**University of Mosul**

**College of Nursing**

**Department of Clinical Science**

**Adult Nursing ( II )**

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**Second Stage**

# Brain Tumors

**Edited By**

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## ❖ Background

- A brain tumor occupies space within the skull, growing as a spherical mass or diffusely infiltrating tissue.
- The effects of brain tumors are caused by inflammation, compression, and infiltration of tissue.
- A variety of physiologic changes result, causing any or all of the following pathophysiologic events:
  - Increased intracranial pressure (ICP) .
  - cerebral edema.
  - Seizure activity & focal neurologic signs
  - Hydrocephalus
  - Altered pituitary function
- Neoplastic lesions in the brain ultimately cause death by increasing ICP and impairing vital functions, such as respiration.
- There are over 100 types of brain tumors with an estimated annual occurrence of 78,000 new cases. These include 25,000 malignant and 53,000 nonmalignant brain tumors.

## ❖ Classification of Brain Tumors

A. Primary brain tumors :

- originate from cells within the brain.
- In adults, originate from glial cells (cells that make up the structure and support system of the brain and spinal cord) and are supratentorial (located above the covering of the cerebellum).
- Primary tumors progress locally and rarely metastasize outside the CNS.
- Developed countries have a higher incidence of primary brain tumors, with rates of 5.1 per 100,000 compared to 3.0 per 100,000 in less developed countries. (due to more frequent diagnosis with improved imaging modalities).
- Risk factors include: exposure to ionizing radiation is the only known modifiable risk factor, genetic factors and genetic syndromes (such as neurofibromatosis) .

B. Secondary, or metastatic, brain tumors:

- Develop from structures outside the brain and are twice as common as primary brain tumors.
- Can occur from the lung, breast, lower gastrointestinal tract, pancreas, kidney, and skin (melanomas) neoplasms.
- The highest incidence of brain tumors in adults occurs in the fifth through seventh decades of life.
- There is a slight male predominance in the incidence of malignant brain tumors.

## ❖ Type of Primary Brain Tumors

Brain tumors may be classified into several groups:

1. Brain tumors arising from the coverings of the brain (e.g., dural meningioma)
2. Brain tumors developing in or on the cranial nerves (e.g., acoustic neuroma), those originating within brain tissue (e.g., glioma).
3. Metastatic lesions originating elsewhere in the body.
4. Tumors of the pituitary and pineal glands and of cerebral blood vessels are also types of brain tumors.

- Relevant clinical considerations include the:
  1. Location.
  2. Histologic character of the tumor.
  3. Tumors may be benign or malignant: ( benign tumor, such as a colloid cyst, can occur in a vital area and can grow large enough to have serious effects )

## ❖ Gliomas

1. In adults, gliomas (principally astrocytoma) account for approximately 30% of symptomatic primary brain tumors ( Glial tumors ).
2. The most common type of intracerebral brain neoplasm, are divided into many categories.
3. Astrocytomas, arising from astrocytic cells, are the most common type of glioma and are graded from I to IV, indicating the degree of malignancy.
4. The grade is based on cellular density, cell mitosis, and degree of differentiation from the original cell type.

5. Grades III and IV tumors are known as glioblastomas and have little resemblance to the original cell type.
6. Astrocytomas infiltrate into the surrounding neural connective tissue and therefore cannot be totally removed without causing considerable damage to vital structures.
7. Oligodendroglial tumors, arising from oligodendroglial cells (about 4% of primary brain tumors) represent about 10% to15% of the gliomas. Most oligodendrogliomas occur in adults ages 50 to 60, are found in men more often than in women, and are categorized as low or high grade (anaplastic).
8. The histologic distinction between astrocytomas and oligodendrogliomas is difficult to make but is important, because oligodendrogliomas are more sensitive than astrocytomas to chemotherapy.
9. Tumors originating from ependymal cells, another type of glial cell, are known as ependymomas and are more common in children than adults.
10. Glial tumors may be treated with a combination of surgery, radiation therapy, and chemotherapy, depending on specific cell and patient characteristics.

## ❖ Meningiomas

1. Represent 15% of all primary brain tumors, are common benign encapsulated tumors of arachnoid cells on the meninges.
2. They are slow growing, occur most often in middle aged adults, and are more common in women.
3. Meningiomas most often occur in areas proximal to the venous sinuses.

4. Manifestations depend on the area involved and are often the result of compression rather than invasion of brain tissue.
5. Preferred treatment for symptomatic lesions is surgery with complete removal or partial dissection, although radiation therapy may be useful for some patients.

## ❖ Acoustic Neuromas

1. Tumor of the eighth cranial nerve ; the cranial nerve most responsible for (hearing and balance) ; It usually arises just within the internal auditory meatus, where it frequently expands before filling the cerebellopontine recess.
2. An acoustic neuroma may grow slowly and attain considerable size before it is diagnosed.
3. The patient usually experiences loss of hearing, tinnitus, and episodes of vertigo and staggering gait. As the tumor becomes larger, painful sensations of the face may occur on the same side as a result of the tumor's compression of the fifth cranial nerve .
4. Many acoustic neuromas are benign and can be managed conservatively.
5. Many that continue to grow can be surgically removed and have a good prognosis .
6. Some acoustic neuromas may be suitable for stereotactic radiotherapy rather than open craniotomy.

## ❖ Pituitary Adenomas

1. Account for about 16% of all primary brain tumors.
2. They can occur at any age, but they are more common in older adults.
3. Women are affected more often than men, particularly during the childbearing years.
4. Pituitary tumors are rarely malignant but cause symptoms as a result of pressure on adjacent structures or hormonal changes.

### ❖ Pressure Effects of Pituitary Adenomas

1. Pressure from a pituitary adenoma may be exerted on the optic nerves, optic chiasm, or optic tracts or on the hypothalamus or the third ventricle if the tumor invades the cavernous sinuses or expands into the sphenoid bone.
2. These pressure effects produce headache, visual dysfunction, hypothalamic disorders (disorders of sleep, appetite, temperature, and emotions), increased ICP, and enlargement and erosion of the sella turcica.

### ❖ Hormonal Effects of Pituitary Adenomas

1. Functioning pituitary tumors can produce one or more hormones normally produced by the anterior pituitary.
2. Hormonal hypersecretion is caused only by pituitary adenomas. Most adenomas (65% to 70%) secrete an excess amount of hormone including prolactin (prolactinomas), growth hormone (GH) producing acromegaly in adults, adrenocorticotropic hormone (ACTH) resulting in Cushing syndrome, or thyroid-stimulating hormone (TSH).
3. Adenomas that secrete TSH or follicle-stimulating hormone and luteinizing hormone occur infrequently, whereas adenomas that produce both GH and prolactin are relatively common.

4. The female patient whose pituitary gland is secreting excessive quantities of prolactin presents with amenorrhea or galactorrhea (excessive or spontaneous flow of milk).
5. Male patients with prolactinomas may present with impotence and hypogonadism.
6. Acromegaly, caused by excess GH, produces enlargement of the hands and feet, distortion of the facial features, and pressure on peripheral nerves (entrapment syndromes).

## ❖ Gerontologic Considerations

1. The most frequent tumor types in the older adult are:
   A. Anaplastic astrocytoma.
   B. Glioblastoma.
   C. Cerebral metastases from other sites
2. The incidence of all brain tumors increases with age.
3. Intracranial tumors can produce : personality changes, confusion, speech dysfunction, or disturbances of gait.

## ❖ Clinical Manifestations

Brain tumors can produce both :

A. Focal: signs and symptoms result from tumors that interfere with functions in specific brain regions

B. Generalized symptoms: reflect by increased ICP.

➢ Increased Intracranial Pressure

1. Signs and symptoms : ( headache, nausea with or without vomiting, and papilledema (swelling of the optic nerve).
2. Personality changes and a variety of focal deficits, including motor, sensory, and cranial nerve dysfunction, are common.

➢ Headache

1. is most common in the early morning and is made worse by coughing, straining, or sudden movement.
2. It is thought to be caused by the tumor invading, compressing, or distorting the pain-sensitive structures or by edema that accompanies the tumor.
3. Headaches are usually described as deep, expanding, dull, but unrelenting.
4. Frontal tumors usually produce a bilateral frontal headache; pituitary gland tumors produce pain radiating between the two temples (bitemporal); in cerebellar tumors, the headache may be located in the suboccipital region at the back of the head.

➢ Vomiting

1. Seldom related to food intake, is usually the result of irritation of the vagal centers in the medulla.
2. Forceful vomiting is described as projectile vomiting. Headache may be relieved by vomiting.

- Visual Disturbances

1. The tumor itself or the surrounding edema can compress the third cranial nerve, causing optic disc swelling or papilledema.

- Seizures

1. Occurring in 60% of patients with brain tumors either initially or throughout their disease process.
2. Seizures can be a result of metabolic factors (electrolyte imbalances, liver failure or kidney disease, radiation or chemotherapy side effects).

- Localized Symptoms

1. Sensory or motor abnormalities, visual alterations, alterations in cognition, or language disturbances (e.g., aphasia).
2. A tumor in the motor cortex of the frontal lobe produces hemiparesis and partial seizures on the opposite side of the body or generalized seizures.
3. A frontal lobe tumor may also produce changes in emotional state and behavior, as well as an apathetic mental attitude.
4. The patient often becomes impulsive, inappropriate in speech, gestures, and behavior.
5. A parietal lobe tumor may cause decreased sensation on the opposite side of the body or generalized seizures.
6. A temporal lobe tumor may cause seizures as well as psychological disorders.
7. An occipital lobe tumor produces visual manifestations: contralateral homonymous hemianopsia (visual loss in half of the visual field on the opposite side of the tumor) and visual hallucinations.

8. A cerebellar tumor causes dizziness; an ataxic or staggering gait with a tendency to fall toward the side of the lesion; marked muscle incoordination; and nystagmus (involuntary rhythmic eye movements), usually in the horizontal direction.
9. A cerebellopontine angle tumor usually originates in the sheath of the acoustic nerve and gives rise to a characteristic sequence of symptoms.
10. Brainstem tumors may be associated with cranial nerve deficits along with complex motor and sensory function impairments .

## ❖ Assessment and Diagnostic Findings

1. Neurologic examination indicates the involved areas of the CNS.
2. Computed tomography (CT) scans, enhanced by a contrast agent, can give specific information concerning the number, size, and density of the lesions, and the extent of secondary cerebral edema. CT can provide information about the ventricular system.
3. A magnetic resonance imaging (MRI) scan is the most helpful diagnostic tool for detecting brain tumors, particularly smaller lesions, and tumors in the brainstem and pituitary regions, where bone is thick . MRI is also useful in monitoring response to treatment.
4. Computer-assisted stereotactic (three-dimensional) biopsy is used to diagnose deep-seated brain tumors and to provide a basis for treatment and prognosis.
5. Positron emission tomography (PET) is used to supplement MRI scanning in centers where it is available. On PET scans, low-grade tumors are associated with hypometabolism, and high-grade tumors show hypermetabolism.

6. An electroencephalogram can detect abnormal brain waves in regions occupied by or adjacent to tumor; it is used to evaluate temporal lobe seizures and to assist in ruling out other disorders.
7. Cytologic studies of the CSF may be performed to detect malignant cells as CNS tumors can shed cells into the CSF resulting in metastasis.

## ❖ Medical Management

### ➢ Surgical Management

1. The objective of surgical management is to :
   - Remove as much tumor as possible without increasing the neurologic deficit (paralysis, blindness).
   - Relieve symptoms by partial removal (decompression).
   - Provides tissue to establish a definitive diagnosis.
2. A variety of surgical approaches may be used which depends on the type of tumor, its location, and its accessibility.
3. This approach is used in patients with meningiomas, acoustic neuromas, cystic astrocytomas of the cerebellum, colloid cysts of the third ventricle, congenital tumors such as dermoid cyst, and some of the granulomas.
4. For patients with malignant glioma, complete removal of the tumor and cure are not possible, but the rationale for resection includes relief of ICP, removal of any necrotic tissue, and reduction in the bulk of the tumor.
5. Most pituitary adenomas are treated by transsphenoidal microsurgical removal .

## ➢ Radiation Therapy

1. Radiation therapy decreases the incidence of recurrence of incompletely resected tumors .
2. Gamma radiation is delivered via an external beam to the tumor in multiple fractions.
3. Brachytherapy (the surgical implantation of radiation sources to deliver high doses at a short distance).
4. Radioisotopes such as iodine 131 (131I) are used to minimize effects on surrounding brain tissue.
5. Stereotactic procedures may be performed using a linear accelerator or gamma knife to perform radiosurgery .

## ➢ Chemotherapy

1. The greatest challenge in chemotherapy of brain tumors is that the blood–brain barrier prevents drugs from getting to the tumor in effective doses without causing systemic toxicity .
2. Malignant glioma is usually treated with 6 weeks of oral temozolomide (Temodar) during radiation therapy, followed by 6 to 12 months of oral temozolomide.
3. Autologous bone marrow transplantation is used in some patients who will receive chemotherapy or radiation therapy, because it can “rescue” the patient from the bone marrow toxicity associated with high doses of chemotherapy and radiation.

## ➢ Pharmacologic Therapy

1. Corticosteroids are useful in relieving headache and alterations in level of consciousness ( such as dexamethasone (Decadron) are thought to reduce inflammation and edema around tumors.
2. Other medications used include osmotic diuretics (e.g., mannitol [Osmitrol] and hypertonic saline) to decrease the fluid content of the brain, which leads to a decrease in ICP.
3. Anticonvulsant medications are used to treat and control seizures.
4. Venous thromboembolic events, such as deep vein thrombosis and pulmonary embolism, occur in about 15% of patients and are associated with significant morbidity.
5. Patients receiving anticoagulant agents must be closely monitored because of the risk of CNS hemorrhage.

## ❖ Nursing Management

1. Headache characteristics should be assessed.
2. Upright positioning and pain medications may be useful in managing pain; nurses should evaluate effectiveness of pain management interventions.
3. Even if seizure history is absent, the patient and family should be educated about the possibility of seizure and the need to adhere to prophylactic anticonvulsant medications, if prescribed.
4. The patient with a brain tumor may be at increased risk for aspiration as a result of cranial nerve dysfunction.
5. Preoperatively, the gag reflex and ability to swallow are evaluated.

6. In patients with diminished gag response, care includes educating the patient to direct food and fluids toward the unaffected side, having the patient sit upright to eat, offering a semisoft diet, and having suction readily available.
7. The nurse performs neurologic checks; monitors vital signs; maintains a neurologic observation record ; spaces nursing interventions to prevent rapid increase in ICP; and reorients the patient when necessary to person, time, and place.
8. The use of corticosteroids to control headache and neurologic symptoms requires astute nursing assessment and intervention because many adverse effects can occur, including hyperglycemia, electrolyte abnormalities, and muscle weakness .
9. Patients with changes in cognition caused by their lesion require frequent reorientation and the use of orienting devices (e.g., personal possessions, photographs, lists, a clock), supervision of and assistance with self-care, and ongoing monitoring and intervention for prevention of injury.
10. Patients with seizures are carefully monitored and protected from injury.
11. Motor function is checked at intervals because specific motor deficits may occur, depending on the tumor's location.

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# Fractures

**Edited By**

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## ❖ Background

❖ **Definition :** is a complete or incomplete disruption in the continuity of bone structure and is defined according to its type and extent.

- Fractures occur when the bone is subjected to stress greater than it can absorb.
- Fractures may be caused by direct blows, crushing forces, sudden twisting motions, and extreme muscle contractions.
- When the bone is broken, adjacent structures are also affected, which may result in soft tissue edema, hemorrhage into the muscles and joints, joint dislocations, ruptured tendons, severed nerves, and damaged blood vessels.
- Body organs may be injured by the force that caused the fracture or by fracture fragments.

## ❖ Types of Fractures

- Fractures types are classified by location :
  A. Proximal.
  B. Midshaft.
  C. Distal.
- Fractures are also described according to the degree of break:
  A. **Greenstick fracture**: refers to a partial break.
  B. **Comminuted fracture**: has more than two fragments:

- Type of fracture according to trauma characteristics

1. **Closed fracture (simple fracture)**: is one that does not cause a break in the skin.
2. **Open fracture (compound, or complex, fracture):** is one in which the skin or mucous membrane wound extends to the fractured bone.
   - Type of wound accompanied with open fractures
     A. Type I is a clean wound less than 1 cm long.
     B. Type II is a larger wound without extensive soft tissue damage or avulsions.
     C. Type III (A, B, C) is highly contaminated and has extensive soft tissue damage. It may be accompanied by traumatic amputation and is the most severe.
3. **Intra-articular fracture:** extends into the joint surface of a bone.

## ❖ Clinical Manifestations

1. Acute pain:

- The pain is continuous and increases in severity until the bone fragments are immobilized.
- Immediately after a fracture, the injured area becomes numb and the surrounding muscles flaccid.
- The muscle spasms that accompany a fracture begin shortly thereafter, within a few to 30 minutes, and result in more intense pain than the patient reports at the time of injury.
- The muscle spasms can minimize further movement of the fracture fragments or can result in further bony fragmentation or malalignment.

2. Loss of function:

- After a fracture, the extremity cannot function properly because normal function of the muscles depends on the integrity of the bones to which they are attached.
- Pain contributes to the loss of function. In addition, abnormal movement (false motion) may be present.

3. Deformity:

- Displacement, angulation, or rotation of the fragments in a fracture of the arm or leg causes a deformity that is detectable when the limb is compared with the uninjured extremity.

4. Shortening of the extremity:

- In fractures of long bones, there is actual shortening of the extremity because of the compression of the fractured bone.
- Sometimes, muscle spasms can cause the distal and proximal site of the fracture to overlap, causing the extremity to shorten.

5. Crepitus.
6. Localized edema and ecchymosis. signs may not develop for several hours after the injury or may develop within an hour, depending on the severity of the fracture.

## ❖ Emergency Management

❖ Immediately after injury:

1. If a fracture is suspected, the body part must be immobilized before the patient is moved.
2. Adequate splinting is essential.
3. Joints proximal and distal to the fracture also must be immobilized to prevent movement of fracture fragments.
4. Immobilization of the long bones of the lower extremities may be accomplished by bandaging the legs together, with the unaffected extremity serving as a splint for the injured one.
5. In an upper extremity injury, the arm may be bandaged to the chest, or an injured forearm may be placed in a sling.
6. The neurovascular status distal to the injury should be assessed both before and after splinting to determine the adequacy of peripheral tissue perfusion and nerve function.

## ❖ Medical Management

❖ **Reduction:** refers to restoration of the fracture fragments to anatomic alignment and positioning.

- Types :

1. Closed reduction : is accomplished by bringing the bone fragments into anatomic alignment through manipulation and manual traction. The extremity is held in the aligned position while a cast, splint, or other device is applied.Traction (skin or skeletal) may be used until the patient is physiologically stable to undergo surgical fixation.

2. Open reduction: a surgical approach by which the fracture fragments are anatomically aligned. Internal fixation devices (metallic pins, wires, screws, plates, nails, or rods) may be used to hold the bone fragments in position until solid bone healing occurs.

❖ **Immobilization**

1. To maintain proper position and alignment until union occurs.
2. Accomplished by external or internal fixation.
3. Methods of external fixation include bandages, casts, splints, continuous traction, and external fixators.

## ❖ Fracture Healing and Complications

Many factors influence the time frame of the healing process .

1. fractures of flat bones (pelvis, sternum, and scapula) heal rapidly.
2. A complex, comminuted fracture may heal slowly.
3. Fractures at the ends of long bones, where the bone is more vascular and cancellous, heal more quickly than do fractures in areas where the bone is dense and less vascular (midshaft).
4. Fractures typically heal more quickly in younger patients.
5. Complications of fractures may be either acute or chronic.
6. Early complications include shock, fat embolism, compartment syndrome, and VTE (deep vein thrombosis [DVT], pulmonary embolism [PE]).
7. Delayed complications include delayed union, malunion, nonunion, AVN of bone, complex regional pain syndrome (CRPS, formerly called *reflex sympathetic dystrophy*), and heterotopic ossification.

## ❖ Factors That Inhibit Fracture Healing

1. Age >40 years
2. Avascular necrosis
3. Bone loss
4. Cigarette smoking
5. Comorbidities (e.g., diabetes, rheumatoid arthritis)
6. Corticosteroids, nonsteroidal anti-inflammatory drugs
7. Extensive local trauma
8. Inadequate immobilization
9. Infection
10. Local malignancy
11. Malalignment of the fracture fragments
12. Space or tissue between bone fragments
13. Weight bearing prior to approval

## Early Complications

1. **Shock**
   - Hypovolemic shock resulting from hemorrhage is more frequently noted in trauma patients with pelvic fractures and in patients with a displaced or open femoral fracture in which the femoral artery is torn by bone fragments.
2. **Fat Embolism Syndrome**
   - describes the clinical manifestations that occur when fat emboli enter circulation following orthopedic trauma, especially long bone (e.g., femur) fractures.
3. **Compartment Syndrome**
   - An anatomic compartment is an area of the body encased by bone or fascia (e.g., the fibrous membrane that covers and separates muscles) that contains muscles, nerves, and blood vessels.
   - The human body has 46 anatomic compartments, and 36 of these are located in the extremities.
   - Compartment syndrome is characterized by the elevation of pressure within an anatomic compartment that is above normal perfusion pressure.
   - Compartment syndrome arises from an increase in compartment volume (e.g., from edema or bleeding), a decrease in compartment size (e.g., from a restrictive cast), or aspects of both.
   - Compartment syndrome occurs more frequently in young adults, and although it may take up to 48 hours for symptoms to present, it typically develops quickly, within 6 to 8 hours after the initial injury or after fracture repair .

**4. Other Early Complications**

- VTE, including DVT and PE, are associated with reduced skeletal muscle contractions and bed rest.
- Patients with fractures of the lower extremities and pelvis are at high risk for VTE.

## Delayed Complications

**1. Delayed Union, Nonunion, and Malunion**

- occurs when healing does not occur within the expected time frame for the location and type of fracture.
- Delayed union may be associated with distraction (pulling apart) of bone fragments, systemic or local infection, poor nutrition, or comorbidity (e.g., diabetes, autoimmune disease).
- Nonunion results from failure of the ends of a fractured bone to unite, whereas malunion is the healing of a fractured bone in a malaligned position

**2. Avascular Necrosis of Bone (AVN; Osteonecrosis)**

- AVN occurs when the bone loses its blood supply and dies.
- It may occur after a fracture with disruption of the blood supply to the distal area.
- It is also seen with prolonged high-dose corticosteroid therapy, radiation therapy, sickle cell disease, rheumatoid arthritis, and other diseases.
- The patient develops pain with movement that progresses to pain at rest.

**3. Complex Regional Pain Syndrome**

- CRPS is a rare condition characterized by chronic pain in a limb, typically after an injury.
- Dysfunctional peripheral and central nervous system responses that mount an excessive response to the precipitating event (e.g., fracture, surgery) are thought to be the cause of the pain.
- Women are affected more often than men, and the average age of diagnosis is 40 years.
- Clinical manifestations of CRPS include severe burning pain, local edema, hyperesthesia, stiffness, discoloration, vasomotor skin changes (i.e., fluctuating warm, red, dry and cold, sweaty, cyanotic), and trophic changes that may include glossy, shiny skin, and changes in hair and nail growth.

**4. Heterotopic Ossification**

- refers to benign bone growth in an atypical location, such as in the soft tissue.
- is categorized as traumatic myositis ossificans usually develops in response to soft tissue trauma (e.g., contusion, sprain).
- It is characterized by pain and joint stiffness that causes decreased ROM.
- It typically occurs in young males after musculoskeletal sports injuries.

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# Head Injury

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## ❖ Background

1. Head injury is a broad classification that encompasses any damage to the head as a result of trauma.
2. A head injury does not necessarily mean a brain injury is present.
3. Traumatic brain injury (TBI) or craniocerebral trauma describes an injury that is the result of an external force and is of sufficient magnitude to interfere with daily life and prompts the seeking of treatment.
4. CDC estimates that there are (2.5) million emergency department (ED) visits in the USA each year, the majority of which are for a mild TBI.
5. As a result of TBI, approximately 52,000 people die (contributing to about 30% of all injury-related deaths), 275,000 are hospitalized, and 80,000 to 90,000 will have long-term disabilities .
6. Approximately 78% of patients are treated in the ED and released.
7. The most common causes of TBIs are falls (35.2%), motor vehicle crashes (17.3%), being struck by objects (16.5%), and assaults (10%). Children 0 to 4 years, adolescents 15 to 19 years, and adults 65 years and older are most likely to sustain a TBI.
8. In every age group, TBI rates are higher for males than for females.
9. An estimated 57 million living with a TBI related disability, producing an annual economic impact of approximately $60 to $75 billion due to medical expenses and the cost of lost productivity.

## ❖ Pathophysiology

1. Damage to the brain from traumatic injury takes two forms:

A. Primary injury :

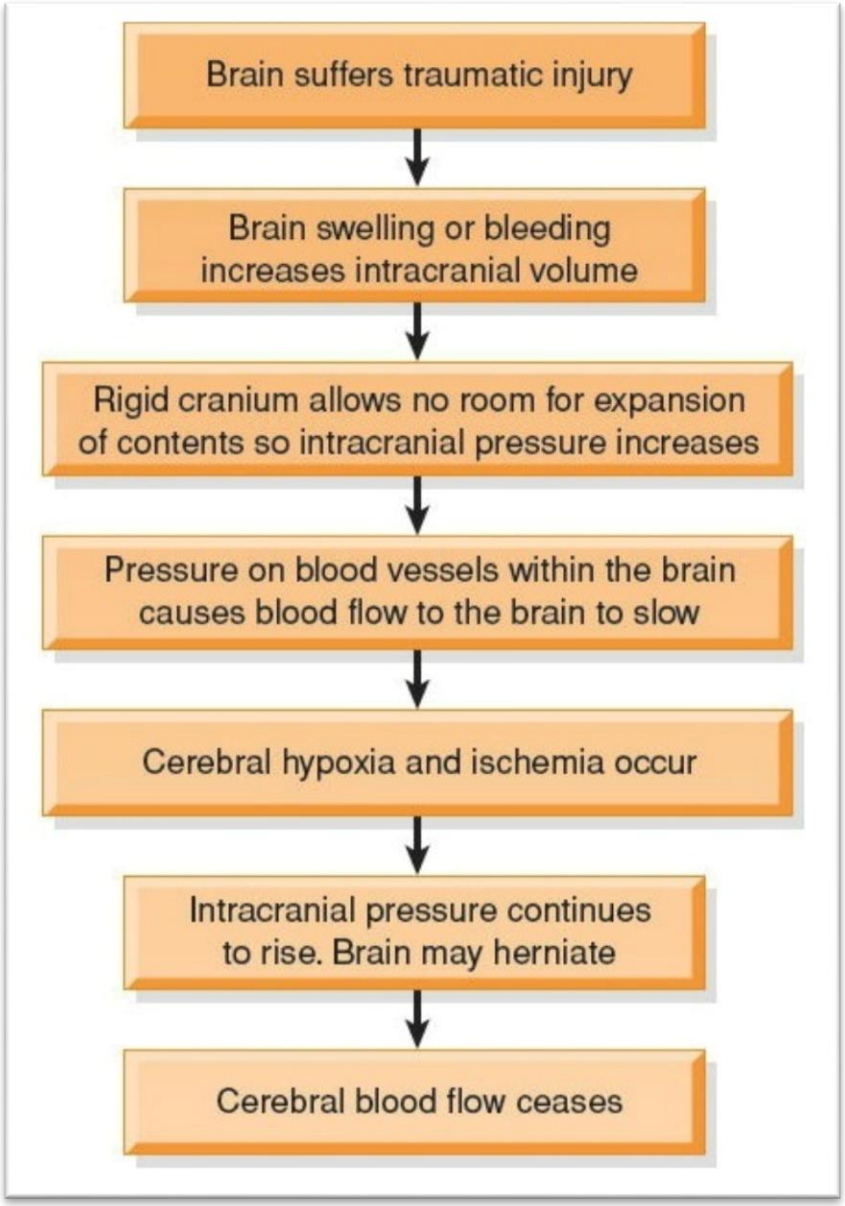
- Definition : as the consequence of direct contact to the head/brain during the instant of initial injury, causing extracranial focal injuries (e.g., contusions, lacerations, external hematomas, and skull fractures).
- Possible focal brain injuries from sudden movement of the brain within the cranial vault (e.g., subdural hematomas (SDHs), concussion, diffuse axonal injury [DAI]).

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.
- Identification, prevention, and treatment of secondary injury are the main foci of early management of severe TBI.

2. Intracranial pathologic processes such as intracranial hemorrhage, cerebral edema, intracranial hypertension, hyperemia, seizures, and vasospasm .
3. Systemic effects from hypotension, hyperthermia, hypoxia, hypercarbia, infection, electrolyte imbalances, and anemia can also be factors which add to the complex biochemical, metabolic, and inflammatory changes that further compromise an injured brain .
4. Any bleeding or swelling within the skull increases the volume of contents within the skull and therefore causes increased intracranial pressure (ICP) .

5. ICP increases can cause displacement of the brain through or against the rigid structures of the skull which causes restriction of blood flow to the brain, decreasing oxygen delivery and waste removal.
6. Cells within the brain become anoxic and cannot metabolize properly, producing ischemia, infarction, irreversible brain damage, and eventually brain death .



## ❖ Scalp Injury

1. Isolated scalp trauma is generally classified as a minor injury.
2. Because its many blood vessels constrict poorly, the scalp bleeds profusely when injured.
3. Trauma may result in an abrasion (brush wound), contusion, laceration, or hematoma beneath the layers of tissue of the scalp (subgaleal hematoma).
4. A large avulsion (tearing away) of the scalp may be potentially life threatening and is a true emergency.
5. Diagnosis of a scalp injury is based on physical examination, inspection, and palpation.
6. 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.
7. Subgaleal hematomas (hematomas below the outer covering of the skull) usually reabsorb and do not require any specific treatment.

## ❖ Skull Fractures

1. 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.
2. Skull fractures are classified by:
   - Types include linear (is a break in the continuity of the bone) , comminuted, ( refers to a splintered or multiple fracture line ) ; and 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).
   - Location fractures include frontal, temporal, and basal skull fractures.
3. 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.

## ❖ Clinical Manifestations

1. Symptoms, apart from those of the local injury, depend on the severity and the anatomic location of the underlying brain injury.
2. Persistent, localized pain usually suggests that a fracture is present.
3. Fractures of the cranial vault may or may not produce swelling in the region of the fracture.
4. 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 hemorrhage 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 sign).

5. Basal skull fractures are suspected when CSF escapes from the ears (CSF otorrhea) and the nose (CSF rhinorrhea).
6. 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 and Diagnostic Findings

1. A computed tomography (CT) scan can be used to diagnose a skull fracture.
2. The ease with which a diagnosis of skull fracture is made depends on the site of the fracture.
3. Magnetic resonance imaging (MRI) scan provides better resolution and clearer pictures of the injured are .

## ❖ Gerontologic Considerations

1. Older patients with head injuries differ from those who are younger in terms of etiology of injury, higher mortality rates, longer lengths of hospital stay, and poorer functional outcomes.
2. Neurologic assessment can be challenging, as the older adult patient with a TBI can have hearing and/or visual deficits or pre-existing dementia or cognitive issues, making establishment of a neurologic baseline difficult.
3. The most common causes of injury in older adult patients are falls and motor vehicle crashes
4. Approximately 61% of all TBIs among adults aged 65 years and older result from falls.

5. Physiologic changes related to aging may place the older adult at increased risk for injury, alter the type and severity of injury that occurs, or lead to complications.
6. Two major factors place older adults at increased risk for hematomas. First, brain weight decreases, the dura becomes more adherent to the skull, and reaction times slow with increasing age ; Second, many older adults take aspirin and anticoagulant agents as part of routine management of chronic conditions.

## ❖ Medical Management

1. Non depressed skull fractures generally do not require surgical treatment.
2. Close observation of the patient is essential.
3. Nursing personnel may observe the patient in the hospital, but if no underlying brain injury is present, the patient may be allowed to return home.
4. Depressed skull fractures usually require surgery with elevation of the skull and débridement, usually within 24 hours of injury.
5. Skull fractures can be a combination of open, compound, closed, or simple.
6. Associated injuries include concurrent scalp laceration, dural tears, and brain injury directly below the fracture from compression of the tissue below the bony injury and from lacerations produced by the bony fragments .

**University of Mosul**

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**Adult Nursing ( II )**

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**Second Stage**

# Osteomyelitis

**Edited By**

**Dr. Saad Hussein Murad Al – Shammary**

## ❖ Background

❖ **Definition** : is an infection of the bone that results in inflammation, necrosis, and formation of new bone.

❖ **Classifications** :

1. Hematogenous osteomyelitis (i.e., due to bloodborne spread of infection).
2. Contiguous – focus osteomyelitis, from contamination from bone surgery, open fracture, or traumatic injury (e.g., gunshot wound).
3. Osteomyelitis with vascular insufficiency, occur most commonly among patients with diabetes and peripheral vascular disease, and commonly affecting the feet.

❖ **Patients at high risk**:

1. Older adults.
2. Poorly nourished.
3. Obese.
4. Impaired immune systems.
5. Chronic illnesses (e.g., diabetes, rheumatoid arthritis).
6. Patient receiving long-term corticosteroid therapy or immunosuppressive agents, and those who use IV drugs.

❖ **Postoperative surgical wound infections**

1. Typically occur within 30 days after surgery.
2. They are classified as:
   A. Incisional (superficial, located above the deep fascia layer).
   B. Deep (involving tissue beneath the deep fascia).
3. Implant used a deep postoperative infections may occur within a year.

4. Osteomyelitis may become chronic and may affect the patient's quality of life.

## ❖ Pathophysiology

1. More than 50% of bone infections are caused by *Staphylococcus aureus* and increasingly of the variety that is methicillin resistant (i.e., methicillinresistant *Staphylococcus aureus* [MRSA]).
2. Surgical site ink markers have been linked to infections by cross contamination between preoperative patients who use their markers; therefore, these items are now considered one patient or one-time use items.
3. Other pathogens include the Gram-positive organisms *streptococci* and *enterococci*, followed by Gram-negative bacteria, including *pseudomonas*.
4. The initial response to infection is inflammation, increased vascularity, and edema.
5. After 2 or 3 days, thrombosis of the local blood vessels occurs, resulting in ischemia with bone necrosis.
6. The infection extends into the medullary cavity and under the periosteum and may spread into adjacent soft tissues and joints.
7. Unless the infective process is treated promptly, a bone abscess forms.
8. The resulting abscess cavity contains sequestrum (i.e., dead bone tissue), which does not easily liquefy and drain. Therefore, the cavity cannot collapse and heal, as it does in soft tissue abscesses.
9. New bone growth, the involucrum, forms and surrounds the sequestrum.
10. Although healing appears to take place, a chronically infected sequestrum remains and produces recurring abscesses throughout the patient's life.This is referred to as chronic osteomyelitis.

## ❖ Clinical Manifestations

❖ When the infection is bloodborne:

1. Onset is usually sudden.
2. Clinical and laboratory manifestations of sepsis (e.g., chills, high fever, rapid pulse, general malaise).
3. The systemic symptoms at first may overshadow the local signs.

❖ As the infection extends through the cortex of the bone:

1. it involves the periosteum and the soft tissues.
2. The infected area becomes painful, swollen, and extremely tender.
3. The patient may describe a constant, pulsating pain that intensifies with movement as a result of the pressure of the collecting purulent material (i.e., pus).
4. When osteomyelitis occurs from spread of adjacent infection or from direct contamination, there are no manifestations of sepsis.
5. The surface area that lies over the infected bone is swollen, warm, painful, and tender to touch.

❖ Patient with chronic osteomyelitis presents with:

1. a nonhealing ulcer that overlies the infected bone with a connecting sinus that will intermittently and spontaneously drain pus.

❖ Diabetic osteomyelitis can occur without any external wounds.

1. Microvascular and macrovascular pathophysiologic changes, along with an impaired immune response by patients with diabetes who have poor glycemic control can exacerbate the spread of infection from other sources

## ❖ Assessment and Diagnostic Findings

❖ In acute osteomyelitis:

1. Early x-ray findings demonstrate soft tissue edema. In about 2 to 3 weeks, areas of periosteal elevation and bone necrosis are evident.
2. Radioisotope bone scans, particularly the isotope-labeled white blood cell (WBC) scan, and MRI help with early definitive diagnosis.
3. Blood studies reveal leukocytosis and an elevated ESR.
4. Wound and blood culture studies are performed, although they are only positive in 50% of cases. Therefore, treatment with antibiotics may be prescribed without isolating the organism.

❖ Chronic osteomyelitis:

1. large, irregular cavities, raised periosteum, sequestra, or dense bone formations are seen on x-ray.
2. Bone scans may be performed to identify areas of infection.
3. The ESR and the WBC count are usually normal.
4. Anemia, associated with chronic infection, may be evident.
5. Cultures of blood specimens and drainage from the sinus tract are frequently unreliable for isolating the organisms involved.
6. An open bone biopsy is indicated as percutaneous aspirations are not reliable for gathering cultures to identify the underlying pathogen .

## ❖ Prevention

1. Elective orthopedic surgery should be postponed if the patient has a current infection (e.g., urinary tract infection, sore throat).
2. During surgery, careful attention is paid to the surgical environment.
3. Prophylactic antibiotics, given to achieve adequate tissue levels at the time of surgery and for 24 hours after surgery, are helpful.
4. Urinary catheters and drains are removed as soon as possible to decrease the incidence of hematogenous spread of infection.
5. Aseptic postoperative wound care reduces the incidence of superficial infections and osteomyelitis.
6. Prompt management of soft tissue infections reduces extension of infection to the bone or hematogenous spread.

## ❖ Medical Management

1. The initial goal of therapy is to control and halt the infective process.
2. General supportive measures (e.g., hydration, diet high in vitamins and protein, correction of anemia) are instituted.
3. The area affected with osteomyelitis is immobilized to decrease discomfort and to prevent pathologic fracture of the weakened bone.

### ❖ Pharmacologic Therapy

1. Antibiotic therapy is longer term than with other infections; typically it continues for 3 to 6 weeks.

❖ **Surgical Management**

1. If the infection is chronic and does not respond to antibiotic therapy, surgical débridement is indicated.
2. The infected bone is surgically exposed, the purulent and necrotic material is removed, and the area is irrigated with sterile saline solution.
3. A sequestrectomy (removal of enough involucrum to enable the surgeon to remove the sequestrum) is performed.
4. In many cases, sufficient bone is removed to convert a deep cavity into a shallow saucer (saucerization).
5. All dead, infected bone and cartilage must be removed before permanent healing can occur.
6. A closed suction irrigation system may be used to remove debris.

## Nursing Process for Patient with Osteomyelitis

❖ **Assessment**

1. The patient reports an acute onset of signs and symptoms (e.g., localized pain, edema, erythema, fever) or recurrent drainage of an infected sinus with associated pain, edema, and low-grade fever.
2. The nurse assesses the patient for risk factors (e.g., older age, diabetes, long-term corticosteroid therapy) and for a history of previous injury, infection, or orthopedic surgery.
3. The gait may be altered as the patient avoids pressure and movement of the area. In acute hematogenous osteomyelitis, the patient exhibits generalized weakness due to the systemic reaction to the infection.

4. Physical examination reveals an inflamed, markedly edematous, warm area that is tender. Purulent drainage may be noted. The patient has an elevated temperature. With chronic osteomyelitis, the temperature elevation may be minimal, occurring in the afternoon or evening.

❖ **Nursing Diagnoses**

1. Acute pain related to inflammation and edema.
2. Impaired physical mobility related to pain, use of immobilization devices, and weight-bearing limitations
3. Risk for infection: bone abscess formation
4. Deficient knowledge related to the treatment regimen

❖ **Planning and Goals**

1. Relief of pain.
2. Improved physical mobility within therapeutic limitations.
3. Control and eradication of infection.
4. Knowledge of the treatment regimen.

❖ **Nursing Interventions**

- Relieving Pain

1. The affected part may be immobilized with a splint to decrease pain and muscle spasm.
2. The nurse monitors the skin and neurovascular status of the affected extremity.
3. The wounds are frequently very painful, and the extremity must be handled with great care and gentleness.
4. Elevation reduces swelling and associated discomfort.
5. Pain is controlled with prescribed analgesic agents and other pain-reducing techniques.

- Improving Physical Mobility

1. Treatment regimens restrict weight-bearing activity. The bone is weakened by the infective process and must be protected by avoidance of stress on the bone.
2. The patient must understand the rationale for the activity restrictions.
3. The joints above and below the affected part should be gently moved through their range of motion.
4. The nurse encourages full participation in ADLs within the prescribed physical limitations to promote general well-being.

- Controlling the Infectious Process

1. The nurse monitors the patient's response to antibiotic therapy and observes the IV access site for evidence of phlebitis, infection, or infiltration.
2. With long-term, intensive antibiotic therapy, the nurse monitors the patient for signs of superinfection (e.g., oral or vaginal candidiasis, loose or foul-smelling stools).
3. The nurse carefully monitors the patient for the development of additional sites that are painful or sudden increases in body temperature.
4. If surgery is necessary, the nurse takes measures to ensure adequate circulation to the affected area (wound suction to prevent fluid accumulation, elevation of the area to promote venous drainage, avoidance of pressure on the grafted area), to maintain needed immobility, and to ensure the patient's adherence to weight-bearing restrictions.
5. The nurse changes dressings using aseptic technique to promote healing and to prevent cross contamination.

❖ **Evaluation**

1. Experiences pain relief
   A. Reports decreased pain at rest
   B. Experiences no tenderness at site of previous infection.
   C. Experiences minimal discomfort with movement
2. Increases in safe physical mobility
   A. Participates in self-care activities within restrictions
   B. Maintains full function of unimpaired extremities
   C. Demonstrates safe use of immobilizing and assistive devices
   D. Modifies environment to promote safety and to avoid falls
3. Shows absence of infection
   A. Takes antibiotic as prescribed.
   B. Reports normal temperature.
   C. Exhibits no edema.
   D. Reports absence of drainage.
   E. Laboratory results indicate normal WBC count and ESR.
   F. Wound cultures are negative
4. Adheres to therapeutic plan
   A. Takes medications as prescribed.
   B. Protects weakened bones.
   C. Demonstrates proper wound care.
   D. Reports signs and symptoms of complications promptly.
   E. Consumes a healthy diet.
   F. Keeps follow-up health care appointments

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# Rheumatoid Arthritis

**Edited By**

**Dr. Saad Hussein Murad Al – Shammary**

## ❖ Background

- is an autoimmune disease of unknown origin that affects 1% of the population worldwide.
- females having a 2.5 times greater incidence than males.
- Commonly occurs between the third and sixth decade of life.
- The incidence of RA increases after the sixth decade of life.
- Risk factors for the development of RA include:
  1. environmental factors such as pollution and smoking,
  2. family history of first-degree relatives.
  3. illnesses such as bacterial and viral illnesses.

## ❖ Pathophysiology

1. The exact mechanism of action for the etiology of RA is unknown.
2. Autoimmune reaction originates in the synovial tissue.
3. Both environmental factors, such as cigarette smoking, and genetic factors coalesce to produce inflammatory and destructive synovial fluid, starting in the more distal joints.
4. This RA synovium breaks down collagen, causing edema, proliferation of the synovial membrane, and ultimately pannus formation.
5. Pannus destroys cartilage and erodes the bone.
6. The consequence is the loss of articular surfaces and joint motion.
7. Muscle fibers undergo degenerative changes.
8. Tendon and ligament elasticity and contractile power are lost.

9. The RA inflammatory process has also been implicated in other disease processes (i.e., arteriosclerosis).
10. the RA disease process somehow interferes with the production of high-density lipoprotein cholesterol, which is the form of cholesterol responsible for decreasing cellular lipids and, therefore, is considered anti atherosclerotic.
11. The nervous system is also affected by the RA inflammatory process; the synovial inflammation can compress the adjacent nerve, causing neuropathies and paresthesias. Axonal degeneration and neuronal demyelination are also possible due to the infiltration of polymorphonuclear leukocytes, eosinophils, and mononuclear cells, causing necrotizing or occlusive vasculitis.

## ❖ Clinical Manifestations

### ❖ Diagnosis Criteria :

1. The criteria are based on a point system where a total score of 6 or greater is required for the diagnosis of RA.
2. The scoring system is based on:
   A. Joint involvement (number of joints affected).
   B. Serology (low positive or high positive rheumatoid factor [RF] or anti-citrullinated peptide antibody [ACPA]).
   C. Abnormall results of the acute phase reactants (erythrocyte sedimentation rate [ESR] or C- reactive protein [CRP]).
   D. duration of symptoms greater than 6 weeks.

3. Patients diagnosed with RA who are excluded from these diagnostic criteria include:
   A. Patients who have one joint with synovitis that is not related to any other clinical disease and who also score at least 6 to 10 points on the scale.
   B. Patients diagnosed with bony erosions on X-ray.

❖ Initial clinical manifestations of RA include:

1. Symmetric joint pain.
2. Morning joint stiffness lasting longer than 1 hour.

❖ Over the course of the disease, clinical manifestations of RA vary:

1. Usually reflecting the stage and severity of the disease.
2. Symmetric joint pain.
3. Swelling.
4. Warmth .
5. Erythema.
6. Lack of function are classic symptoms.
7. Palpation of the joints reveals: ( spongy or boggy tissue. Often, fluid can be aspirated from the inflamed joint. Characteristically, the pattern of joint involvement begins in the small joints of the hands, wrists, and feet.

❖ Disease progresses:

1. The knees, shoulders, hips, elbows, ankles, cervical spine, and temporomandibular joints are affected.
2. The onset of symptoms is usually acute. Symptoms are usually bilateral and symmetric.

❖ Early stages of disease:

1. limitation in function can occur when there is active inflammation in the joints.
2. Joints that are hot, swollen, and painful are not easily moved.
3. The patient tends to guard or protect these joints by immobilizing them.
4. Immobilization for extended periods can lead to contractures, creating soft tissue deformity.
5. Deformities of the hands (e.g., ulnar deviation and swan neck deformity) and feet are common in RA.

## ❖ Assessment and Diagnostic Findings

1. Rheumatoid nodules, joint inflammation detected on palpation, and laboratory findings.
2. The history and physical examination focus on manifestations, such as bilateral and symmetric stiffness, tenderness, swelling, and temperature changes in the joints.
3. Assessment for extra-articular changes include: weight loss, sensory changes, lymph node enlargement, and fatigue.
4. cardiovascular risk assessment should be included in the patient's physical assessment .
5. The ESR and CRP tend to be significantly elevated in the acute phases of RA and are therefore useful in monitoring active disease and disease progression.
6. The complete blood count (CBC) should be assessed to establish a baseline count especially prior to starting medications. Patients may exhibit anemia, and platelets may be elevated due to the inflammatory process.

7. A tuberculin (TB) skin test should be done prior to the initiation of certain medications to rule out tuberculosis.
8. X-ray, ultrasound, or both of the hands, wrists, and feet can be useful in establishing a baseline for joint evaluation, and assessing the joints for erosions and synovitis.

## ❖ Medical Management

❖ The goal of treatment at all phases of the RA disease process is to:

1. Decrease joint pain and swelling.
2. Achieve clinical remission.
3. Decrease the likelihood of joint deformity.
4. Minimize disability.

## ❖ Nursing Management

A. Monitoring and Managing Potential Complications

1. Cardiovascular disease that can lead to complications. due to elevated lipid values, chronic inflammation, dysfunction of the endothelium, and/or abnormal homocysteine levels.