

Neurological Critical Care

November 15th, 2011

7:30 a.m. to 4:00 p.m.

Midway Health Campus: Conley Room

Description/Purpose Statement

Adults with neurologic disorders can be among the most difficult to assess and manage. The purpose of this class is to learn about the assessment and care of patients with neurologic disorders such as increased intracranial pressure, spinal cord injury, cranial surgery, cerebrovascular disease, and seizures.

Target Audience

This class was designed for the novice critical care or telemetry nurse; however, other health care professionals are welcome to attend.

Before You Come to Class

You must complete the **Neurological Critical Care Primer**. If you did not receive the primer with this cover letter, please access the primer from the TCHP website at www.tchpeducation.com under home studies. Please bring your primer post-test to class with you for processing.

Schedule

7:30 - 7:45 a.m.	<i>Registration</i>	
7:45 - 9:15 a.m.	Assessment of the Patient with Neurological System Abnormalities	Aimee Nelson
9:15 - 9:30 a.m.	<i>Break</i>	
9:30 - 10:15 a.m.	Types of Head Injuries and Hematomas, Management of the Head Injured Patient	Aimee Nelson
10:15 - 10:45 a.m.	Cranial Surgery, Increased Intracranial Pressure, Routine Invasive Intracranial Pressure Monitoring	Aimee Nelson
10:45 - 11:45 a.m.	Herniation Syndromes, Brain Death	Aimee Nelson
11:45 - 12:30 p.m.	<i>Lunch</i>	
12:30 - 1:00 p.m.	SIADH/DI	Aimee Nelson
1:00 - 1:30 p.m.	Seizures	Aimee Nelson
1:30 - 2:30 p.m.	Spinal Cord Injury	Aimee Nelson
2:30 - 2:45 p.m.	<i>Break</i>	
2:45 - 4:00 p.m.	Cerebrovascular Disease	Tess Sierzant

Continuing Education Credit

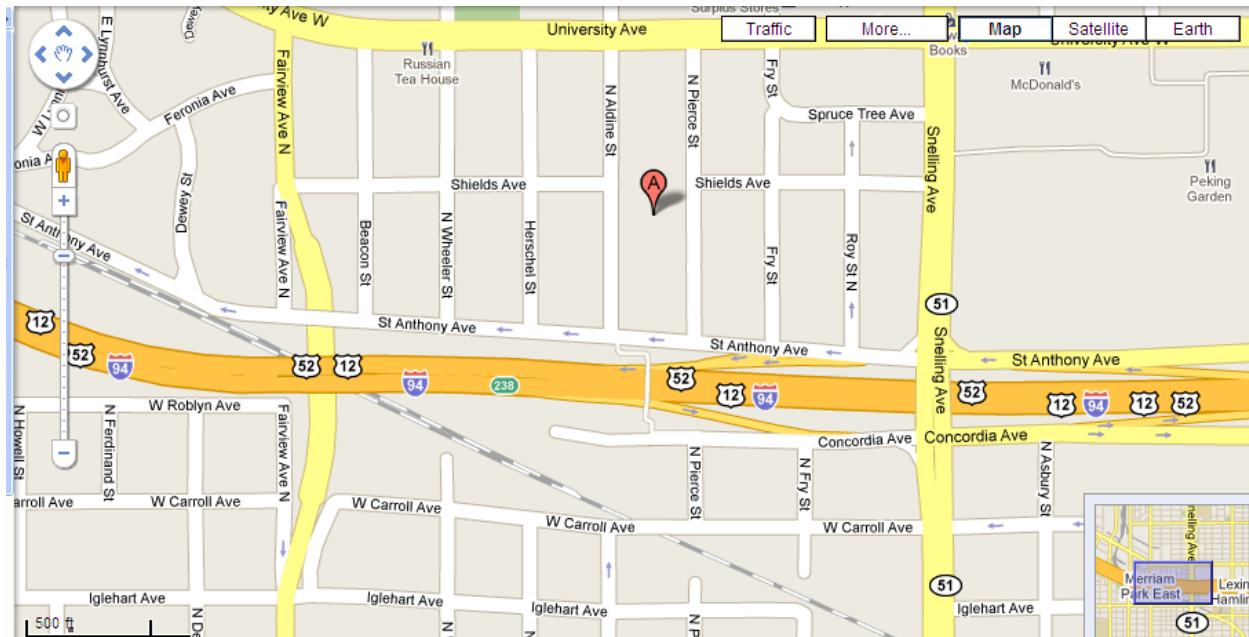
For attending this class , you are eligible to receive:	<p>8.4 Minnesota Board of Nursing contact hours / 7.00 ANCC contact hours.</p> <p>Criteria for successful completion: All participants must attend the program and complete the evaluation form to receive contact hours. If you are an ANCC certified nurse, you must attend the ENTIRE activity to receive contact hours for it.</p> <p>The Twin Cities Health Professionals Education Consortium is an approved provider of continuing nursing education by the Wisconsin Nurses Association, an accredited approver by the American Nurses Credentialing Center's Commission on Accreditation.</p>
If you complete the primer for this class, you are eligible to receive an additional:	<p>2.5 Minnesota Board of Nursing contact hours / 2.08 ANCC contact hours</p> <p>Criteria for successful completion: You must read the primer, complete the post-test and evaluation, and submit it to TCHP for processing.</p>

Please Read!

- Check the attached map for directions to the class and assistance with parking.
- Certificates of attendance will be distributed at the end of the day.
- You should dress in layers to accommodate fluctuations in room temperature.
- Food, beverages, and parking costs are your responsibility.
- If you are unable to attend after registering, please notify the Education Department at your hospital or TCHP at 612-873-2225.
- In the case of bad weather, call the TCHP office at 612-873-2225 and check the answering message to see if a class has been cancelled. If a class has been cancelled, the message will be posted by 5:30 a.m. on the day of the program.
- More complete class information is available on the TCHP website at www.tchpeducation.com.

Midway Health Campus—Conley Room

1700 University Avenue—but NOT on University Ave. despite address
St. Paul, MN 55104
(at Shields Avenue and Aldine Street)



Directions:

Take Interstate 94 to Snelling Avenue exit. Go north approximately 2 blocks to University Avenue and turn left (west). Go to Aldine Street and turn left (south).

Parking: Park in Patient and Visitor lot. This is the first lot on the right (west) side of street. You can also park on surrounding residential streets. Do not park in the ramp unless you are prepared to pay the posted rate—no discount is available.

To get to the room: Enter the Midway building via the entrance that faces Aldine Street. Doors will be open at 7:00 a.m. Sign in at the main desk and then proceed down the hallway to your right. The room is the first door to your right.

Bring with you:

- A lunch or money to purchase food. A microwave is available in the cafeteria. All food and beverages are the responsibility of the individual attending the program.
- Please dress in layers of clothing to accommodate fluctuations in room temperature.
- A pen or pencil to take notes with. A class outline will be provided for you.
- Your home study post-test (if applicable).

TCHP

**Education
Consortium**

Neurological Critical Care Primer

Introduction/Purpose Statement

Adults with neurologic disorders can be among the most difficult to assess and manage. The purpose of this home study is to give you information on the anatomy and physiology of the neurological system, as well as the pathophysiology of many different neurological disorders, including increased intracranial pressure, spinal cord injury, cranial surgery, cerebrovascular disease, and seizures. This information will help understand nursing and medical interventions for these disorders.

Target Audience

This home study was designed for the novice critical care or telemetry nurse; however, other health care professionals are invited to complete this packet.

Content Objectives

1. Describe the normal anatomy and physiology of the brain and spinal cord.
2. Differentiate between the hematomas.
3. Differentiate between the spinal cord injuries.
4. Describe autonomic dysreflexia.
5. Differentiate between SIADH and DI.
6. Differentiate between various seizures.
7. Differentiate between a TIA, RIND, PRIND, progressive, and completed stroke.

Disclosures

In accordance with ANCC requirements governing approved providers of education, the following disclosures are being made to you prior to the beginning of this educational activity:

Requirements for successful completion of this educational activity:

In order to successfully complete this activity you must read the home study, complete the post-test and evaluation, and submit them for processing.

Conflicts of Interest

It is the policy of the Twin Cities Health Professionals Education Consortium to provide balance, independence, and objectivity in all educational activities sponsored by TCHP. Anyone participating in the planning, writing, reviewing, or editing of this program are expected to disclose to TCHP any real or apparent relationships of a personal, professional, or financial nature. *There are no conflicts of interest that have been disclosed to the TCHP Education Consortium.*

Relevant Financial Relationships and Resolution of Conflicts of Interest:

If a conflict of interest or relevant financial relationship is found to exist, the following steps are taken to resolve the conflict:

1. Writers, content reviewers, editors and/or program planners will be instructed to carefully review the materials to eliminate any potential bias.
2. TCHP will review written materials to audit for potential bias.
3. Evaluations will be monitored for evidence of bias and steps 1 and 2 above will be taken if there is a perceived bias by the participants.

No relevant financial relationships have been disclosed to the TCHP Education Consortium.

Sponsorship or Commercial Support:

Learners will be informed of:

- Any commercial support or sponsorship received in support of the educational activity,
- Any relationships with commercial interests noted by members of the planning committee, writers, reviewers or editors will be disclosed prior to, or at the start of, the program materials.

This activity has received no commercial support outside of the TCHP consortium of hospitals other than tuition for the home study program by non-TCHP hospital participants.

If participants have specific questions regarding relationships with commercial interests reported by planners, writers, reviewers or editors, please contact the TCHP office.

Non-Endorsement of Products:

Any products that are pictured in enduring written materials are for educational purposes only. Endorsement by WNA-CEAP, ANCC, or TCHP of these products should not be implied or inferred.

Off-Label Use:

It is expected that writers and/or reviewers will disclose to TCHP when "off-label" uses of commercial products are discussed in enduring written materials. *Off-label use of products is not covered in this program.*

Expiration Date for this Activity:

As required by ANCC, this continuing education activity must carry an expiration date. The last day that post tests will be accepted for this edition is **December 31, 2017**—your envelope must be postmarked on or before that day.

Planning Committee/Editors

Linda Checky, BSN, RN, MBA, Assistant Program Manager for TCHP Education Consortium.

Lynn Duane, MSN, RN, Program Manager for TCHP Education Consortium.

Authors

Lynn Duane, MSN, RN, Program Manager for TCHP Education Consortium.

Karen Poor, MN, RN, Former Program Manager of the Twin Cities Health Professionals Education Consortium

Content Expert

Carol Ann Smith, BAN, RN, CNRN, Program Coordinator of the Traumatic Brain Injury Center at Hennepin County Medical Center.

Contact Hour Information

For completing this Home Study and evaluation , you are eligible to receive:	2.5 MN Board of Nursing contact hours / 2.08 ANCC contact hours <i>Criteria for successful completion:</i> You must read the home study packet, complete the post-test and evaluation and submit them to TCHP for processing. The Twin Cities Health Professionals Education Consortium is an approved provider of continuing nursing education by the Wisconsin Nurses Association, an accredited approver by the American Nurses Credentialing Center's Commission on Accreditation.
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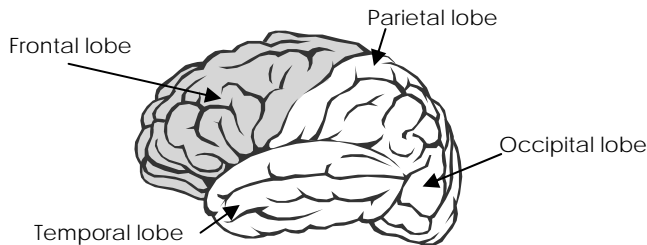
Please see the last page of the packet before the post-test for information on submitting your post-test and evaluation for contact hours.

The Functions Of The Brain

The Cerebrum

The cerebrum is the largest part of the brain. It consists of two cerebral hemispheres, the limbic system, the basal ganglia and the diencephalon.

The **cerebral hemispheres** (telencephalon) are connected by the corpus callosum. They consist of the gyri, sulci and fissures and the cerebral cortex. The cerebral cortex is divided into the frontal, temporal, parietal and occipital lobes.



The **frontal lobe** is responsible for high level cognitive functions such as planning, organizing, sequencing, reasoning, concentration, abstract thinking, regulation of personality, emotional and behavioral control. It also contains the areas for voluntary motor function and provides for storage of information.

The **temporal lobe** is the primary auditory receptive area and is responsible for hearing, the ability to understand the spoken word, memory & learning.

The **parietal lobe** includes the primary sensory cortex and functions to interpret touch, pain and temperature.

The **occipital lobe** is the primary visual cortex and visual association area.

The second section of the cerebrum is called the **diencephalon**. The diencephalon consists of the thalamus and hypothalamus.

The **thalamus** is responsible for relaying and fine tuning information; motor, visual, somatosensory, auditory and gustatory. Portions of the thalamus also regulate circadian rhythms, salivation, GI secretion and motility. It also plays a role in the conscious awareness of pain. It also has a role with the limbic and reticular activating systems.

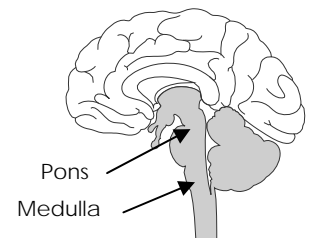
The hypothalamus controls body temperature, thirst, water metabolism, appetite, sexual arousal, visceral and somatic activities, sleep-wakefulness cycle, physical expressions in response to emotions and secretions from the pituitary gland.

The third and final section of the cerebrum is the **limbic** system. The limbic system is a complex system anatomically and functionally connected with many other structures, but primarily involves basic instinctual drives and affective and visceral responses of emotional behavior. It is also associated with learning and new short-term memories.

The **basal ganglia** are masses of gray matter located at the base of the brain that mediates motor effects through the pyramidal motor system, particularly fine motor control of hands and lower extremities.

Brain Stem

The brainstem consists of the midbrain, pons and medulla oblongata. It also contains the cerebral peduncles that connect the brainstem to the cerebellum, and the nuclei for cranial nerves III through XII.



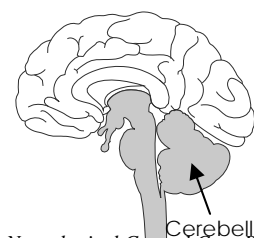
The **midbrain** (mesencephalon) contains neurons that serve ocular and auditory reflexes and the nuclei of cranial nerves III and IV and contain the aqueduct of Sylvius. The midbrain also contains the reticular formation which is related to arousal, consciousness and sleep. It projects to the thalamus and cerebral cortex.

The **pons** (metencephalon) contains the nuclei of cranial nerves V, VI, VII and VIII, the corticospinal (pyramidal) tract, the corticobulbar tract, portions of the reticular formation and is responsible for some respiratory function.

The **medulla oblongata** (myelencephalon) contains the nuclei of cranial nerves IX, X, XI and XII, the lateral spinothalamic tracts, portions of the reticular formation and the center for control of ventilation and respiratory generator.

The Cerebellum

The cerebellum consists of the vermis and two cerebellar hemispheres, which contain the



cerebellar peduncles. The primary functions are regulating muscle tone, coordinating voluntary movements and regulating vestibular reflexes of posture and eye movements.

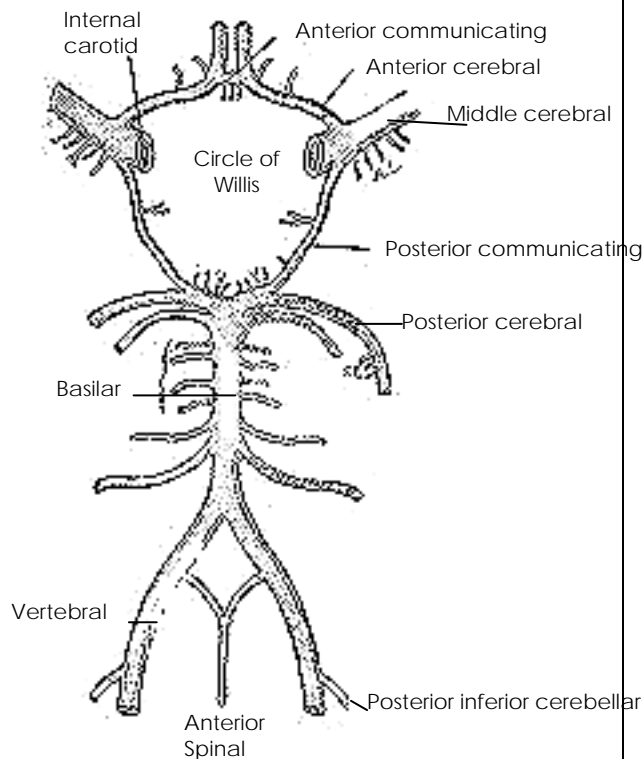
Normal blood flow in the CNS

The brain and spinal cord require three things to survive:

1. oxygenated arterial blood. The nervous system **has** to have oxygen to continue. The brain and the spinal cord cannot store oxygen and cannot use anaerobic metabolism. Functioning stops after about 10 seconds without oxygen.
2. a venous system through which to channel waste products
3. glucose supply. The brain and spinal cord cannot use anything but glucose for an energy source, and cannot store glucose. They depend on the serum level of glucose for their supply.

Arterial Blood Supply

The arterial blood supply to the brain and spinal cord is supplied by two sets of arteries: the internal carotid system and the vertebral system. The **internal carotid arteries** arise from the common carotid arteries and branch into the following arteries:



- The **anterior cerebral** supplies the middle of frontal and parietal lobes and corpus callosum.
- The **anterior communicating** connects the right and left anterior cerebral arteries.
- The **middle cerebral** supplies lateral frontal, temporal, and parietal lobes.
- The **posterior communicating** connects posterior cerebral arteries with internal carotid arteries.

The **vertebral arteries** arise from the subclavian arteries and join at the bottom of the pons to form the basilar artery. The basilar artery branches into:

- The **posterior inferior cerebellar** (PICA) supplies the posterior and inferior cerebellum.
- The **anterior spinal** supplies the front half to three-quarters of the spinal cord and middle of the brain stem.
- The **posterior cerebral** supplies the posterior parietal lobe and inferior temporal and occipital lobes.
- The **superior cerebellar** and **anterior inferior cerebellar** supply the brain stem and cerebellum.

A mechanism designed to ensure collateral circulation to the brain in the event of a blockage is the **Circle of Willis**, an anastomosis of the arteries at the base of the brain.

Venous Blood Return

After oxygenating the cerebral and spinal cord tissues, venous blood is returned to the central circulation through sinuses that lie between the dural layers. The cerebrum has external veins that lie in the subarachnoid space.

Cerebral Blood Flow

The brain and spinal cord are greedy in their need for oxygenated blood. Even though they take up only 2% of the space in the body, they demand 20% of the oxygen. The arterial bed in the brain and spinal cord is able to constrict and dilate as necessary to fill the requirement for oxygenated blood -- a mechanism called autoregulation. Autoregulation works when the systemic mean blood pressure is between 50 and 150 mm Hg. Hypercapnia ($\text{PaCO}_2 > 45$) and hypoxemia ($\text{PaO}_2 < 60$) will cause vasodilation.

What protects the brain instead the skull?

There are three layers of tissue, called meninges, that surround the brain and spinal cord. The meninges are layers of connective tissue that cover the brain and spinal

cord. They consist of the dura mater, arachnoid mater and pia mater.

The outermost layer is called the **dura mater**, which stands for “tough mother.” This layer is attached on the outside to the inside of the cranial bones, and lies on top of a second layer of the meninges -- the arachnoid mater. There are two folds of the dura mater: the falx cerebri, which partially separates the two hemispheres of the cerebrum; and the tentorium cerebelli, a fold which separates the cerebrum from the cerebellum and brain stem.

Between the dura mater and the arachnoid mater lie the meningeal arteries and venous sinuses.

The **arachnoid mater** -- “spider mother” - is a very fine, web-like layer below which lies the subarachnoid space. This space contains the larger blood vessels of the brain, cerebrospinal fluid, and arachnoid villi. The arachnoid villi reabsorbs CSF and transfers it to the venous system and the basilar cisterns which house the CSF.

Finally, the **pia mater** -- “delicate mother” is a fragile layer that adheres to the brain tissue and spinal cord. It is the delicate, innermost layer that covers the entire surface of the brain’s gray matter and dips into the convolutions. It is mesh-like and very vascular, getting blood supply from the internal carotid and vertebral arteries. Fringe-like processes of the pia mater contain the capillaries of the choroid plexus that lines the lateral, third and fourth ventricles and forms CSF.

The dura mater is supplied with blood through **the meningeal arteries**, which are branches of the external carotid arteries. The pia and arachnoid layers receive their blood from the internal carotid and vertebral arteries.

The space between the skull and the outer layer of the dura mater is the **epidural space**. In the vertebral canal, the epidural space is between the periosteum and the spinal dura. The space between the inner dura mater and arachnoid mater is the **subdural space**. Between the dura mater and arachnoid mater lie the meningeal arteries and venous sinuses. The **subarachnoid space** is between the arachnoid mater and the pia mater. It is not a clear space, but spongy with connective tissue. This space contains the cerebrospinal fluid (CSF).

Review of the cranial nerves

The cranial nerves exit the medulla to go to various parts of the body to either provide sensation for or motor operation to all of the face and much of the vital respiratory and cardiac functions. The 12 pairs of cranial nerves are part of the peripheral nervous system.

- I. The **olfactory** nerve (I) extends from the olfactory bulb and functions as a sensory nerve for smell.
- II. The **optic** nerve (II) originates from the bipolar cells of the retina and is a sensory nerve for vision.
- III. The **oculomotor** nerve (III) constricts the pupil and moves the eye and eyelid.
- IV. The **trochlear** nerve (IV) moves the eye downward and inward.
- V. The **trigeminal** nerve (V) has a sensory and motor components, and provides sensation to most of the face, mouth, ear, and sinuses, and innervates the muscles for chewing.
- VI. The **abducens** (VI) nerve abducts the eye.
- VII. The **facial** nerve (VII) has motor, sensory and parasympathetic components. Moves the muscles of facial expression, provides taste sensation on the anterior 2/3 of the tongue and is involved in lacrimation, salivation and nasal secretions.
- VIII. The **acoustic** nerve (VIII), also called the vestibulocochlear nerve, is a sensory nerve with two branches. The cochlear branch is concerned with hearing. The vestibular branch influences balance, maintenance of body position and orientation in space.
- IX. The **glossopharyngeal** nerve (IX) has five branches and enables swallowing and provides sensation to the pharynx, soft palate and tongue; receptors in carotid body and sinus for respiration, BP and HR. It also provides taste receptors from the posterior 1/3 of the tongue, and sensation back of the ear.
- X. The **vagus** nerve (X) has three branches. Motor for phonation, cardiac depression, bronchoconstriction, GI peristalsis and secretion. Sensory to taste of the epiglottis and parasympathetic for the gag reflex.. The vagus nerve moves palatal, pharyngeal, laryngeal muscles; inhibits of HR and adrenal secretions; and stimulates GI peristalsis and secretion. It provides sensation to the palate, pharynx, larynx.
- XI. The **spinal accessory** (XI) nerve elevates shoulders and tilts, turns, and thrusts head forward.
- XII. Finally, the **hypoglossal** (XII) moves the tongue.

Cerebral Hematomas

Mr. Jack Naturale is a 57 year old male who enters the Emergency Room with changes in his level of consciousness. He reportedly fell off a bar stool at the

local bar yesterday. He has a GCS of 12 and is very difficult to arouse. A CT scan is done without contrast, the results of which show that Mr. Naturale has a subdural hematoma.

What is a cerebral hematoma?

There are three types of intracranial hematomas:

- A subdural hematoma is bleeding (usually venous) into the subdural space, **below** the dura mater, but above the brain tissue itself. These are classified as acute, subacute or chronic.
- An epidural hematoma is bleeding (usually arterial) that occurs in the epidural space below the skull but **above** the dura mater.
- An intracerebral (intraparenchymal) hematoma is bleeding into the brain tissue (parenchyma) itself.

CT Scan: Right subdural hematoma with mass effect



What are the causes these hematomas?

Trauma, accompanied by scalp lacerations, skull fractures, or penetrating wounds, is one of the main causes of any of the three types of hematomas. Subdural hematomas may happen spontaneously, especially if the patient is on anti-coagulant therapy. Epidural hematomas are frequently related to linear skull fractures.

Mr. Naturale begins to show signs and symptoms of increasing intracranial pressure.

What is intracranial pressure, and what causes it to increase?

Intracranial pressure is the pressure exerted by the brain, blood, and cerebrospinal fluid (CSF) on the cranium. The cranium (skull) in the adult is a “fixed box” -- it cannot expand to accommodate increased pressure from the contents inside. There are three substances inside the cranial vault:

- **Brain tissue and cell water: 80%**
- **Blood: 10%**
- **CSF: 10%**

Normally, the pressure exerted by these substances is 0-10 mm Hg. This pressure rises when there is an increase in the volume of any of the three substances:

1. Increase in **brain** occurs with cerebral edema caused by drugs or trauma; or with space occupying tumors.
2. Increase in the **blood volume** occurs with intracranial bleeding caused by trauma, surgery, or a blockage in the flow of blood out of the brain (strangulation).
3. Increase in the **CSF** occurs with a blockage in the drainage of CSF, such as may be caused by a tumor.

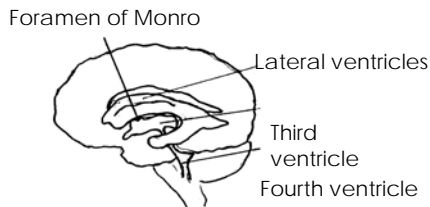
The body tries to compensate for increases in intracranial pressure by a process that was named the *Monroe-Kellie hypothesis*. This hypothesis states that an increase in any one of the substances in the skull will cause a decrease in the other two substances. For example, an increase in brain volume will lead to a decrease in blood or CSF volume.

This compensation will only work up to a certain point; continued rising pressure will eventually lead to herniation. **Herniation** occurs when the pressure inside part or all of the skull is significantly greater than the pressure in another part of the skull or spinal cord. Brain tissue will move to the area which is under lesser pressure.

Another key concept to understand is **cerebral perfusion pressure, or CPP**. It is defined as the net pressure of the blood flow to the brain. It must be maintained within narrow limits because too little pressure could cause brain tissue to become ischemic and too much can raise the intracranial pressure. The mathematical formula is: $CPP = MAP(\text{mean arterial pressure}) - ICP$. Normal is between 70 – 90 mm Hg in an adult.

The purpose of cerebrospinal fluid is...?

Cerebrospinal fluid (CSF) is a clear, colorless, and odorless fluid that cushions the brain and spinal cord and decreases their effective weight. The choroid plexus, a tuft of capillaries, synthesizes approximately 500 ml of CSF per day. There is 125 to 150 ml of CSF in the ventricular system and subarachnoid space at any one time. Most of the CSF is reabsorbed through the arachnoid villi located in the subarachnoid space.



The ventricles in the brain are similar to the ventricles in the heart -- they are holding tanks for fluid. There are four ventricles: two lateral ventricles located on each side of the cerebral hemisphere; the third ventricle lies between the lateral ventricles; and the fourth ventricle lies in the posterior fossa.

The hematoma is evacuated, and Mr. Naturale recovers.

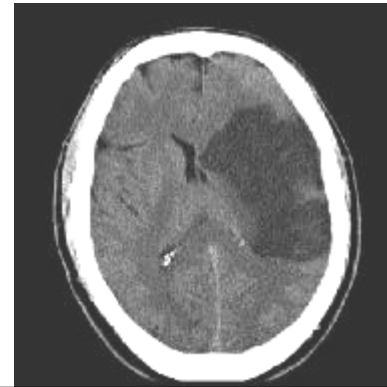
Cerebrovascular Disease

Mrs. Ida Wind is a 69 year old widow who is brought into the ER by her son after she complained of dizziness. Her son noted that she was not able to use her right arm, and that her speech was slurred and slow. She was admitted with an initial diagnosis of r/o CVA.

What is a CVA?

A cerebrovascular accident (CVA) occurs when blood is not delivered to part of the brain. They can be caused by functional constriction or obstruction of a blood vessel. There is a lack of oxygen and glucose delivery to the affected area of the brain, and this results in neurons becoming seriously damaged or dying.

Thrombotic strokes are caused by a blood clot that has formed in a cerebral vessel. About 60 % occur during sleep, possibly because the blood pressure decreases during sleep, which would lead to more sluggish blood flow. Blood abnormalities such as hypercoagulability and polycythemia are another contributing cause. The onset of symptoms is gradual with periods of progression and improvement. Gradual development of the symptoms is related to the edema associated with infarctions, and reaches a peak in one to three days.



Cardiovascular Accident (CVA): Day 5 after a brain infarction or ischemia in the area supplied by the right middle cerebral artery.

A *cerebral embolism* occurs suddenly with no warning. It is not related to activity level and is most often seen in younger patients. The embolus is usually a blood clot, but it can also be a plaque that has broken off from an atherosclerotic blood vessel. In approximately 50% of all cases, the emboli will break up within 12 hours, and symptoms will resolve. Factors contributing to emboli development are often cardiovascular in nature. Atrial fibrillation, mitral or aortic valve vegetation, bacterial or rheumatic endocarditis, and congenital heart disease all can predispose a person to developing a cerebral embolus. Patients with atrial fibrillation are five times more likely to have an embolic CVA.

Terminology

A **TIA** (*transient ischemic attack*) is brief and reversible. Symptoms may last only 2-15 minutes or may last up to 24 hours. TIAs may be associated with the development of collateral communicating vessels that compensate for the occlusion of one artery. TIAs often proceed to stroke at a later time. Common symptoms include:

- contralateral weakness of the face, arms, and legs
- sensory deficits (hemiparesthesias)
- visual impairment. If the ophthalmic artery is involved, the patient may lose sight in one eye for 2-3 minutes (amaurosis fugax)
- confusion or trouble speaking or understanding speech.

RIND stands for *reversible ischemic neurological deficit*. In this pathology, symptoms may last 24 hours to one week.

PRIND is a *partially reversible ischemic neurological deficit*.

A **progressing stroke** is one in which the symptoms are still evolving.

A **completed stroke** means symptoms no longer progressing; the neurological deficits are fixed.

What are the symptoms of a CVA?

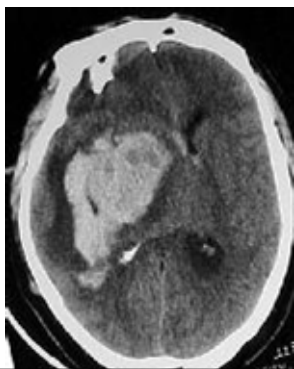
Symptoms of cerebrovascular accident (CVA) and potential for recovery will vary depending on the vessel involved and the degree of occlusion. CVAs have stereotypical deficits depending on whether they are right or left sided. Patients who sustain right CVAs will have more perception problems, memory deficits regarding new spatial information, apraxia, impaired judgment, and increased emotional lability. Left CVAs result in possible expressive and/or receptive aphasia and increased concern over disability. Spatial relationships are generally intact.

Intracranial Bleeding

Trauma, hypertensive bleeds, aneurysms, AVM's and tumors are the top causes of intracranial bleeding. In the case of trauma, falls and motor vehicle crashes are the leading causes. All types of bleeding are exacerbated if the patient is on anti-coagulant therapy.

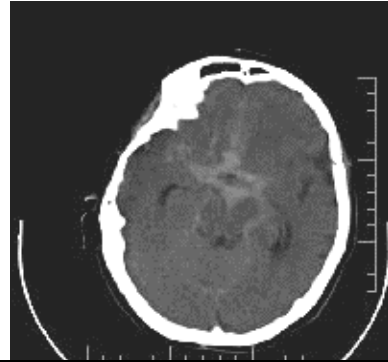
After thrombosis and emboli, intracranial bleeds are the third leading cause of CVAs. The bleed may be intracerebral or subarachnoid. The major cause of spontaneous bleeding into the brain parenchyma is hypertension.

Intracerebral bleeds result from the rupture of a small artery; most often a deep, penetrating vessel. It usually occurs due to sustained pressure within the vessel due to hypertension. This will weaken and eventually rupture the vessel. CSF is usually clear initially, but often blood eventually seeps into the ventricular system.



Hemorrhagic Stroke: Large intracerebral hemorrhage with midline shift.

Subarachnoid hemorrhage (SAH) has a sudden and severe onset. Prodromal warning signs include localized headache, dizziness, visual changes, eye and/or face pain, stiff neck, and cranial nerve III palsy. A patient with SAH will have a severe headache. Nuchal rigidity occurs due to blood in subarachnoid space and meningeal irritation. As ICP decreases, neurologic changes occur. Seizures, nausea/vomiting, and dysrhythmias occur due to sympathetic response.



Subarachnoid hemorrhage (SAH)

A ruptured AVM (arteriovenous malformation) or aneurysm may also bleed into the brain. Symptoms usually evolve abruptly during activity and rapidly progress. There is usually a severe headache, and symptoms may progress to other neurological changes that correspond to the area and extent of involvement.

Cerebral Aneurysms

Aneurysms are small, thin-walled blisters that are usually located at the bifurcation of vessels. The main cause of cerebral aneurysms is a developmental defect in the vessel wall that results in a saccular outpouching at the weakened area. This accounts for 95% of aneurysms that rupture.



Cerebral aneurysm: This CT scan demonstrates a large area of old hemorrhage and encephalomalacia (dark area) in the right temporal lobe as a consequence of a ruptured aneurysm.

Less common than developmental aneurysms are fusiform aneurysms. These develop in response to atherosclerotic degenerative changes of the cerebral vessels; arteries become thin and fibrous. Fusiform aneurysms tend to occur later in life, often as a result of long-term hypertension.

Aneurysms vary by size and form. They can be small (<15 mm) or super giant (> 50 mm). About 15-20 percent of people with aneurysms have multiple aneurysms. Aneurysms can go undetected for many years, only becoming evident when they rupture or compress adjacent nerve tissue causing focal cerebral disturbances (act like a mass lesion). At rupture, blood is often forced into the subarachnoid space. Rupture usually occurs with activity (straining, sports, working, etc.)

Berry aneurysms most frequently form in the Circle of Willis and affect both sexes equally. Rupture usually occurs between 30 and 60 years of age. Fusiform aneurysms generally affect those over 50 years of age.



Berry aneurysm at the Circle of Willis



Fusiform aneurysm



Dissecting aneurysm

Arteriovenous Malformation (AVM)

An arteriovenous malformation is a vascular malformation in which veins and arteries appear to connect without an intermediary capillary bed. The vessel walls are very thin; vessels are tortuous and dilated. The malformed arteries and veins do not allow blood perfusion to the surrounding tissue. AVMs are usually congenital defects that become evident between 10 and 30 years of age. They seem to occur slightly more often in men.

Although AVMs can occur in all parts of the CNS, 40 percent occur in the supratentorial area and involve the cerebral hemispheres. Ten percent occur in the cerebellum and brain stem. They vary in size and commonly lead to degeneration of the brain parenchyma (area between vessels). Small AVMs are < 3 cm; medium are 3-6 cm; and large are > 6 cm.

Symptoms of an arteriovenous malformation (AVM) will vary depending on its size, shape, and location. Usually, the patient presents with seizures and/or headache. The

headache does not respond to drug therapy. There may be a bruit over the AVM, scalp vein dilation, or cutaneous hemangioma. This dilated mass of vessels can increase ICP or cause ischemic steal by pulling blood from other areas of the brain. Cardiomegaly or cardiac decompensation may occur due to the increased blood flow demands.

Because the capillary bed normally provides resistance to blood flow, AVMs are prone to rupture because the blood goes straight from high resistance vessels to low resistance vessels. It is estimated that between 2 and 4 percent of all AVMs hemorrhage. Mortality with initial rupture is 10 percent, and approximately six percent will re-bleed within a year. After the first year post-rupture, risk of re-bleeding stabilizes at about three percent per year with a risk of death at about one percent per year.

Medication can often alleviate general symptoms such as headache, back pain, and seizures caused by. However, the definitive treatment for AVMs is either surgery or focused irradiation therapy.

Primary Brain Tumors

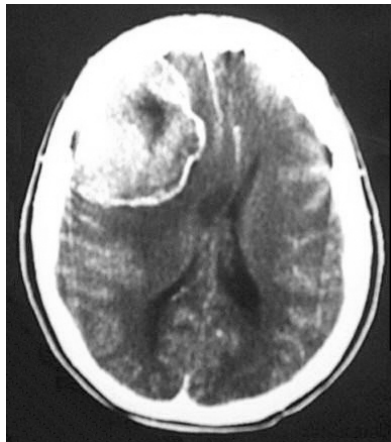
Brain tumors occur in people of all ages; however, peak ages for incidence occur in children under 15 and adults in the 5th, 6th and 7th decades of life. Brain tumors have historically been classified in a variety of ways. The most utilized classification is the system of grading I through IV. The grade I tumor has relatively well differentiated cells similar to normal tissue and is usually considered benign. Grade I tumors rarely spread, and it may be possible to remove the tumor in its entirety. Grade II becomes more atypical, and grades III and IV tumors are increasingly malignant with rapid cell replication.

Benign Tumors

Acoustic Neuromas are slow growing benign tumors of the 8th cranial nerve located in the posterior fossa. This tumor typically occurs in middle-aged adults and accounts for more than 5% of primary brain tumors. Symptoms include hearing loss in one ear, tinnitus, ataxia; if adjacent to the 7th facial nerve, facial paralysis may also occur. These tumors can most often be completely removed surgically. Radiation therapy is also used in the management of these tumors.

Approximately 15% - 20% of all primary tumors are *meningiomas*. They usually occur during middle adult years and are more common in women. Symptoms often include focal seizures; progressive, spastic weakness in

the legs; incontinence; and increased intra-cranial pressure. These slow growing tumors arise from the meningeal covering of the brain and are highly vascular. Meningiomas may be difficult to surgically remove when adjacent to vital structures. In such cases, radiation therapy may be of value.



Brain meningioma:

Nonenhanced CT scan shows a malignant meningioma in the frontal convexity.

Pituitary Adenomas are benign, slow growing tumors that represent about 15% of all intracranial tumors. The pituitary gland is considered to be the “master gland”. It secretes hormones that regulate other glands. These tumors most commonly occur in young or middle aged adults.

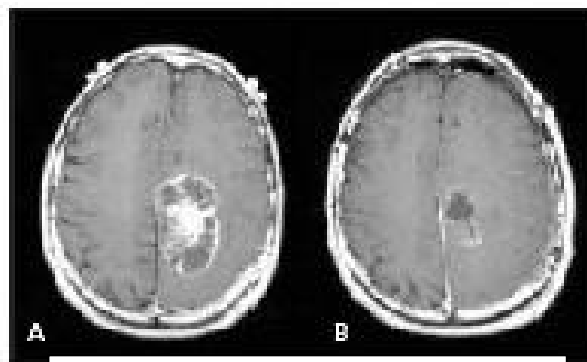
Classification of Pituitary Adenomas

Name	Hormone	Symptoms
Prolactinoma	Prolactin	Women: breast milk production, change in menstrual period. Men: breast enlargement, impotence.
Cushing's disease	ACTH & Cortisone	Weight gain, high blood pressure
Acromegaly	Growth Hormone	Enlarging tissue and organs, diabetes, gigantism
Hyperthyroidism	TSH & Thyroid	Weight loss, irritability, heat intolerance

Treatment includes administering medication that slows tumor growth as well as suppresses the tumor’s secretion of excess hormone. Surgical removal is standard treatment for tumors that are not controlled by drug therapy. Radiation therapy may be necessary for any remaining tumor tissue. Hormonal replacement therapy often follows surgical removal.

Malignant Tumors

Astrocytomas are tumors composed of astrocytes found mainly in the cerebral hemispheres. Astrocytomas are usually classified further into the grade I - IV system. A Grade I astrocytoma is a relatively benign tumor. A Grade IV astrocytoma is called a *glioblastoma multiforme*: a highly malignant tumor which grows rapidly and produces considerable edema. These tumors usually are too invasive to control surgically. Early symptoms include focal or generalized seizures. Headache and increased intra-cranial pressure with vomiting occur later in the disease. Radiation therapy is most often required after biopsy or surgery.



MRI:
Glioblastoma multiforme (GBM) before and after surgical therapy.

Oligodendrogliomas are rare tumors derived from oligodendrocyte cells. The oligodendrocyte cells support and nourish nerve cells. More commonly found in middle aged men, the first symptom is often a seizure. They may also develop increased intra-cranial pressure and headaches. Depending on the tumor location, they may also have visual loss, motor weakness and cognitive deterioration. Surgical removal usually produces a relatively good prognosis.

Ependymomas arise from ependymal cells lining the ventricles. Most tumors of this type are found in patients in their late teens. Hydrocephalus may be the consequence of these tumors and results in such symptoms as headache and nausea and vomiting. Ependymomas are often difficult to completely remove surgically. Standard treatment includes radiation therapy and a shunt procedure; a shunt usually relieves the increased intracranial pressure caused by the hydrocephalus.

Cranial Surgery

Craniotomies for intracranial tumors or other lesions (e.g. hematomas) begin with an incision through the scalp, underlying muscle and periosteum. The tissue is pulled

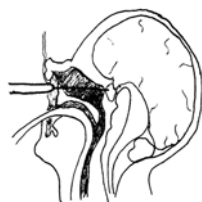
away from the skull to form a flap. Burr holes (drill holes) are used to first enter the skull. **Burr holes may be used alone** when the objective is to evacuate a hematoma, control hemorrhage, perform a biopsy, insert a ventriculostomy or drain an abscess. More extensive craniotomies are accomplished by making a series of burr holes and extending them with a rongeur and using a saw to incise the skull between the burr holes. Once the bone is removed, the underlying dura is cut to expose the brain. Depending on the purpose of the craniotomy, part of the cranium may be removed and a cranioplasty performed. **Cranioplasty** involves replacing the excised bone with synthetic material or transplants of cadaver bone. After the surgical procedure is completed the dura is sutured closed and the cranial bone is held in place with sutures that are sewn through small drill holes in the bone and tissue flap. The skin is then either sutured or stapled closed.

Craniotomies may be classified according to where they are made (temporal, occipital, frontal or parietal) or described as supratentorial or infratentorial.

Supratentorial approaches access lesions above the tentorium in the cerebral hemispheres or the midbrain.

Infratentorial approaches access the cerebellum, medulla and pons.

The *Transsphenoidal* approach is used to remove tumors located within the sella turcica and pituitary adenomas. An incision is made under the upper lip and extended into both sides of the nasal septum. Dissection posteriorly through the floor of the sphenoid sinus allows access to the sella turcica, and then the dura is entered. Surgery is performed with special micro-instruments used via a microscope. A small muscle graft donated from the thigh is used to close the dura. The gum is sutured closed and the nasal septum is packed with petroleum gauze.



Transsphenoidal Approach

Skull Based approaches (combined head and neck with cranial approach) have only recently been considered safe neurosurgical approaches to tumors that lie along the



Supratentorial Approach (along dotted line)



Infratentorial Approach (along dotted line)

internal surface of the base of the brain. This approach usually requires a combined cooperative effort of a neurosurgeon, an otolaryngology surgeon and sometimes a plastic surgeon. The skull base is very difficult to access because it is rich in vital structures and takes great surgical skill to locate and identify. Consequently, these procedures carry a significant morbidity rate.

Seizures

A seizure is a sudden, uncontrolled episode of excessive electrical activity in the brain. This sudden burst of electrical activity produces an alteration of behavior, consciousness, movement, perception, and/or sensation. Under the right set of circumstances, anyone can have a seizure. In fact, 1 in 11 people will have a seizure at some time in their lives. Some people have only one seizure (e.g., a febrile seizure); some will have recurrent seizures.

Epilepsy is a CNS disorder characterized by a tendency to have recurrent seizures. Two million people in the U.S. have epilepsy. Researchers estimate the prevalence at one to two percent of the population. The cause of epilepsy may be genetic, gestational, infectious, traumatic, neoplastic, or vascular.

Primary Seizures = Idiopathic Epilepsy

For primary seizures, no cause can be determined. Heredity is thought to be an influence in development of primary seizures, but the role is not understood. There may be a predisposition for seizures. About one percent of the general population has primary seizures, but that number rises to 6% if one parent has idiopathic epilepsy and 10% if both parents have it. Idiopathic epilepsy is more common in children and teens.

Secondary Seizures = Symptomatic Epilepsy

Secondary seizures (symptomatic epilepsy) result from an isolated cause: metabolic, electrolyte problems; hypo/hyperglycemia; hypoxia, anoxia; drugs; CNS infection; CNS trauma; brain tumor; or sleep deprivation. Symptomatic seizures are more common in adults.

Types of Epileptic Seizures

Seizure types are classified by the physical and electrical activity manifested during the seizure. Differential diagnosis of seizure type is important in properly managing the disorder. The classification system generally recognized for diagnosis of symptom types is the International Classification of Epileptic Seizures (updated 1981). The classification is divided into three sections: generalized, partial, and unclassified. Seventy-

five percent of all seizures can be classified; 25% are unclassified.

Partial seizures (focal or local)

Partial seizures are the most common form of seizures, accounting for the seizures experienced by about 70% of adults and 40% of children with epilepsy. Partial seizures are focal in origin; they derive from a limited portion of the brain. Symptoms correspond to activity of the affected area.

Partial seizures evolving to generalized tonic-clonic seizures

Partial seizures may spread from the original discharge site to other parts of the brain and become a generalized seizure. This type of seizure should be considered focal rather than general, since it is therapeutically useful to classify a seizure based on its initial manifestations. Any seizure preceded by a specific aura is a partial seizure that becomes secondarily generalized. A complex partial seizure that generalizes to a generalized, tonic-clonic seizure is treated differently from a seizure that is generalized from the onset, even though both types of generalized seizures look the same.

Generalized seizures

Generalized seizures involve symmetric (both hemispheres) distribution of abnormal brain discharge; bilateral motor changes. Generalized seizures (convulsive or nonconvulsive) involve the brain bilaterally and are associated with loss of consciousness.

Status epilepticus (SE)

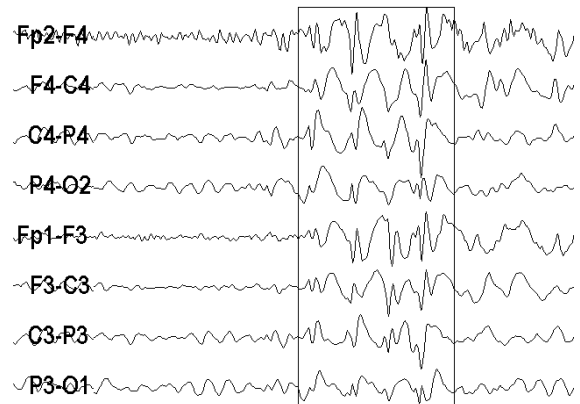
Status epilepticus is a state of continuous epileptic seizure activity (traditionally defined as 30 minutes) without return to full consciousness between seizures.

Approximately 50,000-200,00 people a year have an episode of SE. In absence seizures, SE manifests itself as a fixed stare and unresponsiveness with perhaps a slight flickering of the eyelid (seen in children and adolescents). It is also possible to have status complex partial seizures.

Although medical attention is needed with both these types, they are usually not life threatening. However, the most common type of SE is tonic-clonic (seen in adults). Generalized tonic-clonic (GTC) status epilepticus requires immediate medical attention. It is a state of continuous or repetitive GTC . seizures without an intervening return of consciousness. Under these conditions, the patient usually has respiratory impairment. This is life threatening, putting the patient at severe cardiorespiratory risk.

Just as there are a variety of causes for seizures, there are several possible causes for SE.

- Sudden drop in plasma levels/withdrawal of seizure drugs (most common cause).
- Acute central nervous system disorders: meningitis, encephalitis, or subarachnoid hemorrhage.
- Metabolic disturbances (encephalopathies) and heart failure.
- Chemical withdrawal (drugs or alcohol) -- often refractory to therapy and difficult to treat.



An EEG recording from a patient with primary generalized epilepsy. A burst of bilateral spike and wave discharge is shown in the rectangular area.

First Aid for Seizures

In general, the person should not be restrained. Nothing should be placed in the mouth. Remove all sharp or dangerous objects from the area to prevent injury. Place them on their side so secretions are not aspirated. Seizures that last longer than five minutes are a medical emergency and 911 should be called.

Spinal Cord Injury

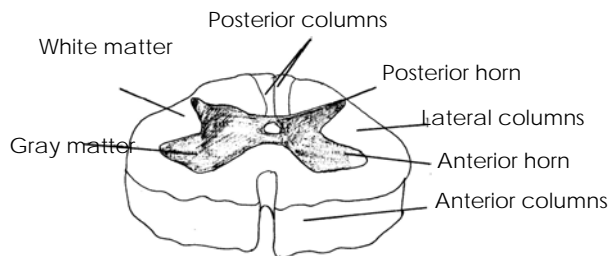
Approximately 11,000 new spinal cord injuries (SCI) occur annually in the US with the majority of victims being males, aged 15 - 25 years old. Motor vehicle accidents, falls, assaults, sports injuries, and diving accidents account for most SCI today. Injury usually occurs in the areas of greatest mobility, e.g. cervical or lumbar regions.

Appropriate handling of the SCI patient in the field can limit neurological deficits. The decreased incidence of quadriplegia compared to paraplegia in the past few years can probably be attributed to improvement in emergency management.

What are the structures of the spinal cord?

The spinal cord is continuous with the medulla in the brain. The main function of the spinal cord is to act as a “highway” for information traveling to and from the brain. There are two parts to the highway: (1) the gray matter, and (2) the white matter. The **gray matter** is in the center of the spinal cord, and contains the anterior horn (motor fibers), the lateral column (autonomic nervous system fibers), and the posterior horn (sensory fibers).

The **white matter** surrounds the gray matter and is separated into three longitudinal columns, also called funiculi. *Think of these columns as lanes in the highway.*



The **ascending tracts** are the lanes through which sensory information is carried up into the brain where it is processed. These tracts have the following responsibilities:

- **Dorsal tract:** Sensation of fine touch, vibration, pressure, proprioception
- **Spinocerebellar tract:** Position sense
- **Spinothalamic (ventral) tract:** Sensation of crude touch and pressure
- **Spinothalamic (lateral) tract:** Sensation of pain and temperature

The **descending tracts** are the lanes through which motor information is carried from the brain through the spinal cord to the nerves which stimulate the muscles. These tracts have the following responsibilities:

- **Corticospinal:** Major motor movement
- **Extrapyramidal (reticulospinal, rubrospinal, vestibulospinal):** Carry information to either facilitate or inhibit motor function and tone

What are the types of spinal cord injuries?

A **Complete SCI** describes an injury where the patient has complete loss of motor and sensory function due to complete interruption of motor and sensory pathways

below the level of injury. There is no function below the level of the injury, and both sides are affected equally. This gives the worst prognosis.

An **Incomplete SCI** is partial interruption of motor and sensory pathways, resulting in loss of some motor and /or sensory function with some sparing of function below the level of injury. They may be able to feel parts of the body that they cannot move or move parts they cannot feel.

Incomplete SCI can further be classified into the following syndromes:

- **Central Cord Syndrome:** The center of the spinal cord is injured. Typical mechanism of injury is hyperextension, with stretching and hemorrhage into the center of the cord. Here the sensory and motor pathways of the upper extremities are more impaired than the lower extremities because they are controlled by the central portion of the cord. The hands and arms are paralyzed, while the legs and lower extremities are normal.
- **Brown Sequard Syndrome:** Just one side of the spinal cord is damaged. Because of the spine’s anatomy and physiology (motor tracts cross over at the brain stem and sensory tracts cross over at the level of entry to the spinal cord) the injury produces an unusual clinical presentation. On the side of the body with the cord lesion, the patient loses motor function and proprioception but can sense pain and temperature. On the opposite side of the body, motor function and proprioception (sense of position in space) are preserved, but pain and temperature sensation are lost. *The extremities that can move can’t feel and those that can feel can’t move.*
- **Anterior Cord Syndrome:** This develops from disrupted blood flow through the anterior spinal artery. Only the dorsal column, which controls proprioception, is fully preserved. The patient with anterior cord syndrome has a mixed loss of sensory and motor function below the level of the cord lesion, but proprioception remains intact. Generally, motor function, pain sensation and temperature sensation is lost below the injury, while touch and proprioception remain intact.

The peripheral nervous system is damaged with a SCI -- what is normal?

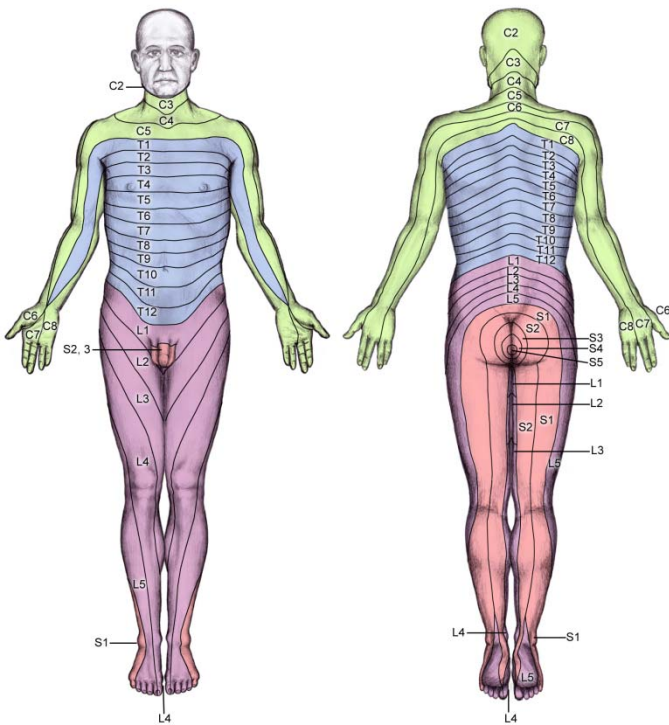
The peripheral nervous system takes up where the central nervous system leaves off. The peripheral nervous

system consists of the nerves that extend out of the brain and spinal cord and serve the limbs and organs.

Information from the spinal tracts and the brain are transmitted through the **spinal nerves** to the end organs; information from the organs and tissues are transmitted through the spinal nerves to the spinal cord and brain for processing. There are thirty-one pairs of spinal nerves which extend from the spinal cord, each having a sensory (dorsal) root and a motor (ventral) root. The spinal nerves extend through the spinal foramina of each vertebra to the peripheral skin, tissues, and organs.

Each spinal nerve supplies sensory information to a certain amount of area; these areas are called **dermatomes**.

Dermatome Map



Part of the peripheral nervous system is the autonomic nervous system. The autonomic nervous system is made up of two opposing divisions: the sympathetic nervous system and the parasympathetic nervous system.

In the **Sympathetic Nervous System**, the axons leave the spinal cord (rather than the brain) at the T1 to L2 levels and travel to the ganglion (pre-ganglionic fibers). At the ganglion, the axon passes through to the different organ systems (post-ganglionic fibers). The SNS axons stimulate action by the chemical mediation of adrenergic receptors which release **norepinephrine**. The

sympathetic nervous system stimulation causes a massive response of all organ systems under its control, causing a “fight or flight” response.

The SNS:

- Increases the heart rate and contractility
- Dilates heart & skeletal muscle blood vessels
- Constricts the blood vessels of the skin, viscera, and external genitalia
- Increases the blood pressure
- Dilates the bronchioles
- Increases respirations
- Decreases peristalsis
- Increases the conversion of glycogen to glucose
- Produces thick, viscous saliva
- Increases perspiration
- Causes the secretion of epinephrine and norepinephrine
- Relaxes the bladder and contract the sphincters
- Dilates the pupil
- Contracts the pilomotor muscles of the skin (goose bumps)

The **Parasympathetic Nervous System** (PNS) fibers originate in the brain stem and in the sacral portion of the spinal cord. The actions of the PNS directly oppose the actions of the SNS. The actions of the PNS are mediated by acetylcholine (cholinergic receptors), and tend to be organ specific, rather than causing a mass effect. The PNS:

- Decreases the heart rate and contractility
- Constricts the pupils
- Contracts the muscles of stomach, intestine and bladder
- Stimulates secretion of most glands

What are the complications of an acute spinal cord injury?

Neurogenic Shock : Dominance of the parasympathetic nervous system develops when the sympathetic nervous system can't send impulses past the spinal cord lesion. The three primary symptoms of neurogenic shock are hypotension, bradycardia and hypothermia. Loss of vasomotor tone results in hypotension from vasodilatation, and increased vagal tone causes bradycardia. The skin stays warm and dry. Neurogenic shock can be differentiated from hypovolemic shock because tachycardia does not occur.

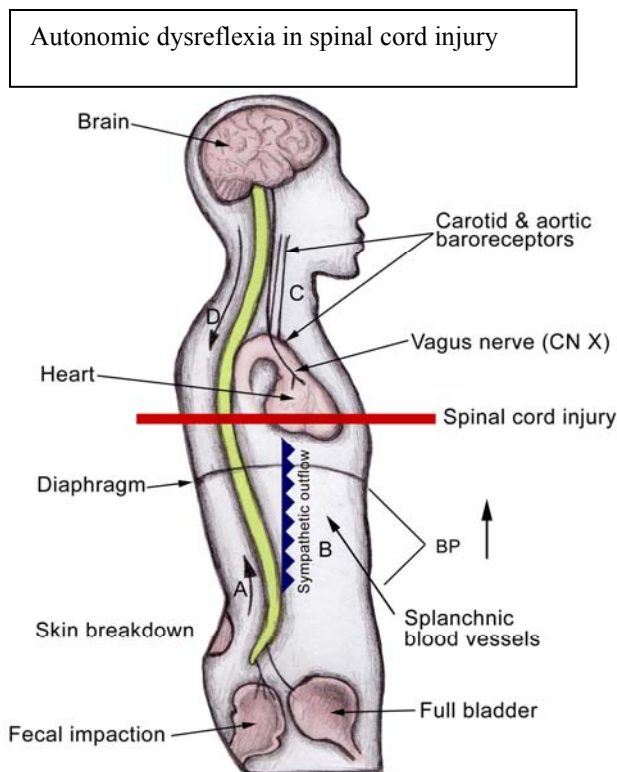
Pulmonary Complications are the number one cause of death after SCI. This is most commonly related to impaired ability to cough resulting in atelectasis, hypoventilation and mucous plugs. The incidence of DVT, resulting in pulmonary embolism, is also extremely

high in SCI related to flaccidity of the lower extremities with loss of skeletal muscle pump.

Autonomic Dysreflexia (hyperreflexia) is a hypertensive crisis that may occur from noxious stimuli. This only occurs in patients with lesions above T6 (the sympathetic outflow level). When noxious stimuli occurs below the level of injury, the sympathetic nervous system is triggered to release massive amounts of catecholamines causing vasoconstriction. Increased BP stimulates baroreceptors in the aortic arch and carotid sinus to send inhibitory signals to the medulla.

These inhibitory impulses cannot cross the cord lesion; instead, vasodilation occurs above the lesion. The patient complains of red, flushed, warm skin; headache; and nasal congestion. Below the lesion the skin is cold, producing goose bumps. Vagal stimulation causes bradycardia as a compensatory mechanism to decrease cardiac output and BP, but it usually can't bring the BP down to normal. Hypertension left unchecked may lead to MI, stroke or subarachnoid hemorrhage.

Offensive stimuli, such as a kinked Foley causing a distended bladder, fecal impaction, or tight clothing, cause this problem.



Disorders of Antidiuretic Hormone Production

Antidiuretic hormone (ADH), also known as arginine vasopressin, is secreted by the posterior pituitary. ADH release by the pituitary is dependent on osmoreceptor stimulation. When blood volume decreases and osmolality increases, osmoreceptors in the hypothalamus trigger the release of ADH from the pituitary. ADH affects the kidney by attaching to receptor sites on the collecting tubules, enhancing permeability and permitting reabsorption of solute free water and decreased water excretion.

The Syndrome of Inappropriate Antidiuretic Hormone (SIADH), results in the inappropriate production or secretion of ADH. SIADH most often develops as a result of injury to the hypothalamic-neurohypophyseal system. Most common causes include: brain tumor or abscess, SAH, head injury, meningitis, encephalitis, or Guillain-Barré syndrome. Although the precise mechanism is not clearly understood, an altered rate of water excretion develops as a result of an abnormally high blood level of ADH in comparison to the serum osmolality.

SIADH is characterized by hyponatremia (sodium <math><135\text{mEq/L}</math>). A low serum osmolality (<math><280\text{ mOsm/kg}</math>) is also present and the urine is concentrated. Treatment may include water restriction, diuretics and the administration of hypertonic saline.

Diabetes Insipidus (DI) is a clinical condition resulting from either a deficiency of ADH or renal unresponsiveness to ADH.

Nephrogenic DI, which is rare, occurs when the kidney is resistant to the water conserving effects of ADH. Nephrogenic DI may be caused by an inherited disorder in males, by kidney disease (such as polycystic kidney disease) and certain drugs such as lithium.

Central (neurogenic) DI develops when there is a lack of circulating ADH and occurs as a result of a dysfunctional hypothalamus or pituitary gland. The most common cause of Central DI is damage to the neurohypophysis (posterior pituitary) from surgery or trauma.

Regardless of the cause, the kidneys are unable to conserve water and this results in frequent urination and pronounced thirst.

The classic sign of DI is polyuria. The patient excretes excessive amounts of urine, regardless of fluid intake. Urine specific gravity is constant at 1.005 or less and

urine osmolality level is low. The serum osmolality and serum sodium levels are increased. The alert patient may complain of general weakness, polydipsia and polyuria. If left untreated, the patient may quickly become extremely dehydrated. DI is usually transient, but can take days to a few weeks to resolve.

Other Neurological Disorders

Myasthenia Gravis is an autoimmune disorder that causes skeletal muscle weakness. Normally, the neurotransmitter substance called acetylcholine transmits the message from the nerve to the muscle and causes the muscle to contract. In myasthenia gravis, antibodies have destroyed the acetylcholine receptors, and this prevents muscle contraction from occurring.

Often, the first noticeable symptom is weakness of the eye muscles. In others, difficulty swallowing and slurred speech may be the first symptoms. The degree of muscle weakness can vary greatly from patient to patient.

Symptoms, which vary in type and severity, may include*:

- a drooping of one or both eyelids (ptosis),
- blurred or double vision (diplopia) due to weakness of the muscles that control eye movements
- unstable or waddling gait
- weakness in arms, hands, fingers, legs, and neck
- a change in facial expression
- difficulty in swallowing
- shortness of breath
- impaired speech (dysarthria)

*(http://www.ninds.nih.gov/disorders/myasthenia_gravis/detail_myasthenia_gravis.htm)

Because weakness is a common symptom and can sometimes be rather vague, diagnosis may be delayed by a year or two. In addition to symptoms, the physician may order a blood test to check for antibodies to acetylcholine receptors or perform electromyography (EMG) to assess the muscle response to electrical stimulation. Another test is called the edrophonium test. Edrophonium is a drug that blocks the breakdown of acetylcholine, and will cause a brief improvement in the symptoms of muscle weakness.

There are several medications available that can help reduce muscle weakness. Neostigmine and pyridostigmine, help to improve neuromuscular transmission and increase muscle strength. Immunosuppressive drugs such as prednisone,

cyclosporine, and azathioprine may also be used. These medications improve muscle strength by inhibiting the production of abnormal antibodies.

A patient may be admitted to ICU when a myasthenic crisis occurs. A myasthenic crisis happens when the muscles that control breathing are so weak, that ventilation is impaired, and the patient experiences respiratory failure.

Today, the prognosis for myasthenia gravis is good. Medications can significantly improve the muscle weakness, and the disorder may even go into remission.

Meningitis is an inflammation of the meninges, the layers of connective tissue that cover the brain. Infection with bacteria or viruses is the most common cause.

Enteroviruses are the most common cause of **viral meningitis** and are spread through direct contact with respiratory secretions.

Two of the most common microorganisms that cause **bacterial meningitis** are *Streptococcus pneumoniae* and *Neisseria meningitidis*. Meningococcal meningitis is the term used to refer to meningitis caused by *Neisseria meningitidis*. Bacterial meningitis has a high mortality rate if not treated.

Some types of bacterial meningitis can be prevented with the following vaccinations:

- Haemophilus influenzae type b (Hib) vaccine
- Pneumococcal conjugate vaccine (PCV7)
- Pneumococcal polysaccharide vaccine (PPV)
- Meningococcal conjugate vaccine (MCV4)

The classic symptoms of meningitis are headache, nuchal rigidity (a stiff neck), fever and mental status changes. When these symptoms are present, it is a medical emergency.

Acute bacterial meningitis requires prompt treatment with intravenous antibiotics to reduce the risk of complications such as cerebral edema, shock, seizures and dehydration.

Since antibiotics are not effective for viral meningitis, treatment usually includes bed rest, plenty of fluids and pain medications to relieve pain and reduce the fever. Often, patients with viral meningitis improve on their own in a week or so.

Encephalitis is generally caused by a viral infection that results in an inflammation of the brain. There are two types of encephalitis: primary and secondary. When the infection starts in the brain, it is called primary encephalitis. Secondary encephalitis occurs when the

viral infection starts elsewhere in the body and travels to the brain.

Some of the more common causes of encephalitis include:

- Herpes simplex virus
- Varicella-zoster virus
- Epstein-Barr virus
- Arboviruses (viruses that are transmitted by mosquitos and ticks)
 - Eastern equine encephalitis
 - Western equine encephalitis
 - St. Louis encephalitis
 - La Crosse encephalitis
 - West Nile encephalitis

Mosquito borne encephalitis can affect anyone, but is more common in the summer months and in areas of the country where these viruses are more prevalent.

In addition to viral infections, bacteria such as borrelia burgdorferi, which causes Lyme disease, can also cause encephalitis.

Symptoms of encephalitis range from mild and non-descript to life threatening. Mild symptoms include headache, irritability and sleepiness. These mild symptoms may be followed by confusion, seizures, fever, severe headache, and nausea & vomiting. The classic symptom is nuchal rigidity (a stiff neck) and should not be ignored. In infants, bulging of the fontanel can occur.

Diagnosis may be accomplished through a lumbar puncture to look for signs of infection, a CT or MRI to determine if there is swelling of the brain or a blood test as in the case of West Nile virus.

Treatment can be difficult because of the limited antiviral drugs available. Two antiviral drugs that may be tried include acyclovir and ganciclovir.

Wearing long-sleeved shirts and pants, applying mosquito repellent and avoiding the outdoors between dusk and dawn when mosquitoes are most active best prevents mosquito-borne encephalitis.

Brain Abscess occurs when there is a bacterial or fungal infection in the brain. The infection usually starts outside of the brain and travels to the brain via the circulatory system. Brain abscesses are uncommon, but do occur more frequently in immune compromised patients.

Symptoms of a brain abscess may develop slowly over a period of 2 weeks or develop suddenly. Symptoms include the following:

- Headache

- Stiff Neck
- Changes in mental status
- Seizures
- Fever/Chills

A brain abscess is a medical emergency, because it causes an increase in intracranial pressure. The increase in intracranial pressure occurs when the brain swells and the abscess puts pressure on the brain tissue.

A brain abscess may be diagnosed with a head CT or MRI. The organism causing the brain abscess may be identified through a needle biopsy.

Treatment generally includes antibiotics or anti-fungal medications. Surgery to drain the abscess may also be indicated.

Guillain-Barré Syndrome is the result of an auto-immune response to an infection or another foreign antigen such as a vaccine. The peripheral nerves become inflamed and a demyelinating neuropathy occurs.

Guillain-Barré is characterized by ascending paralysis. The weakness starts in the legs and moves upward to the arms and face. There may be problems with swallowing and maintaining an airway. Symptoms of Guillain-Barré generally occur 2 – 4 weeks following a respiratory or gastrointestinal illness. Most patients Guillain-Barré require hospitalization and approximately 30% require mechanical ventilation.

Treatment includes:

- Plasma exchange therapy and intravenous immune globulin (IVIG)
- Corticosteroids
- Mechanical ventilation

Following recovery, the patient will likely require intense rehabilitation to regain function and the ability to perform ADL's.

Amvotrophic Lateral Sclerosis (ALS) or Lou Gehrig's Disease is a progressive, fatal degenerative disease of the nervous system. The onset is usually about the age of 50. As the nerve cells that control voluntary muscle movement die, the muscles become weak and atrophied. Ultimately, the patient is unable to control voluntary muscle movement. The etiology of ALS is unknown.

Symptoms for most people starts with limb weakness with the weakness spreading to other body parts as the disease progresses. Other symptoms include difficulty swallowing, difficulty speaking, muscle twitches and difficulty breathing. Ultimately, the result is paralysis, and the patient requires mechanical ventilation. Many

patients chose to forgo mechanical ventilation and ultimately die of respiratory failure or pneumonia.

Although there is no cure for ALS, the medication Riluzole (Rilutek) has been found to reduce the damage to the motor neurons, thus lengthening the time before a patient needs mechanical ventilation and prolonging survival by several months. Other treatments include symptom relief and supportive care such as physical therapy, speech therapy and the possibility of using mechanical ventilation.

Most people die within 3-5 years of diagnosis from the respiratory failure associated with ALS.

Summary

The brain and spinal cord arguably have the most complex anatomy and physiology in the body. Understanding the basics of how the nervous system works can help you in providing care to your patients with brain and spinal cord injuries.

Directions for Submitting Your Post Test for Contact Hours

To obtain a certificate of completion for this home study program, please complete the post-test and evaluation on the next few pages. If you are completing this home study as pre-reading for a TCHP class, please bring your post-test and evaluation to class with you for processing. The date on your certificate of completion will be the date that your home study is received. **Any materials received with a postmark after the expiration will be discarded.**

HealthEast, HCMC, & MVAMC Employees

If you are an employee of HealthEast, HCMC, or MVAMC, you may send the post-test and evaluation to TCHP for processing. Your post-test will be returned to you through your hospital. It cannot be mailed to your home.

Paid Participants

If you are not an employee of one of the TCHP hospitals, please send the post-test and evaluation to TCHP with a check for \$15.00. Please make check payable to **TCHP Education Consortium** and mail to:

**TCHP Education Consortium
Capitol Office Building
525 Park Street, Suite 120
St. Paul, MN 55103**

Your post-test will be returned to you with the certificate of completion.

Neurological Critical Care Primer Post Test

Please print all information clearly and sign the verification statement:

Name _____
(please print legal name above)

Birth date (required)

Format: 01/03/1999

M	M	D	D	Y	Y	Y	Y

For HealthEast, HCMC, or MVAMC, employees only:
Hospital _____ Unit _____

Personal verification of successful completion of this educational activity (required):

I verify that I have read this home study and have completed the post-test and evaluation.

Signature

- 1) Which of the following is a tissue that surrounds the brain?
 - a) meningeal covering
 - b) periosteum
 - c) venous sinus
 - d) falx cerebri
- 2) A subdural hematoma is:
 - a) bleeding that occurs below the dura mater
 - b) bleeding that occurs above the dura mater
 - c) bleeding that occurs within the brain
 - d) none of the above
- 3) What is the normal intracranial pressure?
 - a) 0-10 mm Hg
 - b) 11-20 mm Hg
 - c) 21-30 mm Hg
 - d) 31-50 mm Hg
- 4) According to the Monroe-Kellie hypothesis, if the volume of blood increases in the brain, what will decrease?
 - a) the amount of brain
 - b) the amount of CSF

- c) all of the above
- 5) An example of a malignant brain tumor is:
 - a) meningioma
 - b) glioblastoma multiform
 - c) adenoma
 - d) none of the above
- 6) A complete spinal cord injury is usually described as:
 - a) complete loss of motor and sensory function
 - b) vertebral injury
 - c) injury to the center of the spinal cord
 - d) disrupted blood flow through the spinal arteries
- 7) The major difference between DI and SIADH is:
 - a) the urinary sodium level
 - b) the serum osmolarity
 - c) the serum potassium level
 - d) the specific gravity
- 8) The most common cause of status epilepticus is:
 - a) withdrawal of seizure drugs
 - b) heart failure
 - c) alcohol withdrawal
 - d) meningitis
- 9) Symptoms of a TIA may last:
 - a) less than 30 seconds
 - b) up to 48 hours
 - c) one week
 - d) from two minutes to 24 hours
- 10) The area of the brain that is responsible for hearing, speech, memory and learning is the:
 - a) frontal lobe
 - b) occipital lobe
 - c) temporal lobe
 - d) parietal lobe
- 11) Nuchal rigidity (a stiff neck) is a classic symptom of:
 - a) meningitis
 - b) encephalitis
 - c) both a and b
 - d) none of the above

Expiration date: The last day that post tests will be accepted for this edition is **December 31, 2017**—your envelope must be postmarked on or before that day.

Evaluation: Neurological Critical Care Primer

Please complete the evaluation form below by placing an "X" in the box that best fits your evaluation of this educational activity. Completion of this form is required to successfully complete the activity and be awarded contact hours.

At the end of this home study program, I am able to:	Strongly Agree	Agree	Neutral	Disagree	Strongly Disagree
1. Describe the normal anatomy and physiology of the brain and spinal cord.					
2. Differentiate between the hematomas.					
3. Differentiate between the spinal cord injuries.					
4. Describe autonomic dysreflexia.					
5. Differentiate between SIADH and DI.					
6. Differentiate between the various seizures.					
7. Differentiate between a TIA, RIND, PRIND, progressive, and completed stroke.					
8. The teaching / learning resources were effective. <i>If not, please comment:</i>					

The following were disclosed in writing prior to, or at the start of, this educational activity (please refer to the first 2 pages of the booklet).

	Yes	No
9. Notice of requirements for successful completion, including purpose and objectives		
10. Conflict of interest		
11. Disclosure of relevant financial relationships and mechanism to identify and resolve conflicts of interest		
12. Sponsorship or commercial support		
13. Non-endorsement of products		
14. Off-label use		
15. Expiration Date for Awarding Contact Hours		
16. Did you, as a participant, notice any bias in this educational activity that was not previously disclosed? <i>If yes, please describe the nature of the bias:</i>		

17. How long did it take you to read this home study and complete the post test and evaluation:
 _____ hours and _____ minutes.

18. Did you feel that the number of contact hours offered for this educational activity was appropriate for the amount of time you spent on it?
 _____ Yes
 _____ No, more contact hours should have been offered
 _____ No, fewer contact hours should have been offered.

Expiration date: December 31, 2017