CENTRAL NERVOUS SYSTEM TUMOR

A **central nervous system tumor** (**CNS tumor**) is an abnormal growth of cells from the tissues of the brain or spinal cord. CNS tumor is a generic term encompassing over 120 distinct tumor types.

Types

Tumor classification	Frequency as % of total CNS tumors
Pilocytic astrocytomas	~30%
Diffuse astrocytomas	~12%
Anaplastic astrocytomas	~2%
Glioblastomas	~3%
Oligodendroglial tumors	~1.5%
Ependymal tumor	~9%
Medulloblastomas	~20%
Pineal tumors	~1.5%
Meningeal tumors	~1.2%
Germ cell tumors	~3%

Symptoms

The most common symptoms of CNS tumors are headache, vomiting, and nausea. Symptoms vary depending on the tumor and may include unsteady gait, slowed speech, memory loss, loss of hearing and vision, problems with memory, narrowing of visual field, and back pain. Symptoms may also vary greatly between individuals with the same tumor type.

In paediatric patients, symptoms may include:

- Headache
- Changes in vision
- Nausea and vomiting

- Balance problems
- Seizures
- Behavioral changes
- Abnormal head position
- Delayed puberty
- Abnormal growth
- Excessive thirst
- Reduced consciousness

Some symptoms in adults are specific to the location of the tumor:

- Tumors in the cerebrum, which controls movement, may cause weakness or numbness to the body. This weakness is often limited to one side of the body.
- Tumors in the Broca's area of the cerebrum can cause speech difficulties. In extreme cases, the patient may have problems understanding words.
- Tumors in the premotor cortex and the primary motor cortex, which are located at the front and top of the cerebrum, may cause sloppy movements, trouble walking, difficulties in moving arms, hands, fingers, and legs. In severe cases, such tumors may even cause wallowing and abnormal eye movements.
- Tumors located in the lower part of the cerebrum near the primary visual cortex can cause blurred vision, double vision, or loss of vision.
- Tumors located in the spinal cord usually have symptoms that start with back pain that spreads towards the arms or legs. These tumors can cause trouble urinating or walking.

Causes

The causes of CNS tumors are poorly understood. A few risk factors are known, including radiation exposure, genetic disorder, a family history of CNS tumors, immunodeficiency, stress and a history of previous cancers. As with all cancers, the risk of developing a CNS tumor increases with age.

Inheritance

A number of genetic disorders increase the risk of specific types of CNS tumors. These include tuberous sclerosis, Von Hippel-Lindau disease, Li-Fraumeni syndrome, Gorlin syndrome, Turcot syndrome, Cowden Syndrome and neurofibromatosis types 1 and 2 (NF1/NF2. Patients suffering from NF1 have higher risks of having schwannomas, meningiomas, and some types of gliomas. NF2 is correlated with vestibular schwannomas.

Diagnosis

There are no recommended tests to diagnose CNS tumors.

The tumor is usually found when patients develop symptoms and visit the doctor. The first step in diagnosis is usually a neurological examination, including tests for reflexes, muscle strength, vision and eye movements, and balance and alertness. If the results are abnormal, additional tests carried out by a specialist such as a neurosurgeon or a neurologist may be recommended.

Adults and children both undergo a similar set of tests to diagnose CNS tumors, including:

- Medical history
- Blood test
- Urine test
- Medical imaging
- X-ray
- CT scan
- MRI
- Biopsy

Treatment

CNS tumors are typically treated using one or more of the following options:

- Surgery
- Radiation Therapy
- Chemotherapy
- Targeted therapy

- Active surveillance

Treatment of CNS tumors frequently involves a team of doctors working together, including neurosurgeons, neurologists, medical oncologists, radiation oncologists and endocrinologists.

Surgery

Surgeries are used both to diagnose and to treat CNS tumors. Removal of tumor tissues helps decrease the pressure of the tumor on nearby parts of the brain. The main goal of surgery is to remove as much as possible of the tumor mass while preserving normal brain function, and to relieve the symptoms caused by the tumor such as headache, nausea and vomiting. Some tumors are deep-seated and unsafe to remove, and in these cases the role of surgery may be limited to obtaining a diagnostic biopsy. After the surgery, chemotherapy or radiation therapy may be used to destroy the remaining cancer cells.

Radiation therapy

Radiation therapy uses high energy rays to destroy cancer cells or to shrink tumors. The kinds of rays used are x-rays, gamma rays, electron beams or protons.

According to the National Cancer Institute, there are two types of radiation therapy:

- external radiation therapy
- internal radiation therapy

External radiation therapy or teletherapy uses a machine that sends a focused beam of radiation directed at the location of the tumor in the body. The radiation may be delivered from several angles or in a shaped beam to maximize the dose delivered to the tumor while reducing the harm to healthy parts of the body. Treatment is commonly given daily for 4-8 weeks.

In **internal radiation therapy**, the source of radiation is inserted into the patient's body. This may be done by placing a solid source of radiation adjacent to the tumor in the form of a seed, ribbon or capsule (**brachytherapy**) or by giving the patient a liquid source of radiation that

travels through the body and kills cancer cells (system therapy). In this case the radiation is usually given in the form of injections, ingesting a capsule or through an intravenous line.

Chemotherapy

Chemotherapy is a treatment that uses a tumor-killing drug to prevent the growth of cancer cells by stopping them from dividing. It is often used after surgery or as the first line of treatment. The drug may be given systemically, by injection into a vein or by mouth, or may be injected into the fluid that surrounds the brain and spinal cord to allow the drug to reach the tumor without crossing the blood–brain barrier (intrathecal administration).

Common side effects of chemotherapy include:

- Hair loss
- Mouth sores
- Loss of appetite
- Nausea
- Diarrhoea
- Increased rate of infections
- Easy bruising and bleeding
- Fatigue

Targeted therapy

An increasing number of drugs are available that promise to target a tumor specifically, reducing harm to normal cells. These therapies are matched to the specific tumor, and include antibodies that bind to specific surface molecules found primarily on the tumor, or small molecules that target proteins mutated in the tumor. Targeted therapies may block enzymes or other proteins necessary for cancer cell proliferation, deliver toxic substances directly to cancer cells, help with immune system function, or prevent the tumor from obtaining the nutrients it needs.

For example, bevacizumab is a targeted therapy drug used against various cancers, including glioblastoma, that blocks the blood supply to and therefore the proliferation of cancerous tumors. Checkpoint inhibitors, which prevent the tumor from blocking the action of tumor-killing cells of

the immune system. Although targeted therapy may have fewer side effects than other forms of cancer treatment, side effects are still frequent and may include high blood pressure, fatigue, increased risk of infection, or diarrhoea.

NURSING CARE USING NURSING PROCESS

Nursing care planning goals for patients with brain tumor centers on relieving pain, reducing anxiety, and promoting an understanding of the signs and symptoms of increased ICP and expected changes in body appearance related to the planned cranial surgery.

Here are three (3) nursing care plans and nursing diagnoses **for brain tumor:**

Acute Pain

Acute Pain: Unpleasant sensory and emotional experience arising from actual or potential tissue damage or described in terms of such damage; sudden or slow onset of any intensity from mild to severe with anticipated or predictable end and a duration of <6 months.

May be related to

- Biologic injuring agents

Possibly evidenced by

- Verbal complains of pain
- Headache in the frontal or occipital area that is worse during the morning and becomes worse with straining or if the head is dropped
- Changes in vital signs
- Hostile, tense behavior
- Restlessness

Desired Outcomes

- Child will rate pain as less than (specify pain rating and scale used).

Nursing Interventions	Rationale

Assess the severity and duration of a headache; observe precipitating factors, recurrence, and progressive characteristics.	Provides data about the presence of a tumor as a headache is a most usual symptom in the child.
Ascertain the child's perception of the word "pain" and ask the family what word the child normally uses. Use a pain assessment tool appropriate for age and developmental level to determine the pain intensity.	Promotes better communication between child/family and nurse during the assessment.
Administer analgesic as prescribed.	Used for the treatment of pain due to central nervous system tumors.
Instruct the child to refrain from sneezing, coughing, or straining during defecation.	Avoids straining that trigger or aggravates a headache.
Apply a cool compress on the head for low to moderate pain.	Promotes comfort and ease from a headache, reduces facial edema if present.
Provide toys, games for quiet play.	Provides diversionary activity to detract from pain.
After surgical intervention, opioids such as morphine sulfate may be initially given. Monitor for side effects such as sedation and respiratory depression; use Naloxone to block the effects of opioids.	Rarely side effects occur; opioids can be administered safely with appropriate monitoring.

Form a preventive strategy for pain management around the clock; note for physiologic and behavioral signs of pain.	Allows immediate identification of pain which improves measures for pain relief.
Educate parents and child about analgesics, to administer in anticipation of a headache and type to give (sustained release) and that it will help to control a headache.	Manages pain before it becomes severe.
Assist parents to formulate activities that will not trigger or heighten headache pain.	Promotes stimulation for a child's development needs.

Anxiety

Anxiety: Vague uneasy feeling of discomfort or dread accompanied by an autonomic response.

May be related to

- Change in health status and a threat to self-concept

Possibly evidenced by

- Increased apprehension as the diagnosis is confirmed and the condition worsens
- Expressed concern and worry about postoperative residual tumor and effects, hair removal before surgery
- Social isolation
- Insomnia

Desired Outcomes

- Parent will verbalize reduced anxiety.
- Child will appear relaxed, with an absence of crying or irritability.

Nursing Interventions	Rationale
Assess anxiety level and need for information that will relieve it post surgery.	Provides information about the degree of anxiety and need for measures and support; allow for identification of fear and uncertainty about surgery and treatments and recovery, guilt

	about illness, possible loss of the child, parental role and responsibility.
Allow expression of concerns and ask about the status of a sick patient and possible complications and prognosis.	Provides a chance to release feelings, secure information needed to reduce anxiety.
Encourage parents / next of kin to remain calm; allow participation in the care of infant/child.	Promotes care and support of the patient by parents/ significant order.
Prepare family and/or patient for diagnostic tests and surgery. Clarify to the child any misconceptions about the condition by illustrating a picture of a brain; allow the use of medical play (dolls, puppets, equipment) after procedures.	Promotes understanding which minimizes anxiety; may clarify misconceptions and raise feelings of control.
If surgery planned, orient to the surgery unit, equipment, and staff.	Lessens anxiety caused by fear of the unknown.
Educate patient about hair clipping and reassure that hair will grow back in a brief period of time, to cover head with hats, turbans or scarf temporarily; that there is swelling of the face and eyes post surgery; that an application of dressing will completely cover the head; use of a doll with head wrapped in a bandage may be helpful in describing the post-surgical dressing.	Promotes understanding of postoperative appearance to assert self-image; support self-concept.
Educate patient that post surgery, a headache, and somnolence may be experienced for a few days or even lethargy and coma may be present.	Renders an explanation of what to expect after surgery.
Clarify any information in simple language and utilize age-related aids that are helpful to the child.	Prevents unnecessary anxiety following from misinterpretation or inconsistencies in information.

Risk for Injury

Risk for Injury***:*** Vulnerable for injury as a result of environmental conditions interacting with the individual's adaptive and defensive resources, which may compromise health.

May be related to

- Sensory, integrative, and effector dysfunction

Possibly evidenced by

- Behavioral changes

- Increased ICP
- Neuromuscular changes
- Neurosensory changes
- Seizure activity
- Vital signs changes

Desired Outcomes

- Patient will not exhibit signs of increased intracranial pressure and will participate in teaching about treatment options.

Nursing Interventions	Rationale
Assess vital signs including increased BP, decreased pulse pressure, pulse, and respirations; take one full minute when monitoring pulse and respiration.	Any changes in the vital signs may reveal the presence of brain tumor depending on type and location of the tumor.
Assess for irritability, lethargy, fatigue, sleepiness, loss of consciousness or coma.	Behavioral changes indicating the presence of brain tumor.
Assess changes in vision (visual acuity, strabismus, diplopia, nystagmus), head tilt, papilledema.	Changes in neurosensory status revealing the presence of brain tumor.
Assess changes in gross and fine motor control, spasticity, ataxia, weakness, paralysis or change in balance, coordination.	Symptoms of neuromuscular changes indicating the presence of brain tumor.
Assess for increased ICP including high-pitched cry (infant) or vomiting, poor feeding, irritability, head enlargement, lethargy, diplopia, behavioral changes, change in VS, seizure activity.	Provides data about changes in intracranial pressure as a result of brain distortion or shifting caused by a tumor.
Assess the child's head circumference; Fluid obstruction caused by tumor will increase head size.	Provides information indicating an increase in ICP as the tumor grows with a poorer prognosis because tumor size becomes large before a diagnosis is made.
Maintain a position of comfort with head elevated.	Provides comfort and minimizes increased ICP by promoting venous drainage.
Modify environment by padding bed or crib, decrease light and stimulation.	Prevents injury if seizure activity possible.
Educate parents and child about diagnostic procedures done to assess the presence of a tumor; base information on child's age and past experiences.	Promotes understanding of procedures.
Notify parents that surgery may be performed to remove the tumor as a reinforcement of physician information and that	Prepared for surgery and possible postoperative treatment with information limited to sensitive, hopeful explanation;

radiation and chemotherapy may be offered after surgery.

information about postoperative therapy should be postponed until this
a decision is secured post surgery.

AUTOIMMUNE NERVOUS SYSTEM DISORDERS

Autoimmune nervous system disorders include multiple sclerosis, myasthenia gravis, and Guillain-Barré syndrome.

Multiple Sclerosis

Multiple sclerosis (MS) is an immune-mediated, progressive demyelinating disease of the CNS. Demyelination refers to the destruction of myelin, the fatty and protein material that surrounds certain nerve fibers in the brain and spinal cord; it results in impaired transmission of nerve impulses

MS may occur at any age but typically manifests in young adults between the ages of 20 and 40 years; it affects women more frequently than men.

CAUSES

- The cause of MS is an area of ongoing research.
- Autoimmune activity results in demyelination, but the sensitized antigen has not been identified.
- Multiple factors play a role in the initiation of the immune process.
- Geographic prevalence is highest in Europe, New Zealand, southern Australia, the northern United States, and southern Canada.
- Researchers believe that some environmental exposure at a young age may play a role in the development of MS later in life.
- Genetic predisposition is indicated by the presence of a specific cluster (haplotype) of human leukocyte antigens (HLAs) on the cell wall. Its presence may increase susceptibility to factors, such as viruses, that trigger the autoimmune response activated in MS.
- A specific virus capable of initiating the autoimmune response has not been identified.
- It is believed that deoxyribonucleic acid (DNA) on the virus mimics the amino acid sequence of myelin, resulting in an immune system cross-reaction in the presence of a defective immune system.

Pathophysiology

Sensitized T and B lymphocytes cross the blood–brain barrier; their function is to check the CNS for antigens and then leave. In MS, sensitized T cells remain in the CNS and promote the infiltration of other agents that damage the immune system. The immune system attack leads to inflammation that destroys myelin (which normally insulates the axon) and the oligodendroglial cells that produce myelin in the CNS.

Demyelination interrupts the flow of nerve impulses and results in a variety of manifestations, depending on the nerves affected.

Plaques appear on demyelinated axons, further interrupting the transmission of impulses. Demyelinated axons are scattered irregularly throughout the CNS. The areas most frequently affected are the optic nerves, chiasm, and tracts; the cerebrum; the brain stem and cerebellum; and the spinal cord. The axons themselves begin to degenerate, resulting in permanent and irreversible damage.

Types and courses of multiple sclerosis (MS)

1. Relapsing-remitting (RR) MS is characterized by clearly acute attacks with full recovery or with sequelae and residual deficit

upon recovery. Periods between disease relapses are characterized by lack of disease progression.

2. Primary progressive (PP): MS is characterized by disease showing progression of disability from onset, without plateaus and temporary minor improvements.
3. Secondary progressive (SP) MS begins with an initial RR course, followed by progression of variable rate, which may also include occasional relapses and minor remissions.
4. Progressive- relapsing (PR) MS shows progression from onset but with clear acute relapses with or without recovery.

SIGNS AND SYMPTOMS

The signs and symptoms of MS are varied and multiple, reflecting the location of the lesion (plaque) or combination of lesions.

The primary symptoms most commonly reported are

- Fatigue
- Depression
- Weakness
- Numbness
- Difficulty in coordination
- **Spasticity** (muscle hypertonicity)
- Loss of balance and pain
- Paresthesias
- Dysesthesias
- Proprioception loss
- Visual disturbances due to lesions in the optic nerves or their connections may include blurring of vision, **diplopia** (double vision), patchy blindness (scotoma), and total blindness
- Involvement of the cerebellum or basal ganglia can produce **ataxia** (impaired coordination of movements) and tremor.
- Secondary complications of MS include urinary tract infections, constipation, pressure ulcers, contracture deformities, dependent pedal edema, pneumonia, reactive depression, and osteoporosis. Emotional, social, marital, economic, and vocational problems may also occur.

Assessment and Diagnostic Findings

i. The diagnosis of MS is based on the presence of multiple plaques in the CNS observed with MRI
ii. Electrophoresis of CSF identifies the presence of oligoclonal banding (several bands of immunoglobulin G bonded together, indicating an immune system abnormality).
iii. Evoked potential studies can help define the extent of the disease process and monitor changes. Underlying bladder dysfunction is diagnosed by urodynamic studies.
iv. Neuropsychological testing may be indicated to assess cognitive impairment.
v. A sexual history helps identify changes in sexual function.

Medical Management

No cure exists for MS. An individual treatment program is indicated to relieve the patient's symptoms and provide continuing support, particularly for patients with cognitive changes, who may need more structure and support.

The goals of treatment are to delay the progression of the disease, manage chronic symptoms, and treat acute exacerbations.

Many patients with MS have a stable disease course and require only intermittent treatment, whereas others experience steady progression of their disease. Symptoms requiring intervention include spasticity, fatigue, bladder dysfunction, and ataxia.

Management strategies target the various motor and sensory symptoms and effects of immobility that can occur.

Pharmacologic Therapy

Medications prescribed for MS include those for disease modification and those for symptom management. The disease- modifying therapies available to treat MS include immunomodulating therapies and immunosuppressive agents

Disease-Modifying Therapies

The disease-modifying medications reduce the frequency of relapse, the duration of relapse, and the number and size of plaques observed on MRI. All of the medications require injection. Examples are

- Interferon beta-1a (Rebif) and interferon beta-1b (Betaseron) are administered subcutaneously.
- Another preparation of interferon beta-1a, Avonex, is administered intramuscularly once a week.
- Glatiramer acetate (Copaxone) reduces the rate of relapse in the RR course of MS. It decreases the number of plaques noted on MRI and increases the time between relapses.
- Copaxone is administered subcutaneously daily. It acts by increasing the antigen-specific suppressor T cells. Copaxone is an option for those with an RR course; however, it may take 6 months for evidence of an immune response to appear.
- IV methylprednisolone, the key agent in treating acuterelapse in the RR course, shortens the duration of relapse. It exerts anti-inflammatory effects by acting on T cells and cytokines.

- One gram is administered IV daily for 3 days, followed by an oral taper of prednisone.
- The medication mitoxantrone (Novantrone) is administered via IV infusion every 3 months.
- Novantrone can reduce the frequency of clinical relapses in patients with secondary-progressive or worsening relapsing-remitting MS.

Patients must be very closely monitored for side effects, especially cardiac toxicity.

Symptom Management

Medications are also prescribed for management of specific symptoms.

1. Baclofen (Lioresal), a gamma-aminobutyric acid (GABA) agonist, is the medication of choice for treating spasticity. It can be administered orally or by intrathecal injection for severe
2. Benzodiazepines (Valium), tizanidine (Zanaflex), and dantrolene (Dantrium) may also be used to treat spasticity.
3. Patients with disabling spasms and contractures may require nerve blocks or surgical intervention.
4. Fatigue that interferes with activities of daily living (ADLs) may be treated with amantadine (Symmetrel), pemoline (Cylert), or fluoxetine (Prozac).
5. Ataxia is a chronic problem most resistant to treatment. Medications used to treat ataxia include betaadrenergic blockers (Inderal), antiseizure agents (Neurontin), and benzodiazepines (Klonopin).
6. Bladder and bowel problems are often among the most difficult ones for patients, and a variety of medications (anticholinergic agents, alpha-adrenergic blockers, antispasmodic agents) may be prescribed.
7. Ascorbic acid (vitamin C) may be prescribed to acidify the urine, making bacterial growth less likely. Antibiotics are prescribed when appropriate.

NURSING PROCESS

Assessments

Nursing assessment addresses neurologic deficits, secondary complications, and the impact of the disease on the patient and family. The patient's mobility and balance are observed to determine whether there is risk of falling.

Assessment of function is carried out both when the patient is well rested and when fatigued. The patient is assessed for weakness, spasticity, visual impairment, incontinence, and disorders of swallowing and speech.

Additional areas of assessment include how MS has affected the patient's lifestyle, how the patient is coping, and what the patient would like to improve.

Nursing Diagnoses

Based on the assessment data, the patient's major nursing diagnoses may include the following:

• Impaired bed and physical mobility related to weakness, muscle paresis, spasticity

• Risk for injury related to sensory and visual impairment

• Impaired urinary and bowel elimination (urgency, frequency, incontinence, constipation) related to nervous system dysfunction

• Impaired verbal communication and risk for aspiration related to cranial nerve involvement

• Disturbed thought processes (loss of memory, dementia, euphoria) related to cerebral dysfunction

• Ineffective individual coping related to uncertainty of course of MS

• Impaired home maintenance management related to physical, psychological, and social limits imposed by MS

• Potential for sexual dysfunction related to lesions or psychological reaction

Goals

The major goals for the patient may include

i. Promotion of physical mobility
ii. Avoidance of injury
iii. Achievement of bladder and bowel continence
iv. Promotion of speech and swallowing mechanisms
v. Improvement of cognitive function
vi. Development of coping strengths

vii. Improved home maintenance management, and

viii. Adaptation to sexual dysfunction

NURSING INTERVENTION

Promoting Physical Mobility

Relaxation and coordination exercises promote muscle efficiency.

Progressive resistive exercises are used to strengthen weak muscles, because diminishing muscle strength is often significant in MS.

EXERCISES: Walking improves the gait, particularly the problem of loss of position sense of the legs and feet. If certain muscle groups are irreversibly affected, other muscles can be trained to compensate. Instruction in the use of assistive devices may be needed to ensure their safe and correct use.

MINIMIZING SPASTICITY AND CONTRACTURES: Muscle spasticity is common and, in its later stages, is characterized by severe adductor spasm of the hips with flexor spasm of the hips and knees.

Without relief, fibrous contractures of these joints occur.

Warm packs may be beneficial, but hot baths should be avoided because of risk of burn injury secondary to sensory loss and increasing symptoms that may occur with elevation of the body temperature.

Daily exercises for muscle stretching are prescribed to minimize joint contractures. Special attention is given to the hamstrings, gastrocnemius muscles, hip adductors, biceps, and wrist and finger flexors. Muscle spasticity is common and interferes with normal function. A stretch–hold–relax routine is helpful for relaxing and treating muscle spasticity.

Swimming and stationary bicycling are useful, and progressive weight bearing can relieve spasticity in the legs. The patient should not be hurried in any of these activities, because this often increases spasticity.

ACTIVITY AND REST: The patient is encouraged to work and exercise to a point just short of fatigue. Very strenuous physical exercise is not advisable, because it raises the body temperature and may aggravate symptoms.

The patient is advised to take frequent short rest periods, preferably lying down. Extreme fatigue may contribute to the exacerbation of symptoms.

MINIMIZING EFFECTS OF IMMOBILITY: Because of the decrease in physical activity that often occurs with MS, complications associated with immobility, including pressure ulcers, expiratory muscle weakness, and accumulation of bronchial secretions, need to be considered and steps taken to prevent them.

Measures to prevent such complications include assessing and maintaining skin integrity and having the patient perform coughing and deep-breathing exercises.

Preventing Injury

If motor dysfunction causes problems of incoordination and clumsiness, or if ataxia is apparent, the patient is at risk for falling. To overcome this disability, the patient is taught to walk with feet apart to widen the base of support and to increase walking stability.

If loss of position sense occurs, the patient is taught to watch the feet while walking.

Gait training may require assistive devices (walker, cane, braces, crutches, parallel bars) and instruction about their use by a physical therapist.

If the gait remains inefficient, a wheelchair or motorized scooter may be the solution. The occupational therapist is a valuable resource person in suggesting and securing aids to promote independence.

If incoordination is a problem and tremor of the upper extremities occurs when voluntary movement is attempted (intention tremor), weighted bracelets or wrist cuffs are helpful. The patient is trained in transfer and activities of daily living (ADLs).

Because sensory loss may occur in addition to motor loss, pressure ulcers are a continuing threat to skin integrity. The need to use a wheelchair continuously increases the risk.

Enhancing Bladder and Bowel Control

Generally, bladder symptoms fall into the following categories:
(1) Inability to store urine (hyperreflexic, uninhibited);
(2) Inability to empty the bladder (hyporeflexic, hypotonic); and (3) a mixture of both types. The patient with urinary frequency, urgency, or incontinence requires special support.
The sensation of the need to void must be heeded immediately, so the bedpan or urinal should be readily available.
A voiding time schedule is set up (every 1.5 to 2 hours initially, with gradual lengthening of the interval).

The patient is instructed to drink a measured amount of fluid every 2 hours and then attempt to void 30 minutes after drinking.
Use of a timer or wristwatch with an alarm may be helpful for the patient who does not have enough sensation to signal the need to empty the bladder.
The nurse encourages the patient to take the prescribed medications to treat bladder spasticity, because this allows greater independence.
Intermittent self-catheterization has been successful in maintaining bladder control in patients with MS.
If a female patient has permanent urinary incontinence, urinary diversion procedures may be considered. The male patient may wear a condom appliance for urine collection.
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Enhancing Communication and Managing Swallowing Difficulties

If the cranial nerves that control the mechanisms of speech and swallowing are affected, dysarthrias (defects of articulation) marked by slurring, low volume of speech, and difficulties in phonation may occur.

Dysphagia (difficulty swallowing) may also occur. A speech therapist evaluates speech and swallowing and instructs the patient, family, and health team members about strategies to compensate for speech
and swallowing problems. The nurse reinforces this instruction and encourages the patient and family to adhere to the plan.

Impaired swallowing increases the patient's risk of aspiration; therefore, strategies are needed to reduce that risk.

Such strategies include having suction apparatus available, careful feeding, and proper positioning for eating.

Promoting Sexual Functioning

Patients with MS and their partners face problems that interfere with sexual activity, both as a direct consequence of nerve damage and also from psychological reactions to the disease.

Easy fatigability, conflicts arising from dependency and depression, emotional liability, and loss of self-esteem compound the problem.

Erectile and ejaculatory disorders in men and orgasmic dysfunction and adductor spasms of the thigh muscles in women can make sexual intercourse difficult or impossible. Bladder and bowel incontinence and urinary tract infections add to the difficulties.

Collaboration between the patient, family, and health care provider is essential for supporting intimacy

A sexual counselor can help bring into focus the patient's or partners sexual resources and suggest relevant information and supportive therapy. Sharing and communicating feelings, planning for sexual activity (to minimize the effects of fatigue), and exploring alternative methods of sexual expression may open up a wide range of sexual enjoyment and experiences.

COMPLETE THE OTHERS

MYASTHENIA GRAVIS

Definition:

Myasthenia gravis (my-us-THEE-nee-uh GRAY-vis) is characterized by weakness and rapid fatigue of any of the muscles under your voluntary control. It's caused by a breakdown in the normal communication between nerves and muscles.

Age at Risk:

Though this disease can affect people of any age, it's more common in women younger than 40 and in men older than 60.

Symptoms

Muscle weakness caused by myasthenia gravis worsens as the affected muscle is used. Because symptoms usually improve with rest, muscle weakness can come and go.

However, the symptoms tend to progress over time, usually reaching their worst within a few years after the onset of the disease.

Although myasthenia gravis can affect any of the muscles that you control voluntarily, certain muscle groups are more commonly affected than others.

Eye muscles

In more than half of people who develop myasthenia gravis, their first signs and symptoms involve eye problems, such as:

- Drooping of one or both eyelids (ptosis)
- Double vision (diplopia), which may be horizontal or vertical, and improves or resolves when one eye is closed

Face and throat muscles

In about 15% of people with myasthenia gravis, the first symptoms involve face and throat muscles, which can:

- **Impair speaking.** The speech might sound soft or nasal, depending on which muscles have been affected.
- **Cause difficulty swallowing.** Patient might choke easily, making it difficult to eat, drink or take pills. In some cases, liquids you're trying to swallow come out your nose.
- **Affect chewing.** The muscles used for chewing might wear out halfway through a meal, particularly if you've been eating something hard to chew, such as steak.

- **Change facial expressions.** For example, your smile might look like a snarl.

Neck and limb muscles

Myasthenia gravis can also cause weakness in your neck, arms and legs. Weakness in the legs can affect how the patient walks. Weak neck muscles make it hard to hold up the head.

Causes

1. **Antibodies:** The nerves communicate with the muscles by releasing chemicals (neurotransmitters) that fit precisely into receptor sites on the muscle cells at the nerve-muscular junction.

In myasthenia gravis, the immune system produces antibodies that block or destroy many of the muscles' receptor sites for a neurotransmitter called acetylcholine. With fewer receptor sites available, the muscles receive fewer nerve signals, resulting in weakness.

Antibodies can also block the function of a protein called a muscle-specific receptor tyrosine kinase. This protein is involved in forming the nerve-muscular junction. Antibodies that block this protein can lead to myasthenia gravis.

2. **Thymus gland**

The thymus gland is a part of your immune system situated in the upper chest beneath the breastbone. Researchers believe the thymus gland triggers or maintains the production of the antibodies that block acetylcholine.

Large in infancy, the thymus gland is small in healthy adults. In some adults with myasthenia gravis, however, the thymus gland is abnormally large. Some people with myasthenia gravis also have tumors of the thymus gland (thymomas). Usually, thymomas aren't cancerous (malignant), but they can become cancerous.

3. **Other causes**

Some people have myasthenia gravis that isn't caused by antibodies blocking acetylcholine or the muscle-specific receptor tyrosine kinase. This type of myasthenia gravis is called antibody-negative myasthenia gravis. Antibodies against another protein, called lipoprotein-related protein 4, can play a part in the development of this condition.

Rarely, mothers with myasthenia gravis have children who are born with myasthenia gravis (neonatal myasthenia gravis). If treated promptly, children generally recover within two months after birth.

Some children are born with a rare, hereditary form of myasthenia, called congenital myasthenic syndrome.

Factors that can worsen myasthenia gravis

- Fatigue
- Illness
- Stress
- Some medications — such as beta blockers, quinidine gluconate, quinidine sulfate, quinine, phenytoin, certain anesthetics and some antibiotics
- Pregnancy
- Menstrual periods

Complications

Complications of myasthenia gravis are treatable, but some can be life-threatening.

Myasthenic crisis

Myasthenic crisis is a life-threatening condition that occurs when the muscles that control breathing become too weak to do their jobs. Emergency treatment is needed to provide

mechanical assistance with breathing. Medications and blood-filtering therapies help people to again breathe on their own.

Thymus gland tumors

Some people with myasthenia gravis have a tumor in their thymus gland, a gland under the breastbone that is involved with the immune system. Most of these tumors, called thymomas, aren't cancerous (malignant).

Other disorders

People with myasthenia gravis are more likely to have the following conditions:

- **Underactive or overactive thyroid.** The thyroid gland, which is in the neck, secretes hormones that regulate your metabolism. If your thyroid is underactive, you might have difficulties dealing with cold, weight gain and other issues. An overactive thyroid can cause difficulties dealing with heat, weight loss and other issues.
- **Autoimmune conditions.** People with myasthenia gravis might be more likely to have autoimmune conditions, such as rheumatoid arthritis or lupus.

Pathophysiology of Myasthenia Gravis

The normal neuromuscular junction releases acetylcholine (ACh) from the motor nerve terminal in discrete packages (quanta). The ACh quanta diffuse across the synaptic cleft and bind to receptors on the folded muscle end-plate membrane. Stimulation of the motor nerve releases many ACh quanta that depolarize the muscle end-plate region and then the muscle membrane causing muscle contraction. In acquired myasthenia gravis, the post-synaptic muscle membrane is distorted and simplified, having lost its normal folded shape. The concentration of ACh receptors on the muscle end-plate membrane is reduced, and antibodies are attached to the membrane. ACh is released normally, but its effect on the post-synaptic membrane is reduced. The post-junctional membrane is less sensitive to applied ACh, and the probability that any nerve impulse will cause a muscle action potential is reduced.

The Thymus in Myasthenia Gravis

Thymic abnormalities are clearly associated with myasthenia gravis but the nature of the association is uncertain. Ten percent of patients with myasthenia gravis have a thymic tumor and 70% have hyperplastic changes (germinal centers) that indicate an active immune response. These are areas within lymphoid tissue where B-cells interact with helper T-cells to produce antibodies. Because the thymus is the central organ for immunological self-tolerance, it is reasonable to suspect that thymic abnormalities cause the breakdown in tolerance that causes an immune-mediated attack on AChR in myasthenia gravis. The thymus contains all the necessary elements for the pathogenesis of myasthenia gravis: myoid cells that express the AChR antigen, antigen presenting cells, and immunocompetent T-cells. Thymus tissue from patients with myasthenia gravis produces AChR antibodies when implanted into immunodeficient mice. However, it is still uncertain whether the role of the thymus in the pathogenesis of myasthenia gravis is primary or secondary. Most thymic tumors in patients with myasthenia gravis are benign, well-differentiated and encapsulated, and can be removed completely at surgery. It is unlikely that thymomas result from chronic thymic hyperactivity because myasthenia gravis can develop years after thymoma removal and the HLA haplotypes that predominate in patients with thymic hyperplasia are different from those with thymomas. Patients with thymoma usually have more severe disease, higher levels of AChR antibodies, and more severe EMG abnormalities than patients without thymoma. Almost 20% of patients with myasthenia gravis whose symptoms began between the ages of 30 and 60 years have thymoma; the frequency is much lower when symptom onset is after age 60.

CLASSIFICATION

The Myasthenia Gravis Foundation of America Clinical Classification divides MG into 5 main classes and several subclasses

- Class I: Any ocular muscle weakness; may have weakness of eye closure; all other muscle strength is normal

- Class II: Mild weakness affecting other than ocular muscles; may also have ocular muscle weakness of any severity
    - Class IIa: Predominantly affecting limb, axial muscles, or both; may also have lesser involvement of oropharyngeal muscles
    - Class IIb: Predominantly affecting oropharyngeal, respiratory muscles, or both; may also have lesser or equal involvement of limb, axial muscles, or both
- Class III: Moderate weakness affecting other than ocular muscles; may also have ocular muscle weakness of any severity
    - Class IIIa: Predominantly affecting limb, axial muscles, or both; may also have lesser involvement of oropharyngeal muscles
    - Class IIIb: Predominantly affecting oropharyngeal, respiratory muscles, or both; may also have lesser or equal involvement of limb, axial muscles, or both
- Class IV: Severe weakness affecting other than ocular muscles; may also have ocular muscle weakness of any severity
    - Class IVa: Predominantly affecting limb, axial muscles, or both; may also have lesser involvement of oropharyngeal muscles
    - Class IVb: Predominantly affecting oropharyngeal, respiratory muscles, or both; may also have lesser or equal involvement of limb, axial muscles, or both
- Class V: Defined by the need for intubation, with or without mechanical ventilation, except when used during routine postoperative management. The use of a feeding tube without intubation places the patient in class IVb.

Diagnosis

Neurological examination

The doctor may check the neurological health by testing:

- Reflexes
- Muscle strength

- Muscle tone
- Senses of touch and sight
- Coordination
- Balance

Tests to help confirm a diagnosis of myasthenia gravis might include:

Edrophonium test

Injection of the chemical edrophonium chloride that results in a sudden, temporary improvement in muscle strength might indicate that you have myasthenia gravis. Edrophonium chloride blocks an enzyme that breaks down acetylcholine, the chemical that transmits signals from your nerve endings to your muscle receptor sites.

Ice pack test

If patient is having a droopy eyelid, the doctor might place a bag filled with ice on the eyelid. After two minutes, he removes the bag and analyzes the droopy eyelid for signs of improvement.

Blood analysis

A blood test might reveal the presence of abnormal antibodies that disrupt the receptor sites where nerve impulses signal the muscles to move.

Repetitive nerve stimulation

In this nerve conduction study, doctors attach electrodes to the skin over the muscles to be tested. Doctors send small pulses of electricity through the electrodes to measure the nerve's ability to send a signal to the muscle.

To diagnose myasthenia gravis, doctors will test the nerve repeatedly to see if its ability to send signals worsens with fatigue.

Single-fiber electromyography (EMG)

This test measures the electrical activity traveling between the brain and the muscle. It involves inserting a fine wire electrode through the skin and into a muscle to test a single muscle fiber.

Imaging

The doctor might order a CT scan or an MRI to check if there's a tumor or other abnormality in the thymus.

Pulmonary function tests

These tests evaluate whether your condition is affecting your breathing

Management

Various treatments, alone or in combination, can relieve symptoms of myasthenia gravis. The treatment will depend on the age, how severe the disease is and how fast it's progressing.

Medications

- **Cholinesterase inhibitors.** Medications such as pyridostigmine (Mestinon, Regonal) and neostigmine (Bloxiverz) enhance communication between nerves and muscles. These medications aren't a cure, but they can improve muscle contraction and muscle strength in some people.

  Possible side effects include gastrointestinal upset, diarrhea, nausea, and excessive salivation and sweating.

- **Corticosteroids.** Corticosteroids such as prednisone inhibit the immune system, limiting antibody production. Prolonged use of corticosteroids, however, can lead to serious side

effects, such as bone thinning, weight gain, diabetes and increased risk of some infections.

- **Immunosuppressants.** The doctor might also prescribe other medications that alter the immune system, such as azathioprine (Azasan,, Imuran) mycophenolate mofetil (Cellcept), cyclosporine (Sandimmune) methotrexate (Trexall) or tacrolimus (Astrograf XL, Prograf). These drugs, which can take months to work, might be used with corticosteroids.

  Side effects of immunosuppressants, such as increased risk of infection and liver or kidney damage, can be serious.

Intravenous therapy

The following therapies are usually used in the short term to treat a sudden worsening of symptoms or before surgery or other therapies.

- **Plasmapheresis:** This procedure uses a filtering process similar to dialysis. The blood is routed through a machine that removes the antibodies that block transmission of signals from your nerve endings to your muscles' receptor sites. However, the good effects usually last only a few weeks, and repeated procedures can lead to difficulty accessing veins for the treatment.

  Risks associated with plasmapheresis include a drop in blood pressure, bleeding, heart rhythm problems or muscle cramps. Some people develop an allergic reaction to the solutions used to replace the plasma.

- **Intravenous immunoglobulin (IVIg).** This therapy provides the body with normal antibodies, which alters the immune system response. Benefits are usually seen in less than a week and can last three to six weeks.
- **Monoclonal antibody.** Rituximab (Rituxan) and the more recently approved eculizumab (Soliris) are intravenous medications used in some cases of myasthenia gravis. These drugs are usually used for people who don't respond to other treatments. They can have serious side effects.

Surgery

Some people with myasthenia gravis have a tumor in their thymus gland. If the patient has a tumor, called a thymoma, doctors will surgically remove the thymus gland (thymectomy).

Even if no tumor in the thymus gland, removing the gland might improve the myasthenia gravis symptoms. However, the benefits of thymectomy can take years to develop.

- **Video-assisted thymectomy.** In one form of this surgery, surgeons make a small incision in the neck or a few small incisions in the side of the chest. They then use a long, thin camera (video endoscope) and small instruments to see and remove the thymus gland.
- **Robot-assisted thymectomy.** In this form of thymectomy, surgeons make several small incisions in the side of the chest and remove the thymus gland using a robotic system, which includes a camera arm and mechanical arms.
- These procedures might cause less blood loss, less pain, lower mortality rates and shorter hospital stays compared with open surgery.

Nursing Interventions

1. Monitor respiratory status and ability to cough and deep breathe adequately.
2. Monitor for respiratory failure.
3. Maintain suctioning and emergency equipment at the bedside.
4. Monitor vital signs.
5. Monitor speech and swallowing abilities to prevent aspiration.
6. Encourage the client to sit up when eating.
7. Assess muscle status.
8. Instruct the client to conserve strength.
9. Plan short activities that coincide with times of maximal muscle strength.
10. Monitor for myasthenic and cholinergic crises.
11. Administer anticholinesterase medications as prescribed.
12. Instruct the client to avoid stress, infection, fatigue, and over-the counter medications.
13. Instruct the client to wear a Medic-Alert bracelet.

Discharge and Home Healthcare Guidelines

- Instruct the patient and family on the importance of rest and avoiding fatigue. Be alert to factors that can cause exacerbations, such as infection (an annual flu shot is suggested), surgery, pregnancy, exposure to extreme temperatures, and tonic and alcoholic drinks. Instruct the patient and family about drug actions and side effects, the indications for dosage alteration, and the selective use of atropine for any overdose.
- Stress the importance of taking the medication in a timely manner. It is advisable to time the dose 1 hour before meals for best chewing and swallowing. Explain the potential drug interactions (especially aminoglycosides and neuromuscular blocking agents, which include many pesticides). Encourage the patient to inform the dentist, ophthalmologist, and pharmacist of the myasthenic condition.
- Instruct patients about the symptoms that require emergency treatment, and encourage them to locate a neurologist familiar with MG management for any follow-up needs. Suggest that they collect a packet of literature to take to the emergency department in case the available physician is unfamiliar with this disease. (The Physician's Handbook is available on request from the MG Foundation.)
- Instruct patients to wear MG identification jewelry. Suggest having an "emergency code" to alert family if they are too weak to speak (such as ringing the phone twice and hanging up).
- Instruct the family about cardiopulmonary resuscitation techniques, how to perform the Heimlich maneuver, how to contact the rescue squad, and how to explain the route to the hospital.
- Make a referral to a vocational rehabilitation center if guidance for modifying the home or work environment, such as a raised seat and handrail for the toilet, would be beneficial.

ASSIGNMENTS

DO A PRESENTATION ON

1. ALZHEIMER'S DISEASE
2. DEMENTIA

INFECTIOUS NEUROLOGIC DISORDERS

The infectious disorders of the nervous system include meningitis, brain abscesses, various types of encephalitis, and Creutzfeldt-Jakob and variant Creutzfeldt-Jakob disease.

The clinical manifestations, assessment, and diagnostic findings as well as the medical and nursing management are related to the specific infectious process.

MENINGITIS IS AS DISCUSSED DURING INFECTIOUS DISEASES

BRAIN ABSCESS

Brain abscesses account for less than 2% of space-occupying brain lesions in the United States and are more common in males during the first two decades of life.

Brain abscesses are rare in immunocompetent people; they are more frequently diagnosed in people who are immunosuppressed as a result of an underlying disease or use of immunosuppressive mediations.

Pathophysiology

A brain abscess is a collection of infectious material within the tissue of the brain. Bacteria are the most common causative organisms. The most common predisposing conditions for abscesses among immunocompetent adults are otitis media and rhinosinusitis.

An abscess can result from intracranial surgery, penetrating head injury, or tongue piercing. Organisms causing brain abscess may reach the brain by hematologic spread from the lungs, gums, tongue, or heart, or from a wound or intra-abdominal infection.

Brain abscesses in immunocompromised people may result from various pathogens. To prevent brain abscess, otitis media, mastoiditis, rhinosinusitis, dental infections, and systemic infections should be treated promptly.

Clinical Manifestations

The clinical manifestations of a brain abscess result from alterations in intracranial dynamics (oedema, brain shift), infection, or the location of the abscess. Headache, usually worse in the morning, is the most prevailing symptom. Fever, vomiting, and focal neurologic deficits occur as

well. Focal deficits such as weakness and decreasing vision reflect the area of brain that is involved.

As the abscess expands, symptoms of increased ICP such as decreasing LOC and seizures are observed.

Assessment and Diagnostic Findings

- MRI
- CT Scan
- Aspiration of the abscess
- Blood culture
- EEG

Medical Management

I. Treatment is aimed at controlling increased ICP, draining the abscess, and providing antimicrobial therapy directed at the abscess and the primary source of infection.

II. Large IV doses of antibiotics are administered to penetrate the blood–brain barrier and reach the abscess. The choice of the specific antibiotic medication is based on culture and sensitivity testing and directed at the causative organism.

III. A stereotactic CT-guided aspiration may be used to drain the abscess and identify the causative organism.

IV. Corticosteroids may be prescribed to help reduce the inflammatory cerebral edema if the patient shows evidence of an increasing neurologic deficit.

v. Antiseizure medications (phenytoin, phenobarbital) may be prescribed to prevent or treat seizures.

Nursing Management

- ✓ Assessment of neurologic function is key to monitoring the progression of disease.
- ✓ Comfort measures to reduce headache include dimming the lights, limiting noise and visitors, grouping nursing interventions, and administering analgesic agents.
- ✓ Opioid analgesic medications may mask neurologic symptoms; therefore, they are used cautiously.
- ✓ Seizures and altered LOC require care directed at injury prevention and safety.

- ✓ Nursing care addressing patient and family anxieties is ongoing throughout the illness. Monitoring of blood chemistry test results and urinary output alert the nurse to the presence of renal complications related to antiviral therapy.

ARTHROPOD –BORNE VIRUS ENCEPHALITIS

Arthropod vectors transmit several types of viruses that cause encephalitis. The primary vector in North America is the mosquito.

In cases ofWest Nile virus, humans are the secondary host; birds are the primary host.

Arbovirus infection (transmitted by arthropod vectors) occurs in specific geographic areas during the summer and fall.

Pathophysiology

Viral replication occurs at the site of the mosquito bite. The host immune response attempts to control viral replication.

If the immune response is inadequate, viraemia will ensue. The virus gains access to the central nervous system (CNS) via the cerebral capillaries, resulting in encephalitis. It spreads from neuron to neuron, predominantly affecting the cortical gray matter, the brain stem, and the thalamus.

Meningeal exudates compound the clinical presentation by irritating the meninges and increasing ICP.

Clinical Manifestations

St. Louis and West Nile encephalitis most commonly affect adults. Climate, an environment conducive to arthropod proliferation, and human behavior contribute to the occurrence of St. Louis and West Nile encephalitis.

An arboviral encephalitis begins with early flulike symptoms, but specific neurologic manifestations depend on the viral type.

A unique clinical feature of St. Louis encephalitis is SIADH with hyponatremia.

Signs and symptoms specific to West Nile encephalitis include a maculopapular or morbilliform rash on the neck, trunk, and arms; enlarged lymph nodes and legs; and flaccid paralysis

Both West Nile and St. Louis encephalitis can result in parkinsonian like move movements, reflecting inflammation of the basal ganglia.

Seizures, a poor prognostic indicator, are present in both types of encephalitis but are more common in the St. Louis type.

Assessment and Diagnostic Findings

1. After a brief febrile prodrome, neurologic symptoms reflect the area of the brain that is involved. Neuroimaging and CSF evaluation are useful in the diagnosis of arboviral encephalitis.
2. The MRI scan demonstrates inflammation of the basal ganglia in cases of St. Louis encephalitis and inflammation in the periventricular area in cases of West Nile encephalitis.
3. Immunoglobulin M antibodies to West Nile virus are observed in serum and CSF.
4. Serum cultures are not useful, because the viraemia is brief.
5. Polymerase chain reaction (PCR) evaluation of CSF may demonstrate viral ribonucleic acid (RNA).

Medical Management

No specific medication for arboviral encephalitis includes controlling the seizures and the increased ICP. Interferon may be useful in treating St. Louis encephalitis.

Ribavirin and interferon alpha-2b show some effect against West Nile virus but have not been evaluated in controlled studies

Neuropsychiatric complications, such as emotional outbursts and other behavior changes, occur frequently.

Nursing Management

A. If the patient is very ill, hospitalization may be required.
B. The nurse carefully assesses neurologic status and identifies improvement or deterioration in the patient's condition.
C. Injury prevention is key in light of the potential for falls or seizures. Arboviral encephalitis may result in death or lifelong residual health issues such as neurologic deficits and Seizures.

D. The family will need support and teaching to cope with these outcomes.
E. Public education addressing the prevention of arboviral encephalitis is a key nursing role.
F. Clothing that provides coverage and insect repellents containing 25% to 30% diethyltoluamide (DEET) should be used on exposed clothing and skin in high-risk areas to decrease mosquito and tick bites.
G. Screens should be in good repair in the home, and standing water should be removed.
H. Blood donation centers screen all blood for West Nile virus.
I. Cases of West Nile virus must be reported to the CDC.

READ UP FUNGAL ENCEPHALITIS

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Creutzfeldt-Jakob Disease

Creutzfeldt-Jakob disease (CJD) and variant Creutzfeldt- Jakob disease (vCJD) belong to a group of degenerative, infectious neurologic disorders called transmissible spongiform encephalopathies (TSE). CJD is very rare and has no identifiable cause.

vCJD is the human variation of bovine spongiform encephalopathy (BSE); it results from the ingestion

by humans of prions in infected beef.

TSEs are caused by **prions,** proteinaceous particles that are smallerthan a virus and are resistant to standard methods of sterilization.

Although CJD and vCJD have distinct clinical features, one characteristic they share is a lack of CNS inflammation.

CJD may lie dormant for decades before causing neurologic degeneration.

The incubation period of Vcjd seems to be shorter (less than 10 years). It is not known whether an increased number of cases will appear in the future.

In both diseases, the symptoms are progressive, there is no definitive treatment, and the outcome is fatal.

Pathophysiology

The prion is a unique pathogen because it lacks nucleic acid, which enables the organism to withstand conventional means of sterilization. How the prion replicates in the absence of nucleic acid is unknown.

In both CJD and vCJD, the prion crosses the blood–brain barrier and is deposited in brain tissue and causes degeneration of brain tissue. Cell death occurs, and spongy vacuoles are produced in the brain (**spongiform** changes). The spongiform vacuoles are surrounded by amyloid plaque.

The mode of transmission was linked to the ingestion of beef contaminated with neurologic tissue.

The prion exists in lymphoid tissue and blood in both vCJD and CJD. Both prion diseases are believed to be blood borne.

Clinical Manifestations

- CJD and vCJD have several clinically distinct features.
- Psychiatric symptoms occur early in vCJD, whereas they are a late symptom in CJD.
- The mean age at onset of vCJD is 27 years, whereas the mean age for CJD onset is 50 years.
- The presenting symptoms of vCJD include affective symptoms (ie, behavioral changes), sensory disturbance, and limb pain.
- Muscle spasms and rigidity, dysarthria, incoordination, cognitive impairment, and sleep disturbances follow.
- Patients with sporadic CJD present with mental deterioration, ataxia, and visual disturbance.
- Memory loss, involuntary movement, paralysis, and mutism occur as the disease progresses.

Assessment and Diagnostic Findings

Historically, brain biopsy was used to diagnose CJD. The three diagnostic tests currently used in suspicious clinical presentations to support the diagnosis of CJD are

- Immunologic assessment,
- Electroencephalography, and

- MRI scanning.
- Immunologic assessment of CSF detects a protein kinase inhibitor called 14-3-3. The presence of this inhibitor indicates neuronal cell death, which is not specific to CJD but does support the diagnosis.
- The EEG reveals a characteristic pattern over the duration of the disease. After initial slowing, the EEG shows periodic activity. Later in the course of the disease, the EEG shows burst-suppressions characterized by periodic spikes alternating with slow periods.
- The MRI scan demonstrates symmetric or unilateral hyper-intense signals arising from the basal ganglia.
- Patients with vCJD do not demonstrate EEG or CSF changes, and the MRI scan shows bilateral hyperintensity of the posterior thalamus.
- The prion associated with vCJD has been shown to accumulate in the tonsils and other lymphoreticular tissues; therefore, tonsillar biopsy may be used in the diagnosis of vCJD.

Medical Management

After the onset of specific neurologic symptoms, progression of disease occurs quickly. There is no effective treatment for CJD or vCJD.

The care of the patient is supportive and palliative.

Goals of care include prevention of injury related to immobility and dementia, promotion of patient comfort, and provision of support and education for the family.

Nursing Management

- The nursing care of patients is primarily supportive and palliative.
- Psychological and emotional support of the patient and family throughout the course of the illness is needed.
- Care extends to providing for a dignified death and supporting the family through the processes of grief and loss.
- Hospice services are appropriate either at home or at an inpatient facility.
- Prevention of disease transmission is an important part of nursing care.

- Although patient isolation is not necessary, use of standard precautions is important. Institutional protocols are followed for blood and body fluid exposure and decontamination of equipment.
- In the operating room, it is recommended that disposable instruments be used and then incinerated, because conventional methods of sterilization do not destroy the prion.

The World Health Organization has guidelines that outline the stringent sterilization methods that must be used to destroy prions on surfaces.

TRAUMA OF THE NERVOUS SYSTEM

Trauma involving the central nervous system can be life threatening. Even if it is not life-threatening, brain and spinal cord injury may result in major physical and psychological dysfunction and can alter the patient's life completely.

Neurologic trauma affects the patient, the family, the health care system, and society as a whole because of its major sequelae and the costs of acute and long-term care of patients with trauma to the brain and spinal cord.

HEAD INJURIES

Head injury is a broad classification that includes injury to the scalp, skull, or brain. It is estimated that 1.4 million people sustain a head injury each year in the United States, and approximately 50,000 people die, 235,000 are hospitalized, and 1.1 million are treated and released from an emergency department.

A head injury may lead to conditions ranging from mild concussion to coma and death; the most serious form is known as a traumatic brain injury (TBI).

The most common causes of TBIs are falls (28%), motor vehicle crashes (20%), being struck by objects (19%), and assaults (11%).

People at highest risk for TBI are those in the 15- to 19-year age group. Males are twice as likely as females to sustain a TBI. Adults 75 years of age or older have the highest TBI related hospitalization and death rates, and African Americans also have high mortality rates.

Pathophysiology

Research suggests that not all brain damage occurs at the moment of impact. Damage to the brain from traumatic injury takes two forms: primary injury and secondary injury.

A. **Primary injury** is the initial damage to the brain that results from the traumatic event. This may include contusions, lacerations, and torn blood vessels due to impact, acceleration/deceleration, or foreign object penetration.
B. **Secondary injury** evolves over the ensuing hours and days after the initial injury and results from inadequate delivery of nutrients and oxygen to the cells.

The cranial vault contains three main components: brain, blood, and cerebrospinal fluid (CSF). According to the Monro-Kellie doctrine, the cranial vault is a closed system, and if one of the three components increases in volume, at least one of the other two must decrease in volume, or the pressure increases. Any bleeding or swelling within the skull, increases the volume of its contents and therefore causes increased intracranial pressure (ICP). If the pressure increases enough, it can cause displacement of the brain through or against the rigid structures of the skull. This causes restriction of blood flow to the brain, decreasing oxygen delivery and waste removal. Cells within the brain become anoxic and cannot metabolize properly, producing ischaemia, infarction, irreversible brain damage, and, eventually, brain death.

In summary,

- Brain suffers traumatic injury, Brain swelling or bleeding increases intracranial volume
- Rigid cranium allows no room for expansion of contents so intracranial pressure increases
- Pressure on blood vessels within the brain causes blood flow to the brain to slow
- Intracranial pressure continues to rise.
- Brain may herniate
- Cerebral hypoxia and ischaemia occur
- Cerebral blood flow ceases
- Brain cellular deaths occur.

Other forms of trauma to the nervous system include

Scalp Injury

Isolated scalp trauma is generally classified as a minor injury. Because its many blood vessels constrict poorly, the scalp bleeds profusely when injured. Trauma may result in an abrasion (brush wound), contusion, laceration, or hematoma beneath the layers of tissue of the scalp (subgaleal hematoma).

A large avulsion (tearing away) of the scalp may be potentially life-threatening and is a true emergency. Scalp wounds are potential portals of entry for organisms that cause intracranial infections.

Therefore, the area is irrigated before the laceration is sutured, to remove foreign material and to reduce the risk for infection. Subgaleal hematomas (hematomas below the outer covering of the skull) usually reabsorb and do not require any specific treatment.

Skull Fractures

A skull fracture is a break in the continuity of the skull caused by forceful trauma. It may occur with or without damage to the brain.

Skull fractures can be classified as simple, comminuted, depressed, or basilar.

A **simple (linear**) fracture is a break in the continuity of the bone.

A **comminuted** skull fracture refers to a splintered or multiple fracture line.

Depressed skull fractures occur when the bones of the skull are forcefully displaced downward and can vary from a slight depression to bones of the skull being splintered and embedded within brain tissue.

A fracture of the base of the skull is called **a basilar skull fracture**.

A fracture may be open, indicating a scalp laceration or tear in the Dura (e.g., from a bullet or an ice pick), or closed, in which case the Dura is intact.

Signs and Symptoms

Symptoms, apart from those of the local injury, depend on the severity and the anatomic location of the underlying brain injury. Persistent, localized pain usually suggests that a fracture is present. Fractures of the cranial vault may or may not produce swelling in the region of the fracture; therefore, an x-ray is needed for diagnosis.

Fractures of the base of the skull tend to traverse the paranasal sinus of the frontal bone or the middle ear located in the temporal bone. Therefore, they frequently produce haemorrhage from the nose, pharynx, or ears, and blood may appear under the conjunctiva. An area of ecchymosis (bruising) may be seen over the mastoid (Battle's sign). Basilar skull fractures are suspected when CSF escapes from the ears (CSF otorrhoea) and the nose (CSF rhinorrhoea). Drainage of CSF is a serious problem, because meningeal infection can occur if organisms gain access to the cranial contents via the nose, ear, or sinus through a tear in the Dura.

Assessment of brain trauma

Be alert for the following signs and symptoms:

• Altered level of consciousness

• Confusion

• Pupillary abnormalities (changes in shape, size, and response to light)

• Altered or absent gag reflex

• Absent corneal reflex

• Sudden onset of neurologic deficits

• Changes in vital signs (altered respiratory pattern, widened pulse pressure, bradycardia, tachycardia, hypothermia, or hyperthermia)

• Vision and hearing impairment

• Sensory dysfunction

• Headache

• Seizures

Diagnostic Findings

- ✓ **X-rays** confirm the presence and extent of a skull fracture
- ✓ **A rapid physical examination** and evaluation of neurologic status detects obvious brain injuries.
- ✓ **Computed tomography** (CT) scan
- ✓ **Magnetic resonance imaging (MRI)** is used to evaluate patients with head injury when a more accurate picture of the anatomic nature of the injury is warranted and when the patient is stable enough to undergo this longer diagnostic procedure.
- ✓ **Cerebral angiography** may also be used to identify supratentorial, extracerebral, and intracerebral hematomas and cerebral contusions.

BRAIN INJURY

The most important consideration in any head injury is whether the brain is injured. Even seemingly minor injury can cause significant brain damage secondary to obstructed blood flow and decreased tissue perfusion. The brain cannot store oxygen or glucose to any significant degree. Because the cerebral cells need an uninterrupted blood supply to obtain these nutrients, irreversible brain damage and cell death occur if the blood supply is interrupted for even a few minutes. Clinical manifestations of **brain injury** (injury to the brain that is severe enough to interfere with normal functioning)

Closed (blunt) brain injury occurs when the head accelerates and then rapidly decelerates or collides with another object (eg, a wall, the dashboard of a car) and brain tissue is damaged but there is no opening through the skull and dura.

Open brain injury occurs when an object penetrates the skull, enters the brain, and damages the soft brain tissue in its path (penetrating injury), or when blunt trauma to the head is so severe that it opens the scalp, skull, and dura to expose the brain.

Concussion

A **concussion** after head injury is a temporary loss of neurologic function with no apparent structural damage. A concussion (also referred to as a mild TBI) may or may not produce a brief loss of consciousness. The mechanism of injury is usually blunt trauma from an acceleration-deceleration force, a direct blow, or a blast injury.

If brain tissue in the frontal lobe is affected, the patient may exhibit bizarre irrational behaviour, whereas involvement of the temporal lobe can produce temporary amnesia or disorientation.

There are two types of concussion: **mild** and **classic**.

1. A mild concussion may lead to a period of observed or self-reported transient confusion, disorientation, or impaired consciousness. Commonly, there is a memory lapse at the time of injury and a loss of consciousness lasting less than 30 minutes. Other signs and symptoms of neurologic or neuropsychological dysfunction may include seizures, headache, dizziness, irritability, fatigue, or poor concentration.
2. A classic concussion is an injury that results in a loss of consciousness; characteristically, this usually lasts less than 6 hours. This loss of consciousness is always accompanied by some degree of posttraumatic amnesia. Diagnostic studies may show no apparent structural sign of injury, but the duration of unconsciousness is an indicator of the severity of the concussion.

The patient may be hospitalized overnight for observation or discharged from the hospital in a relatively short time after a concussion. Monitoring includes observing the patient for headache, dizziness, lethargy, irritability, emotional ability, fatigue, poor concentration, decreased attention span, memory difficulties, and intellectual dysfunction that may occur from 1 week to 1 year after the initial injury.

The occurrence of these symptoms after injury is referred to as post-concussion syndrome. Recovery may appear complete, but long-term sequelae are possible.

Contusion

In cerebral **contusion,** a moderate to severe head injury, the brain is bruised and damaged in a specific area because of severe acceleration-deceleration force or blunt trauma. The impact of the brain against the skull leads to a contusion.

Although a contusion may occur in any area of the brain, most are usually located in the anterior portions of the frontal and temporal lobes, around the sylvian fissure, at the orbital areas, and, less commonly, at the parietal and occipital areas.

Contusions are characterized by loss of consciousness associated with stupor and confusion. Other characteristics can include tissue alteration and neurologic deficit without haematoma formation, alteration in consciousness without localizing signs, and haemorrhage into the tissue that varies in size and is surrounded by edema. The effects of injury (haemorrhage and oedema) peak after about 18 to 36 hours.

Patient outcome depends on the area and severity of the injury.

Temporal lobe contusions carry a greater risk of swelling, rapid deterioration, and brain herniation. Deep contusions are more often associated with haemorrhage and destruction of the reticular activating fibers altering arousal

Diffuse Axonal Injury

Diffuse axonal injury (DAI) results from widespread shearing and rotational forces that produce damage throughout the brain—to axons in the cerebral hemispheres, corpus callosum, and brain stem. The injured area may be diffuse, with no identifiable focal lesion.

DAI is associated with prolonged traumatic coma; it is more serious and is associated with a poorer prognosis than a focal lesion or ischaemia.

Diagnosis:

This is made by clinical signs in conjunction with a CT or MRI scan. Recovery depends on the severity of the axonal injury.

Intracranial Haemorrhage

Hematomas are collections of blood in the brain that may be epidural (above the Dura), subdural (below the Dura), or intra-cerebral (within the brain)

Major symptoms are frequently delayed until the hematoma is large enough to cause distortion of the brain and increased ICP.

The signs and symptoms of cerebral ischemia resulting from compression by a hematoma are variable and depend on the speed with which vital areas are affected and the area that is injured

In general, a rapidly developing haematoma, even if small, may be fatal, whereas a larger but slowly developing one may allow compensation for increases in ICP.

Epidural Haematoma

After a head injury, blood may collect in the epidural (extradural) space between the skull and the dura mater. This can result from a skull fracture that causes a rupture or laceration of the middle meningeal artery, the artery that runs between the dura and the skull inferior to a thin portion of temporal bone. Haemorrhage from this artery causes rapid pressure on the brain.

Epidural haematomas are often characterized by a brief loss of consciousness followed by a lucid interval in which the patient is awake and conversant.

During this lucid interval, compensation for the expanding haematoma takes place by rapid absorption of CSF and decreased intravascular volume, both of which help maintain a normal ICP. When these mechanisms can no longer compensate, even a small increase in the volume of the blood clot produces a marked elevation in ICP. The patient then becomes increasingly restless, agitated, and confused as the condition progresses to coma. Then, often suddenly, signs of herniation appear (usually deterioration of consciousness and signs of focal neurologic deficits, such as dilation and fixation of a pupil or paralysis of an extremity), and the patient's condition deteriorates rapidly.

The most common type of herniation syndrome associated with an epidural hematoma is **uncal Herniation** An epidural hematoma is considered an extreme emergency because marked neurologic deficit or even respiratory arrest can occur within minutes. Treatment consists of making openings through the skull (burr holes) to decrease ICP emergently, remove the clot, and control the bleeding.

A craniotomy may be required to remove the clot and control the bleeding. A drain is usually inserted after creation of burr holes or a craniotomy to prevent re-accumulation of blood.

Subdural Haematoma

A subdural hematoma is a collection of blood between the **Dura** and the brain, a space normally occupied by a thin cushion of fluid. The most common cause of subdural haematoma is trauma, but it can also occur as a result of coagulopathies or rupture of an aneurysm. A subdural haemorrhage is more frequently venous in origin and is caused by the rupture of small vessels that bridge the subdural space.

The subdural haematoma that results may be acute, subacute, or chronic, depending on the size of the involved vessel and the amount of bleeding.

Acute and Subacute Subdural Haematoma: Acute subdural haematomas are associated with major head injury involving contusion or laceration. Clinical symptoms develop over 24 to 48 hours. Signs and symptoms include changes in the level of consciousness (LOC), pupillary signs, and hemiparesis.

There may be minor or even no symptoms with small collections of blood. Coma, increasing blood pressure, decreasing heart rate, and slowing respiratory rate are all signs of a rapidly expanding mass requiring immediate intervention.

Subacute subdural haematomas are the result of less severe contusions and head trauma. Clinical manifestations usually appear between 48 hours and 2 weeks after the injury.

Signs and symptoms are similar to those of an acute subdural haematoma.

If the patient can be transported rapidly to the hospital, an immediate craniotomy is performed to open the dura, allowing the subdural clot to be evacuated. Successful outcome also depends on the control of ICP and careful monitoring of respiratory.

The mortality rate for patients with acute or subacute subdural hematoma is high because of associated brain damage.

Chronic Subdural Haematoma: Chronic subdural hematomas can develop from seemingly minor head injuries and are seen most frequently in the elderly. The elderly are prone to this type of head injury secondary to brain atrophy, which is a frequent consequence of the aging process.

Seemingly minor head trauma may produce enough impact to shift the brain contents abnormally. The time between injury and onset of symptoms can be lengthy (e.g. 3 weeks to months), so the actual injury may be forgotten.

A chronic subdural haematoma can resemble other conditions; for example, it may be mistaken for a stroke. The bleeding is less profuse, but compression of the intracranial contents still occurs. The blood within the brain changes in character in 2 to 4 days, becoming thicker and darker. In a few weeks, the clot breaks down and has the color and consistency of motor oil. Eventually, **calcification or ossification** of the clot takes place. The brain adapts to this foreign body invasion, and the clinical signs and symptoms fluctuate.

Symptoms include severe headache, which tends to come and go; alternating focal neurologic signs; personality changes; mental deterioration; and focal seizures. The patient may be labeled neurotic or psychotic if the cause is overlooked.

The treatment of a chronic subdural haematoma consists of **surgical evacuation** of the clot. The procedure may be carried out through multiple **burr holes**, or **a craniotomy** may be performed for a sizable subdural mass that cannot be suctioned or drained through burr holes.

Intra-cerebral Haemorrhage and Haematoma

Intra-cerebral hemorrhage is bleeding into the substance of the brain. It is commonly seen in head injuries when force is exerted to the head over a small area (eg, missile injuries, bullet wounds, stab injuries). These hemorrhages within the brain may also result from the following:

- Systemic hypertension, which causes degeneration and rupture of a vessel
- Rupture of a saccular aneurysm
- Vascular anomalies
- Intracranial tumors
- Bleeding disorders such as leukemia, hemophilia, aplastic anaemia, and thrombocytopenia
- Complications of anticoagulant therapy

Management includes

- ✓ Supportive care
- ✓ Control of ICP
- ✓ Careful administration of fluids, electrolytes, and antihypertensive medications

- ✓ Surgical intervention is by craniotomy

Management of Brain Injuries

1. Assessment and diagnosis of the extent of injury are accomplished by the initial physical and neurologic examinations.
2. CT and MRI scans are the primary neuro-imaging diagnostic tools and are useful in evaluating the brain structure.
3. Positron emission tomography (PET) is available in some trauma centers for assessing brain function.
4. Any patient with a head injury is presumed to have a cervical spine injury until proven otherwise. The patient is transported from the scene of the injury on a board with the head and neck maintained in alignment with the axis of the body.
5. A cervical collar should be applied and maintained until cervical spine x-rays have been obtained and the absence of cervical spinal cord injury documented.
6. All therapy is directed toward preserving brain homeostasis and preventing secondary brain injury, which is injury to the brain that occurs after the original traumatic.
7. Common causes of secondary injury are cerebral oedema, hypotension, and respiratory depression that may lead to hypoxaemia and electrolyte imbalance.
8. Treatments to prevent secondary injury include: stabilization of cardiovascular and respiratory function to maintain adequate cerebral perfusion, control of haemorrhage and hypovolaemia, and maintenance of optimal blood gas values.

Treatment of Increased Intracranial Pressure

As the damaged brain swells with oedema or as blood collects within the brain, an increase in ICP occurs; this requires aggressive treatment.

If the ICP remains elevated, it can decrease the CPP. Initial management is based on the principle of preventing secondary injury and maintaining adequate cerebral oxygenation

Surgery is required for evacuation of blood clots, débridement and elevation of depressed fractures of the skull, and suture of severe scalp lacerations.

ICP is monitored closely; if increased, it is managed by maintaining adequate oxygenation, elevating the head of the bed, and maintaining normal blood volume. Devices to monitor ICP or drain CSF can be inserted during surgery or at the bedside and management of pain and anxiety.
Comatose patients are intubated and mechanically ventilated to ensure adequate oxygenation and protect the airway.
Because seizures can occur after head injury and can cause secondary brain damage from hypoxia, antiseizure agents may be administered.
If the patient is very agitated, benzodiazepines may be prescribed to calm the patient without decreasing LOC. These medications do not affect ICP, making them good choices for the patient with head injury.
A nasogastric tube may be inserted, because reduced gastric motility and reverse peristalsis are associated with head injury, making regurgitation and aspiration common in the first few hours.

Brain Death

When a patient has sustained a severe head injury incompatible with life, the patient is a potential organ donor. The nurse may assist in the clinical examination for determination of brain death and in the process of organ procurement.
All 50 states recognize the Uniform Determination of Brain Death Act that states death will be determined by accepted medical standards and indicates irreversible loss of all brain function, including the brain stem. The three cardinal signs of brain death on clinical examination are coma, the absence of brain stem reflexes, and apnoea.
Adjunctive tests, such as cerebral blood flow studies, electroencephalogram (EEG), transcranial Doppler, and brain stem auditory evoked potential, is often used to confirm brain death.

NURSING PROCESS OF PATIENT WITH A TRAUMATIC BRAIN INJURY

Assessment

Depending on the patient's neurologic status, the nurse may elicit information from the patient, from the family, or from witnesses or emergency rescue personnel. Although all usual baseline data may not be collected initially, the immediate health history should include the following questions:

- ✓ When did the injury occur?

- ✓ What caused the injury?
- ✓ A high-velocity missile?
- ✓ An object striking the head?
- ✓ A fall?
- ✓ What was the direction and force of the blow?

A history of unconsciousness or amnesia after a head injury indicates a significant degree of brain damage, and changes that occur minutes to hours after the initial injury can reflect recovery or indicate the development of secondary brain damage. The nurse should determine if there was a loss of consciousness, the duration of the unconscious period, and if the patient could be aroused.

In addition to asking questions that establish the nature of the injury and the patient's condition immediately after the injury, the nurse examines the patient thoroughly. This assessment includes determining the patient's LOC using the Glasgow Coma Scale (GCS) and assessing the patient's response to tactile stimuli (if unconscious), pupillary response to light, corneal and gag reflexes, and motor function.

The GCS is based on the three criteria of eye opening, verbal responses, and motor responses to verbal commands or painful stimuli. It is particularly useful for monitoring changes during the acute phase, the first few days after a head injury. It does not take the place of an in-depth neurologic assessment.

The Glasgow Coma Scale is a tool for assessing a patient's response to stimuli. Scores range from 3 (deep coma) to 15 (normal).

Eye opening Spontaneous {4}

Response to voice 3

To pain 2

None 1

Best verbal Oriented {5}

Response Confused 4

Inappropriate words 3

Incomprehensible sounds 2

None 1

Best motor Obeys command {6}

Response Localizes pain 5

Withdraws 4

Flexion 3

Extension 2

None 1

Total 3 to 15

NURSING DIAGNOSES

Based on the assessment data, the patient's major nursing diagnoses may include the following:

a. Ineffective airway clearance and impaired gas exchange related to brain injury
b. Ineffective cerebral tissue perfusion related to increased ICP, and possible seizures
c. Deficient fluid volume related to decreased LOC and hormonal dysfunction
d. Imbalanced nutrition, less than body requirements, related to increased metabolic demands, fluid restriction, and inadequate intake
e. Risk for injury (self-directed and directed at others) related to seizures, disorientation, restlessness, or brain damage
f. Risk for imbalanced body temperature related to damaged temperature-regulating mechanisms in the brain
g. Risk for impaired skin integrity related to bed rest, hemiparesis, hemiplegia, immobility, or restlessness
h. Disturbed thought processes (deficits in intellectual function, communication, memory, information processing) related to brain injury
i. Disturbed sleep pattern related to brain injury and frequent neurologic checks Interrupted family processes related to unresponsiveness of patient, unpredictability of outcome, prolonged recovery period, and the patient's residual physical disability and emotional deficit.
j. Deficient knowledge about brain injury, recovery, and the rehabilitation process

OBJECTIVES / GOALS

The goals for the patient may include

1. Maintenance of a patent airway
2. Adequate cerebral perfusion pressure (CPP)
3. Fluid and electrolyte balance
4. Adequate nutritional status
5. Prevention of secondary injury
6. Maintenance of normal body temperature
7. Maintenance of skin integrity
8. Improvement of cognitive function
9. Prevention of sleep deprivation
10. Effective family coping
11. Increased knowledge about the rehabilitation process, and absence of complications

Nursing Interventions

The nursing interventions for the patient with a head injury are extensive and diverse. They include making nursing assessments, setting priorities for nursing interventions, anticipating needs and complications, and initiating rehabilitation.

1. ***Monitoring Neurologic Function***

The importance of ongoing assessment and monitoring of the patient with brain injury cannot be overstated. The following parameters are assessed initially and as frequently as the patient's condition requires. As soon as the initial assessment is made, the use of a neurologic flow chart is started and maintained.

2. **Level of consciousness**

The GCS is used to assess LOC at regular intervals, because changes in the LOC precede all other change in vital and neurologic signs.

The patient's best responses to predetermined stimuli are recorded. Each response is scored (the greater the number, the better the functioning), and the sum of these scores gives an indication of the severity of coma and a prediction of possible outcome. The lowest score is 3 (least

responsive); the highest is 15 (most responsive). A GCS between 3 and 8 is generally accepted as indicating a severe head injury.

Interventions to ensure an adequate exchange of air are including the following:

• Maintaining the unconscious patient in a position that facilitates drainage of oral secretions, with the head of the bed elevated about 30 degrees to decrease intracranial venous pressure

• Establishing effective suctioning procedures (pulmonary secretions produce coughing and straining, which increase ICP)

• Guarding against aspiration and respiratory insufficiency

• Closely monitoring arterial blood gas values to assess the adequacy of ventilation. The goal is to keep blood gas values within the normal range to ensure adequate cerebral blood flow.

• Monitoring the patient who is receiving mechanical ventilation for pulmonary complications such as acute respiratory distress syndrome (ARDS) and pneumonia

Monitoring Fluid and Electrolyte Balance

Brain damage can produce metabolic and hormonal dysfunctions. The monitoring of serum electrolyte levels is important, especially in patients receiving osmotic diuretics, those with syndrome of inappropriate antidiuretic hormone (SIADH) secretion, and those with posttraumatic diabetes insipidus

Promoting Adequate Nutrition

1. Head injury results in metabolic changes that increase calorie consumption and nitrogen excretion. Protein demand increases. Early initiation of nutritional therapy has been shown to improve outcomes in patients with head injury.
2. Patients with brain injury are assumed to be catabolic and nutritional support consultation should be considered as soon as the patient is admitted.
3. Parenteral nutrition via a central line or enteral feedings administered via a nasogastric or nasojejunal feeding tube should be considered
4. If CSF rhinorrhea occurs, an oral feeding tube should be inserted instead of a nasal tube.

COMPLETE THE REMAINING ONCES

COMPLICATIONS OF TRAUMATIC BRAIN INJURY

Based on all the assessment data, the major complications include the following:

- Decreased cerebral perfusion
- Cerebral edema and herniation
- Impaired oxygenation and ventilation
- Impaired fluid, electrolyte, and nutritional balance
- Risk of posttraumatic seizures

SPINAL CORD INJURY (SCI)

Spinal cord injury (SCI) is a major health disorder. Almost 200,000 people in the United States live each day with a disability from SCI, and an estimated 11,000 new injuries occur each year, SCI is primarily an injury of young adult males and 50% of those injured are between 16 and 30 years of age. Patients over the age of 60 years account for 10% of SCIs, and this figure has steadily risen over the past 25 years.

Causes

a. Motor vehicle crashes account for 48% of reported cases of SCI,
b. Falls (23%),
c. Violence primarily from gunshot wounds (14%),
d. Recreational sporting activities (9%), and
e. Other events accounting for the remaining injuries.

Paraplegia: (paralysis of the lower body) and **tetraplegia** (formerly quadriplegia—paralysis of all four extremities) can occur, with incomplete tetraplegia (formerly quadriplegia) the largest category, followed by complete paraplegia, complete tetraplegia, and paraplegia.

RISK FACTORS

The predominant risk factors for SCI include

- Young age,
- Male gender, and
- Alcohol and drug use.

Pathophysiology

Damage in SCI ranges from transient concussion (from which the patient fully recovers), to contusion, laceration, and compression of the spinal cord substance (either alone or in combination), to complete **transection** (severing) of the spinal cord (which renders the patient paralyzed below the level of the injury).

The vertebrae most frequently involved are the 5th, 6th, and 7th cervical vertebrae (C5 to C7), the 12th thoracic vertebra (T12), and the 1st lumbar vertebra (L1). These vertebrae are most susceptible because there is a greater range of mobility in the vertebral column in these areas.

SCIs can be separated into two categories: primary injuries and secondary injuries. Primary injuries are the result of the initial insult or trauma and are usually permanent.

Secondary injuries are usually the result of a contusion or tear injury, in which the nerve fibers begin to swell and disintegrate.

A secondary chain of events produces ischaemia, hypoxia, oedema, and haemorrhagic lesions, which in turn result in destruction of myelin and axons.

The secondary injury is of primary concern for critical care nurses. Experts believe secondary injury is the principal cause of spinal cord degeneration at the level of injury and that it is reversible during the first 4 to 6 hours after injury.

Clinical Manifestations

Manifestations of SCI depend on the type and level of injury. The type of injury refers to the extent of injury to the spinal cord itself.

Incomplete spinal cord lesions (the sensory or motor fibers, or both, are preserved below the lesion) are classified according to the area of spinal cord damage: central, lateral, anterior, or

peripheral. The American Spinal Injury Association (ASIA) provides classification of SCI according to the degree of sensory and motor function present after injury Neurologic level" refers to the lowest level at which sensory and motor functions are normal. Below the neurologic level, there is total sensory and motor paralysis, loss of bladder and bowel control (usually with urinary retention and bladder distention), loss of sweating and vasomotor tone, and marked reduction of blood pressure from loss of peripheral vascular resistance.

A **complete spinal cord lesion** (total loss of sensation and voluntary muscle control below the lesion) can result in paraplegia or tetraplegia.

If conscious, the patient usually complains of acute pain in the back or neck, which may radiate along the involved nerve. However, absence of pain does not rule out spinal injury, and a careful assessment of the spine should be conducted if there has been a significant force and mechanism of injury (ie, concomitant head injury). Often the patient speaks of fear that the neck or back is broken.

Respiratory dysfunction is related to the level of injury. The muscles contributing to respiration are the abdominals and intercostals (T1 to T11) and the diaphragm (C4). In high cervical cord injury, acute respiratory failure is the leading cause of death.

Assessment and Diagnostic Findings

1. A detailed neurologic examination is performed
2. x-rays (lateral cervical spine x-rays) and
3. CT scanning
4. An MRI scan may be ordered as a further workup if a ligamentous injury is suspected

Emergency Management

The immediate management at the scene of the injury is critical, because improper handling of the patient can cause further damage and loss of neurologic function.

Any patient who is involved in a motor vehicle crash, a diving or contact sports injury, a fall, or any direct trauma to the head and neck must be considered to have SCI until such an injury is ruled out.

Initial care must include a rapid assessment, immobilization, extrication, and stabilization or control of life-threatening injuries, and transportation to the most appropriate medical facility.

Immediate transportation to a trauma center with the capacity to manage major neurologic trauma is then necessary.

At the scene of the injury, the patient must be immobilized on a spinal (back) board, with the head and neck maintained in a neutral position, to prevent an incomplete injury from becoming complete.

One member of the team must assume control of the patient's head to prevent flexion, rotation, or extension; this is done by placing the hands on both sides of the patient's head at about ear level to limit movement and maintain alignment while a spinal board or cervical immobilizing device is applied. If possible, at least four people should slide the patient carefully onto a board for transfer to the hospital.

Any twisting movement may irreversibly damage the spinal cord by causing a bony fragment of the vertebra to cut into, crush, or sever the cord completely.

The standard of care is that the patient is referred to a regional spinal injury or trauma center because of the multidisciplinary personnel and support services required to counteract the destructive changes that occur in the first 24 hours after injury

During treatment in the emergency and x-ray departments, the patient is kept on the transfer board. The patient must always be maintained in an extended position. No part of the body should be twisted or turned, and the patient is not allowed to sit up. Once the extent of the injury has been determined, the patient may be placed on a rotating specialty bed.

Pharmacologic Therapy

Administration of high-dose IV corticosteroids or methylprednisolone sodium succinate in the first 24 or 48 hours is controversial.

Despite the ongoing controversy surrounding the practice, the use of IV high-dose methylprednisolone is accepted as standard therapy for SCI in many countries and remains an established clinical practice in most trauma centers

Respiratory Therapy

Oxygen is administered to maintain a high partial pressure of oxygen (PaO_2), because hypoxemia can create or worsen a neurologic deficit of the spinal cord.

If endotracheal intubation is necessary, extreme care is taken to avoid flexing or extending the patient's neck, which can result in extension of a cervical injury.

Other forms of treatment are

I. TENS
II. Use of traction

NURSING DIAGNOSES

- Ineffective breathing patterns related to weakness or paralysis of abdominal and intercostal muscles and inability to clear secretions
- Ineffective airway clearance related to weakness of intercostals Muscles
- Impaired bed and physical mobility related to motor and sensory impairments
- Disturbed sensory perception related to motor and sensory impairment
- Risk for impaired skin integrity related to immobility and sensory loss
- Impaired urinary elimination related to inability to void spontaneously
- Constipation related to presence of atonic bowel as a result of autonomic disruption
- Acute pain and discomfort related to treatment and prolonged mobility

Goals

The goals for the patient may include

1. Improved breathing pattern and airway clearance
2. Improved mobility
3. Improved sensory and perceptual awareness,
4. Maintenance of skin integrity,
5. Relief of urinary retention
6. Improved bowel function,
7. Promotion of comfort, and
8. Absence of complication.

Nursing Interventions

The patient requires extensive rehabilitation, which is less difficult if appropriate nursing management has been carried out during the acute phase of the injury or illness. Nursing care is one of the key factors determining the success of the rehabilitation program. The main objective is for the patient to live as independently as possible in the home and community.

1. **Promoting Adequate Breathing and Airway Clearance**

Possible impending respiratory failure is detected by observing the patient, measuring vital capacity, monitoring oxygen saturation through pulse oximetry, and monitoring arterial blood gases. Early and vigorous attention to clearing bronchial and pharyngeal secretions can prevent retention of secretions and atelectasis. Suctioning may be indicated, but it should be used with caution to avoid stimulating the vagus nerve and producing bradycardia and cardiac arrest.

If the patient cannot cough effectively because of decreased inspiratory volume and inability to generate sufficient expiratory pressure, chest physical therapy and assisted coughing may be indicated. Specific breathing exercises are supervised by the nurse to increase the strength and endurance of the inspiratory muscles, particularly the diaphragm. Assisted coughing promotes clearing of secretions from the upper respiratory tract and is similar to the use of abdominal thrusts to clear an airway.

Proper humidification and hydration are important to prevent secretions from becoming thick and difficult to remove even with coughing. The patient is assessed for signs of respiratory infection (eg, cough, fever, dyspnoea).

Ascending edema of the spinal cord in the acute phase may cause respiratory difficulty that requires immediate intervention.

Therefore, the patient's respiratory status must be monitored closely.

2. **Increasing Mobility**

EXERCISE PROGRAMS. The unaffected parts of the body are built up to optimal strength to promote maximal self care. The muscles of the hands, arms, shoulders, chest, spine, abdomen, and neck must be strengthened in the patient with paraplegia, because he or she must bear full weight on these muscles to ambulate. The triceps and the latissimus dorsi are important muscles used in crutch walking.

The muscles of the abdomen and the back also are necessary for balance and for maintaining the upright position. To strengthen these muscles, the patient can do push-ups when in a prone

position and sit-ups when in a sitting position. Extending the arms while holding weights (traction weights can be used) also develop muscle strength.

Squeezing rubber balls or crumbling newspaper promotes hand strength.

With encouragement from all members of the rehabilitation team, the patient with paraplegia can develop the increased exercise tolerance needed for gait training and ambulation activities. The importance of maintaining cardiovascular fitness is stressed to the patient. Alternative exercises to increase the heart rate to target levels must be designed within the patient's abilities.

MOBILIZATION: After the spine is stable enough to allow the patient to assume an upright posture, mobilization activities are initiated. A brace or vest may be used, depending on the level of the lesion.

A patient whose paralysis is a result of complete transection of the cord can begin weight bearing early, because no further damage can be incurred.

The sooner muscles are used; the less chance there is of disuse atrophy. The earlier the patient is brought to a standing position, the less opportunity there is for osteoporotic changes to take place in the long bones. Weight bearing also reduces the possibility of renal calculi and enhances many other metabolic processes.

Braces and crutches enable some patients with paraplegia to ambulate for short distances. Ambulation using crutches requires a high expenditure of energy.

Motorized wheelchairs and specially equipped vans can provide greater independence and mobility for patients with high-level SCI or other lesions. Every effort should be made to encourage the patient to be as mobile and active as possible.

3. **Preventing Disuse Syndrome**

Patients are at high risk for development of contractures as a result of disuse syndrome due to the musculoskeletal system changes (atrophy) brought about by the loss of motor and sensory functions below the level of injury.

Range-of motion exercises must be provided at least four times a day, and care is taken to stretch the Achilles tendon with exercises.

The patient is repositioned frequently and is maintained in proper body alignment whether in bed or in a wheel chair.

4. **Promoting Skin Integrity**

Because these patients spend a great portion of their lives in wheelchairs, pressure ulcers are an ever-present threat.

Contributing factors are permanent sensory loss over pressure areas; immobility, which makes relief of pressure difficult; trauma from bumps (against the wheelchair, toilet, furniture, and so forth) that cause unnoticed abrasions and wounds; loss of protective function of the skin from excoriation and maceration due to excessive perspiration and possible incontinence; and poor general health (anemia, oedema, malnutrition), leading to poor tissue perfusion.

The person with tetraplegia or paraplegia must take responsibility for monitoring (or directing monitoring) of his or her skin status. This involves relieving pressure and not remaining in any position for longer than 2 hours, in addition to ensuring that the skin receives meticulous attention and cleansing.

The patient is taught that ulcers develop over bony prominences that are exposed to unrelieved pressure in the lying and sitting positions. The most vulnerable areas are identified.

The patient with paraplegia is instructed to use mirrors, if possible, to inspect these areas morning and night, observing for redness, slight edema, or any abrasions. While in bed, the patient should turn at 2- hour intervals and then inspect the skin again for redness that does not fade on pressure.

The bottom sheet should be checked for wetness and for creases.

The patient with tetraplegia or paraplegia who cannot perform these activities is encouraged to direct others to check these areas and prevent ulcers from developing.

The patient is taught to relieve pressure while in the wheelchair by doing push-ups, leaning from side to side to relieve ischial pressure, and tilting forward while leaning on a table. The caregiver for the patient with tetraplegia will need to perform these activities if the patient cannot do so independently.

A wheelchair cushion is prescribed to meet individual needs, which may change in time with changes in posture, weight, and skin tolerance.

A referral can be made to a rehabilitation engineer, who can measure pressure levels while the patient is sitting and then tailor the cushion and other necessary aids and assistive devices to the patient’s needs.

The diet for the patient with tetraplegia or paraplegia should be high in protein, vitamins, and calories to ensure minimal wasting of muscle and the maintenance of healthy skin, and high in fluids to maintain well-functioning kidneys.

Excessive weight gain and obesity should be avoided, because they further limit mobility.

5. **Maintaining Urinary Elimination**

Immediately after SCI, the urinary bladder becomes atonic and cannot contract by reflex activity. Urinary retention is the immediate result. Because the patient has no sensation of bladder distention, overstretching of the bladder and detrusor muscle may occur, delaying the return of bladder function.

Intermittent catheterization is carried out to avoid over-distention of the bladder and UTI. If this is not feasible, an indwelling catheter is inserted temporarily. At an early stage, family members are shown how to carry out intermittent catheterization and are encouraged to participate in this facet of care, because they will be involved in long term follow-up and must be able to recognize complications so that treatment can be instituted.

The patient is taught to record fluid intake, voiding pattern, and amount of residual urine after catheterization, characteristics of urine, and any unusual sensations that may occur.

6. **Improving Bowel Function**

Immediately after SCI, a paralytic ileus usually develops as a result of neurogenic paralysis of the bowel; therefore, a nasogastric tube is often required to relieve distention and to prevent vomiting and aspiration.

Bowel activity usually returns within the first week.

As soon as bowel sounds are heard on auscultation, the patient is given a high-calorie, high-protein, high-fiber diet, with the amount of food gradually increased.

The nurse administers prescribed stool softeners to counteract the effects of immobility and analgesic agents. A bowel program is instituted as early as possible.

Monitoring and Managing Potential Complications

Thrombophlebitis is a relatively common complication in patients after SCI. The patient must be assessed for symptoms of thrombophlebitis and PE.

Chest pain, shortness of breath, and changes in arterial blood gas values must be reported promptly to the physician.

The circumferences of the thighs and calves are measured and recorded daily; further diagnostic studies are performed if a significant increase is noted. Patients remain at high risk for thrombophlebitis for several months after the initial injury. Patients with paraplegia or tetraplegia are at increased risk for the rest of their lives.

Immobilization and the associated venous stasis, as well as varying degrees of autonomic disruption, contribute to the high risk and susceptibility deep venous thrombosis (DVT).

Anticoagulation is initiated once head injury and other systemic injuries have been ruled out. Low-dose fractionated or unfractionated heparin may be followed by long term oral anticoagulation (ie, warfarin) or subcutaneous fractionated heparin injections. Additional measures such as range-of-motion exercises, anti-embolism stockings, and adequate hydration are important preventive measures.

Pneumatic compression devices may also be used to reduce venous pooling and promote venous return. It is also important to avoid external pressure on the lower extremities that may result from flexion of the knees while the patient is in bed.

ORTHOSTATIC HYPOTENSION: For the first 2 weeks after SCI, the blood pressure tends to be unstable and quite low.

It gradually returns to preinjury levels, but periodic episodes of severe orthostatic hypotension frequently interfere with efforts to mobilize the patient. Interruption in the reflex arcs that normally produce vasoconstriction in the upright position, coupled with vasodilatation and pooling in abdominal and lower extremity vessels, can result in blood pressure readings of 40 mm Hg systolic and 0 mm Hg diastolic. Orthostatic hypotension is a particularly common problem for patients with lesions above T7.

In some patients with tetraplegia, even slight elevations of the head can result in dramatic decreases in blood pressure.

A number of techniques can be used to reduce the frequency of hypotensive episodes. Close monitoring of vital signs before and during position changes is essential. Vasopressor medication can be used to treat the profound vasodilatation.

Anti-embolism stockings should be applied to improve venous return from the lower extremities. Abdominal binders may also be used to encourage venous return and provide diaphragmatic support when the patient is upright.

Activity should be planned in advance, and adequate time should be allowed for a slow progression of position changes from recumbent to sitting and upright.
Tilt tables frequently are helpful in assisting patients to make this transition.

COMPLETE THE OTHER MANAGEMENT.