Migraine

A **migraine** is a primary headache disorder characterized by recurrent headaches that are moderate to severe.[1] Typically, the headaches affect one half of the head, are pulsating in nature, and last from a few hours to 3 days.[1] Associated symptoms may include nausea, vomiting, and sensitivity to light, sound, or smell.[2] The pain is generally made worse by physical activity.[12] Up to one-third of people affected have an aura: typically a short period of visual disturbance that signals that the headache will soon occur.[12] Occasionally, an aura can occur with little or no headache following it.[13]

Migraines are believed to be due to a mixture of environmental and genetic factors.[3] About two-thirds of cases run in families.[5] Changing hormone levels may also play a role, as migraines affect slightly more boys than girls before puberty and two to three times more women than men.[4][14] The risk of migraines usually decreases during pregnancy and after menopause.[4][15] The underlying mechanisms are not fully known.[15] They are, however, believed to involve the nerves and blood vessels of the brain.[5]

Initial recommended treatment is with simple pain medication such as ibuprofen and paracetamol (acetaminophen) for the headache, medication for the nausea, and the avoidance of triggers.[10] Specific medications such as triptans or ergotamines may be used in those for whom simple pain medications are not effective.[5] Caffeine may be added to the above.[16] A number of medications are useful to prevent attacks including metoprolol, valproate, and topiramate.[8][9]

Globally, approximately 15% of people are affected by migraines.[11] It most often starts at puberty and is worst during middle age.[1] As of 2016, it is one of the most common causes of disability.[17] An early description consistent with migraines is contained in the Ebers papyrus, written around 1500 BCE in ancient Egypt.[18] The word "migraine" is from the Greek ἡμικρανία (*hemikrania*), "pain on one side of the head",[19] from ἡμι- (*hemi-*), "half", and κρανίον (*kranion*), "skull".[20]

Migraine

Woman with migraine headache

Specialty	Neurology
Symptoms	Headaches, nausea, sensitivity to light, sensitivity to sound, sensitivity to smell[1][2]
Usual onset	Around puberty[1]
Duration	Recurrent, long term[1]
Causes	Environmental and genetic[3]
Risk factors	Family history, female[4][5]
Differential diagnosis	Subarachnoid hemorrhage, venous thrombosis, idiopathic intracranial hypertension, brain tumor, tension headache, sinusitis,[6] cluster headache[7]
Prevention	Metoprolol, valproate, topiramate[8][9]
Medication	Ibuprofen, paracetamol (acetaminophen), triptans, ergotamines[5][10]
Frequency	~15%[11]

Contents

Signs and symptoms

Migraines typically present with self-limited, recurrent severe headache associated with autonomic symptoms.[5][21] About 15–30% of people with migraines experience them with an aura[10][22] and they also frequently have migraines without aura.[23] The severity of the pain, duration of the headache, and

frequency of attacks are variable.[5] A migraine lasting longer than 72 hours is termed status migrainosus.[24] There are four possible phases to a migraine, although not all the phases are necessarily experienced:[12]

- The prodrome, which occurs hours or days before the headache
- The aura, which immediately precedes the headache
- The pain phase, also known as headache phase
- The postdrome, the effects experienced following the end of a migraine attack

Migraines are associated with major depression, bipolar disorder, anxiety disorders, and obsessive compulsive disorder. These psychiatric disorders are approximately 2–5 times more common in people without aura, and 3–10 times more common in people with aura.[25]

Prodrome phase

Prodromal or premonitory symptoms occur in about 60% of those with migraines,[2][26] with an onset that can range from two hours to two days before the start of pain or the aura.[27] These symptoms may include a wide variety of phenomena,[28] including altered mood, irritability, depression or euphoria, fatigue, craving for certain food(s), stiff muscles (especially in the neck), constipation or diarrhea, and sensitivity to smells or noise.[26] This may occur in those with either migraine with aura or migraine without aura.[29] Neuroimaging indicates the limbic system and hypothalamus as the origin of prodromal symptoms in migraine.[30]

Aura phase

An aura is a transient focal neurological phenomenon that occurs before or during the headache.[2] Auras appear gradually over a number of minutes and generally last less than 60 minutes.[31] Symptoms can be visual, sensory or motor in nature and many people experience more than one.[32] Visual effects occur most frequently; they occur in up to 99% of cases and in more than 50% of cases are not accompanied by sensory or motor effects.[32]

Vision disturbances often consist of a scintillating scotoma (an area of partial alteration in the field of vision which flickers and may interfere with a person's ability to read or drive).[2] These typically start near the center of vision and then spread out to the sides with zigzagging lines which have been described as looking like fortifications or walls of a castle.[32] Usually the lines are in black and white but some people also see colored lines.[32] Some people lose part of their field of vision known as hemianopsia while others experience blurring.[32]

Sensory aura are the second most common type; they occur in 30–40% of people with auras.[32] Often a feeling of pins-and-needles begins on one side in the hand and arm and spreads to the nose–mouth area on the same side.[32] Numbness usually occurs after the tingling has passed with a loss of position sense.[32] Other symptoms of the aura phase can include speech or language disturbances, world

spinning, and less commonly motor problems.[32] Motor symptoms indicate that this is a hemiplegic migraine, and weakness often lasts longer than one hour unlike other auras.[32] Auditory hallucinations or delusions have also been described.[33]

Pain phase

Classically the headache is unilateral, throbbing, and moderate to severe in intensity.[31] It usually comes on gradually[31] and is aggravated by physical activity.[12] In more than 40% of cases, however, the pain may be bilateral and neck pain is commonly associated with it.[34] Bilateral pain is particularly common in those who have migraines without an aura.[2] Less commonly pain may occur primarily in the back or top of the head.[2] The pain usually lasts 4 to 72 hours in adults,[31] however in young children frequently lasts less than 1 hour.[35] The frequency of attacks is variable, from a few in a lifetime to several a week, with the average being about one a month.[36][37]

The pain is frequently accompanied by nausea, vomiting, sensitivity to light, sensitivity to sound, sensitivity to smells, fatigue and irritability.[2] In a basilar migraine, a migraine with neurological symptoms related to the brain stem or with neurological symptoms on both sides of the body,[38] common effects include a sense of the world spinning, light-headedness, and confusion.[2] Nausea occurs in almost 90% of people, and vomiting occurs in about one-third.[39] Many thus seek a dark and quiet room.[39] Other symptoms may include blurred vision, nasal stuffiness, diarrhea, frequent urination, pallor, or sweating.[40] Swelling or tenderness of the scalp may occur as can neck stiffness.[40] Associated symptoms are less common in the elderly.[41]

Rarely, an aura occurs without a subsequent headache.[32] This is known as an acephalgic migraine or silent migraine; however, it is difficult to assess the frequency of such cases because people who do not experience symptoms severe enough to seek treatment may not realize that anything unusual is happening to them and dismiss it without reporting any problems.

Postdrome

The migraine postdrome could be defined as that constellation of symptoms occurring once the acute headache has settled.[42] Many report a sore feeling in the area where the migraine was, and some report impaired thinking for a few days after the headache has passed. The person may feel tired or "hung over" and have head pain, cognitive difficulties, gastrointestinal symptoms, mood changes, and weakness.[43] According to one summary, "Some people feel unusually refreshed or euphoric after an attack, whereas others note depression and malaise."[44] For some individuals this can vary each time.

Cause

The underlying causes of migraines are unknown.[45] However, they are believed to be related to a mix of environmental and genetic factors.[3] They run in families in about two-thirds of cases[5] and rarely occur due to a single gene defect.[46] While migraines were once believed to be more common in those of high intelligence, this does not appear to be true.[47] A number of psychological conditions are associated, including depression, anxiety, and bipolar disorder,[48] as are many biological events or triggers.

Genetics

Studies of twins indicate a 34% to 51% genetic influence of likelihood to develop migraine headaches.[3] This genetic relationship is stronger for migraines with aura than for migraines without aura.[23] A number of specific variants of genes increase the risk by a small to moderate amount.[46]

Single gene disorders that result in migraines are rare.[46] One of these is known as familial hemiplegic migraine, a type of migraine with aura, which is inherited in an autosomal dominant fashion.[49][50] Four genes have been shown to be involved in familial hemiplegic migraine.[51] Three of these genes are involved in ion transport.[51] The fourth is an axonal protein associated with the exocytosis complex.[51] Another genetic disorder associated with migraine is CADASIL syndrome or cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy.[2] One meta-analysis found a protective effect from an angiotensin converting enzyme polymorphisms on migraine.[52] The *TRPM8* gene, which codes for a cation channel, has been linked to migraines.[53]

Triggers

Migraines may be induced by triggers, with some reporting it as an influence in a minority of cases[5] and others the majority.[54] Many things such as fatigue, certain foods, and weather have been labeled as triggers; however, the strength and significance of these relationships are uncertain.[54][55] Most people with migraines report experiencing triggers.[56] Symptoms may start up to 24 hours after a trigger.[5]

Physiological aspects

Common triggers quoted are stress, hunger, and fatigue (these equally contribute to tension headaches).[54] Psychological stress has been reported as a factor by 50 to 80% of people.[57] Migraines have also been associated with post-traumatic stress disorder and abuse.[58] Migraines are more likely to occur around menstruation.[57] Other hormonal influences, such as menarche, oral contraceptive use, pregnancy, perimenopause, and menopause, also play a role.[59] These hormonal influences seem to play a greater role in migraine without aura.[47] Migraines typically do not occur during the second and third trimesters of pregnancy, or following menopause.[2]

Dietary aspects

Between 12 and 60% of people report foods as triggers.[60][61] Evidence for such triggers, however, mostly relies on self-reports and is not rigorous enough to prove or disprove any particular trigger.[62] A clear explanation for why food might trigger migraines is also lacking.[60]

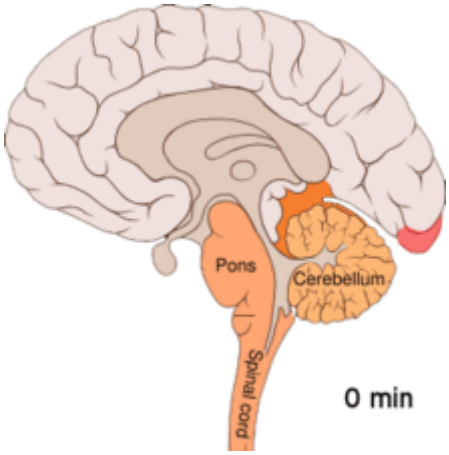
There does not appear to be evidence for an effect of tyramine – which is naturally present in chocolate, alcoholic beverages, most cheeses and processed meats – on migraine.[63] Likewise, while monosodium glutamate (MSG) is frequently reported,[64] evidence does not consistently support that it is a dietary trigger.[65]

Environmental aspects

A review on potential triggers in the indoor and outdoor environment concluded that there is insufficient evidence to confirm environmental factors as causing migraines. They nevertheless suggested that people with migraines take some preventive measures related to indoor air quality and lighting.[66]

Pathophysiology

Migraines are believed to be a neurovascular disorder,[5] with evidence supporting a mechanism starting within the brain and then spreading to the blood vessels.[67] Some researchers believe neuronal mechanisms play a greater role,[68] while others believe blood vessels play the key role.[69] Others believe both are likely important.[70] One theory is related to increased excitability of the cerebral cortex and abnormal control of pain neurons in the trigeminal nucleus of the brainstem.[71]



Animation of cortical spreading depression

Adenosine, a neuromodulator, may be involved.[72] Released after the progressive cleavage of adenosine triphosphate (ATP), adenosine acts on adenosine receptors to put the body and brain in a low activity state by dilating blood vessels and slowing the heart rate, such as before and during the early stages of sleep. Adenosine levels have been found to be high during migraine attacks.[72][73] Caffeine's role as an inhibitor of adenosine may explain its effect in reducing migraine.[74] Low levels of the neurotransmitter serotonin, also known as 5-hydroxytryptamine (5-HT), are also believed to be involved.[75]

Aura

Cortical spreading depression, or *spreading depression* according to Leão, is a burst of neuronal activity followed by a period of inactivity, which is seen in those with migraines with an aura.[76] There are a number of explanations for its occurrence, including activation of NMDA receptors leading to calcium entering the cell.[76] After the burst of activity, the blood flow to the cerebral cortex in the area affected is decreased for two to six hours.[76] It is believed that when depolarization travels down the underside of the brain, nerves that sense pain in the head and neck are triggered.[76]

Pain

The exact mechanism of the head pain which occurs during a migraine is unknown.[77] Some evidence supports a primary role for central nervous system structures (such as the brainstem and diencephalon),[78] while other data support the role of peripheral activation (such as via the sensory nerves that surround blood vessels of the head and neck).[77] The potential candidate vessels include dural arteries, pial arteries and extracranial arteries such as those of the scalp.[77] The role of vasodilatation of the extracranial arteries, in particular, is believed to be significant.[79]

Diagnosis

The diagnosis of a migraine is based on signs and symptoms.[5] 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.[80] It is believed that a substantial number of people with the condition remain undiagnosed.[5]

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, 1 criteria":[12]

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

- Four hours to three days in duration
- Two or more of the following:
  - Unilateral (affecting half the head)
  - Pulsating
  - Moderate or severe pain intensity
  - Worsened by or causing avoidance of routine physical activity
- One or more of the following:
  - Nausea and/or vomiting;
  - 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.[81] 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%.[10] In those with fewer than three of these symptoms the probability is 17%.[10]

Classification

Migraines were first comprehensively classified in 1988.[23] The International Headache Society updated their classification of headaches in 2004.[12] A third version was published in 2018.[82] According to this classification migraines are primary headaches along with tension-type headaches and cluster headaches, among others.[83]

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. Less commonly, an aura can occur without a headache, or with a nonmigraine headache. 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", 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. Now that this mechanism is not believed to be primary, the symptomatic term migraine with brainstem aura (MBA) is preferred.[38]
- 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.
- *Probable migraine* describes conditions that have some characteristics of migraines, but where there is not enough evidence to diagnose it as a migraine with certainty (in the presence of concurrent medication overuse).

- *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. Specifically, greater or equal to 15 days/month for longer than 3 months.[84]

Abdominal migraine

The diagnosis of abdominal migraines is controversial.[85] Some evidence indicates that recurrent episodes of abdominal pain in the absence of a headache may be a type of migraine[85][86] or are at least a precursor to migraines.[23] These episodes of pain may or may not follow a migraine-like prodrome and typically last minutes to hours.[85] They often occur in those with either a personal or family history of typical migraines.[85] Other syndromes that are believed to be precursors include cyclical vomiting syndrome and benign paroxysmal vertigo of childhood.[23]

Differential diagnosis

Other conditions that can cause similar symptoms to a migraine headache include temporal arteritis, cluster headaches, acute glaucoma, meningitis and subarachnoid hemorrhage.[10] Temporal arteritis typically occurs in people over 50 years old and presents with tenderness over the temple, cluster headaches presents with one-sided nose stuffiness, tears and severe pain around the orbits, acute glaucoma is associated with vision problems, meningitis with fevers, and subarachnoid hemorrhage with a very fast onset.[10] Tension headaches typically occur on both sides, are not pounding, and are less disabling.[10]

Those with stable headaches that meet criteria for migraines should not receive neuroimaging to look for other intracranial disease.[87][88][89] This requires that other concerning findings such as papilledema (swelling of the optic disc) are not present. People with migraines are not at an increased risk of having another cause for severe headaches.

Prevention

Preventive treatments of migraines include medications, nutritional supplements, lifestyle alterations, and surgery. Prevention is recommended in those who have headaches more than two days a week, cannot tolerate the medications used to treat acute attacks, or those with severe attacks that are not easily controlled.[10] Recommended lifestyle changes include stopping tobacco use and receding behaviors that interfere with sleep.[90]

The goal is to reduce the frequency, painfulness, and duration of migraines, and to increase the effectiveness of abortive therapy.[91] Another reason for prevention is to avoid medication overuse headache. This is a common problem and can result in chronic daily headache.[92][93]

Medication

Preventive migraine medications are considered effective if they reduce the frequency or severity of the migraine attacks by at least 50%.[94] Guidelines are fairly consistent in rating topiramate, divalproex/sodium valproate, propranolol, and metoprolol as having the highest level of evidence for first-line use.[95] Propranolol has the best evidence in children.[90]

Recommendations regarding effectiveness varied however for gabapentin and pregabalin.[95] Timolol is also effective for migraine prevention and in reducing migraine attack frequency and severity, while frovatriptan is effective for prevention of menstrual migraine.[95] Tentative evidence also supports the use of magnesium supplementation.[96] Increasing dietary intake may be better.[97]

Amitriptyline and venlafaxine are probably also effective.[98] Angiotensin inhibition by either an angiotensin-converting enzyme inhibitor or angiotensin II receptor antagonist may reduce attacks.[99]

Medications in the anti-calcitonin gene-related peptide, including eptinezumab, erenumab, fremanezumab, and galcanezumab, appear to decrease the frequency of migraines by one to two per month.[100] They are, however, expensive: a year of erenumab costs $6,900 as of 2019.[101]

Alternative therapies

Acupuncture has a small effect in reducing the number of migraines, compared to sham acupuncture, a practice where needles are placed randomly or do not penetrate the skin.[102] Physiotherapy, massage and relaxation, and chiropractic manipulation might be as effective as propranolol or topiramate in the prevention of migraine headaches; however, the research had some problems with methodology.[103][104] Another review, however, found evidence to support spinal manipulation to be poor and insufficient to support its use.[105]

Tentative evidence supports the use of stress reduction techniques such as cognitive behavioral therapy, biofeedback, and relaxation techniques.[57] Regular physical exercise may decrease the frequency.[106] Of the alternative medicines, butterbur has the best evidence for its use.[107][108] However, unprocessed butterbur contains chemicals called pyrrolizidine alkaloids (PAs) which can cause liver damage, however there are versions that are PA free.[109] In addition, butterbur may cause allergic reactions in people who are sensitive to plants such as ragweed.[110] There is tentative evidence that coenzyme Q10 reduces migraine frequency.[111]

The supplement melatonin has little evidence to support its use as an add-on therapy for prevention and treatment of migraine.[112] The data on melatonin are mixed and certain studies have had negative results.[112] The reasons for the mixed findings are unclear but may stem from differences in study design and melatonin dosage.[112] Melatonin's possible mechanism of action in migraine are not completely clear, but may include improved sleep, direct action on melatonin receptors in the brain and anti-inflammatory properties.[112][113]

Devices and surgery

Medical devices, such as biofeedback and neurostimulators, have some advantages in migraine prevention, mainly when common anti-migraine medications are contraindicated or in case of medication overuse. Biofeedback helps people be conscious of some physiological parameters so as to control them and try to relax and may be efficient for migraine treatment.[114][115] Neurostimulation uses noninvasive or implantable neurostimulators similar to pacemakers for the treatment of intractable chronic migraines with encouraging results for severe cases.[116][117] A transcutaneous electrical nerve stimulator and a transcranial magnetic stimulator are approved in the United States for the prevention of migraines.[118][119] Migraine surgery, which involves decompression of certain nerves around the head and neck, may be an option in certain people who do not improve with medications.[120]

Management

There are three main aspects of treatment: trigger avoidance, acute symptomatic control, and medication for prevention.[5] Medications are more effective if used earlier in an attack.[5] The frequent use of medications may result in medication overuse headache, in which the headaches become more severe and more frequent.[12] This may occur with triptans, ergotamines, and analgesics, especially opioid analgesics.[12] Due to these concerns simple analgesics are recommended to be used less than three days per week at most.[121]

Analgesics

Recommended initial treatment for those with mild to moderate symptoms are simple analgesics such as nonsteroidal anti-inflammatory drugs (NSAIDs) or the combination of paracetamol (also known as acetaminophen), aspirin, and caffeine.[10] Several NSAIDs, including diclofenac and ibuprofen have evidence to support their use.[122][123] Aspirin can relieve moderate to severe migraine pain, with an effectiveness similar to sumatriptan.[124] Ketorolac is available in an intravenous formulation.[10]

Paracetamol, either alone or in combination with metoclopramide, is another effective treatment with a low risk of adverse effects.[125] Intravenous metoclopramide is also effective by itself.[126][127] In pregnancy, paracetamol and metoclopramide are deemed safe as are NSAIDs until the third trimester.[10]

Triptans

Triptans such as sumatriptan are effective for both pain and nausea in up to 75% of people.[5][128] When sumatriptan is taken with naproxen it works better.[129] They are the initially recommended treatments for those with moderate to severe pain or those with milder symptoms who do not respond to simple analgesics.[10] The different forms available include oral, injectable, nasal spray, and oral dissolving tablets.[5] In general, all the triptans appear equally effective, with similar side effects. However, individuals may respond better to specific ones.[10] Most side effects are mild, such as flushing; however, rare cases of myocardial ischemia have occurred.[5] They are thus not recommended for people with cardiovascular disease,[10] who have had a stroke, or have migraines that are accompanied by neurological problems.[130] In addition, triptans should be prescribed with caution for those with risk factors for vascular disease.[130] While historically not recommended in those with basilar migraines there is no specific evidence of harm from their use in this population to support this caution.[38] They are not addictive, but may cause medication-overuse headaches if used more than 10 days per month.[131]

Ergotamines

Ergotamine and dihydroergotamine are older medications still prescribed for migraines, the latter in nasal spray and injectable forms.[5][132] They appear equally effective to the triptans[133] and experience adverse effects that typically are benign.[134] In the most severe cases, such as those with status migrainosus, they appear to be the most effective treatment option.[134] They can cause vasospasm including coronary vasospasm and are contraindicated in people with coronary artery disease.[135]

Other

Intravenous metoclopramide, intravenous prochlorperazine, or intranasal lidocaine are other potential options.[10][127] Metoclopramide or prochlorperazine are the recommended treatment for those who present to the emergency department.[10][127] Haloperidol may also be useful in this group.[127][132] A single dose of intravenous dexamethasone, when added to standard treatment of a migraine attack, is associated with a 26% decrease in headache recurrence in the following 72 hours.[136] Spinal manipulation for treating an ongoing migraine headache is not supported by evidence.[137] It is recommended that opioids and barbiturates not be used due to questionable efficacy, addictive potential, and the risk of rebound headache.[10] There is tentative evidence that propofol may be useful if other measures are not effective.[138]

Children

Ibuprofen helps decrease pain in children with migraines and is the initially recommended treatment.[139][140] Paracetamol does not appear to be effective in providing pain relief.[139] Triptans are effective, though there is a risk of causing minor side effects like taste disturbance, nasal symptoms, dizziness, fatigue, low energy, nausea, or vomiting.[139] Ibuprofen should be used less than half the days in a month and triptans less than a third of the days in a month to decrease the risk of medication overuse headaches.[140]

Chronic migraine

Topiramate and botulinum toxin (Botox) have evidence in treating chronic migraine.[98][141] Botulinum toxin has been found to be useful in those with chronic migraines but not those with episodic ones.[142][143] The anti-CGRP monoclonal antibody erenumab was found in one study to decrease chronic migraines by 2.4 days more than placebo.[144]

Prognosis

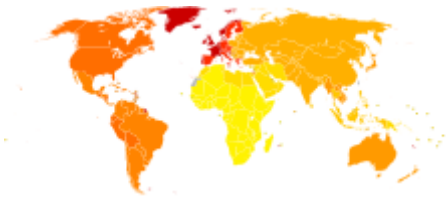
Long-term prognosis in people with migraines is variable.[21] Most people with migraines have periods of lost productivity due to their disease;[5] however typically the condition is fairly benign[21] and is not associated with an increased risk of death.[145] There are four main patterns to the disease: 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.[21]

Migraines with aura appear to be a risk factor for ischemic stroke[146] doubling the risk.[147] Being a young adult, being female, using hormonal birth control, and smoking further increases this risk.[146] There also appears to be an association with cervical artery dissection.[148] Migraines without aura do not appear to be a factor.[149] The relationship with heart problems is inconclusive with a single study supporting an association.[146] Overall however migraines do not appear to increase the risk of death from stroke or heart disease.[145] Preventative therapy of migraines in those with migraines with auras may prevent associated strokes.[150] People with migraines, particularly women, may develop higher than average numbers of white matter brain lesions of unclear significance.[151]

Epidemiology

Worldwide, migraines affect nearly 15% or approximately one billion people.[11] It is more common in women at 19% than men at 11%.[11] In the United States, about 6% of men and 18% of women get a migraine in a given year, with a lifetime risk of about 18% and 43% respectively.[5] In Europe, migraines

affect 12–28% of people at some point in their lives with about 6–15% of adult men and 14–35% of adult women getting at least one yearly.[14] Rates of migraines are slightly lower in Asia and Africa than in Western countries.[47][152] Chronic migraines occur in approximately 1.4 to 2.2% of the population.[153]



Disability-adjusted life year for migraines per 100,000 inhabitants in 2004

no data	145–165
<45	165–185
45–65	185–205
65–85	205–225
85–105	225–245
105–125	>245
125–145	

These figures vary substantially with age: migraines most commonly start between 15 and 24 years of age and occur most frequently in those 35 to 45 years of age.[5] In children, about 1.7% of 7 year olds and 3.9% of those between 7 and 15 years have migraines, with the condition being slightly more common in boys before puberty.[154] During adolescence migraines become more common among women[154] and this persists for the rest of the lifespan, being two times more common among elderly females than males.[155] In women migraines without aura are more common than migraines with aura, however in men the two types occur with similar frequency.[47]

During perimenopause symptoms often get worse before decreasing in severity.[155] While symptoms resolve in about two thirds of the elderly, in between 3 and 10% they persist.[41]

History

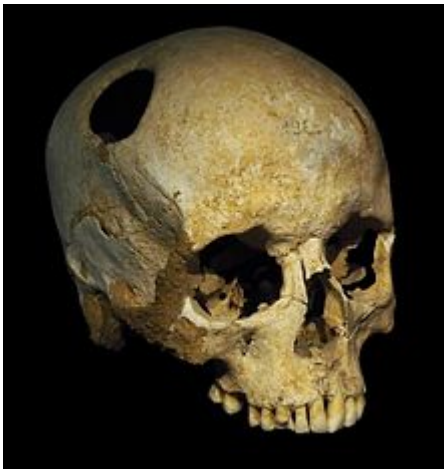
An early description consistent with migraines is contained in the Ebers papyrus, written around 1500 BCE in ancient Egypt.[18] In 200 BCE, writings from the Hippocratic school of medicine described the visual aura that can precede the headache and a partial relief occurring through vomiting.[156]

The Head Ache, George Cruikshank (1819)

A second-century description by Aretaeus of Cappadocia divided headaches into three types: cephalalgia, cephalea, and heterocrania.[157] Galen of Pergamon used the term hemicrania (half-head), from which the word migraine was eventually derived.[157] He also proposed that the pain arose from the meninges and blood vessels of the head.[156] Migraines were first divided into the two now used types – migraine with aura (*migraine ophthalmique*) and migraine without aura (*migraine vulgaire*) in 1887 by Louis Hyacinthe Thomas, a French Librarian.[156]

Trepanation, the deliberate drilling of holes into a skull, was practiced as early as 7,000 BCE.[18] While sometimes people survived, many would have died from the procedure due to infection.[158] It was believed to work via "letting evil spirits escape".[159] William Harvey recommended trepanation as a treatment for migraines in the 17th century.[160]

While many treatments for migraines have been attempted, it was not until 1868 that use of a substance which eventually turned out to be effective began.[156] This substance was the fungus ergot from which ergotamine was isolated in 1918.[161] Methysergide was developed in 1959 and the first triptan,



A trepanated skull, from the Neolithic. The perimeter of the hole in the skull is rounded off by ingrowth of new bony tissue, indicating that the person survived the operation.

sumatriptan, was developed in 1988.[161] During the 20th century with better study-design, effective preventive measures were found and confirmed.[156]

Society and culture

Migraines are a significant source of both medical costs and lost productivity. It has been estimated that they are the most costly neurological disorder in the European Community, costing more than €27 billion per year.[162] In the United States, direct costs have been estimated at $17 billion, while indirect costs — such as missed or decreased ability to work — is estimated at $15 billion.[163] Nearly a tenth of the direct cost is due to the cost of triptans.[163] In those who do attend work with a migraine, effectiveness is decreased by around a third.[162] Negative impacts also frequently occur for a person's family.[162]

Research

Calcitonin gene related peptides (CGRPs) have been found to play a role in the pathogenesis of the pain associated with migraine.[10] CGRP receptor antagonists, such as olcegepant and telcagepant, have been investigated both *in vitro* and in clinical studies for the treatment of migraine.[164] In 2011, Merck stopped phase III clinical trials for their investigational drug telcagepant.[165][166] Several CGRP monoclonal antibodies were approved and introduced in 2018. Transcranial magnetic stimulation shows promise[10][167] as does transcutaneous supraorbital nerve stimulation.[168] There is preliminary evidence that a ketogenic diet may help prevent episodic and long-term migraine.[169][170]

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External links

- Migraine (https://curlie.org//Health/Conditions_and_Diseases/Neurological_Disorders/Headaches/Migraine/) at Curlie
- 2019 Guideline on migraine prevention in children (https://n.neurology.org/content/early/2019/08/13/WNL.0000000000008105)
- 2019 Guideline on migraine treatment in children (https://n.neurology.org/content/early/2019/08/13/WNL.0000000000008095)

External audio

"Sex(ism), Drugs, and Migraines" (https://www.sciencehistory.org/distillations/podcast/sexism-drugs-and-migraines), Distillations Podcast and transcript, Episode 237, January 15, 2019, Science History Institute

Classification	**ICD-10**: G43 (http://apps.who.int/classifications/icd10/browse/2016/en#/G43) · **ICD-9-CM**: 346 (http://www.icd9data.com/getICD9Code.ashx?icd9=346) · **OMIM**: 157300 (https://omim.org/entry/157300) · **MeSH**: D008881 (https://www.nlm.nih.gov/cgi/

	mesh/2015/MB_cgi?field=uid&term=D008881) · **DiseasesDB**: 8207 (http://www.diseasesdatabase.com/ddb8207.htm)
External resources	**MedlinePlus**: 000709 (https://www.nlm.nih.gov/medlineplus/ency/article/000709.htm) · **eMedicine**: neuro/218 (https://emedicine.medscape.com/neuro/218-overview) neuro/517 (http://www.emedicine.com/neuro/topic517.htm#) emerg/230 (http://www.emedicine.com/emerg/topic230.htm#) neuro/529 (http://www.emedicine.com/neuro/topic529.htm#) · **Patient UK**: Migraine (https://patient.info/doctor/migraine-pro)