**COURSE OBJECTIVE:** The purpose of this course is to describe monitoring and care of acute stroke victims in ICU and examine some common complications that may develop.

**LEARNING OBJECTIVES**
Upon completion of this course, you will be able to:

- Discuss neurological assessment and diagnosis of complications in stroke patients
- Recognize common medical complications of acute stroke
- Explain the causes of these complications
- List recommended medical and nursing responses to these complications

**THE STROKE ICU**

Because strokes often leave victims in a medically unstable condition, acute stroke victims must be closely monitored in an ICU. "Experts estimate up to 30% of all stroke patients will deteriorate in the first 24 hours" (Summers et al., 2009). Besides neurological deterioration, non-neurological problems are frequent, and the stroke patient may face myocardial infarction, heart failure, aspiration pneumonia, and pulmonary embolism.

Some stroke patients are sent directly to the stroke ICU for treatment and monitoring. Even stroke patients with minor symptoms but with no radiological evidence of a stroke are typically monitored in a stroke ICU (or an ED observation unit) for 6, 12, or 23 hours, depending on the patient's condition.

Stroke patients who need fibrinolytic treatment are first channeled into the protocol for tissue plasminogen activator (tPA), also called alteplase. Stroke patients with symptoms suggesting the need for neurosurgical intervention are first channeled into the neurosurgical evaluation protocol. However, the end station for both
Watchful monitoring and quick reaction to developing complications are the bases of effective acute care for stroke patients (Oliveira-Filho & Koroshetz, 2009b). At the same time, the stroke team physicians work to establish the specific cause of the stroke and begin to plan a strategy to avoid reoccurrences.

The Use of Clinical Pathways

Nurses take the lead in ICU care by writing a clinical pathway—a clinical plan or care map—for each patient. The clinical pathway is a specific care schedule; it is an individualized version of the ICU’s pre-existing stroke protocol, and it lists a chronology of the tasks for physicians, nurses, rehabilitation specialists, and social workers.

Unlike the overall stroke protocol, a clinical pathway is an evolving document. It is shared with and modified by all members of the stroke team, and it is revised as the patient's condition changes. The clinical pathway is the individualized plan that gives the time line and steps needed for the effective care of a particular patient (Summers et al., 2009).

<table>
<thead>
<tr>
<th>STAFFING A STROKE UNIT</th>
</tr>
</thead>
<tbody>
<tr>
<td>In their Recommendations for Comprehensive Stroke Centers, the Brain Attack Coalition offers these guidelines for staffing a stroke unit:</td>
</tr>
<tr>
<td>[H]igh-quality nursing care is a key factor in determining patient outcomes after a stroke. The majority of nurses caring for stroke patients in an ICU, stroke unit, or ward should be registered nurses. The nurses in a CSC should be familiar with standard neurologic assessments and scales, stroke protocols, care maps, ongoing research projects, and new patient care techniques related to stroke. Nurses who care primarily for stroke patients should attend training sessions sponsored by the CSC (i.e., in-services, seminars, specialized lectures) three times per year. Such nurses should participate in 10 hours of continuing education units (CEUs) activities (or other educational programs) annually that are related to or focused on cerebrovascular disease. Each nurse should have a file that documents his/her participation in the above activities. It is suggested that each CSC nurse (stroke unit or ICU) attend one national or regional meeting every other year that focuses on some aspect of cerebrovascular disease.</td>
</tr>
<tr>
<td>An APN [advanced practice nurse] is a vital team member involved in several important aspects of a CSC such as patient care, care maps, research activities, stroke registries, educational programs, and quality assurance. The designation of APN could include a nurse practitioner, master's-prepared clinical nurse specialist, or American Board of Neuroscience Nurses–certified nurse. It is recommended that a CSC have one APN (or similar personnel) to implement and coordinate [care under the various stroke protocols]. (Alberts et al., 2005)</td>
</tr>
</tbody>
</table>

Monitoring Neurological Functioning

The key to managing complications in the stroke ICU is recognizing them quickly. The deterioration in a...
Patient's neurological status is always a signal to search quickly for a complication.

Stroke ICU nurses are characterized by their experience in performing neurological function assessments. During the first 24 hours, acute stroke patients need a neurological assessment at least every 4 hours. Stroke assessments are usually made along with the check of vital signs (pulse, blood pressure, temperature, oxygen saturation, blood glucose, and respiratory pattern).

Here is a template for identifying problems during a full neurological examination:

<table>
<thead>
<tr>
<th>ASSESSMENT OF NEUROLOGICAL PROBLEMS — SAMPLE CHECKLIST</th>
</tr>
</thead>
<tbody>
<tr>
<td>(Check abnormalities that apply)</td>
</tr>
<tr>
<td><strong>A. MENTAL STATUS</strong></td>
</tr>
<tr>
<td><strong>Responsiveness</strong></td>
</tr>
<tr>
<td>Opens Eyes: □ Not spontaneously, only to voice □ Only to pain □ Not at all</td>
</tr>
<tr>
<td><strong>Behavior</strong></td>
</tr>
<tr>
<td>Overall: □ Agitated □ Combative □ Inappropriate □ Restless</td>
</tr>
<tr>
<td>Motor Response: □ Doesn't follow commands □ Localizing to pain</td>
</tr>
<tr>
<td>□ Flexion to pain □ Extension to pain □ No response to pain</td>
</tr>
<tr>
<td><strong>Speech</strong> (Has trach: □)</td>
</tr>
<tr>
<td>Content: □ Inappropriate words □ Sounds, not words □ No speech</td>
</tr>
<tr>
<td>Clarity: □ Slurred □ Unintelligible</td>
</tr>
<tr>
<td>Aphasia: □ Expressive □ Receptive</td>
</tr>
<tr>
<td>Naming Objects: □ Inaccuracies</td>
</tr>
<tr>
<td><strong>Orientation</strong></td>
</tr>
<tr>
<td>Is Disoriented to: □ Time □ Place □ Person</td>
</tr>
<tr>
<td><strong>Memory</strong></td>
</tr>
<tr>
<td>Memory Problems: □ Short-term □ Long-term</td>
</tr>
<tr>
<td><strong>B. CRANIAL NERVE DEFICITS</strong></td>
</tr>
<tr>
<td><strong>I</strong></td>
</tr>
<tr>
<td>Odors: □ Cannot smell odors □ Not tested</td>
</tr>
<tr>
<td><strong>II</strong></td>
</tr>
<tr>
<td>R eye: □ Decreased acuity □ Field deficit □ No vision</td>
</tr>
<tr>
<td>L eye: □ Decreased acuity □ Field deficit □ No vision</td>
</tr>
<tr>
<td><strong>III, IV, VI</strong></td>
</tr>
<tr>
<td>EOM:</td>
</tr>
<tr>
<td>R eye does not move: □ Down □ Up □ Out □ In □ Down+In</td>
</tr>
<tr>
<td>L eye does not move: □ Down □ Up □ Out □ In □ Down+In</td>
</tr>
<tr>
<td>Reports diplopia: □</td>
</tr>
<tr>
<td>Pupils:</td>
</tr>
<tr>
<td>R: □ Sluggish □ Nonreactive □ Nonreactive pinpoint □ Nonreactive Dilated</td>
</tr>
<tr>
<td>□ No consensual reaction □ Hippus □ Right size &gt; Left size</td>
</tr>
<tr>
<td>L: □ Sluggish □ Nonreactive □ Nonreactive pinpoint □ Nonreactive Dilated</td>
</tr>
<tr>
<td>□ No consensual reaction □ Hippus □ Left size &gt; Right size</td>
</tr>
</tbody>
</table>
Ptosis: □ R □ L
Nystagmus: □ R □ L

V
Touch sensation on face decreased: □ R □ L
For R face, pt reports: □ Pain □ Numbness □ Tingling
For L face, pt reports: □ Pain □ Numbness □ Tingling
Lack of corneal reflex on:
    R: □ Ipsilaterally □ Via consensual pathway
    L: □ Ipsilaterally □ Via consensual pathway
Chewing: □ Impaired □ Cannot chew

VII
Weak eye closure: □ R □ L
Facial droop: □ R □ L

VIII
Hearing impairment: □ R □ L

IX/X
Swallowing: □ Impaired
Gag reflex: □ Reduced

XI
Weak shoulder shrug: □ R □ L

XII
Tongue deviates to: □ R □ L

C. PERIPHERY

Sensory (upper limbs, lower limbs):
Decreased sensation: □ R □ U □ L □ R □ L □ L
Decreased discrimination of sharp from dull: □ R □ U □ L □ R □ L □ L
Decreased position sense: □ R □ U □ L □ R □ L □ L
Pt reports numbness: □ R □ U □ L □ R □ L □ L
Pt reports tingling: □ R □ U □ L □ R □ L □ L

Motor Strength:
(5=normal, 4=reduced, 3=weak against gravity, 2=weak even without gravity, 1=trace contraction, 0=nothing)
    RU □ 4 □ 3 □ 2 □ 1 □ 0
    LU □ 4 □ 3 □ 2 □ 1 □ 0
    RL □ 4 □ 3 □ 2 □ 1 □ 0
    LL □ 4 □ 3 □ 2 □ 1 □ 0
Drift: □ RU □ LU

Specific weakness:
    Hand grasp: □ R □ L
    Upper arm push: □ R □ L
    Upper arm pull: □ R □ L
    Foot dorsiflex: □ R □ L
Foot plantarflex: □ R □ L

**Coordination:**
- Impaired fine motor coordination: □ R hand □ L hand
- Impaired rapid alternating movements: □ R hand □ L hand
- Ataxia: □ RU □ LU □ RL □ LL
- Gait: □ Impaired □ Not tested

**Other:**
- Tremors: □ RU □ LU □ RL □ LL
- Abnormal movements: □ RU □ LU □ RL □ LL

When first admitted to the ICU, acute stroke patients need a full baseline neurological assessment and the determination of their NIHSS and a Glasgow Coma Scale scores (see below). ICU stroke protocols often recommend only a brief neurological examination thereafter unless some neurological deterioration is detected. "Deterioration" of neurological functioning has been defined as an increase of 1 point on the NIHSS (Adams et al., 2007).

### NEUROLOGICAL ASSESSMENT TOOLS

**The NIH Stroke Scale (NIHSS)** is a measure of the severity of neurological deficits and can be used to objectively monitor the improvement or deterioration of the stroke. The NIHSS rates 13 neurological characteristics of a patient. (A sample scoring form with an explanation can be downloaded from NINDS (2003).) Testing takes 5–8 minutes and requires no special equipment. In the NIHSS, points are assigned for neurological deficits, and the final scores range from 0 to 42, with higher scores indicating more severe deficits. The chances of a good recovery fall off dramatically in patients with scores greater than 10. A score >22 is labeled a major stroke.

**The Glasgow Coma Scale (GCS)** has been a part of neurologic practice for 35 years and has proved to be an objective and reproducible way to describe a patient's level of consciousness and arousal. Administering the scale takes 3–5 minutes and requires no special equipment. External stimuli are given to a patient, and the tester rates 3 neurological aspects of the patient's response: eye opening, limb movement, and vocalization. (A sample scoring form can be downloaded from the Internet Stroke Center (ISC, 1974).) Points are given for higher levels of response and consciousness. Final scores can range from 3 to 15, with lower scores indicating more severe neurological deficiency. (This is opposite to the NIHSS, in which higher scores indicate more severe deficits.)

### ACUTE COMPLICATIONS OF A STROKE

A stroke ICU has plans in place for the most common medical complications. Members of the ICU staff will always have different levels of expertise, but pre-written protocols and standardized stroke orders can help to ensure that the best care can always be given without delay and with few mistakes.

The best care practices for stroke are evolving quickly, and ICU protocols should evolve, too. The stroke response team and the stroke ICU staff should regularly review their protocols and standing orders. Stroke care is complex, and frontline stroke ICUs are always learning and improving.
Intracranial Problems

Intracranial problems are the most common causes of neurological deterioration after an acute stroke. Brain edema, additional ischemia, and bleeding are the main culprits: more than 33% of the deteriorations are caused by swelling of ischemic brain tissue, while approximately 20% of deteriorations are caused by an additional occurrence of cerebral ischemia or by new or continued intracerebral hemorrhaging. Seizures are an additional, although less common, intracranial cause of neurological deterioration (Adams et al., 2007).

ISCHEMIC BRAIN SWELLING

Injured brain tissue swells from edema, and sufficient swelling will push the brain against the skull or indistensible edges of the dura. In these situations, the brainstem is often squeezed, and the patient will show signs of cerebral herniation. Cerebral herniation should be suspected when new neurological signs include both cranial nerve problems (especially loss or reduction of pupillary responses) and peripheral motor deficits. Brain herniation is a life-threatening emergency.

Unfortunately, the clinical signs of herniation or of increased intracranial pressure are clearest late in the process (Biros & Heegaard, 2009), and early stages of brain swelling cannot easily be recognized clinically in acute stroke patients, although serial head images can give suggestive clues. Therefore, protection against herniation and the other serious consequences of brain swelling depends on the stroke team already being on the alert for the possibility of edema.

Edema is an occasional consequence of any ischemic stroke, but occlusions of the middle cerebral artery (MCA), multilobar infarcts, and cerebellar infarcts are the ischemic strokes most likely to lead to brain edema. Except for patients with cerebellar infarcts, serious episodes of brain edema typically occur 3 to 5 days after an acute ischemic stroke rather than in the first 24 hours (Summers et al., 2009).

<table>
<thead>
<tr>
<th>WATCH LIST FOR BRAIN SWELLING</th>
</tr>
</thead>
<tbody>
<tr>
<td>Besides these particular ischemic strokes, other factors that put a patient on the watch list for serious brain swelling include:</td>
</tr>
<tr>
<td>• A history of hypertension or heart failure</td>
</tr>
<tr>
<td>• An elevated white blood cell count</td>
</tr>
<tr>
<td>• &gt;50% MCA hypodensity in head images</td>
</tr>
<tr>
<td>• Ischemic changes beyond the territory of the MCA</td>
</tr>
<tr>
<td>• Currently on mechanical ventilation</td>
</tr>
</tbody>
</table>

Source: Adams et al., 2007.

If brain edema is suspected, steps to reduce the swelling include:

• Raise the head of the bed to 20°–30°
• Restrict hypo-osmolar fluids
• Correct hypoxemia and hypercarbia
• Reduce any increases in body temperature
• Avoid vasodilators

Hydrocephalus or posterior fossa swelling is treated with suboccipital craniotomy, and for any significant brain edema, a craniotomy can be done and necrotic tissue removed. Mannitol has been used for temporary reduction of brain swelling until surgery can be performed. In addition, hypothermia (to 33°–34° C) is sometimes tried. Nonetheless, even with timely therapy, significant brain swelling has a greater than 50% mortality rate (Adams et al., 2007).
INCREASED INTRACRANIAL PRESSURE

In adults, normal intracranial pressure (ICP) is ≤15 mm Hg. Neurological problems will develop if the intracranial pressure increases to ≥20 mm Hg.

Both ischemic and hemorrhagic strokes sometimes increase intracranial pressure indirectly as a result of brain edema. Hemorrhagic strokes can also increase intracranial pressure directly by adding extravascular blood to the restricted intracranial space.

During a stroke, an increase in intracranial pressure further reduces cerebral perfusion, which can cause global neurological dysfunction and additional ischemia. Increased intracranial pressure can also cause lethal brainstem compression.

Clinically, elevated ICP presents as headache, vomiting, and a decreased level of consciousness. Papilledema can be seen in a funduscopic exam, and sometimes there is periorbital bruising. The appearance of Cushing’s triad—bradycardia, respiratory depression, and hypertension—is an especially ominous sign.

To keep increasing ICP from becoming life threatening, it should be monitored using direct intracranial measurements; typically, direct measurement is via an intraventricular catheter (a ventriculostomy) (Smith & Amin-Hanjani, 2009). Minimally, ICP measurements should be monitored in stroke patients who have coma (i.e., a Glasgow Coma Scale value of 3–8).

Treatments for increased ICP include positioning the head of the bed at a 20°–30° angle, controlling pain (e.g., with morphine or alfentanil), aggressively treating fever with acetaminophen and mechanical cooling, and avoiding techniques and situations that increase intrathoracic pressure. (Moving some stroke patients to an upright posture will worsen their neurological status, so position changes must be done cautiously.)

Other measures to lower ICP include reducing the extravascular fluid volume with intravenous mannitol, inducing respiratory alkalosis with forced hyperventilation, sedation (e.g., barbiturates or propofol), avoiding hypotension, directly draining some cerebrospinal fluid (CSF), or performing a craniotomy to mechanically decompress the intracranial space (Evans et al., 2007; Smith & Amin-Hanjani, 2009). Hydrocephalus, which is often seen after subarachnoid hemorrhage, can be relieved by a shunt or ventricular drain (Singer et al., 2009).

ADDITIONAL INTRACEREBRAL BLEEDING

Another cause of deteriorating neurological functioning is additional intracerebral bleeding. This problem can be recognized using brain imaging, usually CT scans.

- **Ischemic Strokes.** Approximately 1/3 of acute ischemic strokes produce some bleeding. It is estimated that 5% of acute ischemic brain infarcts spontaneously hemorrhage sufficiently to cause neurological worsening, with cerebellar infarcts the most likely to cause problems. Patients with worsening neurological symptoms may need surgery to remove the clot (Adams et al., 2007).

- **Intracerebral Hemorrhages (ICH).** Between 40% and 70% of acute intracerebral hemorrhages bleed significantly during the first 24 hours, usually at the edges of the initial hematoma. Acutely increased systolic blood pressure can be one force driving the expansion of these hematomas, and studies have reported that cautiously reducing systolic blood pressure toward 140 mm Hg may improve outcomes in some patients. Treatment with clotting factor VIIa has also been tried, but the results have been mixed. Currently, it is not clear that surgery can stop most cases of hematoma expansion in intracerebral hemorrhages (Rordorf & McDonald, 2009; Steiner & Bosel, 2010).

- **Subarachnoid Hemorrhages (SAH).** Rebleeding of an acute subarachnoid hemorrhage happens in more than 1/3 of patients. Approximately 10% of these rebleeds occur in the first 5 days, with the first 24 hours having the greatest risk. Without treatment, rebleeds in the first month are usually fatal. The only effective treatment is prevention by obliterating the ruptured aneurysm, which can be clipped surgically or occluded endovascularly with a coil. Either treatment leaves an approximately 20% chance of rebleeding (Ferns et al., 2009; Ropper & Samuels, 2009; Singer et al., 2009).

SEIZURES
After a stroke, seizure activity has been seen in as many as 1/4 of all patients, although some studies have reported seizures in as few as 3% of stroke victims. Most seizures are partial or nonconvulsive. In the ICU, seizures are usually treated with IV antiepileptic medication (Adams et al., 2007; Rordorf & MacDonald, 2009; Singer et al., 2009).

Airway, Breathing, and Respiratory Problems

ABNORMAL BREATHING PATTERNS

Acute stroke patients commonly have periods of abnormal breathing, especially when the patient has decreased consciousness or a large or serious stroke. Tachypneic (fast breathing) patterns can be a problem if they lower blood levels of CO₂ and thereby reduce cerebral perfusion. Other, more common abnormal respiratory patterns, however, do not signal impending neurological deterioration. Nonetheless, any change in respiration should alert the nurses to check airway patency, vital signs, and neurological functioning (Chalela & Jacobs, 2009a).

The most common abnormal breathing pattern in ICU stroke patients is periodic breathing.

- **Periodic breathing.** Approximately 1/4 of stroke patients have episodes of patterned breathing. The recurrent pattern usually alternates a set of shallow breaths with a set of deep breaths. Patients with subarachnoid hemorrhage are especially prone to developing periodic breathing.

- **Cheyne-Stokes respiration.** This is a specific type of periodic breathing. During Cheyne-Stokes respiration, a patient stops breathing temporarily. When starting to breathe again, the patient takes increasingly deeper breaths. After a peak, the breaths become shallower and shallower until breathing again stops. The cycle then repeats. Cheyne-Stokes respiration is usually a reflection of underlying heart or lung problems; when it occurs, it is a warning that the patient may be hypocapnic or hypoxemic.

NEED FOR MECHANICAL VENTILATION

Self-regulated breathing can be a problem for stroke victims, especially those patients with hemorrhagic stroke or with damage to the brainstem. Patients with breathing dysfunction usually have impaired consciousness or impaired airway reflexes, and an endotracheal tube is inserted if the patient's protective airway mechanisms have been compromised.

Patients who develop aspiration pneumonia, pulmonary edema, stupor with reduced respiratory reflexes, or seizures are likely to require mechanical ventilation. The need for endotracheal intubation is a poor sign—approximately 1/2 of the acute stroke patients who are intubated will die within 30 days.

It can be difficult to wean stroke patients after extended periods of mechanical ventilation. Daily trials of autonomous breathing are recommended to exercise the respiratory muscles and to slow the inevitable muscular atrophy from disuse (Mayer & Schwab, 2010).

DECREASED OXYGENATION

Ischemia from poor oxygenation of brain tissue is a major cause of the neurological deficits of a stroke, and longer periods of oxygen deprivation produce more extensive and irreversible damage. Therefore, to save brain tissue, it is critical to maintain a normal blood oxygen saturation. Currently, there is no evidence that either supplemental or hyperbaric oxygen is helpful for stroke patients who already have a normal blood oxygen saturation (Summers et al., 2009).

In the ICU, oxygen saturation is monitored continuously. Hypoxemia ≤92% is treated with supplemental oxygen at 2–4 L/min. **Continuous pulse oximetry** is required, because patients can be hypoxemic without showing clinical symptoms (Chalela & Jacobs, 2009a).

The appearance of hypoxemia also alerts the nurse to check:

- Airway patency
Cardiovascular Problems

CARDIAC MONITORING

Strokes and heart problems are frequent companions. Hypertension and atherosclerosis are shared precursors of a range of cardiovascular diseases, including stroke; thus, stroke patients often present with existing cardiac problems. Strokes can also be the cause of such heart problems as arrhythmias and myocardial infarctions. In a stroke patient, one must actively search for these problems, because myocardial infarctions concurrent with a stroke are often silent. Therefore, the initial evaluation of acute stroke patients should include a cardiac exam, an ECG, and blood tests for cardiac markers. Later, in the ICU, acute stroke patients need continuous cardiac monitoring (Summers et al., 2009).

ECG abnormalities are seen in 90% of acute stroke patients, 60% to 70% of acute stroke patients have significant cardiac disturbances, and 20% of all stroke victims will have a myocardial infarction within 10 years. By itself, having a stroke gives a patient the same risk for developing an arrhythmia or an acute coronary syndrome as having an established diagnosis of coronary artery disease (Chalela & Jacobs, 2009b; Ropper & Samuels, 2009).

HYPERTENSION

Seventy-three percent of stroke patients have a history of hypertension, and hypertension is the single most common risk factor for stroke (George et al., 2009). Thus, patients with acute stroke often present with high blood pressure; between 40% and 80% of acute ischemic stroke patients have hypertension in the first 24 hours (Kaplan & Rose, 2009). Significant hypertension makes a poor outcome likely after an acute stroke, and ICU stroke patients must have their blood pressures monitored frequently.

Ideally, ischemic stroke patients will have their blood pressures maintained at a systolic pressure of about 180 mm Hg and a diastolic pressure of 105–110 mm Hg during the first 24 hours. Higher blood pressures are treated cautiously. The temptation to immediately reduce high blood pressure should be tempered by two observations:

- Some degree of hypertension is often needed to maintain adequate cerebral perfusion after a stroke.
- In many patients, the acutely elevated blood pressure of a stroke will spontaneously decline during the first 24 hours.

It has been suggested that markedly high blood pressures (>220 mm Hg systolic pressure or >110–120 mm Hg diastolic pressure) be lowered, but the reduction should be made gradually. A recommended course of action is to reduce hypertension only about 15% during the first 24 hours after an ischemic stroke (Oliveira-Filho & Koroshetz, 2009a, c).

Patients with intracerebral hemorrhages are usually treated for hypertension more aggressively than patients with ischemic strokes in an attempt to decrease the blood pressure's contribution to increased intracranial pressure. As with ischemic strokes, the goal is to maintain the patient's blood pressure <180/105 mm Hg during the first 24 hours after an ICH.

Before using antihypertensive drugs to treat high blood pressures, nurses should consider remedying other factors that may be elevating the blood pressure. Pain, nausea, a full bladder, or a loud environment can all raise a patient's blood pressure. Intracranial problems, such as increased bleeding, can also raise blood pressure.

When using antihypertensive drugs, labetalol is commonly used when there is also tachycardia, while nicardipine (a purely peripheral vasodilator) is used when there is bradycardia, congestive heart failure, a history of bronchospasm, or COPD (Oliveira-Filho & Koroshetz, 2009a, c; Summers et al., 2009).
PULMONARY EMBOLISM

After the first 48 hours, one serious complication of stroke is pulmonary embolus, which is responsible for 10% of stroke deaths. Pulmonary emboli are typically generated in lower limb or deep pelvic veins, especially in elderly patients who have been paralyzed or otherwise immobilized. Prevention of deep vein thrombosis begins in the ICU, with early patient mobilization, external compression devices, and anticoagulants (at a safe time) (Adams et al., 2007). Comprehensive prophylaxis against deep vein thrombolysis is considered a critical component of care in the ICU of an accredited stroke center (Summers et al., 2009).

Other Common Problems

Other complications that occur frequently in ICU stroke patients include fevers, hyperglycemia, dysphagia, and infections.

FEVER

Fevers give stroke patients a poorer neurological outcome. Even a 1° C rise in temperature increases patient mortality rates, so fevers are aggressively treated with antipyretic drugs. Fever can be directly caused by a stroke, but stroke patients with a fever are also searched for infections—especially, for pneumonia and urinary tract infection (Oliveira-Filho & Koroshetz, 2009b).

To treat a fever, acetaminophen is begun when the patient's temperature reaches 37.5° C (99.6° F). Faster temperature reduction can be achieved with patient cooling systems (Summers et al., 2009).

HYPERGLYCEMIA

Both hyperglycemia and hypoglycemia are associated with increased brain injury after an acute stroke.

Approximately 1/3 of the patients who present with acute stroke have hyperglycemia (i.e., blood glucose >126 mg/dl) (Oliveira-Filho & Koroshetz, 2009b). Hyperglycemia of >140 mg/dl increases the likelihood of a poor outcome in stroke patients, and carefully administered rapid-acting insulin is recommended to reduce levels of blood glucose when they are >180 mg/dl.

Hypoglycemia is also deleterious to an injured brain. Thus, the effects of insulin administration are closely monitored, and glucose and potassium must be available to buffer the effect of the insulin. In general, it appears that a safe target goal for blood glucose in critically ill neurological patients is between 120 mg/dl and 180 mg/dl (Mayer & Schwab, 2010).

For patients who have received rtPA, their blood glucose level is checked every 1–2 hours. For other patients, blood glucose levels are checked every 6 hours (Summers et al., 2009).

DYSPHAGIA

Within the first 3 days, between 42% and 67% of acute stroke patients have dysphagia (i.e., difficulty swallowing), and dysphagia can lead to aspiration pneumonia. Swallowing difficulties can be outwardly unapparent; therefore, acute stroke patients are NPO (including no water, no ice chips, and no oral medications) until their swallowing ability has been formally evaluated. Formal evaluation is done by a speech language pathologist or a specially trained nurse using a proven assessment protocol (Smith et al., 2005) such as the Massey Bedside Swallowing Screen (Massey & Jedlicka, 2002).

After a patient has been cleared to begin oral intake, a nurse should watch for signs of swallowing difficulty—choking; coughing; a wet voice after liquids; slow or labored eating, drinking, or swallowing; or discoordinated mouth and tongue movements while eating or drinking (Summers et al., 2009). People are more likely to aspirate liquids than semi-solid, textured foods with the consistency of pudding, so oral intake should probably begin with semi-solids. Patients must be alert and fully awake when eating or drinking (Trapl et al., 2007).

INFECTION
In the ICU, a stroke patient is at risk for infection because many levels of natural defenses have been compromised. Protective skin has been punctured with needles, protective mucosal tubes, such as urethras, have been scraped and then bypassed with catheters, and protective airway reflexes have been subdued by medicines or by the stroke itself. Reduced protective mechanisms make aspiration pneumonia and urinary tract infections the two most common infections acquired by ICU stroke patients.

**Aspiration Pneumonia.** New fever or a decreasing level of consciousness is a sign that a nurse must listen to an ICU stroke patient's lungs. One out of 5 acute stroke patients develops pneumonia. If the patient has a nasogastric feeding tube, there is a 44% chance that they will develop pneumonia. Most of these pneumonias are aspiration pneumonia, meaning that they have developed from microbes aspirated from the mouth and throat. Dysphagia and reduced airway protection reflexes, due directly to the stroke or due to a reduced level of consciousness, are common precursors to aspiration pneumonia (Chalela & Jacobs, 2009a).

Dysphagia testing (see above) and oral intake restrictions and cautions are important preventive measures. Nausea and vomiting should be treated quickly. When possible, ventilated patients are kept in a semi-recumbent position and their airways are suctioned. For acute stroke patients who will be in the ICU >3 days, a new intensive prophylactic protocol, topical oropharyngeal decontamination, appears to lead to a small but significant reduction in hospital-acquired pneumonias (Mayer & Schwab, 2010).

**Urinary Tract Infections.** UTIs are another common cause of fever in acute stroke patients. Therefore, a new fever or a change in the patient's level of consciousness should also prompt a urine screen (Adams et al., 2007).

**CARE BEYOND 24 HOURS**

In the United States, the average stroke patient's hospital stay is almost 5 days. For ICU nurses, the first 24 hours of this stay are focused on stabilizing the patient's physiology while closely monitoring for complications. Meanwhile, besides providing acute care, physicians use the first 24–48 hours to identify the cause of the stroke and to formulate a plan to correct the cause or to otherwise reduce the risk of additional strokes.

After the first 24 hours, patient care takes on a less acute care routine.

**Watching for Bowel and Bladder Problems**

Stroke patients often have bowel and bladder problems, and nurses can prepare for these problems from the beginning. For constipation, nurses must take the lead and request, if needed, stool softeners, laxatives, or enemas. For urinary incontinence, the goal is to try to remove indwelling catheters early to reduce the chance of the patient developing a UTI. After removing an indwelling catheter, scheduled intermittent catheterization every 4–6 hours can help to re-establish a regular filling and emptying cycle for the patient's bladder.

**Mobilizing the Patient**

When the progression of a stroke has been stabilized, it is time to mobilize the patient, first getting them to sit and move their limbs in bed and then working on standing. A physical therapist should assess the patient and plan an individually tailored comprehensive mobilization program. Initial mobilization should be cautious and closely monitored, because neurological symptoms can worsen with the introduction of movements and posture changes. However, early mobility is important. Immobility of stroke patients increases the risk of pneumonia, atelectasis, deep venous thrombosis, pulmonary embolism, contractures, muscle atrophy, and bedsores; overall, immobility is a factor in half of the deaths within 30 days after an ischemic stroke (Adams et al., 2007).

On the other hand, with early mobilization comes an increased **risk of falls.** The ICU nurses should warn technicians, aides, the family, and the patient that, unless specifically instructed, the patient must not get out of bed without a capable supporting assistant. For patients who are alert and oriented, call buttons and basic supplies are put within easy reach, and the patient is warned not to go to the bathroom without a nurse or an aide to assist. For patients who might not use good judgment, beds can be enclosed, a sitter can be assigned to the room, or alarms can be set to ensure that the patient does not get out of bed without assistance (Summers et al., 2009).

**Protecting Against Skin Damage**
Neurological deficits, e.g., a reduced level of consciousness or a loss of sensation, put stroke patients at increased risk for skin damage. Nurses must regularly examine skin areas that are subjected to pressure or that are soiled by incontinence. When possible, the patient is repositioned every 1–2 hours to reduce the likelihood of pressure ulcers (Summers et al., 2009).

**Maintaining Hydration and Nutrition**

Dehydration or malnutrition will slow a patient's recovery. Dehydration also predisposes a patient to deep venous thrombosis. (When patients eat or drink, they should initially be watched, even if they have passed a dysphagia assessment.) Patients with dysphagia or impaired mental status may need a feeding tube; for the long term, a percutaneous endoscopic gastrotomy tube is usually preferred to nasogastric or nasoduodenal tubes. Generally, no special nutritional supplements appear to be needed for optimal recovery from a stroke (Adams et al., 2007).

**SUMMARY**

Approximately 1/3 of all stroke patients deteriorate within the first 24 hours, and immediate diagnosis and close monitoring during that period is crucial. Worsening vital or neurological signs are often due to intracranial complications, such as cerebral edema, increased intracranial pressure, or rebleeding. Additional complications may include:

- Airway, breathing, and respiratory problems
- Cardiovascular problems, such as myocardial infarctions or hypertension
- Fevers and infections
- Metabolic dysfunctions, such as hyperglycemia
- Dysphagia

Thus, the ICU care of acute stroke victims requires continual monitoring and the readiness to cope quickly with a wide range of medical issues.

**RESOURCES**

**Sources of Stroke Information**

American Stroke Association (A Division of American Heart Association)
http://www.strokeassociation.org

Brain Aneurysm Foundation
http://www.bafound.org

Brain Attack Coalition
http://www.stroke-site.org

Internet Stroke Center
http://www.strokecenter.org

National Aphasia Association
http://www.aphasia.org

National Institute of Neurologic Disorders and Stroke

National Stroke Association
http://www.stroke.org

**Stroke Scales and Similar Tools**
Lists and information
http://www.strokecenter.org/trials/scales/scales-overview.htm

NIH Stroke Scale Training (free online training course/1 CME)
http://nihss-english.trainingcampus.net/uas/modules/trees/windex.aspx

NIH Stroke Scale Training (free on a mobile phone/1 CME)
http://learn.heart.org/ihtml/application/student/interface.heart2/nihss.html

REFERENCES


