus. These nuclei work with the cerebellum to coordinate fine motions, such as fingertip movements.

Limbic system - is the center of our emotions, learning, and memory. Included in this system are the cingulate gyri, hypothalamus, amygdala (emotional reactions) and hippocampus (memory).

Meninges

The brain and spinal cord are covered and protected by three layers of tissue called meninges. From the outermost layer inward they are: the dura mater, arachnoid mater, and pia mater.

The dura mater is a strong, thick membrane that closely lines the inside of the skull; its two layers, the periosteal and meningeal dura, are fused and separate only to form venous sinuses. The dura creates little folds or compartments. There are two special dural folds, the falx and the tentorium. The falx separates the right and left hemispheres of the brain and the tentorium separates the cerebrum from the cerebellum.

The arachnoid mater is a thin, web-like membrane that covers the entire brain. The arachnoid is made of elastic tissue. The space between the dura and arachnoid membranes is called the subdural space.

The pia mater hugs the surface of the brain following its folds and grooves. The pia mater has many blood vessels that reach deep into the brain. The space between the arachnoid and pia is called the subarachnoid space. It is here where the cerebrospinal fluid bathes and cushions the brain.[2]

Ventricles and cerebrospinal fluid

The brain has hollow fluid-filled cavities called ventricles). Inside the ventricles is a ribbon-like structure called the choroid plexus that makes clear colorless cerebrospinal fluid (CSF). CSF flows within and around the brain and spinal cord to help cushion it from injury. This circulating fluid is constantly being absorbed and replenished.

There are two ventricles deep within the cerebral hemispheres called the lateral ventricles. They both connect with the third ventricle through a separate opening called the foramen of Monro. The third ventricle connects with the fourth ventricle through a long narrow tube called the aqueduct of Sylvius. From the fourth ventricle, CSF flows into the subarachnoid space where it bathes and cushions the brain. CSF is recycled (or absorbed) by special structures in the superior sagittal sinus called arachnoid villi.

A balance is maintained between the amount of CSF that is absorbed and the amount that is produced. A disruption or blockage in the system can cause a build-up of CSF, which can cause enlargement of the ventricles (hydrocephalus) or cause a collection of fluid in the spinal cord (syringomyelia).

Blood supply

Blood is carried to the brain by two paired arteries, the internal carotid arteries and the vertebral arteries. The internal carotid arteries supply most of the cerebrum.[1]

The vertebral arteries supply the cerebellum, brainstem, and the underside of the cerebrum. After passing through the skull, the right and left vertebral arteries join together to form the basilar artery. The basilar artery and the internal carotid arteries "communicate" with each other at the base of the brain called the Circle of Willis. The communication between the internal carotid and vertebral-basilar systems is an important safety feature of the brain. If one of the major vessels becomes blocked, it is possible for collateral blood flow to come across the Circle of Willis and prevent brain damage.

The venous circulation of the brain is very different than the rest of the body. The major vein collectors are integrated into the dura to form venous sinuses. The venous sinuses collect the blood from the brain and pass it to the internal jugular veins. The superior and inferior sagittal sinuses drain the cerebrum, the cavernous sinuses drains the anterior skull base. All sinuses eventually drain to the sigmoid sinuses, which exit the skull and form the jugular veins. These two jugular veins are essentially the only drainage of the brain.

Cells of the brain

The brain is made up of two types of cells: nerve cells (neurons) and glia cells.

Nerve cells

There are many sizes and shapes of neurons, but all consist of a cell body, dendrites and an axon. The neuron conveys information through electrical and chemical signals.

Neurons transmit their energy, or "talk", to each other across a tiny gap called a synapse. A neuron has many arms called dendrites, which act like antennae picking up messages from other nerve cells. These messages are passed to the cell body, which determines if the message should be passed along. Important messages are passed to the end of the axon where sacs containing neurotransmitters open into the synapse. The neurotransmitter molecules cross the synapse and fit into special receptors on the receiving nerve cell, which stimulates that cell to pass on the message.

Glia cells

Glia (Greek word meaning glue) is the cells of the brain that provide neurons with nourishment, protection, and structural support. There are about 10 to 50 times more glia than nerve cells and are the most common type of cells involved in brain tumors.

- Astroglia or astrocytes transport nutrients to neurons, hold neurons in place, digest parts of dead neurons, and regulate the blood brain barrier.
- Oligodendroglia cells provide insulation (myelin) to neurons.
- Ependymal cells line the ventricles and secrete cerebrospinal fluid (CSF).
- Microglia digest dead neurons and pathogens.[2]

The functions of the brain depend on the ability of neurons to transmit electrochemical signals to other cells, and their ability to respond appropriately to electrochemical signals received from other cells. The electrical properties of neurons are controlled by a wide

variety of biochemical and metabolic processes, most notably the interactions between neurotransmitters and receptors that take place at synapses.[2]

Neurotransmitters and receptors

Neurotransmitters are chemicals that are released at synapses when an action potential activates them—neurotransmitters attach themselves to receptor molecules on the membrane of the synapse's target cell, and thereby alter the electrical or chemical properties of the receptor molecules. With few exceptions, each neuron in the brain releases the same chemical neurotransmitter, or combination of neurotransmitters, at all the synaptic connections it makes with other neurons; this rule is known as Dale's principle. Thus, a neuron can be characterized by the neurotransmitters that it releases. [4]

The two neurotransmitters that are used most widely in the vertebrate brain are glutamate, which almost always exerts excitatory effects on target neurons, and gamma-aminobutyric acid (GABA), which is almost always inhibitory. Neurons using these transmitters can be found in nearly every part of the brain. Because of their ubiquity, drugs that act on glutamate or GABA tend to have broad and powerful effects. Some general anaesthetics act by reducing the effects of glutamate; most tranquilizers exert their sedative effects by enhancing the effects of GABA.[4]

There are dozens of other chemical neurotransmitters that are used in more limited areas of the brain, often areas dedicated to a particular function.

Electrical activity

As a side effect of the electrochemical processes used by neurons for signalling, brain tissue generates electric fields when it is active. When large numbers of neurons show synchronized activity, the electric fields that they generate can be large enough to detect outside the skull, using electroencephalography (EEG) or magnetoencephalograph. Each part of the brain shows a mixture of rhythmic and nonrhythmic activity, which may vary according to behavioural state. The cerebral cortex tends to show large slow delta waves during sleep, faster alpha waves when awake but inattentive, and chaotic-looking irregular activity when actively engaged in a task.[4]

Metabolism

Blood–brain barrier allows metabolism inside the brain to operate differently from metabolism in other parts of the body. Glial cells play a major role in brain metabolism by controlling the chemical composition of the fluid that surrounds neurons, including levels of ions and nutrients. Most of the brain's energy consumption goes into sustaining the electric charge (membrane potential) of neurons. The energy consumption of the brain does not vary greatly over time, but active regions of the cerebral cortex consume somewhat more energy than inactive regions. The brain typically gets most of its energy from oxygen-dependent metabolism of glucose (i.e., blood sugar), but ketones provide a major alternative source, together with contributions from medium chain fatty acids (caprylic and heptanoicacids), lactate, acetate, and possibly amino acid.[3]

MIGRAINE

Migraine is an episodic headache associated with certain features such as sensitivity to light, sound, or movement; nausea and vomiting often accompany the headache. Migraine is a benign and recurring syndrome of headache associated with other symptoms of neurologic dysfunction in varying admixtures. Migraine can often be recognized by its activators, referred to as *triggers*.[5]

A classification system developed by the International headache Society characterizes headache as primary or secondary. Migraine is a type of primary headaches. Primary headache are those in which headache and its associated features are the disorder in itself. Other causes of primary headaches are

1) Tension-type
2) Idiopathic stabbing
3) Exceptional
4) Cluster headache.

Secondary headaches are those caused by exogenous disorders. Primary headache often results in considerable disability and a decrease in the patient's quality of life. Mild secondary headache, such as that seen in association with upper respiratory tract infections, is common but rarely worrisome. Common causes of secondary headache are:-

1. Systemic infection
2. Head injury
3. Vascular disorder
4. Subarachnoid haemorrhage
5. Brain tumour

The brain of the migraine patient is particularly sensitive to environmental and sensory stimuli; migraine-prone patients do not habituate easily to sensory stimuli. This sensitivity is amplified in females during the menstrual cycle. Headache can be initiated or amplified by various triggers, including glare, bright lights, sounds, or other afferent stimulation; hunger; excess stress; physical exertion; stormy weather or barometric pressure changes; hormonal fluctuations during menses; lack of or excess sleep; and alcohol or other chemical stimulation. Knowledge of a patient's susceptibility to specific triggers can be useful in management strategies involving lifestyle adjustments.[5]

Pain usually occurs when peripheral nociceptors are stimulated in response to tissue injury, visceral distension, or other factors. In such situations, pain perception is a normal physiologic response mediated by a healthy nervous system. Pain can also result when pain-producing pathways of the peripheral or central nervous system are damaged or activated

inappropriately. Headache may originate from either or both mechanisms. Relatively few cranial structures are pain-producing; these include the scalp, middle meningeal artery, dural sinuses, falx cerebri, and proximal segments of the large pial arteries. The ventricular ependyma, choroid plexus, pial veins, and much of the brain parenchyma are not pain-producing.[5]

The key structures involved in headache appear to be

- The large intracranial vessels and dura mater and the peripheral terminals of the trigeminal nerve that innervate these structures
- The caudal portion of the trigeminal nucleus, which extends into the dorsal horns of the upper cervical spinal cord and receives input from the first and second cervical nerve roots (the trigeminocervical complex)
- Rostral pain-processing regions, such as the ventroposteromedial thalamus and the cortex
- The pain-modulatory systems in the brain that modulate input from trigeminal nociceptors at all levels of the pain-processing pathways

The innervation of the large intracranial vessels and dura mater by the trigeminal nerve is known as the *trigeminovascular system*. These autonomic symptoms reflect activation of cranial parasympathetic pathways, and functional imaging studies indicate that vascular changes in migraine when present are similarly driven by these cranial autonomic systems. Migraine and other primary headache types are not "vascular headaches"; these disorders do not reliably manifest vascular changes, and treatment outcomes cannot be predicted by vascular effects. Migraine is a brain disorder, and best understood and managed as such.[5]

AETIOLOGY

The aetiology of migraine is largely unknown. There is often a family history, suggesting a genetic predisposition. The great female preponderance and the tendency for some women to have migraine attacks at certain points in their menstrual cycle hint at hormonal influences. The relevance of the contraceptive pill in this context is difficult to establish, but it does appear to exacerbate migraine in many patients, and to increase the small risk of stroke in patients who suffer from migraine with aura. In some patients there are identifiable dietary precipitants such as cheese, chocolate or red wine. When psychological stress is involved, the migraine attack often occurs after the period of strain so that some patients tend to have attacks at weekends or at the beginning of a holiday.[5]

PATHOGENISES

The sensory sensitivity that is characteristic of migraine is probably due to dysfunction of monoaminergic sensory control systems located in the brainstem and thalamus.

Activation of cells in the trigeminal nucleus results in the release of vasoactive neuropeptides, particularly calcitonin gene–related peptide (CGRP), at vascular terminations of the trigeminal nerve and within the trigeminal nucleus. Centrally, the second-order trigeminal neurons cross the midline and project to ventrobasal and posterior nuclei of the thalamus for further processing. Additionally, there are projections to the periaqueductal gray and hypothalamus, from which reciprocal descending systems have established antinociceptive effects. Other brainstem regions likely to be involved in descending modulation of trigeminal pain include the nucleus locus coeruleus in the pons and the rostroventromedial medulla.

Migraine genes identified by studying families with familial hemiplegic migraine(FHM) reveal involvement of ion channels, suggesting that alterations in membrane excitability can predispose to migraine. Mutations involving the $Ca_v2.1$ (P/Q)–type voltage-gated calcium channel *CACNA1A* gene are now known to cause FHM 1; this mutation is responsible for about 50% of FHM. Mutations in the Na^+-K^+ATPase *ATP1A2* gene, designated FHM 2, are responsible for about 20% of FHM. Mutations in the neuronal voltage-gated sodium channel *SCN1A* cause FHM 3.[5]

CLINICAL FEATURES

Repeated attacks of headache lasting 4–72 h in patients with a normal physical examination, no other reasonable cause for the headache .Other symptoms are-

1. Unilateral pain
2. Throbbing pain
3. Aggravation by movement
4. Moderate or severe intensity
5. Nausea/vomiting
6. Photophobia and phonophobia.

Symptoms Accompanying Severe Migraine attacks-

1. Nausea
2. Photophobia
3. Lightheadedness
4. Scalp tenderness
5. Vomiting
6. Visual disturbances
7. Paresthesias
8. Vertigo
9. Photopsia
10. Alteration of conciousness
11. Diarrhea
12. Syncope

13. Seizure
14. Confusional state

Vertigo can be prominent; it has been estimated that one-third of patients referred for vertigo or dizziness have a primary diagnosis of migraine.[5]

DIAGNOSIS

The diagnosis of a migraine is based on signs and symptoms. Neuroimaging tests are not necessary to diagnose migraine, but may be used to find other causes of headaches in those whose examination and history do not confirm a migraine diagnosis.

The diagnosis of migraine without aura, according to the International Headache Society, can be made according to the following criteria, the "5, 4, 3, 2, and 1 criteria":

Five or more attacks—for migraine with aura, two attacks are sufficient for diagnosis.

1) Four hours to three days in duration
2) Two or more of the following:
3) Unilateral (affecting half the head);
4) Pulsating;
5) "Moderate or severe pain intensity";
6) "Aggravation by or causing avoidance of routine physical activity"

One or more of the following:

1) Nausea and/or vomiting;
2) Sensitivity to both light (photophobia) and sound (phonophobia)

If someone experiences two of the following: photophobia, nausea, or inability to work or study for a day, the diagnosis is more likely. In those with four out of five of the following: pulsating headache, duration of 4–72 hours, pain on one side of the head, nausea, or symptoms that interfere with the person's life, the probability that this is a migraine is 92%.In those with fewer than three of these symptoms the probability is 17%.[5]

CLASSFICATION

Migraines were first comprehensively classified in 1988.The International Headache Society most recently updated their classification of headaches in 2004. According to this classification migraines are primary headaches along with tension-type headaches and cluster headaches, among others.

Migraines are divided into seven subclasses (some of which include further subdivisions):

Migraine without aura, or "common migraine", involves migraine headaches that are not accompanied by an aura.

Migraine with aura, or "classic migraine", usually involves migraine headaches accompanied by an aura.

Two other varieties are familial hemiplegic migraine and sporadic hemiplegic migraine, in which a person has migraines with aura and with accompanying motor weakness. If a close relative has had the same condition, it is called "familial", and otherwise it is called "sporadic".

Another variety is basilar-type migraine, where a headache and aura are accompanied by difficulty speaking, world spinning, ringing in ears, or a number of other brainstem-related symptoms, but not motor weakness. This type was initially believed to be due to spasms of the basilar artery, the artery that supplies the brainstem.

Childhood periodic syndromes that are commonly precursors of migraine include cyclical vomiting (occasional intense periods of vomiting), abdominal migraine (abdominal pain, usually accompanied by nausea), and benign paroxysmal vertigo of childhood (occasional attacks of vertigo).

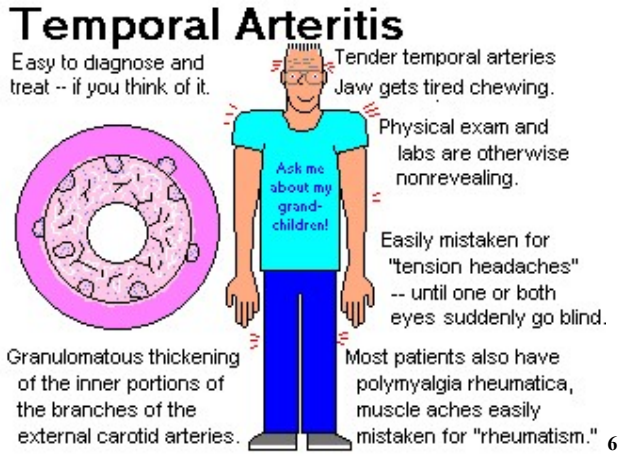
Retinal migraine involves migraine headaches accompanied by visual disturbances or even temporary blindness in one eye.

Complications of migraine describe migraine headaches and/or auras that are unusually long or unusually frequent, or associated with a seizure or brain lesion.

Chronic migraine is a complication of migraines, and is a headache that fulfills diagnostic criteria for migraine headache and occurs for a greater time interval.[5]

DIFFERENTIAL DIAGNOSIS

Temporal arteritis



[6]

Temporal (giant cell) arteritis is an inflammatory disorder of arteries that frequently involves the extracranial carotid circulation. It is a common disorder of the elderly. The average age of onset is 70 years, and women account for 65% of cases. About half of patients with untreated

temporal arteritis develop blindness due to involvement of the ophthalmic artery and its branches; indeed, the ischemic optic neuropathy induced by giant cell arteritis is the major cause of rapidly developing bilateral blindness in patients >60 years. Because treatment with glucocorticoids is effective in preventing this complication, prompt recognition of the disorder is important.

Typical presenting symptoms include headache, polymyalgia rheumatic jaw claudication, fever, and weight loss. Headache is the dominant symptom and often appears in association with malaise and muscle aches. Head pain may be unilateral or bilateral and is located temporally in 50% of patients but may involve any and all aspects of the cranium. Pain usually appears gradually over a few hours before peak intensity is reached; occasionally, it is explosive in onset. The quality of pain is only seldom throbbing; it is almost invariably described as dull and boring, with superimposed episodic stabbing pains similar to the sharp pains that appear in migraine. Most patients can recognize that the origin of their head pain is superficial, external to the skull, rather than originating deep within the cranium (the pain site for migraineurs). Scalp tenderness is present, often to a marked degree; brushing the hair or resting the head on a pillow may be impossible because of pain. Headache is usually worse at night and often aggravated by exposure to cold. Additional findings may include reddened, tender nodules or red streaking of the skin overlying the temporal arteries, and tenderness of the temporal or, less commonly, the occipital arteries.[5]

Cluster headache (Migrainous Neuralgia)

[7]

This is less common than migraine. There is a 5:1 predominance of males and onset is usually in the third decade. The characteristic syndrome comprises periodic, severe, unilateral periorbital pain accompanied by unilateral lacrimation, nasal congestion and conjunctival injection, often with the other features of Horner's syndrome. The pain, whilst being very severe, is characteristically brief (30-90 minutes). Typically, the patient develops these symptoms at a particular time of day (often in the early hours of the morning). The syndrome may occur repeatedly for a number of weeks, followed by a respite for a number of months before another cluster occurs

There is little genetic predisposition, no provoking dietary factors and a male predominance, which suggest a different aetiology from that of migraine, but this remains unknown. Patients are usually heavy smokers with a higher than average alcohol consumption.[5]

Glaucoma

Glaucoma may present with a prostrating headache associated with nausea and vomiting. The headache often starts with severe eye pain. On physical examination, the eye is often red with a fixed, moderately dilated pupil.

Meningitis

Acute, severe headache with stiff neck and fever suggests meningitis. Lumbar puncture is mandatory. Often there is striking accentuation of pain with eye movement. Meningitis can be easily mistaken for migraine in that the cardinal symptoms of pounding headache, photophobia, nausea, and vomiting are frequently present, perhaps reflecting the underlying biology of some of the patients.

Intracranial Hemorrhage

Acute, severe headache with stiff neck but without fever suggests subarachnoid hemorrhage. A ruptured aneurysm, arteriovenous malformation, or intraparenchymal hemorrhage may also present with headache alone. Rarely, if the hemorrhage is small or below the foramen magnum, the head CT scan can be normal. Therefore, lumbar puncture may be required to diagnose definitively subarachnoid hemorrhage.

Tension Headache

8

Typically occur on both sides, are not pounding, and are less disabling. This is the most common type of headache and is experienced at some time by the majority of the population in some form. The pain is usually constant and generalised but often radiates forward from the occipital region. It is described as 'dull', 'tight' or like a 'pressure', and there may be a sensation of a band round the head or pressure at the vertex. In contrast to migraine, the pain may continue for weeks or months without interruption, although the severity may vary, and there is no associated vomiting or photophobia. The patient can usually continue normal activities, and the pain may be less noticeable when the patient is occupied. The pain is characteristically less severe in the early part of the day and becomes more troublesome as

the day goes on. Local tenderness may be present over the skull vault or in the occiput but this should be distinguished from the acute pain precipitated by skin contact in trigeminal neuralgia and the exquisite tenderness of temporal arteritis. Typically, the headache is reported to be poorly responsive to ordinary analgesia. They are caused by excessive contraction of the muscles of the head and neck. Emotional strain or anxiety is a common precipitant to tension-type headache and there is sometimes an associated depressive illness. Anxiety about the headache itself may lead to continuation of symptoms, and patients often become convinced of a serious underlying condition.[5]

Neuralgic Headache

There are a number of rare headache syndromes which produce pains about the eye similar to cluster headaches. These include chronic and episodic paroxysmal hemicrania, and SUNCT (Short-lasting Unilateral Neuralgiform headaches with Conjunctival injection and Tearing).

Primary exertional headache

Primary exertional headache has features resembling both cough headache and migraine. It may be precipitated by any form of exercise; it often has the pulsatile quality of migraine. The pain, which can last from 5 min to 24 h, is bilateral and throbbing at onset; migrainous features may develop in patients susceptible to migraine. Primary exertional headache can be prevented by avoiding excessive exertion, particularly in hot weather or at high altitude.

The mechanism of primary exertional headache is unclear. Acute venous distension likely explains one syndrome, the acute onset of headache with straining and breath holding, as in weightlifter's headache. As exertion can result in headache in a number of serious underlying conditions, these must be considered in patients with exertional headache. Pain from angina may be referred to the head, probably by central connections of vagal afferents, and may present as exertional headache (cardiac cephalgia). The link to exercise is the main clinical clue that headache is of cardiac origin. Pheochromocytoma may occasionally cause exertional headache. Intracranial lesions and stenosis of the carotid arteries are other possible etiologies.

Post-traumatic headache

A traumatic event can trigger a headache process that lasts for many months or years after the event. The term trauma is used in a very broad sense: headache can develop following an injury to the head, but it can also develop after an infectious episode, typically viral meningitis, a flulike illness, or a parasitic infection. Complaints of dizziness, vertigo, and impaired memory can accompany the headache. Symptoms may remit after several weeks or persist for months and even years after the injury. Typically the neurologic examination is normal and CT or MRI studies are unrevealing. Chronic subdural hematoma may on occasion mimic this disorder. In one series, one-third of patients with NDPH reported headache beginning after a transient flulike illness characterized by fever, neck stiffness, photophobia, and marked malaise. Evaluation reveals no apparent cause for the headache. There is no convincing evidence that persistent Epstein-Barr infection plays a role in this syndrome. A complicating factor is that many patients undergo LP during the acute illness; iatrogenic low CSF volume headache must be considered in these cases. Posttraumatic headache may also

be seen after carotid dissection and subarachnoid hemorrhage, and following intracranial surgery. The underlying theme appears to be that a traumatic event involving the pain-producing meninges can trigger a headache process that lasts for many years.

Chronic daily headache

The broad diagnosis of chronic daily headache (CDH) can be applied when a patient experiences headache on 15 days or more per month. CDH is not a single entity; it encompasses a number of different headache syndromes, including headache secondary to trauma, inflammation, infection, medication overuse, and other causes.

Paroxysmal Hemicrania

Paroxysmal hemicrania (PH) is characterized by frequent unilateral, severe, short-lasting episodes of headache. Like cluster headache, the pain tends to be retroorbital but may be experienced all over the head and is associated with autonomic phenomena such as lacrimation and nasal congestion. Patients with remissions are said to have episodic PH, whereas those with the nonremitting form are said to have chronic PH. The essential features of PH are unilateral, very severe pain; short-lasting attacks (2–45 min); very frequent attacks (usually more than five a day); marked autonomic features ipsilateral to the pain; rapid course (<72 h). The male: female ratio in PH is close to 1:1.[5]

Brain tumor

Approximately 30% of patients with brain tumors consider headache to be their chief complaint. The head pain is usually nondescript—an intermittent deep, dull aching of moderate intensity, which may worsen with exertion or change in position and may be associated with nausea and vomiting. This pattern of symptoms results from migraine far more often than from brain tumor. Headache of brain tumor disturbs sleep in about 10% of patients. Vomiting that precedes the appearance of headache by weeks is highly characteristic of posterior fossa brain tumors. A history of amenorrhea or galactorrhea should lead one to question whether a prolactin-secreting pituitary adenoma (or the polycystic ovary syndrome) is the source of headache arising de novo in a patient with known malignancy suggests either cerebral metastases or carcinomatous meningitis, or both. Head pain appearing abruptly after bending, lifting, or coughing can be due to a posterior fossa mass, a Chiari malformation, or low CSF volume.[1]

Facial migraine

Unilateral episodic facial pain associated with symptoms suggestive of either migraine or cluster headache. It can be distinguished from migrainous neuralgia by longer duration of pain, lack of clustering, and frequent episodes of nausea and vomiting.

Retinal migraine

Loss of vision limited to one eye.

MANAGEMENT

Migraine can often be managed to some degree by a variety of approaches. Most patients benefit by the identification and avoidance of specific headache triggers. A regulated lifestyle is helpful, including a healthful diet, regular exercise, regular sleep patterns, avoidance of excess caffeine and alcohol, and avoidance of acute changes in stress levels.

The measures that benefit a given individual should be used routinely since they provide a simple, cost-effective approach to migraine management. Patients with migraine do not encounter more stress than headache -free individuals; over responsiveness to stress appears to be the issue. Since the stresses of everyday living cannot be eliminated, lessening one's response to stress by various techniques is helpful for many patients. These may include yoga, transcendental meditation, hypnosis, and conditioning techniques such as biofeedback.[5]

Acute Attack Therapies for Migraine

The mainstay of pharmacologic therapy is the judicious use of one or more of the many drugs that are effective in migraine. The selection of the optimal regimen for a given patient depends on a number of factors, the most important of which is the severity of the attack. Mild migraine attacks can usually be managed by oral agents; the average efficacy rate is 50–70%. Severe migraine attacks may require parenteral therapy. Most drugs effective in the treatment of migraine are members of one of three major pharmacologic classes: anti-inflammatory agents, 5-$HT_{1B/1D}$ receptor agonists, and dopamine receptor antagonists

Preventive Treatments for Migraine

Patients with an increasing frequency of migraine attacks, or with attacks that are either unresponsive or poorly responsive to abortive treatments, are good candidates for preventive agents. In general, a preventive medication should be considered in the subset of patients with five or more attacks a month. The mechanism of action of these drugs is unclear; it seems likely that the brain sensitivity that underlies migraine is modified. Patients are usually started on a low dose of a chosen treatment; the dose is then gradually increased, up to a reasonable maximum to achieve clinical benefit.

PROGNOSIS

Most people with migraines have periods of lost productivity due to their disease however typically the condition is fairly benign and is not associated with an increased risk of death.[5]

There are four main patterns to the disease: symptoms can resolve completely, symptoms can continue but become gradually less with time, symptoms may continue at the same frequency and severity, or attacks may become worse and more frequent.

Migraines with aura appear to be a risk factor for ischemic stroke doubling the risk. Being a young adult, being female, using hormonal contraception, and smoking further increases this risk. . Migraines without aura do not appear to be a factor. Overall however migraines do not appear to increase the risk of death from stroke or heart disease. Preventative therapy of migraines in those with migraines with auras may prevent associated strokes.[5]

HOMOEOPATHIC THERAPEUTICS

The therapeutic indications of these medicines have been given below as described by **Dr. William Boericke.** Indications of other important medicines have also been described below.

Therapeutic Indications of Prescribed Medicines

- **NUX VOMICA**-Headache in occiput or over eyes, with vertigo; brain feels turning in a circle. Oversensitiveness. Vertigo, with momentary loss of consciousness. Intoxicated feeling; worse, morning, mental exertion, tobacco, alcohol, coffee, open air. Pressing pain on vertex, as if a nail driven in. Vertigo in morning and after dinner. Scalp sensitive. Frontal headache, with desire to press the head against something. Congestive headache, associated with haemorrhoids. Headache in the sunshine. Feels distended and sore within, after a debauch.
- **NATRIUM MURIATICUM**- Blinding headache. Aches as if a thousand little hammers were knocking on the brain, in the morning on awakening, after menstruation, from sunrise to sunset. Feels too large; cold. Anaemic headache of school-girls; nervous, discouraged, broken down. Chronic headache, semi-lateral, congestive, from sunrise to sunset, with pale face, nausea, vomiting; periodical; from eyestrain; menstrual. Before attack, numbness and tingling in lips, tongue and nose, relieved by sleep.
- **PULSATILLA PRATENSIS**- The disposition and mental state are the chief guiding symptoms to the selection of Pulsatilla. It is pre-eminently a female remedy, especially for mild, gentle, yielding disposition. Sad, crying readily; weeps when talking; changeable, contradictory. The patient seeks the open air; always feels better there, even though he is chilly. Mucous membranes are all affected Symptoms ever changing. Thirstless, peevish, and chilly. When first serious impairment of health is referred to age of puberty. Great sensitiveness. Wants the head high. Feels uncomfortable with only one pillow. Lies with hands above head.Wandering stitches about head; pains extend to face and teeth; vertigo; better in open air. Frontal and supra-orbital pains. Neuralgic pains, commencing in right temporal region, with scalding lachrymation of affected side. Headache from overwork. Pressure on vertex.

- **SEPIA OFFICINALIS**-Stinging pain from within outward and upward mostly left, or in forehead, with nausea, vomiting; worse indoors and when lying on painful side. Jerking of head backwards and forwards. Coldness of vertex. Headache in terrible shocks at menstrual nisus, with scanty flow. Open fontanels. Roots of hair sensitive. Vertigo, with sensation of something rolling round in head. Mentally Sep. is indifferent to those loved best. Averse to occupation, to family. Irritable; easily offended. Dreads to be alone. Very sad. Weeps when telling symptoms. Miserly. Anxious toward evening; indolent.
- **BELLADONNA**- Pain; fullness, especially in forehead, also occiput, and temples. Headache from suppressed catarrhal flow. Sudden outcries. Pain worse light, noise, jar, lying down and in afternoon; better by pressure and semi-erect posture. Boring of head into pillow; drawn backward and rolls from side to side. Constant moaning. Hair splits; is

dry and comes out. Headache worse on right side and when lying down; ill effects, colds, etc.; from having haircut. Vertigo, with falling to left side or backwards. Sensitive to least contact. Much throbbing and heat. Palpitation reverberating in head with labored breathing

- **SANGUINARIA CANADENSIS**-Pain head, worse right side, sun headache. Periodical sick headache; pain begins in occiput, spreads upwards, and settles over eyes, especially right. Veins and temples are distended. Pain better lying down and sleep. Headaches return at climacteric; every seventh day. Pain in small spot over upper left parietal bone. Burning in eyes. Pain in the back of head "like a flash of lightning".
- **SPIGELIA ANTHELMIA**-Pain beneath frontal eminence and temples, extending to eyes. Semi- lateral, involving left eye; pain violent, throbbing; worse, making a false step. Pain as if a band around head.
- **SULPHUR**-Constant heat on top of head. Heaviness and fullness, pressure in temples. Beating headache; worse, stooping, and with vertigo. Sick headache recurring periodically.
- **ANACARDIUM ORIENTALE**- Pressing pain head, as from a plug; worse after mental exertion - in forehead; occiput, temples, vertex; better during a meal.
- **BRYONIA ALBA**-Dizzy or Faint on Rising up. Vertigo felt in occiput. Bursting; Splitting or Heavy crushing headache; fronto-occipital agg. Moving eyes, coughing, straining at stools etc. Vertigo; as though all objects are whirling, or as if sinking deep down in the bed, amel. cold. Pain over left eye, pressive, going to occiput, thence spreading over whole body. Scalp very sensitive, cannot bear even a soft brush, every hair pains. Headache; from ironing; when constipated.
- **SILICEA TERRA**- Ascending occipital pains amel. pressure. Periodical headaches. Headache, then blindness. Vertex throbs. Fontanelles open, with distended abdomen. Profuse sweat on head. Headache agg. by exertion, study, noise, motion, jar, light, cold air, talking and straining at stools, and amel. by wrapping warmly, and pressure. Headache while fasting or when not eating at proper time. Migraine. Chronic headaches since some severe disease. Vertigo; ascends from dorsal region agg. looking upwards, closing eyes, lying on left side.
- **CINCHONA OFFICINALIS**-Pain head.Sensation as if the skull would burst.Intense throbbing in the head. Worse by contact, current of air, stepping. Relievedfrompressure, warm room[9].

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