NEUROLOGICAL NURSING

**Module Outcomes**

1. Review the anatomy and physiology of the nervous system.
2. Diagnose, manage and rehabilitate patients with neurological conditions

**Content**

**Neurological conditions**: Head injury, spinal injury, cerebral vascular accident, epilepsy, meningitis, encephalitis, brain abscess, space occupying lesions, Parkinson’s disease, disseminated sclerosis, bell’s palsy, myasthenia gravis, Guillen Barre Syndrome.

**SYMPTOMS AND SIGNS OF NEUROLOGIC DISEASE**

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| 1. **Headaches**: *Headache* is an almost universal experience, and one of the most common symptoms in medical practice. |
| 1. **Difficulty walking and falls:** Change in gait is a common presenting complaint in neurology. 2. **Dizziness, vertigo, blackouts, collapse and fatigue**  * *Vertigo* - an illusion of movement * *Dizziness* covers many complaints, from a vague feeling of unsteadiness to severe, acute vertigo * *Blackout*, like dizziness, is a descriptive term implying either altered consciousness, visual disturbance or falling  1. ***Fatigue*** is another common symptom of neurological disorders 2. **Spasticity** |

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| **Cranial nerves** |

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| **No.** | **Name** | **Main clinical action** |
| I | Olfactory | Smell |
| II | Optic | Vision, fields, afferent light reflex |
| III | Oculomotor | Eyelid elevation, eye elevation, Adduction, depression in Abduction, efferent (pupil) |
| IV | Trochlear | Eye in torsion, depression in Adduction |
| V | Trigeminal | Facial and corneal sensation, muscles of mastication |
| VI | Abducens | Eye Abduction |
| VII | Facial | Facial movement, taste fibres |
| VIII | Vestibular | Balance |
|  | Cochlear | Hearing |
| IX | Glossopharyngeal | Sensation - soft palate, taste fibres |
| X | Vagus | Cough, palatal and vocal cord movements |
| XI | Accessory | Head turning, shoulder shrugging |
| XII | Hypoglossal | Tongue movement |

UNCONSCIOUSNESS AND COMA

* **Consciousness** means a state of wakefulness with awareness of self and surroundings.
* **Clouding of consciousness** - used more in psychiatry than neurology - means reduced wakefulness and/or self-awareness, sometimes with confusion.
* **Confusion** means altered consciousness - the subject is bewildered and misinterprets his/her surroundings.
* **Delirium** is a state of high arousal (typically *delirium tremens*). There is confusion and often visual hallucination.
* **Sleep** is *normal* mental and physical inactivity from which the subject can be roused.
* **Stupor** is an *abnormal*, sleepy state from which the subject can be aroused by vigorously or repeated stimuli - also used for various psychiatric states, e.g. catatonic and depressive stupor
* **Coma** means unrousable unresponsiveness. The Glasgow Coma Scale for head injury is shown below

**READING ASSIGNMENT 1;**

1. How to perform neurological examination.
2. Glasgow coma scale
3. Neurological investigations

NEUROLOGICAL DISORDERS

1. [**HEAD INJURY**](file:///E:\Dan%20notes-2\JUDYS%20NOTES%20SURGERY\Okell-SURGERY\index.htm)

**Definition**: Trauma to the head causing neurological manifestations.

**Etiology:**

Most common causes include motor vehicle accidents (e.g., collisions between vehicles, pedestrians struck by motor vehicles, bicycle accidents), falls, assaults, sports-related injuries, and penetrating trauma.

The male-to-female ratio for TBI is nearly 2:1, and TBI is much more common in persons younger than 35 years.

**CLASSIFICATION OF HEAD INJURIES**

Head injuries can be classified according to. **(SAPP)**

1. Severity of the injury.
2. Anatomical classification
3. Pathological classification-penetrating or blunt injury
4. Primary and secondary brain injury.
5. **SEVERITY OF INJURY**

Severity is assessed by the following methods notably using the Glasgow Coma Scale.

**Score below 8** is considered to represent **severe head injury**

**While 8 to 12** is assessed as **moderate head injury.**

**13 to 15 is mild head injury.**

1. **ANATOMICAL CLASSIFICATION;** Injury can involve one or more of the following.
2. **SCALP ;** This consists of five layers
3. **SKULL INJURIES;** Skull fractures are **simple or compound.**

**Simple if there is no communication between the fracture and the atmosphere**, while the fracture is **compound if there is such communication**. Skull fractures are classified as follows:

**Simple/Closed Fractures (no communication)**

1. **BRAIN INJURY;** Injury to the brain is either **localised or diffuse** and can be either **primary or secondary**.

**PRIMARY BRAIN NJURY**

* **Injury directly due to the insult and occurring at the time of the injury**
* **Brain concussion**-temporary physiological disruption of brain function.
* **Brain contusion**-Small petechie and hemorrhages. A **contusion** is the focal bruising or tearing of cerebral tissue accompanied by parenchymatous hemorrhage and/or local edema.
* **Brain laceration**-obvious deformity

**Localised injury**

Deformation of the brain at the point of impact. Associated with dural laceration and underlying brain contusion or laceration. Usually there is a localized surrounding oedema around the site of the impact.

**Diffuse injury**

This carries a greater risk of damage to the brain and the mechanisms involved in this injury are:

1. **Acceleration/deceleration injury.**

In acceleration injury the **head is put into motion from a stand still position,** as a result of which **the different layers of the brain travels at different velocities with shearing effects and rotation of the brain within the skull.**

The **shearing stresses** between different layers of the brain result in **petechial haemorrhages** as well as **diffuse axonal injury** involving the white matter and brain stem.

**In deceleration injury** the head is brought to a standstill from a moving position as in falls. The same mechanism applies.

The extent of the diffuse injury and the axonal damage determines the outcome.

The more severe the injury is, more brain damage occurs with more axonal injury. This would be associated with higher morbidity and mortality.

1. **Penetrating injury**

**High velocity or slow velocity injury** as a result of penetration with sharp objects.

The base of the skull is thin bone and could easily be penetrated especially in children.

This result in skull base fracture and damage to the brain overlying that area.

1. **Compression injury**

**The head is compressed between two solid objects as in motor vehicle** **accidents**.

The result is **multiple linear fractures particularly in the weak areas of the skull base resulting in multiple cranial nerve injuries.**

**SECONDARY BRAIN INJURY;**

This results as consequence to the primary brain injury and this includes:

1. **INTRACRANIAL HAEMATOMAS**. Include epidural haematoma, subdural hematoma,  **Subarachnoid hemorrhage (**The subarachnoid bleeding itself **does not usually cause neurologic damage, but hydrocephalus and cerebral);**

**Intraventricular haemorrhage**

1. **BRAIN OEDEMA**
2. **INFECTION**
3. **HYDROCEPHALUS;** Hydrocephalus can be caused by blockage of the ventricular system by **blood clot in cases of intra-ventricular haemorrhage**
4. **CSF LEAK;** This is a result of skull fractures crossing the nasal sinuses. In case of ethmoid sinuses -rhinorrhea and fracture internal ear and the middle ear with rupture of tympanic membrane cause otorrhea. Often these leaks are temporary and spontaneous closure within one leak occurs. If it persists then surgical intervention should be considered.

**Cerebral ischaemia**

This is common after severe head trauma and is caused by a combination of either hypoxia or impaired cerebral perfusion.

The brain is unable to autoregulate its blood supply with a decrease in blood pressure.

Glutamate excess and free radical accumulation lead to neuronal damage.

**DIAGNOSIS**

* History; **Detail description of the event leading to injury** to the head either from the relatives or from the patient**.** Aetiology-RTA, Assault, Fall, Missiles, Explosive. Detail of **exact mechanism** leading to head injury., Site of trauma, any wounds Any history of bleeding, Signs of shock-diziness,confusion,sweating ,Any history of loss of consciousness, History of headache, vomiting , Blurring of vision-increased ICP, History of otorrhea or rhinorrhea, Any lateralizing signs-loss of power in the limbs or loss of sensation. History of alcohol or other drug consumption raise the risk of intracranial bleeding and cloud the mental status assessment. The history of previous head injuries-Premorbid illness –DM, HTN, Epilepsy
* **Physical examination**

Suspect significant head trauma in any traumatized patient with cranial hematomas or lacerations or with altered sensorium with or without focal neurologic findings.

Obtain **complete vital signs**

The **GCS** is the mainstay for rapid neurologic assessment in acute head injury. Both initial and worst GCS post resuscitation scores have correlated significantly with 1-year outcomes following severe head injury

**Examine the scalp** carefully for evidence of trauma .Inspect the head, and palpate carefully for scalp lacerations, hematomas, ecchymoses, and deformity.

**Neurologic Examination**

* **INVESTIGATIONS IN HEAD INJURIES;** Plain skull x-ray;  **CT scan,**

**MRI;** This examination is useful **to show long term effects of head injury.** Depending on the availability it also could be used in investigating acute cases.

**Beta transferring;** This is a test for an enzyme which is only found in CSF. It is the optimum test for CSF leak.

**Other Important Baseline Tests;** PCV, Urea and electrolytes, Arterial blood gases, Blood alcohol level, Random blood glucose

**MANAGEMENT**

* 1st is A,B,C,D of resuscitation plus vital signs

**Airway and cervical spine;**

**Maintain cervical spine immobilization** in all unconscious or symptomatic (neck pain or tenderness) patients.

Inspect mouth remove debris by sweeping through

Chin lift/jaw thrust (tongue is attached to the jaw) and always airway in tongue falling back

**Intubations**; keep the neck immobilized in neutral position. Intubate all unconscious patients (GCS < 9) to secure airway.

**Tracheostomy**

**Cricothyrotomy**

**Breathing:** Oxygenation and ventilation;

**Circulation** **and arrest of bleeding**; Shock” is defined as inadequate organ perfusion and tissue oxygenation. In the trauma patient, it is most often due to haemorrhage and hypovolaemia. The diagnosis of shock is based on clinical findings: Hypotension, Hypothermia, Tachycardia , Tachypnoea, Cool extremities, Decreased capillary refill , Pallor, Decreased urine production

* **Resuscitation**

First priority is to **stop any obvious bleeding** by Sub fascial gauze pack placement and Manual compression on the proximal artery. Carefully applied compressive dressing of the entire injured limb can be done.

Fluids: infuse normal saline initially 2L to run as fast as possible through 2 large bore IV lines

* Insert urinary catheter and **monitor the input and output chart** at least 30-50 ml/hour or 0.5/kg/hour of urine flow
* Asses by **vital signs,** pallor, sweating, anxiety ,skin warmth clammy, input and output
* **Blood transfusion** must be considered if the haemoglobin level is less than 7 g/dl and the patient is still bleeding.
* **Maintain HEAD INJURY OBSERVATION CHART;** Monitoring the following in half , hourly or 2 hourly

**Continuous monitor of level of consciousness;** Best eye opening score, Best verbal response score, Best motor response

**Vital signs-**Pulse,Temperature,BP,Respiratory rate

**Pupillary reflexes;** Reaction to light, Size of the pupil,

**Motor examination of limbs;** Spontaneous movement of all the limbs, Paralysis

**Monitor danger signs;** Severe headache, Vomiting, Convulsions/seizure, Drainage of fluids ear or nose

* Give analgesia to relieve pain
* Administer tetanus toxiod
* other general care if patient is unconscious; Bladder care , Bowel care, Physiotherapy chest and limbs, Skin care, Analgesics
* Surgical interventions should be carried depending on severity.

**COMPLICATIONS OF HEAD INJURY**

1. CN palsies
2. Infections
3. Hydrocephalus
4. Convulsive disorder/epilepsy
5. Psychiatric disorders
6. Cerebrospinal fluid fistulae, either in the form of rhinorrhea or otorrhea
7. Posttraumatic movement disorders (Tremor, dystonia, parkinsonism )
8. Post-concussional symptoms e.g. Transient LOC, Bradycardia, Hypertension
9. Cumulative brain damage ('Punch-drunk syndrome')
10. Neuroendocrine & metabolic disturbances e.g. Diabetes insipidus.
11. **SPINAL INJURY**

**Definition**

Spinal cord injury (SCI) is an insult to the spinal cord resulting in a change, either temporary or permanent, in its normal motor, sensory, or autonomic function

Spinal injury may involve soft tissues (muscles and ligaments), bones (vertebrae and discs), and neural tissue (spinal cord and nerves). It is important for the primary assessment to establish the presence of an injury and initiate immediate treatment to avoid worsening the primary injury or secondary.

Patients with spinal cord injury usually have permanent and often devastating neurological deficits and disability.

Causes of spinal cord injuries include

* Road traffic accidents; Assault; Blunt injury; vascular disorders, tumors, infectious conditions, iatrogenic injuries, developmental disorders.
* Penetrating injuries: sharp objects like knives, spears, firearms
* Sports injuries; Falling from a height.

Bone injury can be stable or unstable and may be associated with neurological manifestations like paraplegia or quadriplegia depending on the level of injury. The injury could be a compression fracture with retropulsion of bone fragments into the spinal cord, causing cord compression or the complete transaction of the cord.

**Clinical features**

Spinal injury may present as part of the multiply injured patient and caution is needed not to overlook this condition. Neurogenic shock may be present.

History and physical examination: focus on symptoms related to the vertebral column (most commonly pain) and any motor or sensory deficits.

Evaluated for pain, swelling, bruising, or possible mal-alignment

Head that is in an usual position, numbness of or tingling that spreads down an arm, or leg, weakness, difficulty walking, paralysis (loss of movement) of arms or legs, no bladder or bowel control, shock (pale, clammy skin, bluish lips and fingernails, acting dazed or semiconscious , lack of alertness (unconscious), stiff neck, headache or neck pain.

Spinal shock is defined as the complete loss of all neurological function, including reflexes and recal tone, below a specific level that is associated with autonomic dysfunction.

**MANAGEMENT;**

**MANAGEMENT** **Starts at the site of injury follow:**

Principles of management

* The injury must be recognized.
* Take measures to prevent further damage ("secondary" injury) and to detect deteriorating neurologic function so that corrective measures can be taken.
* The patient must be maintained in optimal condition to allow the greatest possible nervous system repair and recovery.
* Evaluation and rehabilitation of the patient must be actively pursued to maximize the function of surviving but dysfunctional nervous tissue

**Pre-hospital**

* ABC of resuscitation
* Stabilize and immobilize the spine thus cervical column and transport patient on hard board.
* Transportation can by ambulances in short distances but if possible air lift is better to avoid movements that would interfere with splinting.

**Casualty**

* Keep the **cervical collar** and patient on the backboard
* **Resuscitation** following ABCDE should be done.

**Airway**

Clearing of oral secretions and/or debris is essential to maintain airway patency and to prevent aspiration.

Insertion of an oral airway may be all that is required to maintain an airway in some cases. However, intubation may be required in others.

**Breathing**

Includes supplementary oxygen for all patients and chest tube thoracostomy for those with pneumothorax and/or hemothorax

**Circulation**

Judicious fluid replacement with isotonic crystalloid solution to a maximum of 2 liters is the initial treatment of choice.

Adequate perfusion with the following parameters should be maintained

* Systolic blood pressure (BP) should be 90-100 mm Hg. Systolic.
* Heart rate should be 60-100 beats per minute in normal sinus rhythm. Hemodynamically significant bradycardia may be treated with atropine.
* Urine output should be more than 30 mL/h. Placement of a Foley catheter to monitor urine output is essential
* Prevent hypothermia.

NB Associated head injury occurs in about 25% of SCI patients. A careful neurologic assessment for associated head injury is compulsory

**Inpatient management involves**

* Prevention of secondary injury
* Oxygen therapy
* Splints
* Corticosteroid therapy
* Analgesia
* Bladder care-Condom catheter
* Bowel care
* Skin care
* Physiotherapy chest and limbs

**Splints**

Optimal immobilization is obtained by placing the patient supine (face up) on a firm, flat surface (e.g., rigid, long spine board) without a pillow and with lateral motion of the neck restricted by a rigid cervical collar (Philadelphia), lateral neck rolls connected with tape across the forehead, or traction.

Traction is the most effective method.

**Steroids**

* All SCI patients treated within 3 hours of injury with the following steroid protocol: **methylprednisolone 30 mg/kg bolus over 15 minutes** and an infusion of methyl prednisolone at **5.4 mg/kg/h for 23 hours beginning** 45 minutes after the bolus.
* Significant improvement in motor function and sensation in patients with complete or incomplete SCIs noted

**Analgesics**

* Start of opiod analgesia initially then NSAIDS

**Bladder care: Condom catheter**

* Monitor input output of fluids initially. Also if loss of bladder function

**Bowel care** -Manual evacuation by sweeping through the rectum which cause irritation or the use of enemas(warm soap enema)

**NG tube**

* Placement of a nasogastric tube for decompression.
* This may also be used for nutritional support.

**Anti emetics**

* Aspiration pneumonitis is a serious complication in the SCI patient with compromised respiratory function. Antiemetics should be used aggressively

**Skin care**

* Prevent pressure sores-Denervated skin is particularly prone to pressure necrosis.
* Remove belts and back pocket keys or wallets.
* Turn the patient every 1-2 hours.
* Pad all extensor surfaces.
* Use pneumatic mattresses, continuous motion or ripple mattress.
* Good nursing care to prevent wetness-ZNO

**Physiotherapy-both chest and limbs**

* Prevention of contractures and maintenance of range of motion are important in all patients with spinal cord injury and should begin immediately following the injury.
* Chest physiotherapy to prevent pneumostatic pneumonia

**Prognosis**

Patients with a complete spinal cord injury (SCI) have a less than 5% chance of recovery. If complete paralysis persists at 72 hours after injury, recovery is essentially zero. The prognosis is much better for the incomplete cord syndromes. If some sensory function is preserved, the chance that the patient will eventually be able to walk is greater than 50%

**COMPLICATIONS of spinal injury**

* Neurologic deterioration
* Pressure sores; - Careful and frequent turning of the patient is required to prevent pressure sores.
* Aspiration peumonitis;- Nasogastric decompression of the stomach is mandatory.
* Hypothermia; - Prevent hypothermia by using external rewarming techniques and/or warm humidified oxygen
* Pulmonary complications in SCI are common. Pulmonary complications of SCI include the following:

Atelectasis secondary to decreased vital capacity and decreased functional residual capacity

Increased work of breathing because of decreased compliance

Decreased coughing, which increases the risk of retained secretions, atelectasis, and pneumonia

Muscle fatigue

1. **CEREBROVASCULAR ACCIDENT**

A sudden or gradual interruption of blood supply to a vital centre in the brain is a cerebrovascular accident (CVA), also known as a stroke, brain attack, or central vascular accident.

A CVA can cause complete or partial paralysis or death.

A stroke is focal brain dysfunction due to ischemia. The ischemia may arise from atherosclerotic narrowing of a blood vessel, an embolus, hemorrhage, or other causes.

**Risk factors:**

* Hypertension, smoking, diabetes mellitus, sickle cell disease, cardiac dysrhythmias, substance abuse, and atherosclerosis. Smoking increases the risk of developing a CVA by two to six times.
* The risk also increases for smokers who use birth control pills over an extended period of time. Postmenopausal women are more likely to have CVAs than are younger women.

**Types of CVAs**

The two main types of stroke: are ischaemic and haemorrhagic.

**NB:**

* Read and make brief notes on the two types.

**Signs and symptoms;** depend on its cause;

* In some cases of thrombosis and ischemia, the person has had dizzy spells or sudden memory loss for some time before the actual CVA. No pain accompanies these symptoms. So the client ignores them.
* A cerebral hemorrage may give warning. It causes dizziness and ringing in the ears (tinnitus), as well as a violent headache, often with nausea and vomiting.
* Embolism usually occurs without warning, although the person has often has a history of cardiovascular disease.
* A sudden onset CVA is usually the most severe. This victim loses consciousness; the face becomes red; breathing is noisy and strained. The pulse is slow but full and bounding. Blood pressure is elevated, and the person may be in a deep coma.
* Stroke in evolution is a gradual worsening of symptoms of brain ischemia. The coma may deepen progressively until death occurs, or the person may gradually regain consciousness and eventually recover. The longer the time period that the person remains unresponsive, the less likely it is that the person will recover. The first few days after the onset are critical. The responsive person may show sign of memory loss or inconsistence behaviour, he or she may be easily fatigued, lose bowel and bladder control, or have poor balance.

**Results of CVA;** some of the major side effects include the following;

* **Hemiplegia**: paralysis of one side of the body hemiplegia may affect other functions, such as hearing, general sensation, and circulation: the degree of impairment depends on the part of the brain affected.
* **Aphasia and dysphasia**: Aphasia is a result of damage to the brain’s speech centre. Aphasia is a condition in which people are unable to speak. It can be frustrating and frightening because mental functioning usually is unimpaired. Dysphasia is an inability to say what one wishes to say. Many patients regain some power of speech, but others never do. Dysphagia (difficulty swallowing) may also occur.
* **Brain damage**: the extent of brain damage resulting from a CVA determines the client’s chances for recovery. If the damage is slight, recovery will be more rapid and complete.
* **Hemianopsia (hemianopia);** hemianopsia is defined as blindness in half of the visual field of one or both eyes.
* **Pain:** usually, after a CVA the client suffers very little pain. Problems such as infection, kidney or bladder calculi (stones) fecal impaction or emotional disturbances may aggravate any existing pain.
* **Autonomic disturbances;** e.g., dilated pupils, high or low blood pressure, or headache. Disturbance this kind may be treated with atropine like drugs.
* **Personality changes;** may be functional or organic. Functional type results from frustration at being unable to speak or walk or form the attitudes of other people. In either case the individual may feel useless or helpless. Organic changes may result from blockage of blood supply to part of the brain. In this instance the person may cry or became easily excited. These conditions cannot be consciously controlled.

**Medical and nursing management**

* 1st is A, B, C, of resuscitation plus vital signs.
* Thrombolytic therapy to resolve the clot (usually heparin, dicumarol or warfarin)
* Document every sign of improvement or lack of it.
* Control blood pressure.
* Position the unresponsive person on the unaffected side to keep the airway open and prevent aspiration; proper positioning prevents contractures and undue pressure on any part.
* Avoid placing the unresponsive person on the back; the tongue may fall back and occlude the airway; secretions may accumulate in the back of the throat.
* Provide adequate support for the affected limbs; extremity splints are now widely used. Positioning, support, and splints help prevent contractures.
* Elevate the head of the bed. Reduce the chance of increased intracranial pressure.
* Turn the person often atleast once every 2 hours, keeping the body in proper alignment.
* Provide suctioning as necessary. A mechanical airway or tracheostomy may be required. Oxygen will most likely be administered.
* Monitor vital signs carefully; an elevated temperature and lowered pulse and respiration rates are signs of increased intracranial pressure, which you must report.
* Ensure adequate hydration
* Offer nutritional support.
* Surgical intervention is required to remove blood from the brain if bleeding occurred.
* Keep the eyes lubricated with soothing eye drops as ordered.
* Talk to the person and explain everything you do as if he or she were responsive. Although may not respond he or she hears. Hearing is the last sense to be lost
* Provide PROM exercise
* Begin bladder and bowel retraining as the client is ready for its.
* Rehabilitation ; client has to learn how to work again, teach adaptive ADL , such as transferring form bed to chair and to toilet; dressing; and feeding; speech , physical, or occupational therapy also begins as soon as possible.

**Complications;** aspiration, dementia, loss of mobility.

1. **EPILEPSY**

Epilepsy can be defined as a neurological condition characterised by recurrent seizures. It is also referred to as a seizure disorder or a brain functional disorder that may be manifested as an episodic impairment or loss of consciousness.

A seizure is a sudden attack of altered cerebral function.   
An epileptic seizure is the result of altered cerebral function caused by abrupt, abnormal and excessive, uncontrolled   
repetitive electrical discharges of cerebral neurons. A convulsion refers to a series of forceful, involuntary contractions and   
relaxations of the voluntary muscles.

Most epileptic patients experience their first seizure in childhood, but the age of onset varies from one person to another. Infantile spasms commonly start before one year of age, commonly between three to four months. Thereafter, more generalised seizures occur.

**Etiology (causes of seizure)**

* Idiopathic
* Congenital abnormalities and perinatal injuries may result in seizures presenting in infancy or childhood.
* Metabolic disorders: Withdrawal from alcohol or drugs is a common cause of recurrent seizures, and other metabolic disorders such as uremia and hypoglycemia or hyperglycemia may also be responsible.
* Trauma:Posttraumatic epilepsy is more likely to develop if the dura mater was penetrated and generally becomes manifest within 2 years following the injury
* Tumors and other space-occupying lesions
* Vascular diseases
* Degenerative disorders**:** Alzheimer's disease and other degenerative disorders are a cause of seizures in later life.
* Infectious diseases: such as bacterial meningitis or herpes encephalitis, or in patients with more longstanding or chronic disorders such as neurosyphilis or cerebral cysticercosis. In patients with AIDS, they may result from central nervous system toxoplasmosis, cryptococcal meningitis, secondary viral encephalitis, or other infective complications. Also common in brain abscess
* stroke

**Classification of Seizures**

Seizures can be categorized in various ways, but the descriptive classification proposed by the International League Against Epilepsy is clinically the most useful. Seizures are divided into those that are generalized and those affecting only part of the brain (partial seizures)

**A. PARTIAL SEIZURES**

The initial clinical and electroencephalographic manifestations of partial seizures indicate that only a restricted part of one cerebral hemisphere has been activated. The ictal manifestations depend on the area of the brain involved. Partial seizures are subdivided into simple seizures, in which consciousness is preserved, and complex seizures, in which it is impaired. Partial seizures of either type sometimes become secondarily generalized, leading to a tonic, clonic, or tonic-clonic attack.

**1. Simple partial seizures**

Simple seizures may be manifested by focal motor symptoms (convulsive jerking) or somatosensory symptoms (eg, paresthesias or tingling) that spread (or “march”) to different parts of the limb or body depending on their cortical representation. In other instances, special sensory symptoms (e.g. light flashes or buzzing) indicate involvement of visual, auditory, olfactory, or gustatory regions of the brain, or there may be autonomic symptoms or signs (eg, abnormal epigastric sensations, sweating, flushing, pupillary dilation). The sole manifestations of some seizures are phenomena such as dysphasia, dysmnesic symptoms (eg, déjà vu, jamais vu), affective disturbances, illusions, or structured hallucinations, but such symptoms are usually accompanied by impairment of consciousness.

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| **Table 2. Seizure classification.** |
| |  |  |  | | --- | --- | --- | | **Seizure Type** | **Key Features** | **Other Associated Features** | | **Partial seizures** | Involvement of only restricted part of brain; may become secondarily generalized |  | | Simple partial | Consciousness preserved | May be manifested by focal motor, sensory, or autonomic symptoms | | Complex partial | Consciousness impaired | Above symptoms may precede, accompany, or follow | | **Generalized seizures** | Diffuse involvement of brain at onset |  | | Absence (petit mal) | Consciousness impaired briefly; patient often unaware of attacks | May be clonic, tonic, or atonic components (ie, loss of postural tone); autonomic components (eg, enuresis); or accompanying automatisms Almost always begin in childhood and frequently cease by age 20 | | Atypical absences | May be more gradual onset and termination than typical absence | More marked changes in tone may occur | | Myoclonic seizures | Single or multiple myoclonic jerks |  | | Tonic-clonic (grand mal) | Tonic phase: Sudden loss of consciousness, with rigidity and arrest of respiration, lasting < 1 minute Clonic phase: Jerking occurs, usually for < 2–3 minutes Flaccid coma: Variable duration | May be accompanied by tongue biting, incontinence, or aspiration; commonly followed by postictal confusion variable in duration | | **Status epilepticus** | Repeated seizures without recovery between them; a fixed and enduring epileptic condition lasting 30 minutes |  | |

**2. Complex partial seizures**

Impaired consciousness may be preceded, accompanied, or followed by the psychic symptoms mentioned above, and automatisms may occur. Such seizures may also begin with some of the other simple symptoms mentioned above.

**B. GENERALIZED SEIZURES**

There are several different varieties of generalized seizures, as outlined below. In some circumstances, seizures cannot be classified because of incomplete information or because they do not fit into any category.

**1. Absence (petit mal) seizures**

These are characterized by impairment of consciousness, sometimes with mild clonic, tonic, or atonic components (i.e., reduction or loss of postural tone), autonomic components (e.g., enuresis), or accompanying automatisms. Onset and termination of attacks are abrupt. If attacks occur during conversation, the patient may miss a few words or may break off in mid sentence for a few seconds. The impairment of external awareness is so brief that the patient is unaware of it. Absence seizures almost always begin in childhood and frequently cease by the age of 20 years, although occasionally they are then replaced by other forms of generalized seizure. Electroencephalographically, such attacks are associated with bursts of bilaterally synchronous and symmetric 3-Hz spike-and-wave activity. A normal background in the electroencephalogram and normal or above-normal intelligence imply a good prognosis for the ultimate cessation of these seizures.

**2. Atypical absences**

There may be more marked changes in tone, or attacks may have a more gradual onset and termination than in typical absences.

**3. Myoclonic seizures**

Myoclonic seizures consist of single or multiple myoclonic jerks.

**4. Tonic-clonic (grand mal) seizures**

In these seizures, which are characterized by sudden loss of consciousness, the patient becomes rigid and falls to the ground, and respiration is arrested. This tonic phase, which usually lasts for less than a minute, is followed by a clonic phase in which there is jerking of the body musculature that may last for 2 or 3 minutes and is then followed by a stage of flaccid coma. During the seizure, the tongue or lips may be bitten, urinary or

fecal incontinence may occur, and the patient may be injured. Immediately after the seizure, the patient may either recover consciousness, drift into sleep, have a further convulsion without recovery of consciousness between the attacks (status epilepticus), or after recovering consciousness have a further convulsion (serial seizures). In other cases, patients will behave in an abnormal fashion in the immediate postictal period, without subsequent awareness or memory of events (postepileptic automatism). Headache, disorientation, confusion, drowsiness, nausea, soreness of the muscles, or some combination of these symptoms commonly occurs postictally.

**5. Tonic, clonic, or atonic seizures**

Loss of consciousness may occur with either the tonic or clonic accompaniments described above, especially in children. Atonic seizures (epileptic drop attacks) have also been described.

This is followed by the tonic stage, which usually lasts about 10 to 20 seconds. All muscles become rigid, eyelids open, eyes look up and respiration stops temporarily resulting in cyanosis. The tongue is bitten causing bleeding, which can be seen from the mouth.

Next is the clonic stage, which usually lasts about 30 seconds. It begins with muscle relaxation, which completely interrupts tonic muscle contraction. There are brief violent muscle spasms of the whole body , frothing of the mouth and incontinence of urine and sometimes faeces as well.

The final stage is the comatose stage, where the patient goes into deep coma for minutes or hours. On recovery, they look confused and unaware of what has happened.

**OTHER CLASSIFICATIONS;**

This condition can conveniently be divided into three types.

1. **Petit Mal (small sickness)**  
   This epilepsy, commonly seen in children, is characterised by sudden momentary loss of consciousness with only minor colonic jerks. The facial expression during an attack is blank.
2. **Jacksonian Epilepsy**  
   This is a moderate type of epilepsy named after a London neurologist called Dr. John Hunghlings Jackson (1835-1911). It is characterised by unilateral chronic (sporadic muscular rigidity and relaxation) movements that start in one group of muscles and then systematically spread to adjacent groups of muscles reflecting the match of epileptic activity through the motor cortex. Seizures are due to a discharging focus in the contra lateral motor cortex.
3. **Grand Mal (Major Epilepsy)**This type results in loss of consciousness. It always occurs with usually well defined stages. This begins with the aura (warning) stage, which is characterised by certain unusual feelings such as peculiar sensation, funny smell, feeling nauseated, abdominal discomfort (gastric secretions) and flashing light. You should note that only some of these symptoms may be experienced by the patient at any given time.

**Investigations**; the following investigations should be carried out to assist in identifying the causes of epilepsy.

* Begin with a personal history, which must be specific. It should include when the condition started and the frequency of seizures in terms of how many seizures per day or per week. Also find out whether there was any warning such as an abnormal feeling or sensation before the onset of seizures, if there was loss of consciousness, speech interruption and so on, and confirm the duration of seizures.
* Then undertake a physical examination. This too may help to determine the cause of the fit. Any signs of physical injuries following an epileptic attack may also be detected. An electroencephalogram (EEG) is extremely useful in demonstrating the type of fits according to areas of the brain which may function abnormally. However, some epileptic patients may also have normal EEG. You should take a blood test. Venous blood should be sent to the laboratory for urea level and microscopy. You should also undertake a blood pressure assessment and estimate arterial blood pH. Blood glucose level should be checked to exclude hypoglycaemia

**CLINICAL FINDINGS**

**SYMPTOMS AND SIGNS**

* Nonspecific changes such as headache, mood alterations, lethargy, and myoclonic jerking alert some patients to an impending seizure hours before it occurs. These prodromal symptoms are distinct from the aura which may precede a generalized seizure by a few seconds or minutes and which is itself a part of the attack, arising locally from a restricted region of the brain.
* In most patients, seizures occur unpredictably at any time and without any relationship to posture or ongoing activities. Occasionally, however, they occur at a particular time (e. g, during sleep) or in relation to external precipitants such as lack of sleep, missed meals, emotional stress, menstruation, alcohol ingestion (or alcohol withdrawal; see below), or use of certain drugs. Fever and nonspecific infections may also precipitate seizures in known epileptics. In a few patients, seizures are provoked by specific stimuli such as flashing lights or a flickering television set (photosensitive epilepsy), music, or reading.

B. IMAGING

* MRI is performed in patients with clinical evidence of a progressive disorder and in those presenting with new onset of seizures after the age of 20 years, because of the possibility of an underlying neoplasm.
* A chest radiograph

C. LABORATORY AND OTHER STUDIES

* Initial investigations should always include a full blood count, blood glucose determination, liver and renal function tests, and serologic tests for syphilis. The hematologic and biochemical screening tests are important both in excluding various causes of seizures and in providing a baseline for subsequent monitoring of long-term effects of treatment.
* Electroencephalography may support the clinical diagnosis of epilepsy (by demonstrating paroxysmal abnormalities containing spikes or sharp waves), may provide a guide to prognosis, and may help classify the seizure disorder. Classification of the disorder is important for determining the most appropriate anticonvulsant drug with which to start treatment

**TREATMENT**

**A. GENERAL MEASURES**

For patients with recurrent seizures, anticonvulsant drug treatment is prescribed with the goal of preventing further attacks and is usually continued until there have been no seizures for at least 3 years. Epileptic patients should be advised to avoid situations that could be dangerous or life-threatening if further seizures should occur.

**1. Choice of medication**

The drug with which treatment is best initiated depends on the type of seizures to be treated . The dose of the selected drug is gradually increased until seizures are controlled or side effects prevent further increases. If seizures continue despite treatment at the maximal tolerated dose, a second drug is added and the dose increased depending on tolerance; the first drug is then gradually withdrawn.

**Generalized tonic-clonic (grand mal) or partial (focal) seizures:**

Drugs used include; phenytoin, carbamazepine, valproic acid, Phenobarbital, primodone, felbamate, gabapentin, lamotrigine, topiramate

**Absence (petit mal) seizures:** Ethosuximide, valproic acid, clonazepam

**Myoclonic seizures:** valproic acid, clonazepam

**2. Monitoring**

Monitoring serum drug levels has led to major advances in the management of seizure disorders. The most common cause of a lower concentration of drug than expected for the prescribed dose is poor patient compliance. Compliance can be improved by limiting to a minimum the number of daily doses.

All anticonvulsant drugs have side effects

**Discontinuance of medication**

Only when patients have been seizure-free for several (at least 3) years should withdrawal of medication be considered. Unfortunately, there is no way of predicting which patients can be managed successfully without treatment, although seizure recurrence is more likely in patients who initially failed to respond to therapy, those with seizures having focal features or of multiple types, and those with continuing electroencephalographic abnormalities. Dose reduction should be gradual over a period of weeks or months, and drugs should be withdrawn one at a time. If seizures recur, treatment is reinstituted with the same drugs used previously. Seizures are no more difficult to control after a recurrence than before.

**Surgical treatment**

Patients with surgically remediable epilepsy or seizures refractory to pharmacologic management may be candidates for operative treatment, which is best undertaken in specialized centers.

**Vagal nerve stimulation**

Treatment by chronic vagal nerve stimulation for adults and adolescents with medically refractory partial-onset seizures is approved and provides an alternative approach for patients who are not optimal candidates for surgical treatment. The

**Nursing Management**

* Ensure the patient's safety by removing all harmful tools or equipment around.
* **An epileptic seizure is a medical emergency! Remember the "ABC" rule. Ensure a clear Airway, to enable the patient to Breathe and loosen the clothing to facilitate Circulation**
* The head should be protected from injuries by placing a blanket or a folded sheet underneath it.   
  If possible, ensure an airway in the mouth.
* Avoid restraining the patient, because that may cause further injuries, especially of the limbs. Ensure fresh air by removing onlookers
* During an attack you should observe the patient to identify the parts of the body that go into violent contraction or twitching to see how long each seizure takes and other abnormal activities during seizure
* **Care after Seizure**

Change into lateral position to facilitate drainage of respiratory secretions from the mouth. Observe skin, eye and mouth colour. Take and record vital signs to monitor tachycardia and hypertension. Observe the degree of consciousness and mental status, the length of sleep, response to sensory stimulation. Any sensory impairment such as vision and hearing should be reported.

* Medical treatment involves the administration of anti convulsant drugs to control the seizure so that it is not prolonged thus causing physical exhaustion. Common drugs prescribed may be either/or phenobarbitone (luminal) with a dose of 3-6mg/kg body weight or phenytoin sodium (epanutin), with a dose of 10-20mg/kg body weight.

**Status Epilepticus**

This is a very serious neurological condition whereby the patient has repeated seizures or convulsions one after another without recovering consciousness between attacks. If untreated, the patient may die from exhaustion

**Management of status epilepticus;**

* Ascertain the patient has tonic chronic status epilepticus and prolonged or repetitive seizures have occurred.
* Maintain ABC’s
* Obtain intravenous access and initiate drug therapy. E.g. give immediate diazepam 10-20 mg i.v. at 5mg/min and repeat once. If immediate i.v. access is impossible, give diazepam or paraldehyde. Or;
* Give lorazepam i.v. 4 mg at 2 mg/min. Respiratory depression, hypotension and cardiac dysrhythmias may occur.
* Administer oxygen, monitor ECG, BP, routine bloods (include alcohol, calcium, magnesium, drug screen, anticonvulsant levels). Exclude hypoglycaemia: treat if present.
* Give thiamine i.v. (250 mg) if nutrition poor or alcohol abuse suspected
* Monitor vital signs.
* Do blood investigations e.g. serum electrolyte, blood urea and nitrogen, glucose.
* Isotonic saline infusion should be initiated.
* 50 ML of 50 % glucose should be given immediately if hypoglycemis is suspected.
* Monitor blood glucose level.
* Imaging with CT scan is recommended after stabilization of airway and oxygen.
* Lumbar puncture is required to rule out infectious etiologies.
* Obtain history and Perform neurological exam.
* Pharmacological treatment include use of:

Benzodiazepines; e.g. diazepam, lorazepam, midazolam

Phenytoin (lack sedating effect)

Phenobartal

1. **MENINGITIS**

Meningitis is the inflammation of the meninges. Meningitis means inflammation but usually implies serious infection of the meninges .

Microorganisms reach the meninges either by direct extension from the ears, nasopharynx, cranial injury or congenital meningeal defect, or by bloodstream spread. Immunocompromised patients (e.g. HIV, cytotoxic drugs) are at risk of infection by unusual organisms. Non-infectious causes of inflammation include malignant cells/tumors, drugs and blood following subarachnoid haemorrhage

|  |
| --- |
| **Infective causes of meningitis** |

|  |
| --- |
| **Bacteria** ;  *Neisseria meningitidis\** *Streptococcus pneumoniae\**  *Staphylococcus aureus* , Streptococcus Group B *Listeria monocytogenes* Gram-negative bacilli  *Mycobacterium tuberculosis* *Treponema pallidum*  **Viruses** ;  Enteroviruses: ECHO ; Coxsackie ; Mumps ; Herpes simplex ; HIV ;Epstein-Barr virus  **Fungi** ;  *Cryptococcus neoformans* ,*Candida*  (*Coccidioides immitis, Histoplasma capsulatum, Blastomyces dermatitidis* |

**Clinical features**

* Neck stiffness, positive kerning’s sign, altered level of consciousness, headache, fever, vomiting, convulsions, photophobia are common features
* Fever and chills, mental status changes, nausea and vomiting, sensitivity to light (photophobia), severe headache, stiff neck (meningismus)
* Other common symptoms that may occur with the disease: agitation, bulging fontanelles in babies, decreased alertness, poor feeding or irritability in children, rapid breathing, usual posture, with head and neck arched backwards

**Signs and tests**

* Fast heart rate, fever, mental changes, stiff neck
* A lumbar puncture (spinal tap) done to remove a sample of spinal fluid CSF for testing.
* Blood culture
* Chest X-ray;
* CT scan of the head: to diagnose, brain infections
* LP must be done if there is 1 of the following: coma, inability to drink/feed, stiff neck, AVPU= P OR U; bulging fontanelle, fits if age< 6 months, or > 6 years, evidence of partial seizures.

**Treatment:**

Antibiotic therapy

Classify as definite meningitis: give chloramphenicol PLUS penicillin, minimum of 10 days of treatment IV/IM. Steroids are not indicated.

Classify as probable meningitis: give chloramphenicol PLUS penicillin, 7 days IV/IM treatment then oral antibiotics to complete 14 days.

Possible meningitis; IV/IM chloramphenical and penicillin for minimum 3 days or until symptoms resolve.

Viral meningitis is usually not serious, and symptoms should disappear within 2 weeks with no lasting complications.

Antiviral medicine may be given to those with herpes meningitis

Give intravenous fluids

1. **ENCEPHALITIS**

Encephalitis is inflammation of the brain parencyma. The inflammation is caused by an infection invading the brain (infectious); or through the immune system attacking the brain in error (post-infectious/autoimmune encephalitis )

Encephalitis is different from meningitis. Meningitis means inflammation of the protective layers that cove tha brain. Sometimes patients have both meningitis and encephalitis and this is called menigoenncephalitis.

**Causes**

* Viruses; paramyxovirus (measles, mumps)
* Trypanosomiasis

**Clinical features;**

* Frequently begins with flue-like illness or headache. Typically more serious symptoms follow hours to day later. The most serious finding is an alteration in level of consciousness. This can range from mild confusion or drowsiness, to loss of consciousness and coma.
* Other symptoms include: high temperatures, seizures (fits), aversion to bright lights, inability to speak or control movement, sensory changes, neck stiffness, or uncharacteristic behavior.
* Some people may also experience hallucinations and vivid nightmares during the acute period of the encephalitis.

**Diagnosis;**

* Brain scans computerized tomography CT or magnetic resonance imaging.
* Electroencephalogram EEG, which records brain waves, can detect abnormal patterns of activity.
* CSF analysis, and microscopy

**Management**

* Patients in coma should be sedated and ventilated on an intensive care unit.
* Control seizures with diazepam. Treat status epilepticus with phenytoin (using cardiac monitor) or Phenobarbital.
* Acyclovir is effective is effective for herpes simplex virus type I and related viruses
* Offer broad spectrum antibiotic to treat bacterial infections.
* Nutritional support; use nasogastric feeding, in very ill patients.
* Prevent bedsores
* Contractures: encourage the family to keep joints supple; use splints

1. **PARKINSON DISEASE**

Parkinson’s disease is a progressive neurodegenerative disorder that occurs when the neurons within the brain responsible for producing the chemical dopamine became impaired or dies. Dopamine is essential for smooth control and coordination of movement of voluntary muscle groups.

Parkinson's disease is a gradual, progressive, degenerative disease of the basal ganglia (extrapyramidal) motor system

It is involuntary tremulous motion, with lessened muscular power, in part not in action and even when supported: with a propensity to bend the trunk forward, and to pass from a walking to a running pace, the senses and intellect being injured.

Parkinson disease is characterized by loss of dopaminergic cells in the substantia nigra leading to abnormal activity in the basal ganglia as a whole. This leads to

A decrease in the activity of the direct GABAergic pathway-

An increase in the activity of the indirect GABAergic pathway-

**Clinical features**

**T –tremor**

**R- rigidity**

**A-akinesia/Bradykinesia**

**P-postural instability.**

ESSENTIALS OF DIAGNOSIS

* Any combination of tremor, rigidity, bradykinesia, progressive postural instability.
* Seborrhea of skin quite common.
* Mild intellectual deterioration may occur

**TREATMENT:**

**Treatment strategies**

1. The conservative approach

* Avoid all drugs until symptoms are troublesome
* When symptoms become troublesome start amantadine and an anticholinergic.
* When symptoms became disabling introduce L-Dopa or agonist at minimal doses.

1. The neuroprotective approach

* All newly diagnosed cases should be started on selegeline
* When the symptoms become disabling add dopaminergic drugs.

1. The symptomatic approach.

* At diagnosis treatment immediately started with dopaminergic drugs.
* Treatment continually modified in order to maintain maximum function for the maximum amount of time.

**Drug treatment includes:**

1. **Amantadine (mechanist unclear ? dopamine release**

Patients with mild symptoms but no disability may be helped by amantadine. This drug improves all of the clinical features of parkinsonism, but its mode of action is unclear

1. **L-Dopa (**bypass rate limiting stem of dopamine synthesis)
2. **Dopamine agonists** (direct effect on striatum)
3. **COMT inhibitors**( less peripheral inactivation of L-dopa)
4. **Selegeline (MAOb) inhibitor** ? neuroprotective
5. **Muscarinic antagonists** (act on striatal interneurons)
6. **Anticholinergic drugs; M**ore helpful in alleviating tremor and rigidity than bradykinesia

**Other general measures include;**

Physical therapy or speech therapy helps many patients. The quality of life can often be improved by the provision of simple aids to daily living, e.g., rails or banisters placed strategically about the home, special table cutlery with large handles, nonslip rubber table mats, and devices to amplify the voice.

**Surgical measures**

Thalamotomy or pallidotomy may be helpful for patients who become unresponsive to medical treatment or have intolerable side effects from antiparkinsonian agents, especially if they have no evidence of diffuse vascular disease or significant cognitive decline..

**Brain stimulation;** High-frequency thalamic stimulation is effective in suppressing the rest tremor of Parkinson's disease

1. **MYASTHENIA GRAVIS**

Myasthenia gravis, an autoimmune disorder affecting the myoneural junction, is characterized by varying degrees of weakness of the voluntary muscles

Myasthenia gravis (MG) is an uncommon **autoimmune disease** that is caused **by acetylcholine receptor antibodies (AChRA**) at the neuromuscular junction. The antibody binds to the post synaptic acetylcholine receptor sites which makes them unavailable for the transmission of nerve impulses.

It is a condition that causes muscle weakness and extreme fatigueness. The disease is characterised by weakening of the voluntary muscles. The muscles include; the eye muscles that control eye lid movement; facial muscles that help chew, talk, swallow and provide facial expressions; muscles that help breathing; muscles that help in moving the neck and the limbs etc.

**ESSENTIALS OF DIAGNOSIS**

* Fluctuating weakness of commonly used voluntary muscles, producing symptoms such as **diplopia**, **ptosis**, and difficulty in swallowing.
* Activity increases weakness of affected muscles.
* Short-acting anticholinesterases transiently improve the weakness.

**CLINICAL FINDINGS**

**SYMPTOMS AND SIGNS**

The most important diagnostic feature of MG is fatigable muscle weakness

Patients present with **ptosis** (drooping eye lids), **diplopia** (object seen as two objects), **difficulty in chewing or swallowing**, respiratory difficulties, limb weakness, or some combination of these problems.

The disorder follows a slowly progressive course and may have a fatal outcome owing to respiratory complications such as aspiration pneumonia.

**Table 13.4** Symptoms of weakness in myasthenia gravis

|  |  |
| --- | --- |
| **Eyes** | double vision & drooping eye lids |
| **face, mouth** | weakness smiling, chewing & swallowing |
| **speech** | voice weak & easily tires |
| **Limbs** | weakness combing hair  weak hand grip  difficulty arising from chairs or climbing stairs |
| **central muscles** | head drop  weakness sitting up |
| **Respiratory** | shortness of breath |

**Clinical examination**

* Confirms the weakness and fatigability of affected muscles. In most cases, the extraocular muscles are involved, and this leads to ocular palsies and ptosis, which are commonly asymmetric.
* Pupillary responses are normal. The bulbar and limb muscles are often weak, but the pattern of involvement is variable. Sustained activity of affected muscles increases the weakness, which improves after a brief rest. Sensation is normal, and there are usually no reflex changes.

**Diagnosis**

* Can generally be confirmed by the response to a short-acting anticholinesterase. Edrophonium, alternatively, 1.5 mg of neostigmine can be given intramuscularly, and the response then lasts for about 2 hours; atropine sulfate (0.6 mg) should be available to reverse muscarinic side effects.
* **IMAGING**

Lateral and anteroposterior x-rays of the chest and CT scans should be obtained to demonstrate a coexisting thymoma.

* **LABORATORY AND OTHER STUDIES**

Blood tests to check antibodies against acetylcholine response.

Electrophysiologic demonstration of a decrementing muscle response to repetitive 2or 3-Hz stimulation of motor nerves indicates a disturbance of neuromuscular transmission.

Assay of serum for elevated levels of circulating acetylcholine receptor antibodies is useful because it has a sensitivity of 80–90% for the diagnosis of myasthenia gravis.

**Complications**

Involvement of bulbar and respiratory muscles in MG is a neurological emergency. The major complication is respiratory failure.

**TREATMENT**

* Involves the use of cholinesterase inhibitors and immunosuppression . Treatment is very effective at reducing or abolishing weakness but requires scrupulous attention to detail.
* **Cholinesterase inhibitors:**

Neostigmine, pyridostigmine, or both can be used, the dose being determined on an individual basis. The usual dose of neostigmine is 7.5–30 mg (average, 15 mg) taken four times daily; of pyridostigmine, 30–180 mg (average, 60 mg) four times daily. Overmedication may temporarily increase weakness, which is then unaffected or enhanced by intravenous edrophonium

While anticholinesterases may suppress the symptoms they do not alter the disease and hence the need for immunosuppression.

* **Thymectomy** ( surgical removal of thymus gland) usually leads to symptomatic benefit or remission and should be considered in all patients younger than age 60, unless weakness is restricted to the extraocular muscles. If the disease is of recent onset and only slowly progressive, operation is sometimes delayed for a year or so, in the hope that spontaneous remission will occur.
* **Immunosuppression;** these drugs help control the immune system from attacking the muscle cells.

Alternate day steroids are the treatment of choice. These are indicated in most cases. The patient should be admitted to hospital and started on prednisolone 10 mg/po/alternate days increasing slowly by 10 mg increments per dose (every second day) until 1.5 mg/kg or 100 mg is reached whichever is the lower dose. This should be maintained until the patient is stable in remission. Improvement begins after 2-4 weeks and maximises at 6-12 months. Then prednisolone is reduced by 10 mg every 4 weeks until the patient is on 40 mg alternate days, and by 5 mg every 4 weeks until on 20 mg and

* In patients with major disability in whom conventional treatment is either unhelpful or contraindicated, **plasmapheresis** or **intravenous immunoglobulin therapy may be beneficial**. It may also be useful for stabilizing patients before thymectomy and for managing acute crisis..
* Cytotoxic medications have also been used, although the precise mechanism of action in myasthenia is not fully understood. Medications such as azathioprine (Imuran), cyclophosphamide (Cytoxan), and cyclosporine reduce the circulating antiacetylcholine receptor antibody titers. Side effects are significant; therefore, these agents are reserved for patients who do not respond to other forms of therapy.
* A number of medications are contraindicated for patients with myasthenia gravis because they worsen myasthenic symptoms. Risks and benefits should be weighed by the physician and the patient before taking any new medications, including antibiotics, cardiovascular medications, antiseizure and psychotropic medications, morphine, quinine and related agents, beta-blockers, and nonprescription medications. Procaine (Novocain) should be avoided, and the patient’s dentist is so advised.
* **PLASMAPHERESIS**

Plasma exchange (plasmapheresis) is a technique used to treat exacerbations. The patient’s plasma and plasma components are removed through a centrally placed large-bore double-lumen catheter. The blood cells and antibody-containing plasma are separated; then the cells and a plasma substitute are reinfused. Plasma exchange produces a temporary reduction in the titer of circulating antibodies.

**Nursing Management**

* Because myasthenia gravis is a chronic disease and most patients are seen on an outpatient basis, much of the nursing care focuses on patient and family teaching.
* Educational topics for outpatient self-care include medication management, energy conservation, strategies to help with ocular manifestations, and prevention and management of complications.
* To minimize the risk of aspiration, mealtimes should coincide with the peak effects of anticholinesterase medication In addition; rest before meals is encouraged to reduce muscle fatigue.
* The patient is reminded of the importance of maintaining health promotion practices and of following health care screening recommendations.

**Prognosis:**

The prognosis is good. With proper treatment, many patients show improved muscle strength. Most patients undergo a temporary phase remission phase. A few may even have a permanent remission, especially after thymectomy. However, one should keep in mind there is no cure for myasthenia gravid.

The symptoms vary throughout life, but following proper treatment will help one live a near normal life.

**MANAGING MYASTHENIC AND CHOLINERGIC CRISES**

* Respiratory distress and varying degrees of dysphagia (difficulty swallowing), dysarthria (difficulty speaking), eyelid ptosis, diplopia, and prominent muscle weakness are symptoms of myasthenic and cholinergic crisis.
* The patient is placed in an intensive care unit for constant monitoring because of associated intense and sudden fluctuations in clinical condition.

1. **BELL ’S PALSY (FACIAL PARALYSIS)**

Bell’s palsy causes sudden weakness in facial muscles. This makes half of ones face appear to droop. The smile is one-sides, and eye on that side resists closing.

Bell’s palsy (facial Paralysis) is due to peripheral involvement of the seventh cranial nerve on one side, which results in weakness or paralysis of the facial muscles.

**ESSENTIALS OF DIAGNOSIS**

* Sudden onset of lower motor neuron facial palsy.
* Hyperacusis or impaired taste may occur.
* No other neurologic abnormalities

This is a paralysis of one side of the muscles surround the eye. This is due to damage of the nerve supplying the muscles, the seventh nerve.

* It normally develops over 2 days, and hearing is largely unaffected.
* It presents acutely with loss of blinking and weak facial muscles on one side
* There may be associated hearing changes and face numbness.

**Causes**

* 50% of cases are idiopathic but possible causes may include:
* Viral disease; Herpes simplex zoster, herpes simplex
* Vascular ischemia
* Autoimmune disease

**Clinical features**

* Patient experience speech difficulties and unable to eat on the affected side owing to weakness.
* Acute onset of unilateral upper and lower facial paralysis (over a 48 h period), posterior auricular pain, decreasing tearing, hyperacusis, taste disturbances, otalgia, weakness of the facial muscles, poor eyelid closure, aching of the ear or mastoid, alteration of taste, herperacusis, tingling or numbness of the cheek/mouth, epiphora, ocular pain, blurred vision.
* The onset of Bells’s palsy is typically sudden, and symptoms tend to peak in less than 48 hours. This sudden onset can be frightening for patients who often fear they have had a stroke or have a tumor and that the distortion of their faical appearance will be permanent.

**Investigations**

* Test for lyme disease if living in a lyme disease area.
* The ear and hearing should be examined carefully, in case there is another cause.
* If other cranial nerves are affected further investigations such as MRI scan may be needed.

**Treatment**

The management of Bell's palsy is controversial. Approximately 60% of cases recover completely without treatment, presumably because the lesion is so mild that it leads merely to conduction block. Considerable improvement occurs in most other cases, and only about 10% of all patients have permanent disfigurement or other long-term sequelae.

* The objectives of management are; to maintain facial muscle tone and to prevent or minimize denervation.
* Corticosteroid therapy (prednisone) may be initiated to reduce inflammation and edema, which reduces vascular compression and permits restoration of blood circulation to the nerve.

Early administration of corticosteroids appears to diminish severity, relieve pain, and minimize devervatio.

* Facial pain is controlled with analgesic agents or heat applied to the involved side of the face.
* Additional modalities may include electrical stimulation applied to the face to prevent muscle atrophy, or surgical exploration of the facial nerve.
* Surgery may be performed if a tumor is suspected, for surgical decompression of the facial nerve, and for surgical rehabilitation of the paralyzed face.
* Eye care

If the eye does not close properly it will probably get dry. If the cornea gets very dry it may become infected and scarred and even perforate.

The eye must be kept lubricated. If there is still some power in the eye muscles and some blinking does occur, lubricants such as viscotears four times a day may be needed.

Slightly worse cases need thicker ointment such as a simple eye ointment or lacrilube three times a day. Such patients will need to tape their eye shut at night.

The eye must be examined ., even more protection is needed. Some patients need their eye closing with stitched (tarsorraphy) or even an injection or bolulinum toxin. Bolulinum works for 6 weeks but may cause a little doubt with vision. By then the eye muscles may have started to recover.

* Prednisolone should be started preferably within 24 hours.

This may be suitable if immunocompromised….but may be fine if antivirals are used. 1mg/kg/day (maximum 80mg) for the first week, tapering in the second week. Give steroid advise. Avoid contact with people with infections e.t.c

**Nursing management**

Patients need reassurance that a stroke has not occurred and that spontaneous recovery occurs within 3 to 5 weeks in most patients. Teaching patients with Bell’s palsy to care for themselves at home is an important nursing priority.

**Teaching eye care**

Because the blink reflex is diminished, the involved eye may not close completely and needs to be protected to prevent corneal irritation and ulceration. Inform the patient of potentially complications, including corneal irritation and ulceration, overflow of tears, and absence of blink reflex. Key teaching point include:-

* Cover the eye with a protective shield at night.
* Apply eye ointment to keep eyelids closed during sleep.
* Close the paralysed eyelid manually before going to sleep.
* Wear wrap-around sunglasses or goggles to decrease normal evaporation from the eye.

**Teaching about maintaining muscle tone.**

* Show the patient how to perform facial massage with gentle upward motion several times daily when the patient can tolerate the massage.
* Demonstrate facial exercises, such as wrinkling the forehead, blowing out the cheeks, and whistling, in an effort to prevent muscle atrophy.
* Instruct the patient to avoid exposing the face to cold and drafts.
* Remind the patient and family of the importance of participating in health promotion activities and recommended health screening practices.

1. **GUILLEN BARRE SYNDROME (GBS)**

Guillain-Barré syndrome is an autoimmune attack of the peripheral nerve myelin. The result is acute, rapid segmental demyelination of peripheral nerves and some cranial nerves, producing ascending weakness with **dyskinesia** (inability to execute voluntary movements), hyporeflexia, and **paresthesias** (numbness).

CNS disorder characterized by bilateral, symmetrical, polyneuritis to ascending muscle weakness/paralysis;

It causes muscle weakness, loss of reflexes, and numbness or tingling of arms, legs, face, and other parts of the body.

This acute or subacute polyradiculoneuropathy sometimes follows infective illness, inoculations, or surgical procedures. There is an association with preceding Campylobacter jejuni enteritis. The disorder probably has an immunologic basis, but the precise mechanism is unclear

ESSENTIALS OF DIAGNOSIS

* Acute or subacute progressive polyradiculoneuropathy (an inflammatory disorder that affects the peripheral nerves and the spinal nerve roots. The onset and progression of the disease is variable with severe cases resulting in premature death)
* Usually ascending, symmetric weakness.
* Paresthesias are more variable.
* Acute dysautonomia may be life-threatening; i.e. autonomic dysfunction

Causes: idiopathic

**Predisposing factors;** Autoimmune**;** Antecedent viral infection**;** Immunization such as flu vaccine

**Pathophysiology**

Myelin is a complex substance that covers nerves, providing insulation and speeding the conduction of impulses from the cell body to the dendrites. The cell that produces myelin in the peripheral nervous system is the Schwann cell. In Guillain-Barré the Schwann cell is spared, allowing for remyelination in the

recovery phase of the disease. Guillain-Barré is the result of a cell-mediated immune attack on peripheral nerve myelin proteins (Ho & Griffin, 1999). The best-accepted theory is that an infectious organism contains an amino acid that mimics the peripheral nerve myelin protein. The immune system cannot distinguish between the two proteins and attacks and destroys peripheral nerve myelin. Studies indicate that an exact location within the peripheral nervous system, the ganglioside GM1b, is the most likely target of the immune attack (Yuki, Ang, Koga et al., 2000). With the autoimmune attack there is an influx of macrophages and other immune-mediated agents that attack myelin, cause inflammation and destruction, and leave the axon unable to support nerve conduction.

**Clinical presentation**

* Clumsiness- initial sign (awkward in movement or action; lacking physical coordination,
* Dysphagia
* Ascending muscle weakness a paralysis
* Decreased DTRs (deep tendon reflexes)
* Alternative hypertension and hypotension: most feared complication: arrhythmias.
* Autonomic changes: increased sweating and lacrimation, increased salivation, constipation.

**CLINICAL FINDINGS**

**1. Symptoms and signs**

The main complaint is of weakness that varies widely in severity in different patients and often has a proximal emphasis and symmetric distribution. It usually begins in the legs, spreading to a variable extent but frequently involving the arms and often one or both sides of the face. The muscles of respiration or deglutition may also be affected. Sensory symptoms are usually less conspicuous than motor ones, but distal paresthesias and dysesthesias are common, and neuropathic or radicular pain is present in many patients. Autonomic disturbances are also common, may be severe, and are sometimes life-threatening; they include tachycardia, cardiac irregularities, hypotension or hypertension, facial flushing, abnormalities of sweating, pulmonary dysfunction, and impaired sphincter control.

**2. Laboratory investigations**

The cerebrospinal fluid characteristically contains a high protein concentration with a normal cell content, but these changes may take 2 or 3 weeks to develop.

**DIFFERENTIAL DIAGNOSIS**

Diphtheritic, or toxic (heavy metal, hexacarbon, organophosphate) neuropathies. Poliomyelitis and botulism must also be considered as they cause weakness of acute onset.

**Medical Management**

* Because of the possibility of rapid progression and neuromuscular respiratory failure, Guillain-Barré is a medical emergency, requiring intensive care unit management.
* Respiratory therapy or mechanical ventilation may be necessary to support pulmonary function and adequate oxygenation. Mechanical ventilation may be required for an extended period.
* The patient is weaned from mechanical ventilation when the respiratory muscles can again support spontaneous respiration and maintain adequate tissue oxygenation.
* Other interventions are aimed at preventing the complications of immobility. These may include the use of anticoagulant agents and thigh-high elastic compression stockings or sequential compression boots to prevent thrombosis and pulmonary emboli.
* Plasmapheresis and IVIG are used to directly affect the peripheral nerve myelin antibody level. Both therapies decrease circulating antibody levels and reduce the amount of time the patient is immobilized and dependent on mechanical ventilation. Studies indicate that IVIG and plasmapheresis are equally effective in treating Guillain-Barré (Bella & Chad, 1999; Winer, 2002). The cardiovascular risks posed by autonomic dysfunction require continuous ECG monitoring.
* Tachycardia and hypertension are treated with short-acting medications such as alpha-adrenergic blocking agents.
* Hypotension is managed by increasing the amount of IV fluid administered. The use of short-acting agents is important because autonomic dysfunction is very labile.

**Nursing management**

1. Maintain patent airway and ventilation: assist in mechanical ventilation
2. Maintain side rails (paralysis)
3. Prevent complications of immobility
4. Institute NGT feeding
5. Administer medication as ordered:

Anticholnergics- atropine sulfate

Corticosteroids – to suppress immune response

Anti – arrhythmic agents; lidocain (xylocaine), bretyllium – blocks norepinephrine,

Quinidines – anti arrhythmic

1. Assist in plasmaparesis
2. Prevent complications: arrhythmias, respiratory arrest.

**PROGNOSIS**

Most patients eventually make a good recovery, but this may take many months, and 10–20% patients of are left with persisting disability

1. **BRAIN ABSCESS**

A brain abscess is a collection of pus, immune cells, and other materials in the brain, or fungal infection.

Brain abscess presents as an intracranial space-occupying lesion and arises as a sequela of disease of the ear or nose, may be a complication of infection elsewhere in the body, or may result from infection introduced intracranially by trauma or surgical procedures. The most common infective organisms are streptococci, staphylococci, and anaerobes; mixed infections are not uncommon

**Clinical Findings**

**SYMPTOMS AND SIGNS**

Headache, drowsiness, inattention, confusion, and seizures are early symptoms, followed by signs of increasing intracranial pressure and then a focal neurologic deficit. There may be little or no systemic evidence of infection.

Decreased movement

Decreased sensation; numbness and tingling

Aphasia (decreased speech)

Other: fever and chills, headache, language difficulties, loss of coordination, loss of muscle function, typically on one side; seizures, stiff neck, vision changes, vomiting.

**INVESTIGATIONS:**

Blood culture;

MRI of the head

Complete blood count.

**IMAGING**

A CT scan of the head

MRI

**ESSENTIALS OF DIAGNOSIS**

* Symptoms and signs of expanding intracranial mass.
* May be signs of primary infection or congenital heart disease.
* Fever may be absent.

**TREATMENT**

* Give intravenous antibiotics
* Give antifungal medications if the infection is likely caused by a fungus.
* Combined with surgical drainage (aspiration or excision) if necessary to reduce the mass effect, or sometimes to establish the diagnosis.
* Abscesses smaller than 2 cm can often be cured medically. Broad-spectrum antibiotics are used if the infecting organism is unknown. A common regimen is penicillin G (2 million units every 2 hours intravenously) plus either chloramphenicol (1–2 g intravenously every 6 hours), metronidazole (750 mg intravenously every 6 hours), or both.

Antimicrobial treatment is usually continued parenterally for 6–8 weeks, followed by orally for a further 2–3 weeks.

* The patient should be monitored by serial CT scans or MRI every 2 weeks and at deterioration.
* Corticosteroid therapy; Dexamethasone (4–25 mg four times daily, depending on severity, followed by tapering of dose, depending on response) may reduce any associated edema, but intravenous mannitol is sometimes required.

**Possible complications**

* Brain damage, meningitis that is severe or life threatening; return (recurrence ) of infections, seizures.

1. **Bell’s Palsy**

Bell’s palsy (facial paralysis) is due to peripheral involvement of the seventh cranial nerve on one side , which results in weakness or paralysis of the facial muscles.

**Aetiology;**

* The cause is unknown but possible causes include;
* Vascular ischemia, viral disease (herpes simplex, herpes zoster), autoimmune disease, or a combination.

**Clinical presentation:**

* Bell’s palsy may represent a type of pressure paralysis, in which ischemic necrosis of the facial nerve causes a distortion of the face, increased lacrimation (tearing) and painful sensation in the face, behind the ear, and in the eye. The patient may experience speech difficulties and may be unable to eat on the affected side owing to weakness.

**Medical management**

The objectives of management are to maintain facial muscle tone and to prevent or minimize denervation. Steroidal therapy may be initiated to reduce inflammation and edema, which reduces vascular compression and permits restoration of blood circulation to the nerve. Early administration of corticosteroids appears to diminish severity, relieve pain, and minimize denervation. Facial pain is controlled with analgesics agents or heat applied to the involved side of the face. Additional modalities may include electrical stimulation applied to the face to prevent muscle atrophy, or surgical exploration of the facial nerve. Surgery may be performed if a tumor is suspected, for surgical decompression of the facial nerve and for surgical rehabilitation of a paralyzed face.

**Nursing management**

Patients need reassurance that a stroke has not occurred and that spontaneous recovery occurs within 3 to 5 weeks in most patients. Teaching patients with Bell’s palsy to care for themselves at home is an important nursing priority.

**Teaching eye care**

Because the blink reflex is diminished, the involved eye may not close completely and needs to be protected to prevent corneal irritation and ulceration. Inform the patient of potentially complications, including corneal irritation and ulceration, overflow of tears, and absence of blink reflex. Key teaching point include:-

* Cover the eye with a protective shield at night.
* Apply eye ointment to keep eyelids closed during sleep.
* Close the paralysed eyelid manually before going to sleep.
* Wear wrap-around sunglasses or goggles to decrease normal evaporation from the eye.

**Teaching about maintaining muscle tone.**

* Show the patient how to perform facial massage with gentle upward motion several times daily when the patient can tolerate the massage.
* Demonstrate facial exercises, such as wrinkling the forehead, blowing out the cheeks, and whistling, in an effort to prevent muscle atrophy.
* Instruct the patient to avoid exposing the face to cold and drafts.
* Remind the patient and family of the importance of participating in health promotion activities and recommended health screening practices.

1. **DISSEMINATED SCLEROSIS**

Also known as multiple sclerosis

It is a chronic, degenerative, progressive disease of the central nervous system characterized by small patches of demyelination in the brain and spinal cord. Demyelination (destruction of myelin) results in impaired transmission of nerve impulses.

MS is a chronic autoimmune nerve disorder caused by destruction of the insulating layer (myelin) surrounding neurons in the brain and the spinal cord. Myelin helps electrical signals pass quickly and smoothly between the brain and the rest of the body. When the myelin is destroyed, nerve messages are sent more slowly and less efficiently. Patches of scar tissue, called plaques, form over the affected areas, further disrupting nerve communication. The symptoms of MS occurs when the brain and spinal cord nerves no longer communicate properly with other parts of the body.

**Cases:**

* Is an autoimmune disease. (For unknown reasons the immune cells attacks and destroys the myelin sheath that insulated neurons, in the brain and spinal cord.
* Viral infections e.g. AIDs

**Pathophysiology**

The cause of MS is not known, but a defective immune response probably plays a major role. In MS, sensitized T cells inhabit the CNS and facilitate the infiltration of other agents that damage the immune system. The immune system attack leads to inflammation that destroys the myelin and oligodendroglial cells that produce myelin in the CNS. Plaques of sclerotic tissue appear on demyelinated axons, further interrupting the transmission of the impulses. Demyelination interrupts the flow of nerve impulses.

MS is more common in people living in northern temperature zones. It is one of the most disabling neurological diseases of young adults (20 to 40 years) and it affects more women than men.

**Disease course; MS has various courses:**

* Relapsing-remitting course, with complete recovery between relapses.
* Chronic primary progressive course from the onset, with a progressive decline in function and the potential development of quadriparesis, cognitive dysfunction, visual loss and brain-stem syndromes.
* Benign course with a normal life span; symptoms are so mild that patients do not seek treatment.

**Clinical manifestations;**

* Signs and symptoms are varied and multiple and reflect the location of the lesion (plaque) or combination of lesions.
* Primary symptoms: pain, fatigue, weakness numbness, difficulty in coordination, and loss of balance.
* Visual disturbances: blurring of vision, double vision, patchy blindness (scotoma), or total blindness.
* Spastic weakness of the extremities and loss of abdominal reflexes; ataxia and tremor.
* Sensory dysfunction.
* Cognitive and psychosocial problems; depression, and emotional lability, and euphoria.
* Bladder, bowel, and sexual problems possible.

**Secondary manifestations related to complications.**

* Urinary tract infections, constipation
* Pressure ulcers, contractures deformities, dependent pedal edema
* Pneumonia
* Reactive depressions and decreased bones mass
* Emotional, social, marital, economic and vocational problems.

**Exacerbations and remissions**

* Relapses may be associated with periods of emotional and physical stress.
* There is evidence that remyelination occurs in some patients.

**Assessment and diagnostic findings**

* Magnetic resonance imaging (MRI) (primary diagnostic tool) to visualize small plaques, evaluate course and effect of treatment.
* Electrophoresis study of the cerebrospinal fluid (CSF); abnormal immunoglobulin G antibody (oligoclonal bonding) appears in the CSF in up to 95% of patients
* Neurolopsychological testing as indicated to assess cognitive impairment.
* Sexual history to identify changes in sexual function.
* Evoked potential studies and urodynamic studies.

**Medical management**

Because no cure exists for MS, the **goal of treatment are to delay the progression of the disease,** manage chronic symptoms, and treat acute exacerbations. And individualized treatment program is indicated to relieve symptoms (spasticity, fatigue, bladder dysfunction, and ataxia) and provide support. Management strategies target, the various motor and sensory symptoms and effects of immobility that can occur. Radiation therapy may be used to induce immunosuppression.

**Pharmacologic therapy**

* ‘’ABC’’ drugs; interferon-beta- la (Avonex) and beta -1b. (Betaseron) and glatiramer acetate (Copaxone) for relapsing-remitting MS.
* Mitoxantrone (Novantrone) is an intineoplastic agent recently approved to treat secondary progressive MS.
* Immunotherapeutic medications to modulate the immune response and reduce the rate at which the disease progresses and the frequency and severity of exacerbations (azathioprine, interferon, cyclophosphamide)
* Corticosteroids (e.g. intravenous methylpredinsolone) and adrenocorticotropic hormone (ACTH) as anti-inflammatory agents and to improve nerve conduction.
* Amantadine (symmetrel), pemoline (Cylert), or Fluoxetine (Prozac) to treat fatigue.
* Medications used to treat ataxia include (beta-adrenergic blockers(inderal), antiseizure agents (Neurontin), and benzodiazepines (Klonopin, valium)

**Management of related bowel and bladder problems**

Anticholnergics, alpha-adrenergic blockers, or antispasmodic agents may be used to treat problems related to elimination, and patients may be taught to perform intermittent self-catheterization as well. Additional measures include assessment, of urinary tract infections; ascorbic acid to acidify urine ; antibiotics when appropriate.

**Nursing care;**

* Promote physical mobility; encourage exercises.
* Prevent injury; teach patient to walk with feet wide apart
* Enhance bladder and bowel control; teach intermittent self-catheterization. , provide adequate fluids, dietary fibre.
* Improve sensory and cognitive function;
* Managing speech and swallowing difficulties.
* Promote sexual function. Refer to sexual counselor.

1. **SPACE OCCUPYING LESIONS**

A space occupying lesion is any abnormal tissue found on or in an organism, usually caused by disease or trauma.

A space occupying lesion of the brain is usually due to a malignancy but it can be caused by other pathology such as an abscess or a haematoma.

**Symptoms**

* Increased ICP – Headache (worse on walking, lying down, bending forwards or coughing), vomiting, papilloedema (seen in 50% of tumours), reduced GCS.
* Seizures – seen in <50%. Need to exclude SOL in all adult onset seizures, especially if focal.
* Evolving focal neurology – VI nerve palsy commonest due to its long intracranial course.

The symptoms are also dependent on the area of the brain affected:

* Temporal lobe – dysphasia, contralateral homonymous hemianopia, amnesia
* Frontal lobe – Hemiparaesis, personality change, executive dysfunction
* Parietal lobe – Hemisensory loss, astereogenesis (can’t recognise objects by touch alone), reduced 2-point discrimination
* Occipital lobe – contralateral visual field defects, palinopsia (see things again once stimulus has left field of vision)
* Cerebellum – DASHING: Dysdiadochokinesis, Ataxia, Slurred speech, Hypotonia, Intention tremor, Nystagmus, Gait abnormalities
* Personality change – irritability, lack of concentration, socially inappropriate behaviour.

**Causes**

* Tumour – primary (astrocytoma, meningioma, glioblastoma multiforme, oligodendroglioma, ependymoma) or metastatic (30%, breast, lung, melanoma)
* Aneurysms
* Abscess
* Chronic subdural haematoma
* Granuloma (TB)
* Cyst – 3rd ventricle colloid cyst

**Epidemiology;** The epidemiology depends on the underlying cause

**Diagnosis**

***Differential diagnosis****:* Stroke, head injury, venous sinus thrombosis, vasculitis, encephalitis, MS, post-ictal

*I****nvestigations****:*

* CT+/-MRI
* Biopsy
* ! Avoid LP before imaging as risk of herniation !

**Treatment**

The treatment depends on the underlying cause:

* Benign – removal if accessible
* Malignant – excision if possible, ventriculo-peritoneal shunt to reduce hydrocephalus
* Chemo-radiotherapy for metastatic/inoperable tumours
* Analgesia for headache

**Prognosis;** The prognosis depends on the underlying cause.

**Prevention**

* Again much like prognosis, this depends on the underlying cause