RESEARCH/RECHERCHE

Headache

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Among patients consulting their optometrist headache is often the chief complaint.

Headaches are symptoms rather than disease entities. In most cases objective signs are few.

The differential diagnosis of headache is based to a large extent on an adequate health-illness history. The various features of the headache itself together with any associated signs and symptoms often provide a guide to the probable cause or indicate the need for additional tests.

Popular wisdom is not a reliable guide to probable cause, potential seriousness or suitable treatment.

It is hoped that the attached table will supplement the optometrist's clinical assessment and help to identify the type and probable cause of many headaches.

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Table 1

Headache Class.	Pain Type	Pain Location	Pain Frequency	Pain Duration	Probable Cause	Helped by:	Comments
Migraine, classical type, migraine simplex. (Classical migraine occurs in about 3% of the population and constitutes 10% to 20% of all migraines.) (About 80% have a positive family history. A hereditary factor is present in more than 50% of those with migraine; possibly autosomal dominant inheritance.) (See common migraine and other forms of migraine.)	Sick headache; pain is dull, boring, throbbing. The contracting and expanding images of the aura alternate at the same rate as the brain wave rhythm, about 10 per second. More than 20% of all migraine episodes are not preceded by an aura, but an aura is usual in classical migraine. (Scintillating scotomas have occurred in patients taking digitalis. They also notice changes in color vision, both effects suggest that the dose is excessive.)	Frontal, behind the eye; often on the same side in each episode. Usually begins unilaterally but in 33% spreads all over the head. Head pain begins on the side opposite from the scotoma in most cases. The headache develops gradually about 20 minutes after the aura begins. (Traction on the retina can produce a sensation of sparkling or flashing lights which might be confused with the scintillating scotoma of migraine.)	Commonly begins in the teen-age years. Headache-free interval between episodes is usually more than two weeks and attacks generally occur at regular intervals. Very rarely develop status migrainosus i.e. almost daily headache. If episodes occur very frequently suspect over-use of ergotamine or that the headache is not migraine or not migraine alone. Headache frequency or severity may increase in early pregnancy. Many women but not all become free of their migraine during the second and third trimester of pregnancy.	Twelve to twenty-four hours. Onset often in early morning and tends to ease off at the end of the day. Three stages: (a) prodromal (b) heaache, nausea (c) post headache. May end with sleep or after vomiting or after a sudden increase in mental or physical activity. Some discomfort lingers after the acute phase probably because neurokinin or other mediators are released in the area of tissue edema. Increased plasma levels of gamma aminobutyric acid and cyclic adenosine monophosphate. Headaches tend to become less severe after 50 years of age especially in women.	Transient depression of the electrical activity in the brain. Vaso-motor instability. Increased vascular permeability. Vasoconstriction of branches of the internal carotid causes cerebral ischemia and produces the aura. Followed by dilatation of branches of the external carotid which produces the pain. Serotonin released from platelets causes intracerebral vasoconstriction and extracerebral vasodilatation. Prostaglandins especially E_1 cause extracranial vasodilatation and thromboxane causes cerebral vasoconstriction. Stres or mild trauma can precipitate on attack.	Ergotamine tartrate if given in the aura stage i.e. within first half hour. Rebreathing from a paper bag so as to increase the carbon dioxide level in the blood may help in the aura stage. Some drugs are useful as prophylactics e.g. cyproheptadine, or methysergide, or beta blockers including propranolol. Stop birth control pills, stop smoking. For status migrainosus may need narcotics, ergotamine, steroids, sedatives, major tranquilizers. Calcium blockers help by stabilizing the tone of the blood vessels. Correct the refractive error and ocular muscle imbalance.	Prodromes consisting of depression, fatigue, hyperactivity or mood changes often precede the attack. Aura usually visual (teichopsia, fortification figures) precedes headache by 20 to 30 minutes. Closing the eyes does not cause the scintillating scotoma to disappear. Headache, nausea, vomiting, diarrhea, and polyuria follow. Unilateral paresthesias occur in about 30% and some have other neurological signs or symptoms, e.g. aphasia and mental clouding. Conjunctival hyperemia, photophobia, anisocoria, excess lacrimation, blur, transient scotomas (usually-homonymous) and an enlarged blind spot are often present. Micropsia and macropsia have been reported.

Table 2

Headache Class.	Pain Type	Pain Location	Pain Frequency	Pain Duration	Probable Cause	Helped by:	Comments
Migraine, common type. (Constitutes about 25% of all severe headaches and 70% to 85% of all migraines.) (Migraine is probably the second most common cause of severe headache.) (At least 10% of the population have some type of migraine.) (A family history of migraine is reported by 80% of those with migraine.) (Epilepsy occurs more frequently in families with migraine. Migraine occurs more frequently in families with epilepsy.) (A migraine-like headache occurs in 70% of patients undergoing renal dialysis but the mechanism for this headache is uncertain.) (There are many variants of migraine. See classical and other forms of migraine.)	Sick headache; pain is dull, boring, throbbing. The pounding headache often begins slowly during the REM period of sleep. Migraine sufferers have been described as tense, obsessional, meticulous, perfectinioistic, intelligent, neat, sensitive, ambitious, emotionally restrained etc. but there are many exceptions to this type of categorization. Migraineurs are also said to have a low basal metabolic rate and to be relatively infertile. Cerebral edema leads to the gastrointestinal disturbance.	Unilateral or bilateral, anywhere in head often in the temple or frontal area but can alternate beginning on one side one time and on the other side in the next episode. Often seems to be all over the head not just unilateral.	Usually begins in the teen-age years. Occurs once every two weeks or less often. Headache episodes occur at less regular intervals than in classical migraine. Migraine is slightly more common in women than in men. In women with migraine 60% of the attacks occur in association with menstruation. Headache attacks tend to decrease in frequency after 50 years of age especially in women. Migraine usually diminishes after the menopause but rarely it worsens. Migraine almost never begins after 40 years of age.	Twelve to twenty-four or more hours. May awaken the patient in the morning. Many patients feel particularly well or energetic just before an attack. Pain-free interval between episodes. Locally released chemical substances lower the pain threshold near the blood vessels so some discomfort lingers after the acute phase. The headache usually ends with sleep.	Altered neurogenic activity or vasomotor instability. Increased amplitude of pulsation of cranial vessels. Studies show no changes in cerebral blood flow before, during or after an attack. Typically migraine requires a genetic predisposition, a specific personality pattern and a triggering event. Precipitating factors include: fatigue, trauma, stress, estrogens (the pill), "eyestrain", low-tension glaucoma, hunger, flashing lights, noise, and certain odors. During an attack the blood has less endorphins and 50% less serotonin but more platelet abnormalities.	Ergotamine tartrate if given early. Some drugs are useful as prophylactics e.g. cyproheptadine, methysergide, or propranolol and other beta blockers. Pressure on the common carotid eases the pain transiently. Stop birth control pills. The migraine sufferer seeks a quiet dark room and lies down with a cold cloth over the eyes. Migraine headaches often disappear during the middle trimester of pregnancy. Migraine headaches usually diminish with major illnesses or surgery. Correct the refractive error and ocular muscle imbalance.	Drowsiness or mood changes often precede the attack. Generally no visual aura but may have vague, constitutional disturbances hours or days before which the patient learns to interpret as a warning. Nausea, vomiting, light headedness, and polyuria accompany the headache. A few suffer amnesia, confusion, or even psychoses. Most notice fluid retention before the attack and 20% have diarrhea during the episode. Conjunctival hyperemia, photophobia, scotomas, and rarely vasospasm of retinal arterioles and blurred vision. A transient Horner syndrome sometimes appears. Migraine patients are sensitive to lights, sounds and smells and more apt to notice spatial disorientation when adapting to new glasses.
Ophthalmic migraine, retinal migraine, sometimes called ocular migraine, occipital migraine, or even senile migraine equivalent. (Do not confuse with ophthalmoplegic migraine or with Roy's ocular migraine.) (In some abortive forms the visual aura is not followed by a headache, this may be called acephalic migraine).	Unilateral visual impairment precedes the headache. (Jagged figures, Moore's lightning streaks, are seen by some elderly people with degenerative changes in their vitreous. These vertical streaks can be confused with the fortification figures of migraine.)	Periorbital, frontal, behind the eyes. Unilateral or bilateral.	Rare but some have repeated episodes. May develop in older patients who have had migraine for years or appear in younger patients with no family history of migraine.	About 30 minutes. (Do not confuse with transient ischemic attacks which are mostly due to emboli. TIAs are monocular events which last for only a few minutes.)	Vascular causes probably account for 80% of all chronic head-pain complaints. If the disturbance affects vessels supplying the anterior portion of the visual pathway the visual phenomena are monocular. Vasoconstriction involving the vessels supplying the calcarine cortex causes binocular visual effects which do not disappear when the eyes are closed.	Propranolol is effective. Be sure no other cause exists then correct the refractive error and ocular muscle imbalance. (Retinal detachment can cause the eye to see sparks, shooting stars, a fountain of spots, lights or colors which might be confused with the aura of migraine.)	Young adult females. Unilateral retinal dysfunction occurs especially during the aura phase. Scintillating scotomas but no fortification figure. Sometimes have hemorrhages in their conjunctiva, retina or rarely in the vitreous; ischemic papillitis. Retinal hypoxia causes transient loss of vision lasting up to 10 minutes. Eventually pigmentary changes appear in the retina.

Table 3

Headache Class.	Pain Type	Pain Location	Pain Frequency	Pain Duration	Probable Cause	Helped by:	Comments
Roy's ocular migraine, latent vertical phoria syndrome. (This less well known type was described by Raymond Roy.) (Do not confuse with ophthalmic migraine or ophthalmoplegic migraine.) (See muscle contraction headache.)	Headache may be mild, or severe. A pulling feeling in the eyes may precede the headache.	Unilateral or bilateral, anywhere in the head and almost always extends into the neck muscles.	Daily in afternoon or evening but in a few cases wakens the patient in the early morning.	Chronic, usually manifests before patient is 20 years old. Often these patients have sought help from many different practitioners. Migraine of some type accounts for nearly 33% of all lost work days due to sickness.	Latent hyperphoria, May have hypertophthalmia i.e. one eye is anatomically higher in the head than the other eye. Develop postcervical tension from head tilt. Stress results from the induced hyperphoria. May be inherited as a dominant characteristic.	Be sure no organic cause exists. Uncovering and correcting the hyperphoria. The hyperphoria can be uncovered by the monocular occlusion technique of Marlow, Jacques, and Charnwood. Prescribe absorptive lenses and base-in prism if necessary.	Chronic fatigue, nausea, motion sickness, torticollis, stiff neck, nervousness, depression, insomnia. Severe photophobia; tend to suppress or close one eye, blink frequently. Poor stereopsis, poor readers, subnormal amplitude of accommodation, transient scotomas.
Abdominal migraine, juvenile migraine, cyclical vomiting, pediatric migraine, childhood migraine, cyclic infantile colic. (Compare with other types of migraine.)	Abdominal discomfort. Abdominal symptoms are more prominent than headache until about ten years of age then the throbbing paroxysmal headache develops in the second decade. Sixty percent of adults with migraine report that they had motion sickness during childhood.	Pain in abdomen in childhood but later in life manifests as headache. However, some adults have episodes of abdominal symptoms even into middle age.	Abdominal symptoms begin on the average about the age of six years. Headache begins after ten years of age.	Each episode lasts 6 to 12 hours. Cyclical vomiting usually disappears by 8 to 10 years of age but is followed by development of headache episodes in the teen age years.	Familial tendency. Childhood migraine may be secondary to or confused with systemic conditions including hypoglycemia, anemia, upper respiratory tract infections, fever, meningitis.	ASA, rest and sleep. Rarely ergotamine at half the adult dose for a child over the age of 6 years, or methysergide 2 to 4 mg a day. Cyproheptadine 4 to 8 mg at bedtime. Some respond to anticonvulsant drugs e.g. phenobarbital 30 mg t.i.d. but not to ergotamine tartrate. Correct the refractive error and the ocular muscle imbalance.	Affects more boys than girls. Affected children often have motion sickness commonly called car sickness. Some have a visual aura, or dizziness, most have nausea and cyclical vomiting. In a few cases the child becomes confused during the episode.

Table 4

Headache Class.	Pain Type	Pain Location	Pain Frequency	Pain Duration	Probable Cause	Helped by:	Comments
Dietary migraine and allergic migraine. These are really two separate types of migraine. (See toxic and infectious headaches.)	Headache usually with nausea and visual problems. Specific foods precipitate an attack in 30% of those with migraine. In the Chinese restaurant syndrome due to monosodium glutamate (MSG) those affected have a transient episode of throbbing headache with light headedness and increased muscle tension in jaw, neck and shoulders.	Bilateral or rarely unilateral. Mostly frontal but can extend to the vertex or occiput. Flushing of the skin of the face and chest occurs.	If a specific food is responsible the problem appears each time that food is ingested. Seasonal in those cases where some forms of allergy are associated. Up to 25% of the population may be sensitive to MSG.	Allergic type may be worst when patient first awakens.	Exposure to specific foods but much disagreement exists as to cause. Commonest causes are wheat, citrus fruits (octopamine), eggs, nuts, meats, yeast, nitrites, nitrates, alcohol, red wine, aged cheese, milk, seafoods, pickled herring, or ingestion of other tyramine-containing foods or the beta phenylethylamine in chocolate. Monosodium glutamate and nitrates act as vasodilators. Fasting or hypoglycemia may be factors in some patients.	Appropriate diet will prevent the headache in 30 to 80% of these patients. Be wary of food faddists or self-appointed nutritionists. Clonidine is often helpful if a rise in blood pressure is involved. Correct the refractive error and ocular muscle imbalance.	Be sure there is no organic cause. Be wary of placebo effects. Migraine attacks may alternate with episodes of urticaria, asthma or angioneurotic edema. Susceptible patients often have have nasal congestion, rhinorrhea, sneezing, visual problems, photophobia and conjunctival hyperemia. Recent studies indicate that MSG may not be solely responsible for the skin flush.
Complicated migraine, hemiplegic migraine, hemisensory migraine, aphasic migraine, ophthalmoplegic migraine, periodic oculomotor paralysis, Mobius's disease. (Probably accounts for 1% of migraines.) (Shows an inherited tendency, autosomal dominant.) (See basilar artery migraine, ischemic cerebrovascular disease, retinal migraine, subarachnoid hemorrhage and the Tolosa-Hunt syndrome.)	The abrupt onset, dull, migraine-type headache usually develops before the ophthalmoplegia or other forms of motor or sensory dysfunction. The headache in most other types of migraine does not begin abruptly. Ophthalmoplegic migraine is rare but is considered to be a more severe or ominous variant of classical migraine.	Unilateral ache above or behind one eye. Headache plus numbness in limbs or face or paralysis of certain muscles. Symptoms of motor or sensory dysfunction. The weakness and sensory disturbances can be unilateral or bilateral, and are mostly ipsilateral to the headache. Unilateral paresthesias occur in 30% of migraineurs.	This rare form of periodic migraine tends to be familial and probably begins in childhood, but is most likely to be seen in the young adult. Usually have irregularly recurring episodes over many years. The interval between attacks can be as long as five years.	Each episode lasts for only a few hours. All neurological signs usually clear within an hour if no organic lesion is present. Third nerve palsy of this type in a child usually clears in six weeks. Incomplete recovery is possible in hemiplegic migraine and the condition is then called complicated migraine and provides more cause for concern.	Edematous arterial walls impinge on nearby nerves. A predisposing cause seems to be cerebral ischemia due to focal arterial spasm. Suspect an aneurysm until this is ruled out. Ipsilateral herniation of the hippocampal gyrus of the temporal lobe through the incisura tentorii is one cause. There is an elevated level of gamma-aminobutyric acid in the cerebrospinal fluid and the platelets are hyperaggregable.	Be sure there is no organic cause. A CT scan often provides useful diagnostic information but cannot reveal very small aneurysms. May need an angiogram especially if epilepsy occurs too but the risks associated with this procedure are even greater in these patients. Prednisone helps if administered promptly. Correct the refractive error and ocular muscle imbalance.	Mostly young adults. No nausea. Ocular palsy occurs in 17% of all migraine sufferers and of these 80% involve the third nerve which is close to the internal carotid and middle cerebral arteries, or is compressed between the posterior cerebral and superior cerebellar arteries. Can affect cranial nerves III, IV, VI or any part of V. Transient speech defects or impairment of the sense of smell occur only rarely. Some have field losses, ptosis, diplopia, muscle weakness or paralysis, pupillary dysfunction. More than 33% have a fixed dilated pupil.

Table 5

Headache Class.	Pain Type	Pain Location	Pain Frequency	Pain Duration	Probable Cause	Helped by:	Comments
Cluster headache, Harris' periodic neuralgia, histaminic cephalalgia, migrainous neuralgia, Horton's syndrome, Bing's syndrome, Kunkle's cluster headache. (Cluster headaches affect 0.5% of the population and constitute up to 4% of the severe headaches. About 33% have a family history of similar headaches.) (See Charlin's syndrome, both may be called ciliary neuralgia.) (See Sluder's syndrome, both may be called sphenopalatine ganglion neuralgia.) (See carotidynia, Raeder's syndrome and chronic paroxysmal hemicrania.)	The abrupt onset headache is intense, boring, burning. Victim may cry, moan, yell, pace the floor, bang the head, even roll on the floor. More severe pain than in most migraine headaches. Occipital pain may be carried by CNIX and X and the upper cervical nerves. (An atypical cluster headache may be mild or severe, and last hours, days, or even persist for weeks. Some of these patients have hypertension, arteriosclerosis, or infections. Trauma is a cause of some headaches of this type.)	Unilateral near one temple. Pain seems to follow the distribution of the external carotid artery. Nearly always pain is on the same side of the head in each attack. When the pain is supraorbital the condition is referred to as the upper syndrome. In the lower syndrome the pain is infraorbital. The nervus intermedius (Wrisberg) a part of CNVII is the main conduit for cluster headaches, it carries parasympathetic fibers. (In the atypical type the pain follows the distribution of the trigeminal nerve, CNV.)	Attacks tend to develop late in the second decade of life. They have two to five episodes a day for several consecutive weeks, then a clear interval of months or years followed by another cluster of attacks. The mean remission period between clusters is one year. A few people have little or no pain-free interval between headache episodes so have atypical or chronic cluster headaches.	Most of these headache episodes last less than one hour. Often they awaken the patient from sleep between midnight and two a.m. They are associated with the REM period of sleep.	The upper syndrome is due to dilatation of the external carotid and its branches. The lower syndrome is due to dilatation of other large arteries e.g. internal carotid, vertebral, basilar. An "autonomic storm" involving visceromotor centers in the brain has been blamed. Most are smokers, and use alcohol. Worsened by alcohol ingestion or by lying down. Seasonal and allergic factors but few emotional ones have been reported. Is growth hormone related? Cluster headaches are more common in those with hazel irides.	Methysergide is a useful prophylactic. Ergotamine tartrate in low doses can also be useful prophylactically but unless given IV or as an aerosol it is too slow acting to treat an acute episode. Some use prednisone, oxygen, vasoconstrictors, indomethacin, or lithium carbonate. The pain is transiently eased by pressure on the ipsilateral temporal artery. Stop alcohol intake, stop smoking. Be sure it is not an episode of angleclosure glaucoma. Correct the refractive error and ocular muscle imbalance.	Mostly affects men 25 to 45 years of age. No prodromes, no trigger areas, no scotomata, no nausea. May have hyperalgesia of the face and scalp and tenderness over the dilated temporal artery. More risk of peptic ulcers. Said to be squarejawed, have coarse, rough, thick, facial skin and furrowed forehead. Have ipsilateral rhinorrhea and skin hyperemia, ipsilateral conjunctival hyperemia and chemosis. Edema around the eyes, increased lacrimation, sometimes miosis and ptosis. Often an ipsilateral, postganglionic Horner syndrome with the sweating mechanism intact. The small pupil is supersensitive to phenylephrine.

Chronic paroxysmal hemicrania is a variant of cluster headache.	Milder than cluster headache.	Unilateral.	More frequent episodes than are usual in cluster headache.	Episodic.	See cluster headache.	Indomethacin.	Rare, mostly affects young to middle-aged women.
Basilar artery migraine, vertebro-basilar migraine, Bickerstaff's migraine, basilar artery insufficiency syndrome, cramiocervical syndrome. (Over 40% of those with vertebro-basilar artery disease have headaches.) (See Ménière's disease, complicated migraine, post-traumatic headache, and ischemic cerebro-vascular disease.)	With occlusive disease of the vertebral-basilar system 50% develop a severe, throbbing headache. The visual disturbance is often followed by loss of consciousness.	Occipital and parietal; may have tender areas, stiff neck, back pain. May have facial numbness on one side and a motor loss on the other side of the body. An aneurysm of the basilar-vertebral vessels does not produce pain in the ocular region. The vertebro-basilar system supplies blood to the occipital cortex.	Episodic. Usual age of onset is the second or third decade. Often associated with menstruation but can occur in children. Transient ischemic attacks associated with vetebro-basilar insufficiency cause loss of muscle coordination, and homonymous hemianopia.	Brief paroxysms lasting 10 to 30 minutes. Persisting manifestations of these or similar symptoms suggest chronic vertebro-basilar insufficiency. Posterior cerebral insufficiency causes homonymous hemianopia with sparing of fixation. Basilar artery insufficiency causes dizziness, vertigo, drop attacks, diplopia and nystagmus but seldom causes death.	Ischemia of the reticular formation produced by spasm or occlusive disease of the basilar-vertebral arteries. Involvement of brainstem, pontine tegmentum, hypothalamus, thalamus, and occipital lobe. Can follow a whiplash injury or emotional stress. Can be worsened by turning head to one side. Complete thrombosis of the basilar artery can cause death.	Difficult to treat. Some patients are helped by anticonvulsants. Inhalation of 5% carbon dioxide has been recommended. Occlusion of the branches of the basilar artery which supply the pontine region produces the Raymond Céstan syndrome with quadriplegia, anesthesia and nystagmus.	Vasospasm is more apt to occur in young women. Some develop ataxia, tinnitus, vertigo, dysarthria, paresthesias, drop attacks, dysphagia, confusion as well as sensory and visual disturbances. Nausea and vomiting often occur and so can loss of consciousness or hemiplegia. Bilateral graying or blurring of vision, scotomas, bilateral homonymous hemianopia, usually lasting under 5 minutes. Vertical nystagmus, ptosis, transient diplopia, reduced accommodation, cortical blindness.

Table 6

Headache Class.	Pain Type	Pain Location	Pain Frequency	Pain Duration	Probable Cause	Helped by:	Comments
Quincke's angioneurotic edema. (Sometimes inherited as an autosomal dominant.) (See the Melkersson-Rosenthal syndrome.)	Headache.	Cerebral edema is responsible for the headache. Lid edema can cause ptosis.	Sudden onset in young patients.	Brief recurring episodes at irregular intervals but condition may reappear for years.	Cause obscure. Patients fail to synthesize an inhibitor of complement C_1 or produce an abnormal protein.		Edema of skin and mucous membranes. Can involve the respiratory and gastrointestinal tracts. Bradycardia, facial paralysis, deafness, mental confusion, loss of consciousness, paralysis of CNIII. Pupillary anomalies, uveal involvement and rarely papilledema.
Headache with hypertension. (High blood pressure causes less than 5% of all headaches.) (Only 50% of those with hypertension have headaches.) (Hypertension often accompanies arteriolar-sclerosis, diabetes mellitus, kidney disorders and toxemia of pregnancy.)	Headaches are dull, diffuse, throbbing and only rarely paroxysmal. Become severe when cerebral edema develops. A few report a sensation of a tight band. Headaches of this type are most likely to be reported with a pheochromocytoma or with eclampsia.	Generalized, unilateral, frontal, occipital or at vertex; often at base of skull. Poorly localized.	Headache is worst in early morning and peaks just before the patient gets up. Often awakens the patient. Some prefer to sleep in a sitting position or to elevate the head of their bed.	Tends to wear off during the morning after patient gets up thereby helping blood to drain from the large intracranial venous sinuses. Some patients have this type of headache for years. With a pheochromocytoma 80% have a paroxysmal headache in the early morning which persists for about 30 minutes.	Elevated blood pressure. Only when diastolic pressure reaches about 120 mmHg is headache almost sure to occur. Often have cardiac or renal disease. Worsened by straining, coughing or stooping. Some develop sustained muscle contraction too.	Diuretics, clonidine, and beta blockers to lower blood pressure; more exercise, less salt, stopping smoking, losing weight; also eased by rest and relaxation. Transiently eased by pressure on the common carotid.	Many people with elevated blood pressure have no symptoms. A few report dizziness, light headedness or tinnitus. Hypertension is the cause of 10% of the muscle palsies which occur in older patients. Often retinal signs, especially arteriolar constriction or retinopathy or in severe cases papilledema. A venous pulse is not visible on the disc if the intracranial pressure is elevated.
Headache with temporal arteritis, cranial arteritis, giant cell arteritis, also called Horton's syndrome, the Hutchinson-Horton-Magath-Brown syndrome or Rumbold's disease. (See cluster headache.) (Polymyalgia rheumatica is a related condition which has only rarely been responsible for loss of vision.)	Severe, burning, steady or throbbing headache occurs in some but not all patients with this condition. Burning pain is an unusual feature for a vascular type headache. Headache of recent onset in an older patient should suggest temporal arteritis as one possibility.	Temple area, unilateral or bilateral, sometimes occipital. Scalp tenderness. Temporal artery is tender, enlarged, and sensitive. Pain when chewing due to jaw claudication. Jaw pain, trouble swallowing.	Temporal arteritis or giant cell arteritis is a rare but serious condition. Pain is usually worse at night.	Persistent headache is usual but the condition tends to clear in less than two years. Evidence of temporal arteritis is found in 1/1000 patients at autopsy but many had no related symptoms.	An immunologic vasculitis. The temporal artery is usually but not always involved. An involved temporal artery has a pulse which is less evident even though the artery appears to be prominent. Headache made worse by lying down, eased by upright position. The ischemic optic neuropathy is the result of occlusion of the posterior ciliary arteries not occlusion of the central retinal artery.	A true ocular emergency requiring immediate treatment. Steroids are required promptly. Prednisone 40 to 60 mg a day then taper off after about six weeks. Headache worsened by exposure to cold. A biopsy may be needed to confirm the diagnosis and strangely has been said to give some relief itself. The arterial involvement is patchy so a biopsy can be misleading. Temporal arteritis can kill.	Mostly females over 60 years of age. Ipsilateral temporal artery red, swollen and tender in 50% of cases. Anorexia, weight loss, arthralgia, elevated sedimentation rate, fever, weakness, malaise, fatigue, photophobia. Diplopia occurs in 10 to 15% and can be the result of damage to CNIII or VI. Temporal arteritis patients can lose vision in 1 to 4 weeks after onset of the disease. About 50% have severe visual loss which can become bilateral and permanent.

Table 7

Headache Class.	Pain Type	Pain Location	Pain Frequency	Pain Duration	Probable Cause	Helped by:	Comments
Headache with ischemic cerebrovascular disease. (About 30% have headaches.) (See basilar artery migraine, complicated migraine, carotid artery syndrome, stroke headache and carotidynia).	Some are throbbing, some are steady. Intensity varies but is often mild. If a visual hallucination occurs it will disappear when the eyes are closed if the circulation to the eye is impaired but not if the basilar-vertebral arteries are affected.	Frontal and ipsilateral to the affected vessel if the carotid is involved. Fleeting weakness of an arm or leg or transient sensory disturbances can occur with or without a vision disturbance.	Headache may precede or accompany the ischemic condition. Transient ischemic attacks usually indicate cerebrovascular disease, emboli, or impaired circulation but can be due to hematological disorders or elevated intraocular pressure.	Minutes, hours or days. Transient dimming or graying of vision, if it occurs, lasts for not over 10 minutes and can occur with no pain and no other motor or sensory disturbance.	Atherothrombiotic disease affecting the internal carotids or the vertebral arteries or their branches causes headache in 30% of those affected. If the carotid is affected look for a prominent pulse in the temporal artery.	Ophthalmodynamometry is used to aid in the diagnosis. Some can be helped by surgery, endarterectomy. Risk of stroke is high. Listen for a carotid bruit. Idiopathic orthostatic hypotension does not usually cause a headache.	Do not confuse these unilateral transient ischemic attacks (TIAs) with the aura of migraine. With a TIA some have slowly pulsating photopsias and develop confusion without loss of consciousness. The eye on the side of the affected carotid may show cholesterol crystals in retinal vessels or develop occlusion of arteries or veins.
Headache with a "stroke". Strokes are often called cerebrovascular accidents. (See carotid artery syndrome and ischemic cerebrovascular disease.)	Severe, sudden onset headache occurs in 25% of strokes. The lenticular striate artery, a branch of the middle cerebral artery has been called the artery of cerebral apoplexy.	Thrombosis of the internal carotid artery produces a sudden, unilateral headache. A thrombotic stroke affecting the internal capsule causes extensive paralysis but little intellectual impairment. A thrombotic lesion near the cerebral cortex causes less paralysis but always causes more intellectual impairment.	Occlusion of the anterior cerebral artery causes hemiplegia and hemiparesthesia of the contralateral leg. Occlusion of the middle cerebral artery can cause contralateral hemiplegia, hemiparesthesia of the contralateral face, head or arm, and homonymous hemianopia with splitting of fixation and usually anisocoria. The lower quadrants of the visual field are most likely to be affected.	Occlusion of the posterior cerebral artery causes a transient ischemic attack and homonymous hemianopia with sparing of fixation and sometimes causes an aura but seldom death.	About 50% are due to thrombotic occlusion of vessels, about 33% are due to hemorrhage and 10% are due to embolism.	Treatment is controversial. Permanent neurological damage usually follows and one third die soon after the episode.	Stiff neck, continuous vomiting, contralateral paralysis of one side of the body, speech disorders, blood in the spinal fluid, hemiparesthesia of the contralateral face, hand and arm.

Table 8

Headache Class.	Pain Type	Pain Location	Pain Frequency	Pain Duration	Probable Cause	Helped by:	Comments
Headache with the carotid artery syndrome, vascular insufficiency syndrome. (See stroke, carotidynia, ischemic cerebrovascular disease, ophthalmoplegic migraine and the Tolosa-Hunt syndrome.)	Severe unilateral headache with transient weakness or loss of sensation. Sensory or motor defects develop on the contralateral side. If the dominant hemisphere is involved speech will be impaired. If a visual hallucination occurs it will not be seen when the eyes are closed. Ophthalmic manifestations occur in 40% of cases.	Pain is facial and ocular. Aneurysms of the internal carotid cause unilateral, frontal or orbital headache. Such an aneurysm occurs more commonly in females and generally causes paralysis of CNIII.	Carotid artery stenosis causes transient ischemic attacks, unilateral hemiplegia, pain due to ischemia. Impaired circulation in the ophthalmic artery causes amaurosis fugax, ischemic optic neuropathy, and sometimes serious interruption of blood flow in the short posterior ciliary arteries or in the central retinal artery as well as rubeosis iridis and unilateral cataract.	Cavernous sinus thrombosis produces orbital effects i.e. exophthalmos, edema of lids and as well as papilledema, reduced acuity and impaired pupillary reflexes.	Ischemia. Frequently due to atherosclerosis.	A carotid bruit suggests carotid stenosis. An ocular bruit suggests a fistula. Surgery helps some of those affected, usually fistulas can be occluded. Ophthalmodynamometry can help to indicate the responsible artery.	Mostly affects males over 50 years of age. Dizzy, confused, may have a seizure. The amaurosis fugax attacks last for 5 to 15 minutes and are unilateral. Some have transient, monocular, contralateral loss of vision, hypoxic retinopathy, anterior uveitis, chemosis. Look for cholesterol crystals in retinal vessels (Hollenhorst plaques), raised IOP, pulsating exophthalmos, diplopia, paresis of lateral rectus. Pupil may become non reactive or be a Horner pupil.
Head pain with carotidynia. (See cluster headache, Charlin's syndrome, Sluder's syndrome, carotid artery syndrome and ischemic cerebrovascular disease.)	Usually a dull, throbbing headache but not always present. Not as severe as migraine. A similar ache develops within 48 hours after carotid endarterectomy.	Unilateral, neck pain in region of the common carotid radiating to face and ear. Cranial nerve V. Trigger area in floor of mouth.	When present varies in intensity.	Chronic for many years but pain appears in episodes and persists for 24 hours.	Caused by painful dilatation of the common carotid artery. Supranormal pulse in temporal artery. Worsened by pressing on the artery or by yawning, coughing or sneezing.	Steroids usually help. Ergotamine and propranolol have been used prophylactically. Listen for a carotid bruit.	Tends to affect women aged 40 to 50 years. Patients with atherosclerosis of their internal carotid artery are apt to suffer transient ischemic attacks or a stroke.

Cavernous sinus syndrome, cavernous sinus fistula syndrome. Foix syndrome, cavernous sinus thrombosis.	Unilateral.	Facial and ocular.	Rare.		Can be due to thrombosis, tumors, or trauma (75%) in the young, but is often due to an aneurysm in the elderly. Mucormycosis can cause rapid development of a cavernous sinus thrombosis when the fungi spread from the nose to the sinus.	With cavernous sinus thrombosis most have fever and they need large doses of antibiotics.	A cavernous sinus fistula causes chemosis, conjunctival congestion, lid swelling, pulsating exophthalmos, diplopia and reduced vision due to corneal exposure, secondary glaucoma and ischemia. Thrombosis of the cavernous sinus can cause paralysis of CNIII, IV, V, and VI as well as periorbital edema, bilateral proptosis, and chemosis.

Table 9

Headache Class.	Pain Type	Pain Location	Pain Frequency	Pain Duration	Probable Cause	Helped by:	Comments
Headache with diabetic neuropathy. (Many have hypertension, kidney disease, atherosclerosis and arteriolarsclerosis.)	About 50% have some head pain. Many do not know they are diabetic. Diabetes can cause painful ophthalmoplegia.	Forehead or retrobulbar area, i.e. area served by the first division of the fifth cranial nerve. Ischemia may affect cranial nerves III, V or VI.		Chronic.	Diabetes mellitus produces occlusive disease of small vessels. The result is impaired circulation and the formation of small aneurysms and hemorrhages.	Insulin or oral antidiabetic drugs to control the diabetes. Cyclosporin is being tried for type I diabetes. Propranolol may help prevent the pain as well as promote insulin release.	The three classical manifestations are polydipsia, polyuria and polyphagia. Some have paralyses of CNIII, with pupil sparing but nearly 50% show pupillary involvement. Sudden paralysis of CN VI may occur but usually recovers. Children have subnormal accommodation as an early sign. The typical fundus signs include aneurysms, hemorrhages, and exudates.
Wallenberg's syndrome, posterior inferior cerebellar artery syndrome. (See acute cerebellar hematomas.)	Severe attack of vertigo causing the patient to fall.	Ipsilateral loss of pain and temperature sensation in face and throat and contralateral loss of pain and temperature sensation in trunk and extremeties.			Vascular disease of the brain stem, occlusion of the posterior inferior cerebellar artery.		Appears in the fifth decade. Ataxia, muscular hypotonia, vertigo, nausea, trouble swallowing, trouble speaking. Ipsilateral Horner syndrome, nystagmus, diplopia, ptosis.
Headache with intracranial tumors, abscesses, hematomas, shunts, angiomas, aneurysms. (Among those with cerebral aneurysms as many as 25% have migraine too.) (Fewer than 1% of patients with a headache have a brain tumor. Up to 40% of those with a brain tumor have no headache. Ten percent of patients with brain tumors first seek advice concerning their eyes.) (See sub-arachnoid hemorrhage, Bruns's syndrome, headache due to increased intracranial pressure and the headache with various tumors.)	Deep, dull, steady ache, mild to severe. Severity often changes when head position is altered. Severity is not a reliable guide to seriousness. Those with an organic problem may move slowly, walk with legs wide apart, do not swing their arms, and tend to have speech difficulties. Unruptured intra-cranial aneurysms are usually asymptomatic. Rupture of an intracranial aneurysm produces an intense headache.	Localized or generalized. A unilateral frontal or orbital headache with paralysis of CNIII can be caused by an aneurysm of the intradural portion of the carotid artery or the posterior communicating artery. Headache is localized over or near the site of the tumor in 33% of cases. The function of CNIII or CNV is frequently affected. (Subacute bacterial endocarditis is mostly due to streptococci or staphylococci and causes fever, headache, heart murmurs and hemorrhages in conjunctiva and retina, Roth spots.)	Intermittent or more or less constant. Type, location and frequency of headache depend on the location of the abscess, aneurysm or tumor. Often worst early in the day. Headaches are the initial symptom in 30% of patients with intracranial tumors. About 1 in 300 patients die of brain tumor.	Occur every day but vary in intensity. Beware if headache is of recent onset and is increasing in severity. An aneurysm of the internal carotid can cause an intermittent headache for years. The pain is ipsilateral and comes in episodes each lasting about one day.	Tumors and aneurysms cause headache by traction on or displacement of intracranial structures not by the increase in intracranial pressure. Rarely an aneurysm near the junction of the posterior cerebral and superior cerebellar arteries can cause paralysis of CNIII and usually causes some hemiparesis. The associated headache is not referred to the ocular region. Those with an intracranial abscess often have a history of infection, ear disease, sinusitis, lung abscess.	Treatment of the underlying condition. May need codeine or morphine for the pain but often ASA is adequate. Worsened by coughing, straining, exercise, defecating, sexual intercourse, weight lifting or by a sudden change in head position. Among those with a cerebral aneurysm a bruit may be heard. (About 15% of patients with multiple sclerosis have headache as a complaint in the initial bout of this illness.)	The photopsias, if any, pulsate more slowly than those of migraine. Slowly progressive weakness on one side of the body. Convulsions, tinnitus, ataxia, loss of balance, mental changes, visual disturbances, nystagmus, field losses, papilledema, and no visible venous pulse on the disc. With an aneurysm, the pupil on the affected side is almost always dilated and fixed. With paralysis of CNIII the affected eye turns down and out and has ptosis and mydriasis.

Table 10

Headache Class.	Pain Type	Pain Location	Pain Frequency	Pain Duration	Probable Cause	Helped by:	Comments
Tumors affecting the chiasma. Pituitary adenomas erode the sella in 80% of cases. Can be intrasellar or suprasellar.	Some but not all have headache. Headache occurs in 20% of patients with a pituitary adenomas.	Cranio-pharyngiomas may be suprasellar or intrasellar. Meningiomas may be tubeculum sellae (suprasellar), olfactory groove (presellar), or sphenoidal ridge (parasellar).	The pituitary gland is in the middle cranial fossa.		Craniopharyngiomas occur here. A tumor may erode into the paranasal sinuses and cause acute headache with oculomotor paralyses.		Pituitary adenomas cause bitemporal hemianopia and also impair the vision in 90% of these patients. A few suffer unilateral blindness and contralateral hemianopia.

Frontal lobe tumors are supratentorial.	These tumors frequently remain silent for a long time but eventually raise the intracranial pressure and cause headache.	The frontal lobes are in the anterior cranial fossa. If the tumor presses downward it causes the Foster-Kennedy syndrome with papilledema in one eye and optic neuritis followed by optic atrophy in the other eye. The Foster-Kennedy syndrome is not pathognomomic.			Likely tumors include medullablastomas, meningiomas, astrocytomas, glioblastomas or metastases from tumors in lung or breast.	Abscesses or aneurysms are able to produce similar signs and symptoms.	In the later stages frontal lobe tumors cause contralateral hemiparesis, anosmia and personality changes. Mental symptoms are common with these tumors. About 50% have papilledema, 5 to 10% have irregular nystagmus and 30% have visual field losses.
Temporal lobe tumors are supratentorial. (See Raeder's paratrigeminal syndrome.)	Headache is not a prominent feature in most of these patients. Intracranial pressure becomes elevated.	Tumors in the dominant lobe interfere with speech. Tumors in the left lobe impair memory and the ability to write and calculate. Tumors in the right lobe affect visual memory and the ability to read and learn.	The temporal lobes are in the middle cranial fossa.		Most of these tumors are gliomas, some are meningiomas, and only a few are angiomas, astrocytomas, medullablastomas or metastases from lung or breast.	With temporal lobe tumors patients may see organized visual hallucinations, objects, or persons instead of the visual aura associated with classical migraine.	Seizures are the initial sign in 40%. Look for exophthalmos, extraocular muscle paralyses, diplopia, ptosis, ipsilateral mydriasis with a fixed pupil and photophobia. Many have an incongruous, contralateral, upper-quadrant hemianopia and usually papilledema.
Parietal lobe tumors are supratentorial.	Focal tumors in this region cause visuospatial disorientation with a tendency to ignore the opposite side of the body. Intracranial pressure is elevated.	The parietal lobes are the upper central lobes of the cerebral hemispheres.			Probable tumors include medullablastomas, meningiomas (30%), astrocytomas, gliomas or metastases from lung or breast. If the tumor is anterior it causes focal sensory seizures. If the tumor is posterior it causes dyslexia, visual agnosia and primitive hallucinations.		Asymmetric optokinetic nystagmus is not uncommon. If the trigeminal nerve is affected, look for reduced corneal sensitivity and facial paralysis on the side opposite from the tumor and this combination is pathognomomic. Papilledema is rare but homonymous hemianopia is common.

Table 11

Headache Class.	Pain Type	Pain Location	Pain Frequency	Pain Duration	Probable Cause	Helped by:	Comments
Occipital lobe tumors are supratentorial. They are uncommon, about 25% are gliomas and 25% meningiomas.	Headache is not a prominent feature.	The occipital lobes constitute the posterior portion of the cerebral hemispheres and contain the visual cortex.	Tumors here are uncommon but if present cause epilepsy, mild mental disturbances and sometimes alexia, agraphia, micropsia, metamorphopsia and agnosia.		Tumors here may also be medullablastomas, astrocytomas, hemangioblastomas or metastases from lung or breast. Lesions affecting the supranuclear pathway cause loss of involuntary conjugate eye movements.		Seizures. Papilledema is usual and often extreme. About 85% have contralateral, homonymous, hemianopsia with macular sparing but sometimes the macula is also affected.
Supratentorial tumors can also develop from the corpus callosum, the pineal body or the midbrain region.	Midbrain tumors cause increased intracranial pressure.	When the cerebral peduncle and third nerve nucleus are affected there is paralysis of CNIII and contralateral facial palsy, Weber's syndrome.	When the red nucleus is affected there can be paralysis of CNIII with tremor and jerky movements of the contralateral side of the body, Benedikt's syndrome.	Patients with Nothnagel's syndrome show paralysis of CNIII, ataxia and gaze palsies. The lesion affects the superior cerebellar peduncle, red nucleus and fibers of CNIII.	Pineal tumors can cause ataxia, deafness, limitation of upward gaze, papilledema, transient amaurosis.		Parinaud's syndrome, can result from a midbrain tumor. They have paralysis of conjugate upward gaze as well as vertigo, ataxia and marked papilledema. The lids may show ptosis or be retracted. Pupils are dilated. These tumors produce disturbances of ocular motility but have no direct effect on the visual tracts.
Cerebellar tumors are infratentorial. (See also cerebellar hematomas.)	These patients often have a bifrontal headache and elevated intracranial pressure.	Pain in orbital, midfrontal, and neck regions. Among patients under 15 years of age 65% of all brain tumors are in the cerebellum.	The cerebellum is in the posterior cranial fossa.		Tumors can displace the cerebellar tonsils through the foramen magnum and cause impingement on the dorsal roots of the cervical nerves.	Surgery and radiation are required.	Classical cerebellar signs are ataxia, loss of balance, hypotonia and nystagmus. Seizures are common, nausea and facial paralysis often occur. With elevated intracranial pressure patients show weakness of conjugate gaze, nystagmus and papilledema with no pulse visible on the disc.

Cerebellopontine angle tumors are infratentorial.	Intermittent headache and raised intracranial pressure. These tumors may occur with neurofibromatosis, von Recklinghausen's syndrome.	Behind the ear. When an acoustic neurinoma develops on CNVIII it causes similar manifestations and can cause paralysis of CNVI. Contralateral hemiplegia is usual.		Chronic.	Cranial nerve pareses are to be expected. Facial pareses (CNVII). Tinnitus, deafness, and vertigo (CNVIII). Corneal anesthesia, and loss of sneeze reflex (CNV). Dysphagia (CNX).	Vascular lesions can produce similar effects.	Hypotonia, nausea, ataxia, contralateral hemiplegia, facial palsy, and vestibular nystagmus. Paralysis of the lateral rectus, corneal anesthesia, dry eyes and papilledema.
Infratentorial tumors in the pons and medulla.	Headache and raised intracranial pressure, but rarely papilledema.	Tumors of the medulla tend to cause early death so eye signs are infrequently reported.	The pons and the medulla are in the posterior cranial fossa.		Gliomas are the most common tumors here. They are likely to affect the nuclei of the cranial nerves and produce paralysis of CNV, VI, VII or VIII.	Early signs are cardiac irregularities, vertigo and trouble swallowing. The gaze centres are located in the pons.	Vestibular nystagmus is often present. Papilledema is uncommon, but can develop along with paralysis of the tongue in the late stages of medullary tumors. Pontine tumors rarely cause papilledema.

Table 12

Headache Class.	Pain Type	Pain Location	Pain Frequency	Pain Duration	Probable Cause	Helped by:	Comments
Infratentorial tumors in the pons and medulla oblongata.	These patients develop headache with raised intracranial pressure but only a few show papilledema.	Tumors of the medulla tend to cause early death so eye signs are infrequently reported.	The pons and the medulla are in the posterior cranial fossa.		Gliomas are the most common tumors here. They are likely to affect the nuclei of the cranial nerves and produce paralysis of CNV, VI, VII, or VIII.	Early signs are cardiac irregularities, vertigo and trouble swallowing. The gaze centers are located in the pons.	Vestibular nystagmus is often present. Although papilledema is uncommon it can occur along with paralysis of the tongue in the late stages of medullary tumors. Pontine tumors rarely cause papilledema.
Headache with raised intracranial pressure. (Not all patients with elevated intracranial pressure have a headache.) (See intracranial tumors, Bruns' syndrome, hematomas, aneurysms and hemorrhages.)	Deep, dull, ache, may change when head position is changed. Tumors affecting the post chiasmal visual pathways generally cause increased intracranial pressure and headache. These tumors may be supratentorial or infratentorial.	Pseudotumor cerebri is another potential cause especially in young obese females. The cerebral edema and narrowed small ventricles and increased intracranial pressure cause papilledema.			Tumors, aneurysms, occlusions, hemorrhages and disturbances of the circulation of the cerebrospinal fluid. Cerebral edema occurs in many conditions. In infants the fontanelles will bulge. Consiousness may fluctuate.	Worsened by coughing, straining, exercise, sexual intercourse, defecating, weight lifting.	May suffer blackouts, projectile vomiting without nausea, anorexia, convulsions, ataxia, loss of balance, circulatory and respiratory changes, psychic disturbances. Some show reduced corneal sensitivity, disturbed function of extraocular muscles, nystagmus, field losses, papilledema, and have no visible pulse on the disc. A few develop bilateral exophthalmos.
Bruns' syndrome, postural change syndrome. (See intracranial tumors.)	Severe headache but discomfort varies with head position.	Affects cranial nerves III, II and others.	Free of symptoms between attacks.	Paroxysmal attacks of vertigo, headache and projectile vomiting on change of head posture.	Tumors or other lesions of third, fourth, or lateral ventricles which obstruct flow of cerebrospinal fluid. Some are due to cysticercus infection.	Signs and symptoms can be altered by changing head position. Partial ophthalmoplegia can be precipitated by postural changes.	Ataxia, nausea, vomiting, vertigo, tachycardia and syncope. Paralysis of CNIII often occurs. Transient blindness, flashes of light, papilledema.
Headache with an acute cerebellar hematoma. (Cerebellar hematomas constitute 10% of all hemorrhages into brain tissues.) (See cerebellar tumors and the Wallenberg syndrome.)	Acute pain in the head.	Occipital.	Sudden onset.		Hemorrhage into the cerebellum. Traction on or displacement of pain sensitive structures. The posterior inferior cerebellar artery has been called the artery of thrombosis. It is a branch of the vertebral artery.	Prompt treatment, surgical drainage. A cerebellar hematoma is life threatening.	Many have hypertension, ataxia, brain stress signs, nausea, vomiting. Look for papilledema.

Table 13

Headache Class.	Pain Type	Pain Location	Pain Frequency	Pain Duration	Probable Cause	Helped by:	Comments
Terson's syndrome, multiple cerebral aneurysms. Subarachnoid hemorrhage syndrome. (At least 40% die if they have a major bleeding episode.) (See stroke headache, ischemic cerebrovascular disease, and effort headache.)	Severe, throbbing pain in the head. Sudden and severe supraorbital pain. Headache is often the chief symptom but 10% suffer seizures too. A congenital cerebral aneurysm is usually asymptomatic until it ruptures, this can occur in a migraine attack or following trauma.	Sometimes occipital but often begins above one eye. Pain in the back and legs develops later. An aneurysm of the supraclinoid, internal carotid, or posterior communicating arteries causes paralysis of extraocular muscles and pain in the area supplied by the ipsilateral trigeminal nerve.	Constant. Aneurysms of the posterior communicating artery can cause head pain before the subarachnoid hemorrhage occurs. Trauma can precipitate the hemorrhage.	Sudden onset and severe for about a week; all gone in two months providing the victim survives. Recurrent hemorrhages, if they occur, are likely to cause death.	Traction on pain-sensitive structures produces the headache. Sub-arachnoid hemorrhage is often due to rupture of an aneurysm. The aneurysm is usually located at the junction of the internal carotid and posterior communicating arteries. The stroke of the young patient. Death can occur rapidly.	Surgical drainage but usually not treatable in the acute stage of a head injury. Diagnosis of subarachnoid hemorrhage is aided by spinal puncture.	Symptoms of arteriovenous malformations often resemble those of migraine but the aura flickers more slowly than in migraine. Some become drowsy or comatose. Fever, stiff neck, bilateral partial or complete paralysis of CNIII, ptosis, vitreous hemorrhages, ipsilateral mydriasis and photophobia. Papilledema if it occurs is a late sign.
Headache with subdural hematomas. (Subdural hemorrhages are more common than extradural hemorrhages.)	Most have a severe headache which develops rapidly. All the signs tend to fluctuate. Loss of consciousness occurs in the acute type.	Pain at first is on the side of the injury but later all over the head.	More apt to occur in chronic alcoholics.	Acute type or chronic type can follow head injury. Pain tends to become constant.	Trauma affecting the middle meningeal artery. Bleeding may be from a vein close to the sphenoidal ridge. Hematomas can occur spontaneously and produce chronic headache.	Surgical drainage. Signs may not appear until weeks after the trauma. Prognosis is poor for infants and better for adults.	Chronic type causes: dizziness, drowsiness, seizures, confusion, hemiplegia, diplopia, fixed dilated pupil on the side of the lesion (Hutchinson's pupil), papilledema, field defects, and vestibular nystagmus.
Headache with extradural hemorrhages.	First become unconsious then have a lucid interval of minutes to hours, followed by headache, nausea and vomiting.				Follow injury to the anterior branch of the middle meningeal artery; often the result of a linear fracture of the skull.	Surgery is required.	This is an emergency. Patients usually enter a coma, and can die promptly.
Headache with meningeal irritation, chronic meningitis.	Headache is dull to severe, gradual onset. More rapid onset if due to intracranial bleeding. Increased intracranial pressure.	Bilateral or generalized or over the vertex and radiating down the neck. Cranial nerves V, VII, IX and X and the upper cervical nerves are sensitive to pain.	More or less constant. Child is much more uncomfortable if moved.	Until the inflammation subsides.	Meningeal infections including those associated with syphilis, tuberculosis, meningococcal or other infections including viral.	Treatment of the underlying condition. May need codeine or morphine to ease the pain and i.v. antibiotics to control the infection.	Likely to be acutely ill with fever, nausea, vomiting, cranial nerve palsies, delirium, lethargy and a stiff neck. Photophobia and papilledema.

Table 14

Headache Class.	Pain Type	Pain Location	Pain Frequency	Pain Duration	Probable Cause	Helped by:	Comments
Ramsay-Hunt syndrome, geniculate neuralgia, otic neuralgia, Hunt's syndrome, herpes zoster oticus. (See Melkersson-Rosenthal syndrome.)	Severe neuralgic pain. The geniculate ganglion is the sensory ganglion of CNVII. (Similar acute and prolonged local pain usually precedes herpes zoster vesicles on the skin.)	Deep pain in ear. Cranial nerve VII. Herpes viruses may lodge in the geniculate ganglion or in peripheral ganglia of CNIII, IX or X.	Paroxysms of pain occur in the ear when the geniculate ganglion, and the nervus intermedius (Wrisberg) of the seventh nerve are involved.	Pain precedes vesicle formation by several days and may persist after the lesions have disappeared, postherpetic neuralgia.	Herpetic infection of the geniculate ganglion. Herpes zoster (HSV-3) causing an eruption on the tympanic membrane, the external auditory meatus and the concha of the auricle.	Corticosteroids, antidepressants, lithium and phenothiazines have all given help.	Facial paralysis. Hearing may be affected; tinnitus and sometimes vertigo and depression. Dry eyes, dry throat, reduced corneal sensitivity, risk of corneal ulceration, nystagmus.
Melkersson-Rosenthal syndrome, geniculate ganglion syndrome, facioplegic migraine. (See the Ramsay-Hunt syndrome, tic douloureux and Quincke's edema.)	This form of facial palsy is often associated with a migraine type headache.	Face. Facial palsy may be unilateral or bilateral. Cranial nerve VII and its geniculate ganglion are affected.	Periodic.	Chronic. Begins in childhood or early adult life.	Cause obscure but often inherited as an autosomal dominant trait. Possibly the result of a viral infection damaging the parasympathetic cells or fibers in the geniculate ganglion.	Corticosteroids and analgesics are used to treat this type of angioneurotic edema.	Recurrent or chronic painful facial edema, and paralysis; swelling of the lips; congenitally fissured tongue. Some have lagophthalmos, increased lacrimation, blepharochalasis, corneal ulcers, corneal opacities, retrobulbar neuritis and exophthalmos.

Tic douloureux, gasserian neuralgia, trigeminal neuralgia, Fothergill's neuralgia, trifacial neuralgia. One type is called supraorbital neuralgia. (See Raeder's syndrome and Rosen's neuralgia.)	Sharp, stabbing, high intensity pain and burning sensation with spasms of facial muscles. The gasserian (trigeminal or semilunar) ganglion is the sensory ganglion of the trigeminal nerve CNV.	Pain in the face in the eye region, slightly more often on the right side. Pain is limited to one side of the face in the area supplied by the second and less commonly that supplied by the first and third divisions of CNV.	Episodic. Worst in March and October. Pain episodes recur for two or three months at a time. Pain is more constant in the rare cases where a tumor is responsible.	Brief 20 second stabs of pain, repeated for up to an hour. Condition may recur for years but usually remits spontaneously.	Cause may be a blood vessel impinging on a branch of the fifth nerve. Precipitated by stimulation of a trigger area 3 or 4 mm in diameter near the nose or mouth. May accompany multiple sclerosis or follow a herpes infection. Be sure no tumor is present.	Anticonvulsant drugs help but the rationale is not clear, e.g. diphenylhydantoin sodium (Dilantin) 100 mg b.i.d. or t.i.d. or carbamazepine 200 mg b.i.d. or t.i.d. will control the pain in 66% of cases. Some use baclofen, amitryptyline, diazepam, or surgery including radiofrequency heating.	Rare, (estimated to affect 15 people in 100,000) slightly more common in men. Usually affects those over 50 years of age. Most have a trigger area. Generally no nausea, no vomiting, no scotomata. Rarely increased lacrimation and ipsilateral conjunctival hyperemia. The neuro-paralytic keratitis can lead to corneal ulceration or perforation.
Glosspharyngeal neuralgia. (See tic douloureux, Sluder's syndrome, Raeder's syndrome, Costen's syndrome, Charlin's syndrome, and tympanic plexus neuralgia.)	Sharp, stabbing, severe, unilateral, neuralgic pain. The superior and inferior (petrosal) glossopharyngeal ganglia contain cell bodies of the afferent fibers of the glossopharyngeal nerve.	Begins in the back of the throat and radiates to the angle of the jaw, the tongue and the neck and extends to the ear. Cranial nerve IX.		Paroxysmal episodes come in groups.	Obscure. Possibly viral. Some have a trigger area in the tonsillar region.	Treatment is similar to that of trigeminal neuralgia, tic douloureux. Is not a self limited condition. May require surgery of the glossopharyngeal nerve.	Affects about one person in a million. Most of these patients are over 50 years of age. Some have cardiac slowing which can lead to syncope.

Table 15

Headache Class.	Pain Type	Pain Location	Pain Frequency	Pain Duration	Probable Cause	Helped by:	Comments
Tympanic plexus neuralgia, Reichert's syndrome. (Compare with Rosen's neuralgia). (See glossopharyngeal neuralgia.)	Sharp neuralgic pain. The chorda tympani nerve originates from the facial nerve and carries parasympathetic and sensory fibers.	In the vicinity of the external auditory meatus but spreads to the face and tongue and behind the ears.		Paroxysmal.	Possibly viral. Involvement of the tympanic branch of the glossopharyngeal nerve, cranial nerve IX.	Treated by sectioning the nervus intermedius, (a part of CNVII), the chorda tympani or CNIX.	Rare. (Rosen's neuralgia is tic douloureux of the chorda tympani.)
Ménière's syndrome, Ménière's disease, vestibular migraine, eighth nerve neuralgia. (Lermoyez's syndrome is similar but in the Lermoyez syndrome hearing improves during the vestibular attack.) (See Cogan's syndrome and basilar artery migraine.)	Headache varies in severity as do the vertigo and tinnitus. Patient may fall down when the attack begins. Ménière's syndrome may be confused with labyrinthitis.	Abrupt onset of an occipital headache (on the side of the tinnitus) or a tight sensation in the back of the head and neck. Cranial nerve VIII. Deafness may be unilateral or rarely bilateral and is not total.	Typically occurs in episodes appearing every 4 or 5 weeks and separated by symptom-free periods of months or years.	Each episode lasts up to one hour but more than one attack can occur in a single day.	Disorder of the endolymphatic system of the inner ear. Paroxysmal labyrinthine hydrops. Neuralgia of CNVIII. Distended arteries in the posterior fossa impinge on cranial nerves V, VII, VIII, IX and X. Local autonomic dysfunction.	Diazepam i.v., dimenhydrinate orally or i.m., meclizine orally or i.m., cyclizine orally or i.m. or scopolamine transdermally.	Most apt to appear in males in their fifties. Vertigo, tinnitus, nausea, some permanent hearing loss. Room seems to revolve even when the eyes are open. Cheek on the affected side feels dead. Nystagmus with the fast component toward the normal ear. Rarely diplopia and corneal anesthesia.
Sluder's syndrome, Sluder's lower-half headache, sphenopalatine ganglion neuralgia, atypical facial neuralgia, (See cluster headaches. Both may be called sphenopalatine ganglion neuralgia.) (See Charlin's syndrome and vidian neuralgia.)	Continuous pain, burning, deep-seated, nagging and fairly severe. The sphenopalatine (pterygopalatine) ganglion is a parasympathetic ganglion in the pterygoid fossa and receives preganglionic fibers from the facial nerve CNVII.	Usually unilateral or rarely bilateral, in eye, nose, orbit, temple and upper jaw. Pain radiates from the lower face e.g. palate up to the ear. Some have pain in the mastoid region. Rarely pain extends to the occiput. Cranial nerves III, V or VII can be affected.	Frequent episodes; freedom from pain between attacks. Attacks do not appear in groups as they do in cluster headaches, glossopharyngeal neuralgia, tic douloureux or vidian neuralgia.	Episodes last for minutes to days. Condition may persist for years. Residual tenderness of the neck muscles tends to persist.	Probably due to vasodilation of branches of the external carotid, possibly the sphenopalatine branch of the internal maxillary artery. Some blame intranasal or sinus pathology or viral infections. Worsened or precipitated by alcohol, tobacco, or a change in climate. Hysterical mechanisms seem to be involved.	Ergotamine may be helpful but typically treatment is difficult. Beware overuse of analgesics. Correct refractive errors and ocular muscle imbalance.	More women than men. Younger patients. Not a well-defined syndrome, may be a group of similar conditions. Tender spot 3 to 5 cm behind the mastoid but no trigger area. No Horner's syndrome, no gastrointestinal upset. There is a vascular type with unilateral, throbbing, midfacial pain in which facial flushing, rhinorrhea and excessive lacrimation occur.
Vidian neuralgia, Vail's syndrome. (See Sluder's syndrome.)	Fairly severe neuralgic type pain. The vidian nerve is formed by the union of the great superficial petrosal and the deep petrosal nerves.	Usually unilateral. Pain in nose, face, eye, ear, head, neck and shoulders.	Pain appears in acute attacks often during the night.	Episodic.	The vidian nerve passes close to the sphenoid sinus on its way to the sphenopalatine ganglion. Irritation or inflammation of the vidian nerve may be due to a viral infection.		

Table 16

Headache Class.	Pain Type	Pain Location	Pain Frequency	Pain Duration	Probable Cause	Helped by:	Comments
Raeder's paratrigeminal syndrome, the paratrigeminal neuralgia syndrome. A similar symptom complex has been called Symond's type of migraine. (See Horner's syndrome, tic douloureux, cluster headaches, and glossopharyngeal neuralgia.)	Headache is severe, neuralgic or throbbing.	Unilateral, fronto-orbital headache and ipsilateral periocular pain. Facial pain in the region supplied by the first division of the trigeminal nerve CNV. Affects not only the sympathetic fibers but sometimes CNII, III, IV, VI and any division of V.	Recurrent morning headaches. Headache often wakens the patient from sleep. Some have a cluster of headaches, others have a single episode lasting hours to weeks. These two types are benign.	Generally clears spontaneously in a few weeks. Transitory but prognosis is guarded. These headaches are ominous if any other neurological signs develop.	Obscure, possibly meningitis or an aneurysm of the internal carotid affecting CNV and its sympathetic fibers. Some are due to a neoplasm in the middle cranial fossa. A few have fibromuscular dysplasia along the carotid artery. Many have high blood pressure.	Check for a possible neoplasm. Treat the high blood pressure if it is present. Some obtain relief of the headache by oral prednisone.	Mostly middle-aged males. Weakness of jaw muscles, nausea, vomiting but no aura. Suddenly develop an incomplete Horner's syndrome, (ptosis and miosis) but the sweating mechanism is preserved. Severe ocular pain, excessive lacrimation, decreased corneal sensitivity, rarely diplopia and scotomas.
Charlin's syndrome, the nasociliary syndrome, migrainous neuralgia, Harris' neuralgia. (See cluster headache. Both may be called ciliary neuralgia). (See Sluder's syndrome.)	Severe neuralgic pain. The ciliary ganglion lies deep in the orbit and is a parasympathetic ganglion receiving preganglionic fibers from the oculomotor nerve CNIII.	Unilateral in eye, orbit, temple, cheek, jaw and at the root of the nose. Sharp pain at the inner angle of the eye where the nasociliary nerve exits on the side of the nose. CNV.	Long intervals between attacks.	Paroxysms of pain last for minutes to hours.	Neuritis of the nasociliary nerve or of the ciliary ganglion. A virus could cause this type of ciliary neuralgia.	Anesthetic applied to the nerve in the anterior portion of the nasal fossa gives temporary relief.	More common in men. Inflammation of the anterior segment of the eye on the ipsilateral side. Excessive lacrimation, rhinorrhea, blepharospasm, superficial keratitis, photophobia, iritis.
Headache with Bell's palsy, idiopathic facial paralysis.	Local ache. The petrosal and the stylomastoid arteries supply the nerve in the Fallopian canal.	Pain in ear and mastoid; tingling or numbness in cheek or mouth. Face feels stiff and is pulled to one side. Paralysis of CNVII.	Sudden onset in adults.	Generally they recover gradually. Pain eases after a few days. Full recovery in 80%.	Unknown, possibly viral; have inflammation and edema of the facial nerve. Edema in the Fallopian (facial) canal or in the stylomastoid foramen.	Corticosteroids but some studies show that these drugs provide little help. Electrical stimulation often helps restore facial muscle function.	A few have permanent facial paralysis. Hyperacusia, changes in ability to taste, corneal ulcer, ectropion, lagophthalmos, nystagmus, epiphora, ocular irritation.
Horner's syndrome, Bernard-Horner syndrome. (Forms part of many other syndromes.) (Often occurs with cluster headaches and Raeder's syndrome.) (See the Barré-Liéou syndrome.)	Usually no pain but have an oculosympathetic paralysis. Horner's syndrome with heterochromia iridum is called Passow's syndrome.				Impairment of the sympathetic supply to the eye can follow damage to any of the three neurons in the chain. Can be due to birth injury, follow thyroid surgery, or be due to other causes including bronchogenic carcinoma.	Identifying the site of the lesion and treating accordingly. If developing in a middle aged smoker check for cancer of the upper lobe of the lung.	Ptosis, miosis and ipsilateral anhydrosis. If due to birth trauma the iris will be pale in the eye on the affected side. Heterochromia iridum. Rarely an affected eye develops an abnormally shaped pupil.

Table 17

Headache Class.	Pain Type	Pain Location	Pain Frequency	Pain Duration	Probable Cause	Helped by:	Comments
Tolosa-Hunt syndrome, Hunt's syndrome, superior orbital fissure syndrome, painful ophthalmoplegia syndrome. (See complicated migraine and the carotid artery syndrome.)	Pain can be severe and is steady. In the syndrome of the apex of the orbit, (Rollet's syndrome) CNII is also involved.	Unilateral, retro-orbital pain in the area supplied by the first division of CNV. Followed by paralysis of cranial nerves III, IV, or VI.	Attacks recur at intervals of months or years.	Symptoms persist for days or weeks. Disease is usually self-limited.	A non-specific granulomatous inflammation involving any or all of the nerves traversing the superior orbital fissure or cavernous sinus. Trauma, aneurysms or neoplasms are potential causes.	Systemic steroids may be needed for months. Check for diabetes, carotid aneurysms, or tumors. Have an elevated erythrocyte sedimentation rate.	Those affected are usually over 40 years old. Mild exophthalmos. The painful unilateral ophthalmoplegia recovers slowly. Ptosis, sluggish pupillary reactions (in 2%), scintillating scotomas; corneal sensitivity is reduced.
Guillain-Barré syndrome and its variants such as the Miller-Fisher syndrome and Landry's acute ascending paralysis.	Pain or paresthesia precede the flaccid paralysis. Paralysis of any cranial nerve especially VII, IX, or X.	Weakness, pain and progressive paralysis in legs. Some have areas of relative anesthesia. Loss of tendon reflexes.			Acute polyradiculoneuronitis with facial paralysis following an infection. Excess protein in cerebrospinal fluid.	Usually make a complete recovery within a few months but a few die from respiratory paralysis.	Paroxysmal hypertension, ataxia, weakness. Sometimes fixed dilated pupils, ophthalmoplegia, lagophthalmos, optic neuritis, papilledema, optic atrophy.

Cogan's syndrome, Cogan's non-syphilitic interstitial keratitis syndrome. (See Ménière's syndrome.)	Abrupt onset of ocular pain or vertigo and tinnitus with hearing impairment.	Earache and some pain in the eye. Ocular or aural symptoms may be first to appear. Cranial nerve VIII.	This is a rare condition which usually results in some permanent damage to both hearing and labyrinthine function.	Begins on the average at 29 years of age, and persists for a few months.	Considered to be an immune vasculitis. Risk of vascular occlusions. May be related to periarteritis nodosa.	Topical corticosteroids e.g. dexamethasone phosphate (0.1%) one drop every 2 hours for 7 to 14 days. To prevent profound deafness requires systemic administration of prednisone 80 mg a day.	Mostly young adults. Vestibular and auditory dysfunction with vertigo and tinnitus. Some hearing reduction is permanent. Bilateral, subepithelial interstitial keratitis and stromal edema. Ciliary hyperemia; photophobia, blepharospasm, episcleritis, nystagmus. Transient reduction in visual acuity.
Effort or exertional headache; cough headache; post-coital headache; benign orgasmic cephalalgia. (See Terson's syndrome.)	Throbbing, severe. Heart rate and blood pressure increase during intercourse. (Similar headaches can accompany the Valsalva maneuver, forced expiratory effort against a closed airway.)	Usually frontal, behind the eyes but some have an occipital ache.	Occurs in some individuals with orgasm, or immediately after similar exertion. In eyes with controlled narrow-angle glaucoma sexual intercourse in the prone position can precipitate an attack of glaucoma.	Usually brief, up to an hour or so but sometimes does not develop until 2 hours later. A cough is likely to aggravate any headache especially one with brain tumor or elevated intracranial pressure or sinusitis.	Exertional or effort headache can be precipitated by weight lifting or by running or by sexual intercourse. Some have a malignant type associated with a tear in subarachnoid membranes or an actual hemorrhage.	Ergotamine tartrate or methysergide prophylactically is indicated for effort headache. Subarachnoid hemorrhage can be diagnosed by spinal puncture.	Men are more often affected. Effort, or straining raise the blood pressure but this particular headache is not dependent on having chronic high blood pressure and has no associated neurological signs or symptoms unless there is a subarachnoid hemorrhage.

Table 18

Headache Class.	Pain Type	Pain Location	Pain Frequency	Pain Duration	Probable Cause	Helped by:	Comments
Headache with Paget's disease, osteitis deformans. (There is also a condition of congenital hyper-phosphatasemia, juvenile Paget's disease.)	Headache is mild, burning and only rarely severe, however many of those with Paget's disease have no headache.	Localized or generalized. Tender areas on scalp. Eventually pain in back and limbs.	Intermittent or constant.	Prolonged. (The hereditary autosomal recessive type has its onset before 4 years of age. The affected child suffers, dwarfism, osteoporosis, skull thickening, kyphoscoliosis, bowed legs, and has angioid streaks in the fundus, optic atrophy, macular changes and blue scleras.)	Cause of the adult type is unknown. Rate of bone resorption and formation both increased in the spinal column and skull; the result is compression of the brain and the cranial nerves. Many die of cardiovascular disease, some have hypertension, and bleeding in their gastrointestinal tract.	Analgesics and sedatives offer some help for the headache. Calcitonin or mithramycin help control Paget's disease. Patients have high serum levels of alkaline and acid phosphatase.	Mostly males over 40 years of age. Skull increases in size, becomes tender and thickened, but bones become more porous. Skin pigmentation increases. EOM palsies, proptosis, optic atrophy and in 10% angioid streaks. Paget's disease with angioid streaks is called Terry's syndrome.
Post-traumatic, or post-concussion headache, occipital neuralgia. Craniocervical or punch-drunk syndrome. Friedmann's syndrome is traumatic encephalitis. (Kummell's disease is due to traumatic damage to the vertebrae.) (Cervical disc disease or injury often follows an accident.) (A post-traumatic headache occurs in at least 33% of all who are hospitalized for head injury.) (See depression headaches, and the Barré-Liéou syndrome.)	Variable intensity. Type 1 have a steady pressure sensation or band-like ache. Type 2 have a type 1 ache plus a local tender zone. Type 3 have a throbbing, unilateral ache in attacks.	Localized to the site of the injury or generalized or in the forehead and temples. Back and neck pains are often associated. Type 3 is often unilateral. Affects CNV and CNVIII. (Lesions of the cervical spine cause the Barré-Liéou syndrome also called the posterior cervical sympathetic syndrome.)	Variable frequency. Can change with barometric pressure. Type 1. Intermittent but recurs for years. Type 2 continuous or intermittent. Type 3 recurs in short spasms.	Signs and symptoms usually develop within two days of the trauma but in a few patients appear weeks later. Variable duration. Most such headaches fade in two months. Some complain of discomfort until all legal actions are settled. Even years later a few continue to have headaches and dizziness. Whip-lash type injuries often produce prolonged discomfort.	Even minor injury can cause this headache. Hyperextension or hyperflexion of the cervical spine. Type 1 is related to muscle contraction. Type 2 to local tissue damage including scarring, fibrosis, adhesions, disc lesions, hemorrhages. Small focal hemorrhages in the cerebrum and/or cerebellum. Type 3 is related to dilation of branches of the external carotid, a vascular mechanism. Psychological factors are important.	Time, massage, warmth, analgesics, propranolol, antidepressants. Phenobarbital provides relatively short periods of relief. Worsened by some head positions, by drinking alcohol, by emotional upsets or by changes in barometric pressure. Type 3 can be helped by cold compresses and by ergotamine. All need patient understanding and often psychotherapy as the depression tends to persist.	Many become tense, anxious, irritable, dizzy, photophobic, and unable to do prolonged reading. Vestibular (CNVIII) dysfunction occurs in over 50% of cases. Personality changes, memory impairment, resentment and emotional reactions. Patients with type 3 often have anxiety, depression and sometimes nausea. Look for vertical nystagmus, impaired convergence, reduced amplitude of accommodation and sometimes Horner's syndrome, but in type 3 the pupils are dilated, not miotic.
Lumbar puncture headache, leakage headache, post-spinal-tap headache, low pressure headache.	Severe or rarely mild headache. Dull, deep and can be throbbing type.	All over the head, especially frontal or occipital and neck regions. Often extends into a backache.	Constant. Begins within 24 hours of lumbar puncture in about 50% of these patients.	Twenty-four to 48 hours but longer if a spinal anesthetic or a contrast medium has been injected into the spinal column.	Follows withdrawal of 20 ml or more of spinal fluid. Leakage of spinal fluid allows traction or displacement of intracranial structures. Muscle contraction also occurs. Strong osmotic agents can produce a similar headache.	Recumbent position, no pillow. Hydration. Analgesics or ergotamine or DHE45. Use of a fine needle when performing the puncture and employing an oblique entry decrease the leakage of spinal fluid.	Lumbar puncture often causes dizziness, nausea and vomiting and sometimes blurred vision. A lumbar puncture is dangerous if the intracranial pressure is elevated. The cranial structures can herniate into the foramen magnum.

Table 19

Headache Class.	Pain Type	Pain Location	Pain Frequency	Pain Duration	Probable Cause	Helped by:	Comments
Headache with toxic or deficiency states, uremia, alcoholism, nephritis, and anemia. Carbon monoxide or other poisons, e.g. lead, arsenic, nitrites, morphine. Infections, mononucleosis, gastroenteritis or any condition causing fever. May accompany drug abuse or hangover or follow a seizure. (See dietary migraine.)	Moderate to severe, pulsating headache. A high fever in children can cause seizures and vomiting.	Generalized. Headaches with fever are dull and seem deeply situated. Headache and fever accompany subacute bacterial endocarditis. (See headache with intracranial tumors, abscesses etc.) Headache with meningitis can be severe.	Can develop at any age. Often daily. Constant. Following a seizure many develop a headache.	Depends on cause.	Fever and septicemia are the third most common causes of headache. Distention of cerebral and pial arteries is the principal mechanism. History of exposure to toxin. Can also be due to hypoglycemia, anemia, hypoxia, noxious fumes, hangover. The mechanism by which ice cream in the mouth causes a headache is not known, ischemia (?).	Controlling or eliminating the cause. Worsened by exercise or exertion or by lying down. Some people overdose themselves with analgesics so what they need is a reduction in their headache medications.	Headache usually accompanies a fever especially in children. Look for other signs caused by the noxious agent. Sodium nitrite is in hot-dogs or can be contacted in industrial situations. Monosodium glutamate (MSG) in many foods can cause a headache. The Chinese restaurant syndrome headache develops in susceptible individuals about 20 min. after the MSG compound is ingested.
Coffee hunger headache, caffeine withdrawal headache.	Definitely unpleasant.	Frontal.	Often on Sunday morning or other occasions when the usual intake of coffee is delayed.	Until coffee or caffeine containing soft drinks are consumed.	Is associated with dilatation of intracranial and apparently extracranial arteries as a result of caffeine withdrawal.	Coffee acts as a vasoconstrictor and frequently helps to relieve other types of headache.	Very common. An overdose of caffeine can cause a similar headache, with confusion, anxiety, tremors, vomiting and diarrhea.
Periodic headaches, also called cyclical headaches, menstrual headache. (See also migraine.)	Mild to moderate.	No specific location. Usually have abdominal discomfort in the form of cramps.	Once a month. Some women have a headache at both midcycle and just before or during menstruation.	Usually one or two days.	Related to ovulation-menstruation cycle. Possibly an estrogen-pituitary imbalance. Premenstrual tension. Premenstrual syndrome, PMS.	Mild analgesics. Some non-steroidal anti-inflammatory drugs are particularly helpful e.g. mefenamic acid or naproxen sodium for premenstrual headache.	The headache occurring at mid cycle, i.e. at ovulation time is called Mittelschmerz. Fluid retention usually occurs just before menstration.

Table 20

Headache Class.	Pain Type	Pain Location	Pain Frequency	Pain Duration	Probable Cause	Helped by:	Comments
Primary muscle contraction or anxiety-tension headache. Secondary muscle contraction headaches: spastic, myositic or myalgic headache, and cervical arthritis. (Stress, anxiety and emotional tension are the most common causes of headache. The resulting muscle tension causes at least 60% of all headaches.) (See psychogenic headache, post-traumatic headache, depression headache, vasomotor, rhinitis, combined headaches and Roy's ocular migraine.)	Feeling of a tight band around the head. Dull ache, gradual onset, not severe, only rarely pulsating. The acute type is mild and usually self-limited. The chronic type requires treatment. Usually no family history of a similar headache.	Usually bilateral but can be unilateral, vertex, fronto-occipital, temporal and generally extends into neck and shoulders. A real pain in the neck. Often have local tender areas in scalp, neck, or trapezius muscles.	Chronic recurring pain. Often occurs daily, especially toward the end of the day. Sometimes appears in the early hours of the morning but rarely awakens the patient. May increase in frequency at menopause.	Individual episodes last for hours or days but condition is prolonged for months or years. Condition may be acute or chronic. Trouble getting to sleep. Patient may become a hypochondriac.	Emotional factors, repressed hostility, stress, or let down from stress. Holding the head in an unusual position. Disorders of the cervical spine. Muscle tension and tension on the aponeurosis of the frontalis and occipital muscles causes scalp ischemia so the mechanism is vascular. Muscle contraction usually develops with any headache. Pain or stress can cause muscle tension and muscle tension can cause pain.	Patient needs to learn to understand the mechanism. Massage, warmth, gentle exercise, ASA, diazepam, propranolol or muscle relaxants. Helped by biofeedback techniques, by tricyclic antidepressants e.g. amitriptyline and by coffee. Worsened by ergotamine tartrate or by cold. Be sure no organic problem exists and that refractive errors and ocular muscle imbalances are properly corrected.	Often begins in patients between 20 and 40 years of age. Affects more men than women, rare in children. No prodromata, nausea or vomiting but may have anorexia. A common complaint is chronic fatigue. Some have a stiff neck. Stress probably plays a role in all types of headache. Depression often coexists and requires treatment. Overactivity of the sympathetic system causes dilated pupils and photophobia.
Barré-Liéou syndrome, cervical migraine, the posterior cervical sympathetic syndrome. (See the post-traumatic headache.)	Headache.	Occipital and pain in eyes and ears. Affects cranial nerves V and VIII.			Arthritis or trauma of the cervical spine irritate the sympathetic plexus around the vertebral arteries.		Vertigo, tinnitus, vasomotor disorders, facial spasm, stiff neck. Transient diplopia or loss of vision.
Psychogenic, emotional, conversion-hysteria headaches. (Also called tension headaches.) (About 50% of psychiatric patients report pain of some type.) (See combined headaches, depression headaches and post-traumatic headaches.)	Frequently the headache is constant, vise-like, hat-band or stabbing type. Unvarying intensity is suspicious. Headache descriptions tend to be vague or imaginatively detailed. They seek attention.	Bitemporal, vertex, or generalized. Reported distribution of pain does not follow anatomical features. May have a stiff neck.	Daily, constant. Unpredictable onset. Usually begins in third decade and affects more women than men.	Prolonged but in many cases the headache does not interfere with sleep or play. Often associated with a hypochondriacal state. Headache shows little or no change with the normal physiological changes such as puberty, pregnancy or menopause.	Insecurity, stress of daily living, anxiety, even headache from another cause can induce this kind of headache. Mechanism may be muscular, vascular or hormonal. Even if the cause is obscure the pain is real and not based on a conscious attempt to deceive. Depression is likely to develop.	Ruling out organic causes. May need more than elementary psychotherapy. Some need tranquilizers, antidepressants, or phenobarbital. They do not respond well to the usual analgesics. Avoid overuse of analgesics. These patients are reluctant to admit relief.	Suffer from anxiety but often appear outwardly calm and relaxed. Most have trouble getting to sleep. Emotional lability. Some become hypochondriacs. Usually a long history including complaints of fatigue, backache and anorexia. Bizarre symptoms are sometimes reported. Pupils may be large.

Table 21

Headache Class.	Pain Type	Pain Location	Pain Frequency	Pain Duration	Probable Cause	Helped by:	Comments
Depression headache. (Up to 90% of all headaches seen by internists and family practitioners are associated with mental depression.) (See post-traumatic headache, combined headaches and psychogenic headaches.)	Various types of headache; can be hatband type or dull, all around the head or a mild constricting type headache.	May be all over the head or more at the back of the neck or the base of the skull. A stiff neck is a common complaint.	Almost daily. Worse on weekends and holidays. Often occurs between 4:00 and 8:00 a.m. or p.m. Some anxiety is always present. These patients tend to awaken early and lie in bed worrying.	Onset at any age. Persists for weeks or months; rarely entirely free of pain.	May be caused by the presence of a headache due to some other cause. Can follow trauma. A deficiency of catecholamines in the brain has been reported.	Be certain no organic disorder exists. Antidepressants e.g. amitriptyline are often required. Eased by getting up and moving about. Correct refractive errors and ocular muscle imbalance after other care has been initiated.	Affects both sexes equally. Sometimes headache is the only complaint. Often appear outwardly calm and relaxed but report inability to concentrate, fatigue, loss of drive, feel sad, have poorer memory, and frequently have gastrointestinal problems.
Sinus headache. (Sinus infection is the commonest infective cause of headache, but actually causes less than 2% of headaches.) (See vasomotor rhinitis.)	Pain is dull, deep not throbbing. Children with Citelli's sinusitis syndrome have diffuse pain which may be referred to the occiput, mastoid or temples. Complain of loss of memory, inability to concentrate and somnolence.	Jaw, forehead or teeth. Fifth cranial nerve, 2nd division. Frontal but in chronic cases often becomes occipital. If the sphenoid sinus is involved the pain is deep in the head or at the vertex. If the ethmoid sinus is infected the pain is over the bridge of the nose or behind the eyes. Steady pain is felt over the infected sinus if the frontal or maxillary sinuses are involved.	Periodic or constant. May develop or change in association with changes in barometric pressure or humidity.	Worst in the morning except when the maxillary sinus is the offender, then it is worst at the end of the day. Pain is a prominent symptom with acute sinusitis but not with chronic sinusitis.	Inflammation of the turbinates, ostia and ducts. Obstruction can develop in frontal, ethmoid, sphenoid or maxillary sinuses. Usually preceded by severe upper respiratory tract infection or accompanied by an allergic reaction. A deflected nasal septum can lead to a "vacuum" headache originating from the frontal sinus.	Maxillary sinuses drain best when the patient lies down. The others drain best when the patient is vertical. Need ASA, acetominophen or codeine; vasoconstrictors often help. In rare cases surgery is indicated.	Nasal discharge. Low grade fever. Untreated can progress to orbital cellulitis. Pain is worse in some head positions, e.g. bending forward. Worsened by shaking the head or by percussion over the area. Photophobia, increased lacrimation, tender area at trochlea. Look for lid edema and dark circles around the eyes.
Headache with vasomotor rhinitis. (See muscle contraction headaches, sinus headaches and allergic headaches.)	Mild headache.	Frontal with nasal discomfort.	Periodic.	Hours.	Stress leading to congestion and edema of nasal mucosa without infection or allergy; has a vascular basis.	Accurate diagnosis. Symptoms can be brought on by fatigue, chilling, anxiety or anger.	May be confused with sinusitis, allergic reactions or a common cold. Lid edema is common and a few develop scotomas.

Table 22

Headache Class.	Pain Type	Pain Location	Pain Frequency	Pain Duration	Probable Cause	Helped by:	Comments
Pain due to middle-ear infections. (Otitis media and mastoiditis are now rare.)	Severe, stabbing, throbbing pain.	Localized, often begins in occiput then becomes frontal. A type of trigeminal neuralgia. Pain in the ophthalmic branch of CNV. Gradenigo's syndrome (the temporal syndrome) involves the petrous bone and mastoid. (See trigeminal neuralgia.)	Constant. With Gradenigo's syndrome patients have painful palsy of CNVI and ipsilateral hearing loss.	Persisting and often worse at night.	Infection, but trauma and hemorrhages are other possible causes. Gradenigo's syndrome begins as a middle ear infection but is now rarely seen. It causes palsy of CNVI or VII and sometimes CNIII in addition to a unilateral headache.	Treating the underlying cause. Need codeine every 4 hours to suppress the pain and antibiotics to control the infection. Surgery is not employed in treatment of these conditions as often as it was formerly.	Hearing loss, tinnitus, fever. Can cause facial paralysis (CNVII), often affects CNVI and rarely CNIII or IV. Can progress to meningitis. Photophobia, increased lacrimation, reduced corneal sensitivity, paralysis of lateral rectus, diplopia, esotropia.
Pain due to dental disorders, abscessed teeth, malocclusion, temperomandibular joint disorders, mandibular joint neuralgia. (See muscle tension headache, temporal arteritis and glossopharyngeal neuralgia.)	Headache is usually moderate. A referred pain. However in Costen's syndrome the pain is severe, a glossopharyngeal neuralgia. This pain seems to be supraorbital even though CNIX is involved.	Pain in ear, jaw, throat, tongue and nose. Usually unilateral, frontal or temple, less commonly in vertex or occiput and extending to the neck and shoulders. Tender area over muscles of mastication. Some have a supraorbital pain associated with branches of CNV.	Constant but worse if chewing is attempted. Limitation of jaw movement. We clench our teeth when under stress.	Often constant with some fluctuations.	Dental malocclusion, or muscle spasm associated with psychological factors. Irritation of sensory branches of CNV and later sustained muscle contraction in head and neck. (Jaw claudication often develops with temporal arteritis.)	Dental treatment. Fewer than 5% of cases are due to arthritis of these temperomandibular joints. Costen's syndrome may have a large psychological component.	Temperomandibular joint disorders are more common in older patients especially women. Some hear a clicking or grinding when the jaw is moved, many suffer impaired hearing, tinnitus, low grade fever, dizziness; rarely photophobia, excess lacrimation, conjunctival hyperemia and nystagmus.
Headaches due to combined mechanisms, mixed headaches. (These headaches blur the distinction between migraine and muscle tension headaches, however any combination of causes can occur and complicate the diagnostic problem.)	Features of two or more types. Some people appear to progress from migraine type episodes to chronic, persisting headaches.	Various. Not very informative in terms of suggesting specific causes.	Daily or irregularly or following two patterns.	Years.	About 33% of those with migraine develop a muscle tension ache too. Muscle contraction can impair blood flow and lead to a local ischemic problem or even to a rebound vascular headache.	Reducing daily use of analgesics, and trying prophylactic agents, psychotherapy and biofeedback. Avoiding situations which precipitate or aggravate the discomfort.	In about 25% of cases a headache has two or more causes, or a person has two types of headache more or less concurrently. Any type of headache can be combined with or induce depression and muscle contraction especially when the headache persists.

Table 23

Headache Class.	Pain Type	Pain Location	Pain Frequency	Pain Duration	Probable Cause	Helped by:	Comments
Headache due to refractive errors or disorders of binocularity. (Cause less than 10% of headaches.) (Possibly 1% to 3% of headaches apparently localized in the ocular region have an ocular cause.) (See Roy's ocular migraine and ophthalmoplegic migraine.)	Headache is usually dull if due to refractive problems or disorders of binocular vision.	Rarely one-sided, supraorbital, frontooccipital and temporal. Pain in the distribution of the ophthalmic branch of CNV, but may be occipital with ocular muscle imbalance.	If due to refractive problems, pain appears daily, toward end of day or after intensive near work.	Until close work is stopped or the problem is corrected.	Undercorrected hyperopia, astigmatism, or disorders of binocularity including aniseikonia. Contraction of muscles of head and neck. Squinting and frowning have been described as use of the auxiliary muscles of accommodation.	Refractive correction, visual training, absorptive lenses, prisms where needed. Be sure no organic problem exists.	Photophobia often accompanies headaches whether they are of ocular origin or not and nearly always occurs with iritis and with corneal damage.
Uveitis.	Pain is usually dull but can be marked if due to anterior uveitis (or scleritis).	Orbit, nose, eye, temples.	Worse at night except for the photophobia which is worse in bright light.	Prolonged and becoming more severe.	Primary or secondary iritis. (Scleritis or other ocular conditions can cause similar discomfort.)	Uveitis requires analgesics, cycloplegics and steroids.	Flare and cells in anterior chamber, miosis, lower IOP, photophobia. Risk of posterior synechiae and secondary glaucoma.
Glaucoma. (Older patients with chronic headache should always have their intraocular pressure measured and their visual fields assessed.)	Pain can be severe if due to angle-closure glaucoma. Referred pain.	In the area supplied by the ophthalmic branch of CNV.	Intraocular pressure tends to increase during the day so that an angle-closure attack may develop at twilight or at a movie for example, when the pupil becomes dilated.	Usually until pressure is relieved but a few have short episodes and a rare person has no pain even with very high intraocular pressure.	Glaucoma. Field loss can progress even if intraocular pressure is only slightly elevated. Field losses, fundus changes and IOP show poor correlations in some glaucoma patients.	Immediate treatment is by drugs, pilocarpine, glycerol and possibly a carbonic anhydrase inhibitor but surgery (iridectomy) is required for adequate control of angle closure type glaucoma.	With an acute attack patients have nausea, vomiting, steamy cornea, a semi-dilated pupil and reduced vision. With open-angle glaucoma symptoms are minimal but field loss develops progressively.

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ANNOUNCEMENTS/NOUVELLES

COMING EVENTS

1987

May 24 - 27

Association for the Care of Children's Health (ACCH) 22nd Annual Conference
World Trade and Convention Centre
Halifax, NS

Information: ACCH
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20016-3007
USA
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1-800-565-7166
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October 22 - 25

12th Scandinavian Optometric Congress
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Hotel Novotel
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Information: Skandinavisk Optometri Kongres
c/o Borje Zakrisson
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1988

April 30 - May 3

Optica '88
World Optical Trade Fair
Cologne, West Germany

Information: KolnMesse
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M5G 1V2
Tel. (416) 598-3343

April 30 - October 30

Expo Down Under (World Expo 88)
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Information: The Communications Division
World Expo 88
234 Grey Street
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May 25 - 28

British Contact Lens Association International Contact Lens Centenary Congress and Exhibition
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Information: Conference Associates
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1987 OPTOMETRIST'S DESK REFERENCE — CORRECTION

Astute reviewers of the 1987 Optometrist's Desk Reference have pointed out that seventeen of Nova Scotia's optometrists have vanished at the end of p.117. A typesetting computer's glitch inadvertently located them on p.92, directly after the Winnipeg, Manitoba listings.

CAO apologizes for any inconvenience caused to the practitioners of either province.