CHAPTER 8 – CENTRAL NERVOUS SYSTEM

First Nations and Inuit Health Branch (FNIHB) Clinical Practice Guidelines for Nurses in Primary Care.
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ASSESSMENT OF THE CENTRAL NERVOUS SYSTEM

The following characteristics of each symptom should be elicited and explored:

- Onset (sudden or gradual)
- Chronology
- Current situation (improving or deteriorating)
- Location
- Radiation
- Quality
- Timing (frequency, duration)
- Severity (use a pain scale, for example, 0–10)
- Precipitating and aggravating factors
- Relieving factors
- Fever, shortness of breath, headache, abnormal motor activity, change in vital signs[1]
- Associated symptoms
- Effects on daily activities
- Previous diagnosis of similar episodes
- Previous treatments
- Efficacy of previous treatments
- Medication use, dose, timing and frequency (for example, neuroleptics, benzodiazepines)[2]
- Precipitating events (for example, trauma)[2]

CARDINAL SYMPTOMS

In addition to the general characteristics outlined above, additional characteristics of specific symptoms should be elicited, as follows:

General Cerebral Function

- Changes in memory
- Changes in mood, affect and behaviour[3]
- Changes in concentration
- Changes in speech pattern[2]

Cranial Nerve Function

- Changes in vision, drooping eyelids
- Facial weakness
- Speech impediment
- Hearing loss, unusual noise in ears, difficulties with balance
- Impairment of sense of smell or taste
- Change in pupil reaction[2]

Headaches

- Onset, age at onset
- Pattern, changes in pattern
- Location, description, whether pulsating
- Time of day, present on awakening
- Duration
- Precipitating factors
- Associated symptoms: nausea, vomiting, sensory disturbances, photophobia

Changes in Level of Consciousness

- Dizziness
- Fainting, (syncope, or near syncope)[4]
- Convulsions, previous seizure disorder[4]
- History of head injury associated with loss of consciousness
- Refer to Glasgow Coma Scale for determinants of level of consciousness (see the Glasgow Coma Scale at: http://www.unc.edu/~rowlett/units/scales/glasgow.htm). General rule: 90% of patients with a score ≤ 8 will be in a coma. A coma is defined as not opening eyes, not obeying commands and not uttering understandable words[2]

Motor Function

- Muscle weakness, paralysis, stiffness
- Generalized, proximal or distal weakness[4]
- Clumsiness, ataxia
- Staggering gait with wide-base stance
- Tremor
- Involuntary movements, difficulty with gait or balance, restlessness of the legs[5]

Sensory Function

- Loss of or decrease in sensation
- Sensation of “pins and needles,” tingling
- Burning sensation

Other Associated Symptoms

- Bowel or bladder dysfunction
- Impotence
- Pain
- Fever, changes in vital signs[3]

MEDICAL HISTORY (SPECIFIC TO CENTRAL NERVOUS SYSTEM)

- Seizures
- Head trauma
- Metabolic disease (for example, diabetes mellitus, thyroid problems)
- Cardiac disease (for example, hypertension, heart block)
- Transient ischemic attack
- Demyelinating diseases (for example, multiple sclerosis, Parkinson's disease)
- Alcoholism, illicit drug use
- Chronic headaches (migraine, tension)
- Psychiatric illness (for example, depression, bipolar disorder)
- Bell's palsy
- Recent infection (for example, herpes zoster, meningitis)[2]
- Medications (identify those associated with CNS side effects)

FAMILY HISTORY (SPECIFIC TO CENTRAL NERVOUS SYSTEM)

- Seizures
- Metabolic disease (for example, diabetes mellitus)
- Cardiac disease (for example, hypertension, myocardial infarction, stroke)
- Demyelinating diseases (for example, multiple sclerosis, Parkinson's disease)
- Migraine headaches
- Cerebral aneurysms, arteriovenous (AV) malformations
- Psychiatric illness

PERSONAL AND SOCIAL HISTORY (SPECIFIC TO CENTRAL NERVOUS SYSTEM)

- Alcoholism
- Drug abuse
- Occupational exposure to neurotoxins

EXAMINATION OF THE CENTRAL NERVOUS SYSTEM

- Apparent state of health
- Appearance of comfort or distress
- Colour (for example, flushed, pale, cyanotic)
- Nutritional status (emaciated or obese)
- Match between appearance and stated age

SCREENING EXAMINATION

The screening examination will reveal areas of difficulties. If deficits are discovered, a more in-depth examination is required.

To Be Assessed During History-Taking

- Level of consciousness (ease of arousal, state of awareness, orientation to person, place and time)
- Mental status
- Speech (clarity, content, volume, rate)

Cranial Nerves

See Table 1, "Screening Tests for Cranial Nerves."

Table 1 – Screening Tests for Cranial Nerves

Cranial Nerve	Test
I – Olfactory	Smell (test only if there is a specific complaint)
II – Optic	Visual acuity Visual fields Funduscopic examination
III – Oculomotor IV – Trochlear VI – Abducent	Pupillary response (direct or consensual)
	Extraocular eye movements
V – Trigeminal	Motor function: clench teeth, open jaw
	Sensory function: pain (sharp stimulus); light touch (cotton wisp); sensation on forehead, cheek, chin
	Corneal reflex (omit if client is conscious)
VII – Facial	Facial symmetry: raise eyebrows, frown, close eyes tightly against resistance, show teeth, puff cheeks, smile
VIII – Acoustic (Vestibulocochlear)	Hearing (watch ticking, whisper)
	Rinne and Weber tests
IX – Glossopharyngeal X – Vagus	Movement of palate, uvula, pharyngeal wall
	Gag reflex and swallowing
	Hoarseness
XI – Spinal accessory	Shoulder shrug against resistance
	Head turn against resistance
XII – Hypoglossal	Stick out tongue, push tongue against each cheek

Motor Function, Sensory Function and Reflexes

Assess motor function, sensory function and reflexes together, as follows:

Arms and Hands

- Grip strength
- Raise both arms and hold (assess for palmar drift)
- Finger-nose test (assess for eye-hand coordination)
- Blunt and sharp pin prick
- Reflexes (biceps, triceps, brachioradialis [supinator])

Legs

- Straight-leg raising
- Bowstring test
- Quadriceps test
- Heel-to-toe walk
- Heel-shin test
- Romberg test
- Blunt and sharp pin prick
- Reflexes (Achilles' tendon, patellar, plantar)

Meningeal Irritation

Test for meningeal irritation if indicated:

- Neck stiffness
- Brudzinski's sign
- Kernig's sign

COMMON PROBLEMS OF THE CENTRAL NERVOUS SYSTEM

BELL'S PALSY

Bell's palsy, or idiopathic facial paralysis, is the most common cause of non-iatrogenic paralysis of the seventh cranial nerve[6] causing sudden, painless, unilateral paralysis of facial muscles due to inflammation and swelling. The condition usually resolves spontaneously.

CAUSES

- Largely unknown
- Possibly viral infection of facial nerve (commonly herpes simplex virus)
- May be related to Lyme disease and HIV infection
- Hereditary and vascular factors may be contributory

Risk Factors

- Pregnancy (third trimester)
- Positive family history
- Hypertension
- Diabetes mellitus

HISTORY

- Key classic symptoms include: acute unilateral facial weakness, post-auricular pain and unilateral lacrimation[7]
- Symptoms develop within 24–48 hours[7]
- Sudden onset of unilateral facial weakness
- Progression to complete paralysis within a few hours
- Inability to close eye on affected side
- Excessive tearing of affected eye may be present
- Taste sensation may be altered
- Hypersensitivity to sound
- Pain in or behind ear may occur on affected side just before onset of facial weakness
- Landmark for recovery usually 3 months after initial symptoms and patients whose recovery is delayed beyond this time experience significant sequelae[8]

PHYSICAL FINDINGS

- Client appears anxious
- Flat nasolabial fold
- Client unable to close eye, raise eyebrow or smile on affected side
- Eyeball rolls upward when client attempts to close eyelid
- Drooling may be present
- Sensation to light touch and pin prick may be reduced over affected area
- Taste affected over anterior 2/3 of tongue[7]
- Inability or difficulty smiling, whistling or grimacing[7]
- May present with fever, tinnitus and mild hearing deficit[9]

DIFFERENTIAL DIAGNOSIS

- Stroke (brain stem)
- Cerebral tumour
- Parotid gland tumour
- Middle ear or mastoid infection
- Meningitis
- Head or facial trauma with fracture
- Lyme disease
- Herpes zoster oticus
- Guillain-Barré syndrome
- Multiple sclerosis
- Ramsay Hunt syndrome[7]
- Trauma (temporal bone fracture)[8]

COMPLICATIONS[10]

- Corneal abrasion, ulceration and blindness
- Keratitis
- Chronic facial weakness
- Facial muscle contracture
- Impaired nutrition secondary to paralysis

DIAGNOSTIC TESTS

- Bell's palsy is a diagnosis of exclusion and 90% of cases can be diagnosed without use of diagnostic tests[7]
- If after consultation there is uncertainty about the diagnosis, or the patient has gradual progression of symptoms over the follow-up period, discuss referral to a neurologist for further diagnostic testing[8]
- Obtain a Lyme (Borrelia burgdorferi) title if there is a history of exposure to ticks and individual resides in a geographic location where this disease is present[11]

MANAGEMENT

Pregnant women who develop Bell's palsy should be monitored closely for hypertension and preeclampsia. Most cases occur in the third trimester or postpartum.[11]

Goals of Treatment

- Protect the eye from injury
- Prevent complications
- Alleviate pain

Management is directed toward the symptoms and depends on the time and severity of presentation.

Appropriate Consultation

Consult a physician immediately. If within 72 hours of onset and the client is at high risk for denervation (for example, full unilateral facial paralysis, > 50 years of age), drug therapy may be indicated *(see "Pharmacologic Interventions").*

Nonpharmacologic Interventions

Reassure client that full recovery can be expected, in most cases in 6–8 weeks.

Client Education

- Recommend adequate nutritional intake and suggest that client direct food and liquids to unaffected side of mouth to prevent drooling and to promote proper mastication
- Recommend adequate oral hygiene after meals to prevent collection of food and liquids within affected cheek
- Suggest protection of affected eye to prevent corneal abrasions (for example, wearing sunglasses during the day to prevent dust particles from entering eye, taping the eye closed at night)
- Recommend facial exercises and massage, to be performed 2 or 3 times daily to prevent muscle atrophy (wrinkle forehead, blow out cheeks, purse lips, close eyes)
- Recommend and educate to report any eye pain or visual problems due to increased risk for corneal abrasion and ulceration[7]
- Suggest facial exercises including opening and closing mouth and moving jaw laterally 3 times a day and prn[7]
- Encourage the avoidance of exposing face to cold drafts[7]
- Physical therapy may be beneficial including heat therapy, electrical stimulation or massage[11]

Pharmacologic Interventions

If there is ocular involvement, recommend frequent use of artificial tears to prevent drying of eye during the day and eye ointment and a patch at night[12] Corticosteroids are the mainstay of drug therapy[13] and should be ordered by a physician. When started within 72 hours of the onset of symptoms, prednisolone (equivalent to prednisone) significantly increases the proportion of patients who recover full facial function within 3 months.[13]

prednisone 60–80 mg/day (that is, 1 mg/kg/day) PO x 5 days[12]

Antiviral drugs with activity against herpes simplex virus (acyclovir, famciclovir, valacyclovir) may be prescribed by some physicians as an adjunct to corticosteroids.[12] However, antiviral drugs are ineffective when used alone for Bell's palsy and are, at best, of marginal benefit when used with corticosteroids.[12] A recently published, large, randomized trial showed that acyclovir was ineffective when administered alone or in combination with prednisolone.[13]

Counsel client about appropriate use of prescribed medications (dose, frequency, side effects).

Analgesia may be required during the first few days:

acetaminophen (Tylenol), 325 mg, 1–2 tabs PO q4h prn (maximum 4 g/day)

or

ibuprofen (Motrin), 200–400 mg PO q6h prn

Monitoring and Follow-Up

- Arrange daily follow-up for several days
- Patients with facial paralysis or weakness related to Bell's Palsy should be seen several times a week for the first 2–3 weeks to ensure symptoms are not progressing[14]

Referral

- Refer to a physician if complications are suspected or detected or if condition does not resolve within 4 weeks
- Consider referral without delay if any eye symptoms or signs develop[14]

HEADACHES: GENERAL PRINCIPLES

Headaches can be defined as diffuse pain in various parts of the head where the pain is not confined to the area of distribution of a nerve. Headaches are among the most common complaints in primary practice.[15] It is diagnostically and therapeutically useful to divide headaches into two categories:

Primary: benign, usually recurrent and not associated with an underlying structural defect or organic disease. Primary headache disorders include:

- Migraine
- Tension-type headache
- Cluster headache

Secondary: caused by underlying structural defects or organic diseases, less than 2–10% of headaches are secondary.[14,16] Most headaches (90%) are benign.

There is a wide variety of causes of headaches, ranging from abnormalities of the head and neck to systemic illness. Other causes include drugs, alcohol or chemicals.

When a client presents with headache, an accurate, precise description of the pain must be obtained:

- Age at onset of headaches, if recurrent
- Location
- Quality
- Duration
- Time of occurrence (first time, recurrence, onset in morning, evening or during the day)
- Frequency (daily, monthly, occasionally)
- How headache begins
- How headache progresses
- Interference with daily activities
- Symptoms associated with the onset and progression of pain (for example, loss of consciousness, aura, nausea, vomiting, photophobia, sensory changes and fever)
- Aggravating and relieving factors (foods, allergies, medication, position of head, noise, light, alcohol consumption, stress)
- Lifestyle factors that precipitate headache (at home, at work, anytime)
- Occupation
- Family history of headaches (including type)
- Pre-existing medical conditions (for example, glaucoma, hypertension, infection, anxiety, depression, seizures)
- Medication history: dose, duration, frequency of prescription and nonprescription drugs

DIFFERENTIAL DIAGNOSIS

Primary

- Migraine
- Tension (muscle contracture)
- Cluster
- Other
  - Benign cough-related
  - Benign exertional
  - Post-traumatic

Secondary

Disorders of the Cerebral Parenchyma

- Brain tumour
- Brain abscess
- Intracranial hemorrhage
- Cerebral trauma
- Hydrocephalus
- Benign intracranial hypertension

Disorders Involving the Meninges

- Meningitis
- Subarachnoid bleeding

Disorders Involving the Extracranial Structures

- Dental abscess
- Paranasal sinusitis
- Temporomandibular joint syndrome
- Closed-angle glaucoma
- Trigeminal neuralgia
- Herpes zoster infection
- Retro-orbital disease process

Metabolic Causes

- Food additives (for example, nitrites, monosodium glutamate, alcohol)
- Toxins
- Medication (for example, nitrates, oral contraceptives, calcium channel blockers)
- Fever
- Hypercapnia (increased carbon dioxide levels)

Vascular Causes

- Hypertension
- Vasculitis
- Embolic or thrombotic events

Features Suggestive of a Serious Cause of Headache

- Advanced age
- Worst headache ever experienced
- Onset with exertion
- Decreased alertness or cognition
- Radiation of pain between the shoulder blades (which suggests spinal arachnoid irritation)
- Association with nuchal rigidity
- Any history or physical finding suggestive of infection (for example, fever)
- Headache worsening under observation

TENSION HEADACHE

Most common headache type, occurring in both genders and all age groups. Characterized as bilateral, often slowly progressing, non-throbbing, mild to moderate pain. Physical activity does not worsen pain.[14] Diffuse pain in the head.

- Episodic: usually associated with some stressful event, of moderate intensity, self-limited and responds to nonprescription medication
- Chronic: often occurs daily (must be present for at least 15 days per month for 6 months to be considered chronic); pain often bilateral, usually occipito-frontal and associated with contraction of muscles of the neck and scalp

CAUSES

- Stress or anxiety
- Poor posture
- Jaw clenching
- Intramuscular vasoconstriction of scalp muscles
- Depression, anxiety

Risk Factors

- Excess caffeine intake or caffeine withdrawal (headache is the most common symptom of caffeine withdrawal)[17]
- Medications (for example, overuse of analgesics – all drugs used for acute treatment of headache, including butalbital, acetaminophen, opioids, aspirin and other NSAIDs, triptans and ergotamine are associated with medication overuse headache)[18]
- Obstructive sleep apnea
- Family history
- Gender (higher prevalence in females)

HISTORY

- History may be vague
- No obvious relieving or precipitating factors identified
- Medication use: type, frequency, amount, effect
- Often associated with abuse or overuse of medications
- 40% of patients have positive family history
- Pain may become more constant and severe over time
- Stressful events may aggravate symptoms

Features of Headaches

- Generalized
- Constant
- Dull, tight sensation
- Occasionally throbbing
- Present on rising in the morning
- Wax and wane during the day
- Prevent client from falling asleep, but never awaken client from sleep
- Medication affords only minimal or no relief

Associated Symptoms

- Nausea
- Anorexia
- Weight loss
- Dyspepsia
- Diarrhea
- Fatigue
- Early morning awakening
- Concentration impaired
- Libido reduced (as in depression)

PHYSICAL FINDINGS

- Client in no distress, although may complain of headache at time of presentation
- Results of neurologic examination completely normal
- Muscular tightness or tenderness in the neck, upper trapezius, occipital and frontal scalp muscles

DIFFERENTIAL DIAGNOSIS

Although most chronic headaches are benign, it is important to rule out other more serious problems:

- Caffeine dependency
- Nonprescription drug dependency (for example, acetaminophen with or without codeine)
- Dental disease
- Post-traumatic headache
- Temporomandibular joint dysfunction
- Depression
- Cervical spondylosis
- Chronic sinusitis
- Temporal arteritis
- Migraine headache
- Eye problem
- Middle ear disease
- Severe anemia
- Hypoxia
- Hypertension
- Intracranial infection (meningitis)
- Intracranial tumour

COMPLICATIONS

- Interference with daily activities
- Dependence on analgesic medication
- Absenteeism from work or school
- Depression

DIAGNOSTIC TESTS

None.

MANAGEMENT

Goals of Treatment

- Identify symptoms suggestive of serious pathology
- Relieve symptoms

Appropriate Consultation

Consult physician if symptoms suggest serious pathology (for example, neurologic deficit). Otherwise, treat conservatively and follow.

Nonpharmacologic Interventions

- Provide supportive environment
- It is important for success of therapy that caregiver be nonjudgmental
- Explore current life situation: encourage client to talk about worries, concerns, fears
- Discover areas of difficulty that could contribute to headaches
- Evaluate stress level
- Ice packs may help
- Massage therapy may help
- Rest in dark, quiet room may help
- Recommend gradual decrease in use of caffeinated products

Client Education

- Counsel client about appropriate use of medications (dose, frequency, avoidance of overuse)
- Suggest stress management strategies (for example, relaxation techniques)
- Encourage individual to keep a headache diary to identify potential triggers and risks[14]

Pharmacologic Interventions

Analgesics:

acetaminophen (Tylenol), 325 mg, 1–2 tabs PO q4-6h prn (maximum 4 g/day)

or

ibuprofen (Motrin), 200–400 mg PO q6-8h prn

Monitoring and Follow-up

Follow up in 1–2 weeks to assess response to interventions. Suggested follow-up for individuals with cluster or tension type headaches is 2–4 weeks after patient is placed on therapy to determine efficacy.[14]

Referral

Refer to a physician if there is failure to respond to therapy or if there is concern about an underlying disorder.

CLUSTER HEADACHE

Diagnostic criteria for cluster headaches include at least five attacks with:

1. Severe unilateral orbital or suborbital and/or temporal pain lasting 15 minutes to 3 hours.
2. Headache associated with at least one of the following signs on the pain side:
   a. conjunctival injection
   b. lacrimation
   c. nasal congestion
   d. rhinorrhea

e. forehead and facial sweating
f. miosis
g. ptosis
h. eyelid edema

3. Headaches occur with a frequency of one to eight attacks per day.[19]
4. Recurrent attacks of severe unilateral headaches around the eye and temple. Attacks last approximately 30–120 minutes and occur one to three times per day, at the same time of day, for up to 12 weeks; this pattern is typically followed by 1–24 months without an attack.

CAUSES

Unknown.

Risk Factors

– Male > 30 years of age
– Possible relationship to previous head injury
– May be triggered by alcohol, nitroglycerin, disturbance in sleep cycle, emotion (anger), excessive physical activity

HISTORY

– Client usually male, older than mid-20s
– Cyclic or seasonal pattern to attacks
– Sudden onset of unilateral pain
– Headache usually begins without warning, often during sleep
– Begins as dull ache, which quickly increases to severe pain
– Pain intensity peaks within 15 minutes
– Pain steady, boring, piercing and centered about one eye (retro-orbital)
– No aggravating or relieving factors
– Pain extends into adjacent cheek, temple, forehead
– Usually resolves within 30–120 minutes, leaving client fatigued
– Pain recurs later the same day or at same time next day
– Cycle repeats itself until "cluster" ends

Associated Symptoms During Attack

– Agitation, pacing
– Red, tearing eye
– Drooping eyelid
– Nose on unaffected side runs profusely
– Nausea (vomiting is rare)
– Perspiration

PHYSICAL FINDINGS

– Heart rate elevated during attack
– Bradycardia may be present

During Attacks

– Acute distress
– Pale
– Diaphoretic
– Restless
– Ipsilateral rhinorrhea
– Ptosis of affected eyelid
– Conjunctival redness and excessive tearing of affected eye

Between Attacks

– Client feels well (that is, completely asymptomatic)
– Results of neurologic examination normal

DIFFERENTIAL DIAGNOSIS

– Temporal arteritis
– Subarachnoid hemorrhage (initial presentation)
– Episodic, long-lasting tension headaches
– Trigeminal neuralgia
– Acute glaucoma
– Sinusitis

COMPLICATIONS

– Interference with daily activities
– Absenteeism from work or school
– Weight loss during "cluster"
– Depression
– Potential for drug abuse (for example, analgesics)

DIAGNOSTIC TESTS

None.

MANAGEMENT

Goals of Treatment

– Relieve pain
– Prevent recurrence

Appropriate Consultation

– Consult a physician for acute attack
– If symptoms are significant during an initial attack, serious pathology must be ruled out

Adjuvant Therapy

Oxygen (high flow), 100% by non-rebreather mask, 6–10 L/min during an attack is helpful in up to 70% of clients.

Nonpharmacologic Interventions

Client Education

- Explain expected course of disease and prognosis and how to avoid precipitants
- Recommend avoidance of alcohol, bright light, anger, stressful activity or undue excitement during a cluster
- Avoid smoking during a cluster
- Counsel client about smoking cessation

Pharmacologic Therapy

Do not give analgesics to a client with an undiagnosed headache until you have consulted a physician, as they may mask the progression of neurologic symptoms.

Treatment of an Acute Attack

First-line treatment for acute cluster headache is a serotonin receptor agonist ("triptan").[20]

Subcutaneous sumatriptan 6 mg (maximum 2 doses within 24 hours) is effective in about 75% of patients within 20 minutes. (Oral administration of triptans is only modestly effective for acute cluster headache.)[20]

Triptans should be avoided in patients with ischemic heart disease, Prinzmetal's (vasospastic) angina, uncontrolled hypertension, stroke or pregnancy.[20]

Prophylaxis

Prophylaxis (for example, verapamil) should be started as soon as possible at the onset of a cluster episode.[20] Corticosteroids (for example, prednisone) are also useful for short term prophylaxis.[20] Triptans are not effective for the prophylaxis of cluster headaches. Consult a physician.

Counsel client about appropriate use of medications (dose, frequency, compliance, avoidance or abuse of analgesics).

Monitoring and Follow-Up

- Monitor medication compliance
- Assess effectiveness of prophylaxis
- Assess for depression
- Assess for analgesic abuse or dependence

Referral

- Refer all previously undiagnosed clients as soon as possible to a physician during an acute attack
- Clients with chronic recurrence of cluster headaches should be evaluated by a physician if symptoms are not controlled by prophylaxis

MIGRAINE HEADACHES

Recurrent headaches due to vascular disturbances. Symptoms are similar to tension headaches and manifestations of individual migraine attacks vary between and among individuals.[21]

CAUSES

- Constriction and dilatation of intracranial and extracranial arteries
- Individual attacks may be triggered by specific foods (for example, chocolate, cheese, smoked meats, alcohol), missing meals, menstrual cycle, oral contraceptives, fatigue, excessive sleep, stress or relief of stress, excessive or flickering light
- Associated with epilepsy, hereditary hemorrhagic telangiectasia, Tourette's syndrome, ischemic stroke, depression[16]

Risk Factors

- Female
- Young age (10–30 years)
- Family history of migraine

HISTORY

- Regular or near regular perimenstrual or periovulatory timing
- Lessened by sleep
- Prodrome may be present: irritability, mood swings, changes in energy level, food cravings, fluid retention
- Aura (including visual defects and sensory losses) may be present: precedes headache, lasts approximately 5–30 minutes, recedes with onset of headache (although sometimes aura and headache may overlap); 80% of migraines occur without aura

Pain of Headache

- Unilateral or diffuse
- Moderate to severe intensity
- Peaks within 1 hour

– Pulsating in nature (at onset or any time during attack)
– Rest in a dark, quiet room helps
– Bending forward or moving head increases pain

Associated Symptoms

– Photophobia (aversion to light)
– Phonophobia (aversion to noise)
– Osmophobia (aversion to odours)
– Nausea and vomiting
– Diarrhea, constipation
– Chills, tremor, sweating

PHYSICAL FINDINGS

During Attack

– Moderate distress
– Pale
– Diaphoretic
– Scalp arteries may be distended
– Photophobia
– Scalp tenderness
– Results of neurologic exam usually normal during and between attacks

International Headache Society Criteria for Diagnosing Migraine without Aura[22]

1. At least 5 attacks fulfilling criteria 2, 3, 4 and 5
2. Each attack, untreated or treated unsuccessfully, lasts 4–72 hours
3. Each attack has at least 2 of the following characteristics:
   – Unilateral most often, but 30% to 40% have bilateral pain
   – Pulsating quality (occurring at any time during the attack); 50% of those with migraines report non-throbbing pain; headache quality may vary over the course of the attack
   – Moderate or severe intensity, enough to interfere with daily activities
   – Pain aggravated by physical activity such as walking up or down stairs
4. During an attack at least one of the following symptoms is present:
   – Nausea and/or vomiting
   – Photophobia and phonophobia
5. There is no evidence from the client's history or physical examination of any other disease that might cause headaches

Criteria for Diagnosing Migraine with Aura[22]

The criteria are the same as for migraine without aura, with the exception of requiring at least 2 attacks, and include symptoms of neurologic dysfunction (including visual disturbances) before or during attack, not attributed to another disorder.

DIFFERENTIAL DIAGNOSIS

– Disorders or infections of head and neck
– Systemic illness
– Toxic effects of drugs, alcohol, chemicals
– Intracranial lesion
– Stroke
– Drug-seeking behaviour
– Refer also to above criteria for secondary causes of headaches

COMPLICATIONS

– Family and marital dysfunction if headaches frequent
– Absenteeism from work or school
– Depression
– Drug addiction (for example, to prescription opioid analgesics)

DIAGNOSTIC TESTS

None.

MANAGEMENT

Goals of Treatment

– Therapy will vary depending on the severity of each attack[21]
– Identify and modify trigger factors
– Relieve symptoms
– Prevent recurrences

Appropriate Consultation

Consult physician if an acute attack is moderate to severe (on a pain scale of 0 to 10, pain ≥ 5) and is unresponsive to first-line drug therapy, or if attacks recur and are not controlled with current prophylactic regimen.

Severe Attack

Consult physician for medication orders.

Nonpharmacologic Interventions

Mild or Moderate Attack

- Rest in dark, quiet room
- Ice packs
- Pressure massage of the scalp
- Relaxation therapy
- Cognitive behavioural therapy (for example, stress management training)

Severe Attack

- Bed rest in dark, quiet room
- Nothing by mouth temporarily if vomiting is significant

Client Education

- Recommend that patient keep a diary to record headache characteristics, use of medications, potential triggers and response to therapy[21]
- Explain expected disease course and prognosis
- Counsel client about appropriate use of medications (dose, frequency, avoidance of overuse or abuse)
- Recommend regular rest and activities, appropriate diet
- Help client to identify trigger factors and then to attempt to reduce or eliminate them
- Help client to identify and avoid other causative factors (for example, coffee, chocolate, alcohol, certain foods, oral contraceptives, nuts, cheese)

Adjuvant Therapy

Moderate Attack

Assess state of hydration and consider IV access for hydration and medication administration.

Severe Attack

For severe attack only, start IV therapy with normal saline; adjust rate according to state of hydration.

Pharmacologic Therapy

Symptomatic Therapy for Acute Attacks

Nonsteroidal Anti-Inflammatory Drugs

Single dose treatment for acute attack:[23]

ibuprofen 400–1200 mg PO

naproxen 500–1000 mg PO

Oral acetaminophen (Tylenol) has not been shown to be effective for the treatment of acute migraine headache[24] and it is not recommended. It may however, be used in the pregnant migraineur.[25]

Triptans such as sumatriptan are effective and specific drugs that are indicated for the treatment of moderate to severe acute attacks. Triptans should be avoided in patients with ischemic heart disease, Prinzmetal's (vasospastic) angina, uncontrolled hypertension, stroke or pregnancy.[26]

Use of opioid analgesic-containing preparations (for example, acetaminophen, codeine and caffeine) may be suitable for the treatment of moderate to severe migraine, but should be limited because of the risk of rebound headache and the potential for dependency.[24] A Canadian study concluded that patients who received opioids as first-line therapy for migraine were more likely to return to the emergency department with a headache within seven days, that management of acute migraine did not follow consensus[27] guidelines and that a concerted effort was needed to replace narcotics with evidence-based first-line treatments.

Antiemetics are often given as adjunctive therapy in the treatment of moderate to severe migraine, particularly if accompanied by nausea or vomiting:[24]

metoclopramide 10 mg IM/IV

Prophylactic Therapy

The goals of prophylactic therapy for migraine are to reduce the frequency, severity and

duration of attacks; improve the responsiveness to treatment of acute attacks and improve function and reduce disability resulting from acute attacks.

Prophylaxis is indicated when:[28]

- headaches are frequent and the patient expresses a preference for prophylaxis
- recurring migraine attacks interfere with daily routines despite acute treatment
- acute therapies fail, are overused, contraindicated, too expensive or are associated with adverse events

Note: The American Academy of Neurology considers the number of attacks per unit time (that is, 2 or 3 per month) to be an arbitrary criterion that does not account for patient needs. For this reason it no longer uses specific frequency as a criterion for prophylaxis.[25]

Drug classes that are potentially useful for the prophylaxis of migraine include antihypertensives, anticonvulsants and antidepressants. Drugs associated with the best evidence for efficacy and acceptable tolerability include propranolol, metoprolol, nadolol, verapamil, amitriptyline, nortriptyline, topiramate and valproic acid.[28]

Consult a physician before starting prophylactic treatment.

Instruct any female client to report if she becomes pregnant or is contemplating pregnancy, as some prophylactic drug therapies will have to be stopped.

Monitoring and Follow-Up

Mild or Moderate Attack

Encourage regular follow-up until headaches are effectively controlled; frequency of follow-up should be individualized to each person's unique circumstances.

Severe Attack

Monitor response to therapy and vital signs.

Referral

Mild or Moderate Attack

- Arrange follow-up with physician to discuss prophylactic therapy if headaches are frequent or severe enough to interfere with daily activities
- Referral for a neurologic examination may be needed if optimum first-line therapy and prophylaxis fail to control attacks

Severe Attack

Medevac may be required if attack is prolonged and unresponsive to therapy. In ultrasevere cases, (a condition known as status migrainous), there is prolonged (for more than 72 hours) inability to function in any useful capacity.[21]

TEMPORAL (GIANT CELL) ARTERITIS

Giant cell arteritis (GCA) is the most common idiopathic vasculitis of large- and medium-sized vessels. It is thought to be a systemic condition involving inflammation of the arteries that originate from the aortic arch, including, characteristically, the temporal arteries.[29]

CAUSES

Possibly autoimmune.

HISTORY

- Age > 50 years
- Flu-like symptoms initially
- Pain slight and transient initially
- Pain becomes more severe (throbbing or boring) and constant over several days
- Headache unilateral or bilateral
- Headache located in temporal or periorbital area
- Onset gradual or sudden
- Not relieved by over-the-counter analgesics
- Previous diagnosis of polymyalgia rheumatica
- Pain associated with chewing[30]

Associated Symptoms

- Malaise
- Night sweats
- Fever
- Shoulder and back pain
- Reduced vision of eye on affected side

PHYSICAL FINDINGS

- Scalp tenderness[29]
- Claudication of extremities and jaw[29]
- Bruits over carotid, axillary, brachial and subclavian arteries[29]
- Diminished or absent pulses and hypoperfusion of extremities[29]
- Temperature may be mildly elevated
- Client appears mildly-to-moderately ill
- Visual acuity may be reduced on affected side
- Problem with visual acuity may progress to other eye
- Range of motion of shoulder(s) may be reduced; shoulder movement may be painful
- Shoulder joint may be tender
- Temporal artery may be firm, nodular, non-compressible, tender
- Temporal artery may have diminished or absent pulsation

DIFFERENTIAL DIAGNOSIS

- Other disorders of head and neck
- Systemic illness

COMPLICATIONS

- Aortic aneurysm occurs in 1 of 5 patients with GCA; aortic aneurysm with aortic dissection occurs in 1 of 16 patients[29]
- Blindness on affected side
- Progression to blindness of other eye
- Stroke
- Coronary occlusion
- Arterial insufficiency of upper extremities

DIAGNOSTIC TESTS

- Erythrocyte sedimentation rate (ESR), if available (ESR will be > 50 mm/hour)
- ESR may be normal in 5% to 25% of patients with GCA[29]
- In patients with suspected GCA, a temporal artery biopsy should always be performed to confirm diagnosis[29]

MANAGEMENT

Goals of Treatment

- Confirm the diagnosis
- Prevent complications

Appropriate Consultation

Consult a physician immediately if GCA is suspected. Delaying treatment could lead to vision loss. GCA is a medical emergency that can lead to stroke or blindness.[30]

Pharmacologic Therapy

Oral prednisone may be initiated by a physician if transfer to hospital will be delayed.

Referral

Arrange transfer to hospital for further investigation and treatment as soon as possible (biopsy of temporal artery is needed to confirm diagnosis).

TRANSIENT ISCHEMIC ATTACK

Transient ischemic attack (TIA) is a neurologic deficit attributed to focal cerebral, retinal or spinal ischemia without acute infarction. Onset is rapid, and symptoms are of variable duration, typically lasting 2–15 minutes but rarely as long as 24 hours. Most transient ischemic attacks (TIAs) last less than 1 hour.[31]

TIA is an important omen of impending stroke:

- 1% to 15% of patients have a stroke within 3 months, with half of these occurring within 48 hours[32]
- One-third of patients have a stroke within 5 years of the first event

CAUSES

- Temporary reduction or interruption of cerebral blood flow in a focal area
- Underlying problem: atherosclerosis of carotid or vertebrobasilar arteries
- Atrial fibrillation[32]

Risk Factors

- Older age
- Hypertension
- Diabetes mellitus
- Heart disease
- Cardiac arrhythmias (atrial fibrillation)
- Carotid artery disease
- Smoking
- Family history
- Sedentary lifestyle, poor dietary habits[33]
- Stressful environment[33]
- Alcohol consumption (more than 2 drinks/day or ≥ 14 drinks/week in men, ≥ 9 drinks/week in women)[33]

HISTORY

- Usually one of above risk factors is present
- Characterized by sudden onset of neurologic deficits (weakness, incontinence, loss of vision, aphasia, dysarthria, headaches or vertigo)[33]
- Attacks may occur several times a day or once or twice a year
- Symptoms generally similar during repeat attacks
- Identify previous evidence of peripheral vascular disease, coronary artery disease
- Symptoms acute at onset
- Symptoms resolve completely in 24 hours
- Client remains conscious throughout attack
- Symptoms depend on affected blood vessel:
  - Carotid artery: unilateral symptoms, ipsilateral blindness, contralateral weakness or paresthesia, aphasia, headache (may follow attack)

  - Vertebrobasilar arteries: confusion, vertigo, binocular blindness or diplopia, weakness or paresthesia of extremities, drop attacks in which client remains conscious but suddenly collapses
- Slurred speech may be present

PHYSICAL FINDINGS

Because TIA may be brief, the results of a physical examination may be entirely normal. Careful examination of the neurologic and cardiovascular systems is required. Look for evidence of atherosclerosis (for example, peripheral vascular disease and heart disease).

- Blood pressure is often normal but hypertension may be present
- Heart rate is often normal, but may be irregular (because of underlying atrial fibrillation)
- Client usually looks well
- Muscular weakness on affected side may be obvious or subtle
- Visual acuity may be reduced
- Balance may be slightly affected
- Confusion may be evident
- Look for old surgical scars from previous heart surgery
- Focal sensory deficits
- Focal motor deficits
- Deep tendon reflexes may be increased or decreased for up to 24 hours after attack
- Carotid artery thrill may be present
- Carotid bruit(s) may be present
- Other peripheral arterial bruits may be present (for example, aortic, iliac)
- Diminished peripheral pulses if peripheral vascular disease is present
- Heart murmur may be present

DIFFERENTIAL DIAGNOSIS

Differential diagnosis includes anything that can cause decreased cerebral blood flow with cerebral ischemia or transient impairment of cerebral function.

- Hypotension
- Bell's palsy
- Dissecting aortic aneurysm
- Heart disease
- Focal seizure
- Cerebrovascular accident
- Hypoglycemia
- Anemia
- Compressive neuropathy[32]
- Anxiety[32]
- Vasovagal syncope[32]

COMPLICATIONS

- Future cerebrovascular accident or myocardial infarction
- Unstable angina[31]
- Ventricular arrhythmia[31]

DIAGNOSTIC TESTS

- Electrocardiography
- Look for evidence of atrial fibrillation

MANAGEMENT

Goals of Treatment

- Modify risk factors
- Prevent recurrence of TIA or stroke

Appropriate Consultation

Consult a physician as soon as possible.

Nonpharmacologic Interventions

Client Education

- Blood pressure monitoring at each health care encounter[33]
- Explain disease course and expected outcome
- For clients receiving anticoagulant therapy, stress importance of avoiding injuries
- Lifestyle counselling on ways to reduce risk factors such as control of hypertension, smoking cessation, weight reduction, reduction of dietary fat, regular exercise
- Lifestyle and risk factor interventions include the following:[33]
  - Exercise: moderate exercise (an accumulation of 0–60 minutes) 4–7 days per week
  - Smoking: smoking cessation, nicotine replacement therapy and behavioural therapy
  - Diet: low fat (especially saturated fat) and sodium and high in fruit and vegetables
  - Weight: goal to maintain a BMI of 18.5–24.9 kg/m² and a waist circumference < 88 cm for women and < 102 cm for men
  - Alcohol consumption: moderate to no alcohol consumption
  - Stress management: individualized cognitive-behavioural interventions reflecting relaxation techniques

Pharmacologic Therapy

Antiplatelet Therapy

ASA 50–325 mg PO daily[34,35]

Patients with allergy or intolerance to ASA:

clopidogrel 75 mg PO daily

The optimal dosage of ASA is uncertain and there is no evidence that any specific dose is more effective than another, but fewer gastrointestinal side effects and bleeding occur with lower dosages (≤ 325 mg/day).[34]

Enteric-coated ASA does not protect against clinically relevant gastrointestinal bleeding, because injury severe enough to cause bleeding is a reflection of systemic rather than topical (local) effects of the drug.[36] Thus it is not essential to prescribe enteric-coated ASA.

It is common practice to switch patients from ASA to either clopidogrel or combination dipyridamole/ASA 200/25 mg if experiencing ischemic events while on low-dose ASA.[37]

In patients with cerebral ischemia (including TIA) the combination of ASA and clopidogrel is no more effective than either drug given alone, but is associated with an increased incidence of bleeding events.[38]

Counsel client about appropriate use of medications (dose, frequency, total amount, long-term use, side effects and precautions).

Monitoring and Follow-Up

Follow up regularly to monitor symptoms and track progress in reducing risk factors; frequency of follow-up will depend on severity of symptoms and number of risk factors.

Referral

- If neurologic deficits are identified at the time of presentation, manage as a stroke in progress (see "Cerebrovascular Accident (Stroke) in the section "Emergency Problems of the Central Nervous System") and medevac to hospital as soon as possible
- Elective referral to a physician for investigation of underlying pathology can take place if the client is completely asymptomatic at presentation and the event is historical only

EMERGENCY PROBLEMS OF THE CENTRAL NERVOUS SYSTEM

DIFFERENTIAL DIAGNOSIS OF ACUTE UNCONSCIOUSNESS

METABOLIC DISTURBANCES (MNEMONIC "AEIOU AND SOMETIMES S")

- A for anoxia
- E for ethanol intoxication
- I for insulin excess (hypoglycemia)
- O for overdoses (drugs)
- U for uremia
- S for seizure

HYPOPERFUSION OF THE BRAIN

- Stroke
- Hypotension
- Hypovolemia
- Arrhythmias
- Head trauma
- Meningoencephalitis[39]
- Acute hypoxic-ischemic neuronal injury suffered during cardiopulmonary arrest[39]
- Endstage neurodegenerative diseases (Alzheimer's, Parkinson's)[39]

For detailed information on coma, *see "Coma Not Yet Diagnosed" in the chapter, "General Emergencies and Major Trauma."*

MENINGITIS

- Infection of meninges around the brain and spinal cord
- Described as an inflammation of the cranial and spinal leptomeninges[40]. Despite the availability of antimicrobial therapy meningitis-related case fatality rates remain high[41]

CAUSES

- Bacterial meningitis most often develops following bacteremia but can also occur via extensions from surrounding structures (mastoiditis, sinusitis)[40]
- Viral or bacterial infection
- Most common bacterial causes in adults: Haemophilus influenzae, Neisseria meningitides, Streptococcus pneumoniae
- Via direct inoculation during neurosurgery or an open head trauma[40]

Risk Factors

- Predisposing factors for the development of community-acquired meningitis[41]
- Diabetes mellitus[41]
- Otitis media[41]
- Alcoholism
- Sinusitis
- Mastoiditis
- Head injury (open or closed)
- Pneumococcal pneumonia
- Recurrent meningitis
- Age > 60 years
- Immunocompromised

HISTORY

- Often preceded by infection of upper respiratory tract
- High fever
- Cranial nerve palsy[41]
- Headache, which becomes increasingly severe
- Headache made worse with movement, especially bending forward
- Vomiting, with or without nausea
- Photophobia
- Changes in level of consciousness that progress from irritability, through confusion, drowsiness and stupor to coma
- Seizures may develop

PHYSICAL FINDINGS

Perform a full head and neck examination to identify a possible source of infection.

- General feature include a triad of fever, neck stiffness and altered mental state[41]
- Temperature elevated
- Heart rate elevated or bradycardia with raised intracranial and intraocular pressure
- Blood pressure normal (low if client is in septic shock)
- Client in moderate-to-acute distress
- Client flushed
- Altered level of consciousness
- Focal neurologic signs
- Photophobia
- Petechiae may be present
- Cervical nodes may be enlarged
- Nuccal rigidity
- Brudzinski's sign
- Kernig's sign

DIFFERENTIAL DIAGNOSIS

- Bacteremia
- Sepsis
- Brain abscess
- Encephalitis
- Seizure

COMPLICATIONS

- Subdural effusion or empyema[40]
- Cerebritis or ventriculitis[40]
- Venous sinus thrombosis[40]
- CNS infarction leading to hemiparesis, quadriparesis or spinal cord infarction[40]
- Seizure
- Coma
- Shock
- Blindness
- Deafness
- Palsies of cranial nerves III, VI, VII, VIII
- Brain abscess
- Diabetes insipidus
- Syndrome of inappropriate antidiuretic hormone release
- Hydrocephalus
- Cognitive difficulties
- Hemiparesis, quadriparesis
- Death

DIAGNOSTIC TESTS

- Draw blood sample for complete blood count
- Draw three blood samples (15 minutes apart) for culture
- Examination of the cerebral spinal fluid (CSF) is essential to make a diagnosis when meningitis is suspected[40]

It is important to do multiple cultures before initiating antibiotic therapy in meningitis. This increases the chance of isolating the organism.

- Obtain urine for urinalysis (routine and microscopy, culture and sensitivity)
- Obtain a throat swab for culture and sensitivity

MANAGEMENT

Goals of Treatment

- Control infection
- Prevent complications: manage and minimize acute and chronic complications including permanent neurologic damage[40]

Appropriate Consultation

Consult a physician immediately. If unable to contact physician, within a reasonable time frame, follow the guidelines below for intravenous (IV) antibiotics. Do not delay start of antibiotics if this diagnosis is suspected.

Nonpharmacologic Interventions

- Bed rest
- Nothing by mouth
- Insert indwelling urinary catheter (optional if client is conscious)

Adjuvant Therapy

Start IV therapy with normal saline; adjust rate according to state of hydration. Do not overload with fluids, as this could cause brain edema.

Pharmacologic Therapy

Antipyretics to control fever:

acetaminophen (Tylenol), 325 mg, 1–2 tabs PO q4-6h prn (maximum 4 g/day)

For adults, antibiotics[40,42] (if unable to contact physician, within a reasonable time frame):

ceftriaxone (Rocephin) or cefotaxime 2 g IV stat

plus

vancomycin 1 g IV stat

For immunocompromised clients (cancer, HIV, age > 50 years or alcoholic):

ceftriaxone (Rocephin) or cefotaxime 2 g IV stat

plus

vancomycin 1 g IV stat

plus

ampicillin 2 g IV stat (unless penicillin allergic)

Monitoring and Follow-Up

- Monitor ABC (airway, breathing, circulation) and vital signs q30-60min or more frequently as required
- Monitor carefully for development of neurologic symptoms
- Monitor intake and hourly urine output

Referral

Medevac as soon as possible.

SEIZURE DISORDER (CHRONIC)

Sudden, temporary brain dysfunction due to abnormal electrical activity in the brain. A seizure is a manifestation of excessive, hypersynchronous activity of neurons in the brain. Individuals who have seizures due to a clear provocation (such as an acute head trauma, alcohol withdrawal or hyponatremia) are not considered to have epilepsy unless seizures continue beyond the initial acute illness or cerebral insult.[43]

TYPES

- Generalized tonic, clonic (grand mal)
- Focal
- Absence (petit mal)
- Complex partial
- Partial
- Myoclonic
- Infantile spasm
- Unclassified (characterized by eye movements or chewing)
- Status epilepticus

CAUSES

- Epilepsy
- Drugs (noncompliance, withdrawal syndromes, including alcohol, overdose, drug abuse)
- Hypoxia
- Brain tumour

- Cerebral infection (for example, meningitis)
- Metabolic disturbance (for example, hypoglycemia, uremia, liver failure, electrolyte disturbance)
- Head injury
- Stroke, TIA
- Malignancy
- Narcolepsy[43]
- Movement disorders[43]
- Convergent disorder (nonepileptic or psychogenic seizures)[43]

HISTORY

- One of the causes listed above usually present
- Family history of seizure disorder
- History of febrile seizures in childhood[43]
- Age at onset, frequency of seizure activity
- Sudden loss of consciousness or loss of motor control (or both)
- Description of seizure activity variable (depends on type); talk to an eye-witness, if possible
- Loss of bowel and bladder control during active seizure (for example, grand mal)
- History of aura before onset of seizure may be present
- Drugs (prescription, alcohol and illicit) noncompliance with seizure medication
- Infection
- History of stroke, head trauma, hypoxia, neurologic infection, exposure to toxins, developmental problems
- History of chronic sleep deprivation[43]

PHYSICAL FINDINGS

After Acute Seizure

- Temperature normal unless infection is present
- Heart rate elevated
- Blood pressure variable
- Postictal state if seizure has occurred recently (for example, drowsiness, confusion, behavioural changes)
- Evidence of trauma (for example, tongue laceration, dislocated shoulder)
- Results of neurologic examination and examination of other systems depend on specific cause of seizure
- If post initial seizure, assess for presence of skin abnormalities (suspicious birthmarks significant of a genetic syndrome)[43]

When Not in Active Seizure State

The results of neurologic examination are usually normal.

DIFFERENTIAL DIAGNOSIS

- Epilepsy
- Drug-related problem (noncompliance with prescribed regimen, withdrawal syndromes, overdose, multiple drug use)
- Hypoxia
- Brain tumour
- Cerebral infection
- Metabolic disturbance (for example, hypoglycemia, uremia, liver failure, electrolyte disturbance)
- Alcohol withdrawal
- Head injury
- Stroke
- Degenerative disease[44]

COMPLICATIONS

- Injuries during seizure or from a fall
- Hypoxia during seizure
- Status epilepticus
- Interference with normal lifestyle (for example, work, driving, social interactions)

DIAGNOSTIC TESTS

- CBC, glucose, electrolytes, calcium, renal and hepatic functions[45]
- Chest x-ray if metastatic disease is a consideration[45]
- Toxicology screen upon physician consultation[45]
- Lumbar puncture if presenting features suggest intracranial infection[45]
- Electroencephalogram

MANAGEMENT

Management depends on underlying cause and severity of symptoms.

Goals of Treatment

- Control seizures
- Prevent recurrence
- Improve quality of life
- Achieve good adherence to treatment with minimal side effects
- Eventually minimize or discontinue medications with continued control of seizures

Appropriate Consultation

- If client is not in active seizure on arrival: consult physician immediately for clients with previously undiagnosed seizure and for clients with history of breakthrough seizures
- If client is in active seizure on arrival, see the section "Status Epilepticus (Acute Grand Mal Seizure)"

Nonpharmacologic Interventions

- Assist client to identify and reduce or avoid trigger factors (for example, alcohol use)
- Recommend regular meals and balanced nutrition
- Encourage stress reduction
- Recommend pacing of activities to avoid overexertion
- Suggest relaxation therapy
- Avoid sleep deprivation[45]
- Avoid or minimize alcohol consumption[45]
- Avoid cocaine and amphetamines due to proconvulsant properties[45]

Pharmacologic Therapy

Anticonvulsants are tailored to the specific type of seizure. Monotherapy is ideal, but 10% to 15% of patients need two or more medications. Poor compliance is the major cause of seizure recurrence.

In certain circumstances, some communities may consider keeping an emergency supply of a particular anticonvulsant if a patient is identified as being at risk of seizures as a result of missing doses during ongoing therapy.

Monitoring and Follow-Up

- Follow up every 6 months if seizures are well controlled, more frequently if client is having breakthrough seizures
- Assess adherence to medication regimen
- Monitor serum drug levels every 6 months if stable, more frequently if necessary

Referral

- Refer electively for review by a physician at least annually if seizures are well controlled
- Refer urgently if client is having breakthrough seizures
- Consider neurologic follow-up if symptoms are not controlled on current medications

STATUS EPILEPTICUS (ACUTE GRAND MAL SEIZURE)

Epileptic seizure lasting more than 30 minutes or repeated seizures without regaining consciousness. If the seizure lasts more than 60 minutes and is untreated, status epilepticus is associated with significant morbidity and mortality. Impending status epilepticus should be treated aggressively in the first 30 minutes.[45]

CAUSES

- Unknown
- Inadequate absorption of anticonvulsants
- Noncompliance with medications
- Dosage of anticonvulsants reduced too rapidly
- Variable (see above under causes of seizures)

HISTORY

- Attack begins as seizure
- Episodes of tonic and clonic movements occur repeatedly without client regaining consciousness
- May go on for hours or days

PHYSICAL FINDINGS

- Temperature normal unless underlying infection is present
- Heart rate elevated, pulse may be irregular
- Respirations irregular (absent during seizure, present between seizures)
- Blood pressure elevated or low
- Oxygen saturation may be normal or decreased
- Unconscious
- Pale or cyanotic
- Loss of bowel and bladder control
- Repeated episodes of tonic and clonic movements
- Foaming at mouth
- Blood around or in mouth if client has bitten tongue
- Evidence of trauma

COMPLICATIONS

- Hypoxia
- Brain injury begins after approximately 30–45 minutes of uncontrolled seizure activity[45]
- Cardiac arrhythmia
- Brain damage
- Death

DIAGNOSTIC TESTS

- Monitor electrocardiogram (ECG) if client > 50 years of age
- Assess random blood glucose level
- Obtain urine for urinalysis (routine and microscopy, culture and sensitivity)
- Consult for toxicology screening

MANAGEMENT

Goals of Treatment

- Protect airway
- Stabilize cardiorespiratory function
- Stop seizures

Nonpharmacologic Interventions

Time	Management
0–5 min	History, physical exam
	Oral airway, oxygen
	Consider intubation
	Obtain blood sample (see diagnostics)
	Monitor ECG, pulse, blood pressure
5–10 min	Start 2 large-bore IV saline infusions
	50 mL dextrose IV
	Thiamine 50–100 mg IM
	Lorazepam 2 mg/min IV to a max dose of 0.1 mg/kg OR diazepam 5 mg/min to max dose of 0.25 mg/kg[46]

- Ensure airway is clear and patent
- Suction as necessary
- Insert oral pharyngeal airway
- Assist ventilation as needed with Ambu bag

Adjuvant Therapy

- Give oxygen 6–10 L/min; keep oxygen saturation > 97% to 98%
- Start IV therapy with normal saline; adjust rate according to state of hydration

Pharmacologic Therapy (h5)

Benzodiazepines:[46,47]

lorazepam (Ativan), 2 mg IV, administered over 1 min; repeat dose at 4–5 min intervals if seizure activity persists (maximum dose 8 mg)

or

diazepam (Valium), 5 mg IV; repeat dose at 4–5 min intervals if seizure activity persists (maximum dose 30 mg[48])

Lorazepam and diazepam are equally effective in terminating seizures. After IV administration diazepam has a more rapid onset of action (as soon as 10 to 20 seconds compared with up to 2 minutes with lorazepam). Lorazepam has the advantage of a longer duration of action than diazepam (as long as 4 to 6 hours compared with < 20 minutes with diazepam).[49] For this reason, lorazepam may be preferred in the nursing station setting.

Monitor for respiratory depression. Administer benzodiazepines with caution to clients who have received barbiturates, as the side effects of respiratory depression are additive.

Appropriate Consultation

Consult a physician as soon as possible after emergency treatment because long-acting anticonvulsants (for example, phenytoin [Dilantin]) may be needed to stop seizure and prevent recurrence.

Phenytoin (Dilantin), 20 mg/kg IV (at a maximum rate of 50 mg/min); mix with normal saline (500 mg/50 mL for adults).

Monitoring and Follow-Up

- Identify focal neurologic deficits
- Observe for return to normal level of consciousness
- Monitor vital signs
- Monitor for continued seizure activity

Referral

Medevac as soon as possible.

CEREBROVASCULAR ACCIDENT (STROKE)

Stroke is the clinical term for acute loss of perfusion to vascular territory of the brain resulting in ischemia and corresponding loss of neurologic function.[50] Stroke is the third leading cause of death and a leading cause of morbidity and long-term disability. Broadly classified as ischemic stroke (80% to 85%) and hemorrhagic stroke (15% to 20%).[51] Eighty percent of strokes are ischemic, and about 25% are caused by cerebral emboli.

CAUSES

Infarction from Thrombus or Emboli

- Progressing stroke: unstable, progressing neurologic deficits
- Completed stroke: stable, nonprogressing neurologic deficits

Risk Factors

- Age (> 65 years)[51]
- Atrial fibrillation
- History of TIA
- Smoking
- Hypertension
- Hyperlipidemia
- Diabetes mellitus
- Valvular heart disease (especially mitral stenosis and mitral prolapse)
- Coronary artery disease, previous myocardial infarction, congestive heart failure
- Recent myocardial infarction
- Ventricular aneurysm
- Carotid stenosis
- Peripheral vascular disease
- Injection drug use (for example, cocaine, amphetamines)
- Alcohol abuse[51]
- Physical inactivity (obesity)[51]

Intracranial Hemorrhage

- Intracerebral hemorrhage: hemorrhage in or around brain
- Subarachnoid hemorrhage: accounts for 5% to 10% of strokes

Risk Factors

- Hypertension
- Bleeding disorders
- Medications: anticoagulants (warfarin) or antiplatelets (acetylsalicylic acid [ASA], clopidogrel)
- Arteriovenous malformations
- Recent trauma

HISTORY

- Presence of one of the causes listed above
- Abrupt onset is suggestive of infarction, but must rule out brain abscess, tumour and subdural hematoma

Progressing Stroke

- Neurologic dysfunction evolving painlessly over several hours or days
- Headache absent
- Involves progressively more of the body
- Progression stepwise, with periods of stability; may be continuous

Consciousness may be reduced or altered.

Completed Stroke

- Abrupt onset
- Symptoms maximal in a few minutes
- One-sided neurologic deficits
- Consciousness may be reduced or altered

Intracranial Hemorrhage

- Suggested by coma, vomiting, severe headache, history of warfarin therapy, history of vascular anomaly (for example, aneurysm, angioma), systolic blood pressure > 220 mm Hg
- Subarachnoid hemorrhage suggested by new-onset, severe headache that may be followed by nausea and vomiting and loss of consciousness (transient or coma); however, client may have only headache and normal results on physical exam
- There is no acute phase intervention of proven value for intracerebral hemorrhage. Post-acute treatment of primary intracerebral hemorrhage is similar to ischemic stroke except that antithrombotic drugs are avoided[36]

PHYSICAL FINDINGS

- Heart rate may be elevated, pulse irregular
- Blood pressure may be normal, elevated or low
- Client in moderate-to-acute distress
- Client may be unconscious
- Mental confusion may be present
- One-sided weakness
- Aphasia may be present
- Bladder and bowel incontinence or retention may be present
- Sensation may be reduced on affected side
- Muscle weakness on affected side
- Reflexes on affected side may be reduced or hyperactive
- Clonus may be present
- Carotid bruits may be present
- Heart murmur may be present

DIFFERENTIAL DIAGNOSIS

- Seizure disorder
- Complicated migraine
- Drug toxicity (for example, lithium, phenytoin)
- Hypertensive encephalopathy
- Bell's palsy
- Hypoglycemia
- Subdural hematoma
- Head injury
- Tumour
- Psychiatric (conversion disorder)
- Meningitis[50]
- Cerebral venous thrombosis[50]
- Brain abscess[50]
- Epidural hematoma[50]
- Viral encephalitis[50]

COMPLICATIONS

- Inadequate ventilation
- Aspiration
- Seizures
- Disturbances in communication
- Acute urinary retention or urinary incontinence
- Bowel incontinence
- Deep vein thrombosis
- Skin breakdown (decubitus skin ulcers)
- Death

DIAGNOSTIC TESTS

- Random blood glucose
- ECG may be helpful
- Look for atrial fibrillation
- Draw blood for complete blood count (CBC), international normalized ratio (INR) and partial thromboplastin time (PTT), glucose, electrolytes, urea, creatinine, liver function tests, albumin, fasting glucose, cholesterol panel[35]
- Cardiac enzymes[50] (for example, troponins and creatine kinase)
- Toxicology screen[50]
- Chest x-ray[35]

MANAGEMENT

Goals of Treatment

- Protect airway
- Ensure adequate ventilation and oxygenation

Nonpharmacologic Interventions

- Insert oral pharyngeal airway (if unconscious)
- Suction secretions prn
- Ventilate with Ambu bag at 12 bpm prn
- Nothing by mouth if stroke affects level of consciousness or impairs swallowing mechanism
- Insert urinary catheter if level of consciousness impaired
- Induce hypothermia and maintain symptomatic treatment of pyrexia (elevated body temperature associated with poor outcome after stroke)[35]
- Maintain strict blood glucose control[36]

Adjuvant Therapy

- Give oxygen 6–10 L/min or more prn; keep oxygen saturation > 97% to 98%
- Start IV therapy with normal saline; adjust rate according to age, pre-existing medical problems, state of hydration and client's ability to take fluids
- Do not overload with volume, especially if cerebral hemorrhage is suspected

Appropriate Consultation

Consult a physician as soon as the client is stable.

Pharmacologic Therapy

- None specifically in the acute phase
- Do not attempt to reduce blood pressure, as elevated blood pressure is often compensatory, and a sudden drop in blood pressure could increase severity of stroke
- If hypoglycemic, administer 50% IV glucose immediately

Monitoring and Follow-Up

- Monitor vital signs, fluid intake and hourly urine output
- Monitor level of consciousness, changes in neurologic status
- Monitor for complications
- Monitor for decompensation of pre-existing medical problems

Referral

Medevac as soon as possible.

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