Hemorrhagic Stroke:

The KILLER Headache

Intended Audience

This course is designed for licensed stroke team members, including:

- Nurses (RNs/LPNs)
- CCCs and Social Workers in Care Management
- Physical Therapists, Occupational Therapists, and Speech & Language Pathologists
- Pharmacists
- Clinical Dieticians

Prerequisites/Special Considerations

- There are no prerequisites for taking this course
- There are no special considerations

Goal

- Contribute to increased knowledge about hemorrhagic stroke in members of the stroke team

Objectives

By the end of this CBL, the participant will be able to:

- Define hemorrhagic stroke, including ICH and SAH
- Discuss risk factors, possible causes, and pathophysiology of hemorrhagic strokes
- Review treatments and potential complications for patients with hemorrhagic stroke

Key Terms

- Hemorrhagic stroke
- Intracranial hemorrhage (ICH)
- Subarachnoid hemorrhage (SAH)
- Cerebral aneurysm
- Arteriovenous malformation (AVM)

Menu

Lesson 1: Hemorrhagic stroke

Lesson 2: Intracranial hemorrhage

Lesson 3: Subarachnoid hemorrhage

A. Cerebral Aneurysm

B. Arteriovenous Malformation

Lesson 1: Hemorrhagic Stroke

1. Define hemorrhagic stroke
2. Discuss risk factors for hemorrhagic stroke
3. Review cerebral circulation and most common sites of hemorrhagic stroke

Hemorrhagic Stroke

- DEFINITION: Escape of blood from a ruptured blood vessel within the brain or between the layers covering the brain, causing damage to nearby brain tissue

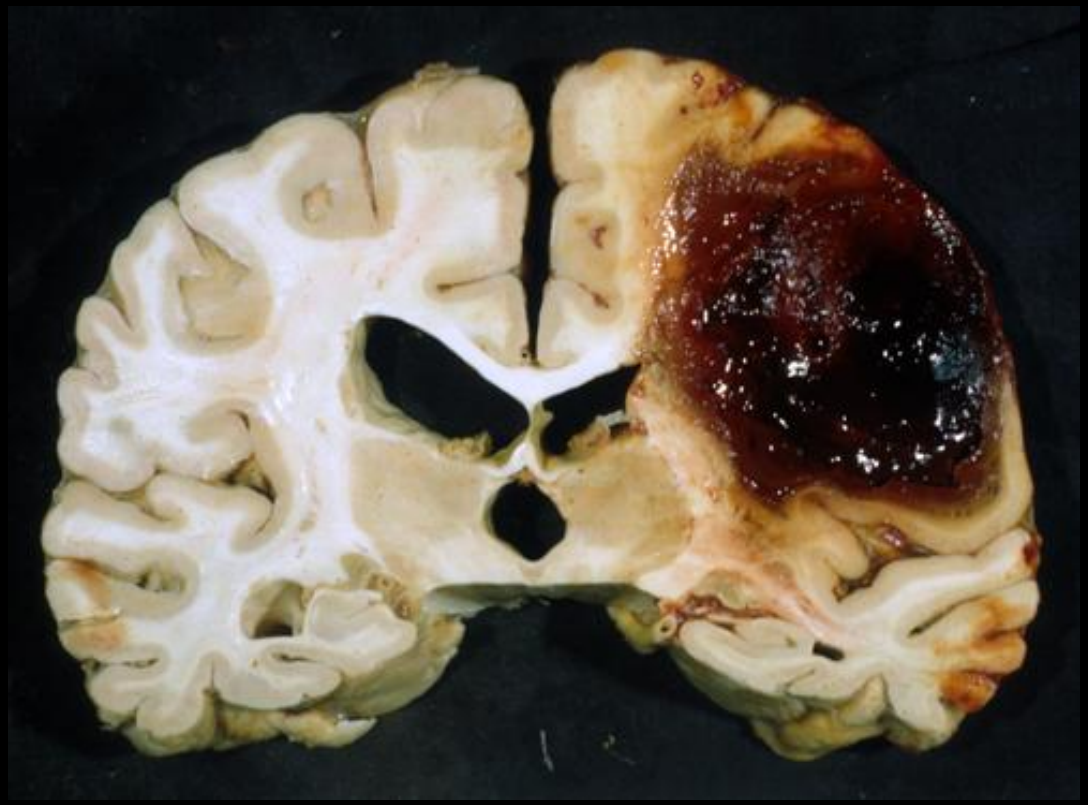
Morbidity & Mortality

- Intracranial hemorrhage (ICH) represents 15% of all strokes
- More than 75,000 new hemorrhagic strokes occur each year
- 30-day mortality rate is 2x – 6x higher than that of ischemic strokes
- More than half will die in the first 2 days
- 6% die before reaching the hospital

Stroke is the 4th leading cause of death and the #1 cause of disability in the U.S.

- Although hemorrhagic stroke represents only a small fraction of total strokes, the morbidity is more severe and mortality rates are higher than in ischemic stroke.
- Only 20% of patients with hemorrhagic stroke will regain their functional independence

Autopsy Cut – Hemorrhagic Stroke



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Uncontrollable Risk Factors for Hemorrhagic Stroke Include:

- Age
- Gender
- Ethnicity
- Genetics (familial aneurysms)

Age

- *Older Adults:* People most at risk for stroke are older adults, particularly those with high blood pressure, who are sedentary, overweight, smoke, or have diabetes.

- *Younger Adults:* Younger people are not immune. About 28% of stroke victims are under the age of 65.

Gender

- In most age groups except in older adults, stroke is ***more common in men than in women.***

- However, it ***kills*** more women than men at a ratio of 3:2.

Ethnicity

- All minority groups, including Native Americans, Hispanics, and African-Americans, face a significantly higher risk than Caucasians.
- The risk is also higher in Asian Americans and the Japanese population.
- The differences in risk among all groups diminish as people age.
- Younger African-Americans are two to three times more likely to experience and four times more likely to die from stroke.

Other Risk Factors Include:

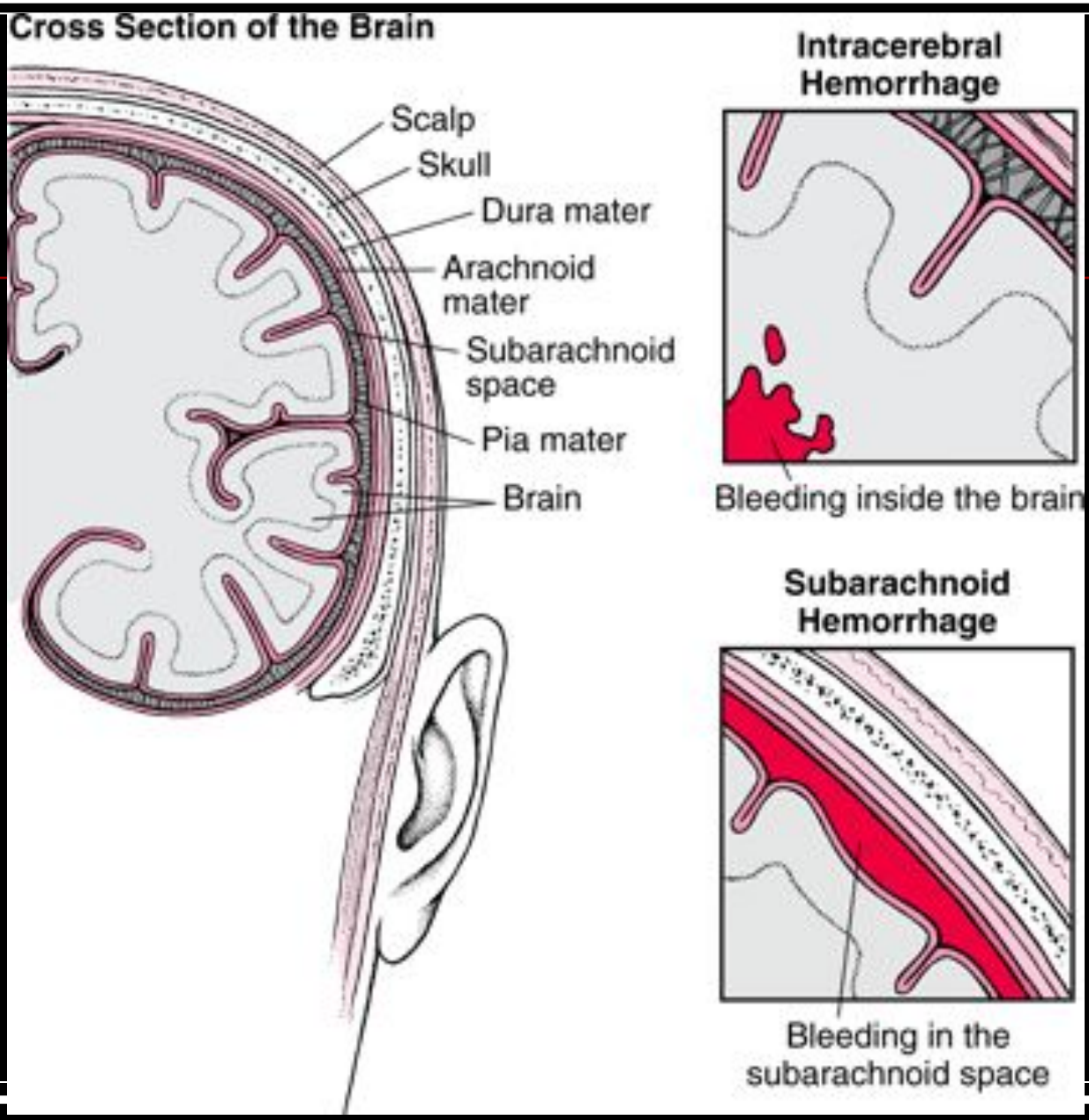
- Hypertension
- Smoking
- Diabetes
- Obesity
- Illicit drug use
- Stress
- Sickle cell anemia

Pathophysiology

- The pathophysiology of a hemorrhagic stroke is associated with an immediate rise in ICP, ischemic cellular responses, cerebral edema, and compromised cerebral perfusion – possibly leading to herniation of the brain

2 Categories of Hemorrhagic Stroke

- **ICH:** Intracranial Hemorrhage – bleeding into brain tissue as a result of bleeding or rupture of a small, deep cortical artery that is damaged by chronic hypertension
- **SAH:** Subarachnoid Hemorrhage – the result of bleeding into the subarachnoid space, most often in relation to a ruptured aneurysm or Arteriovenous Malformation (AVM)



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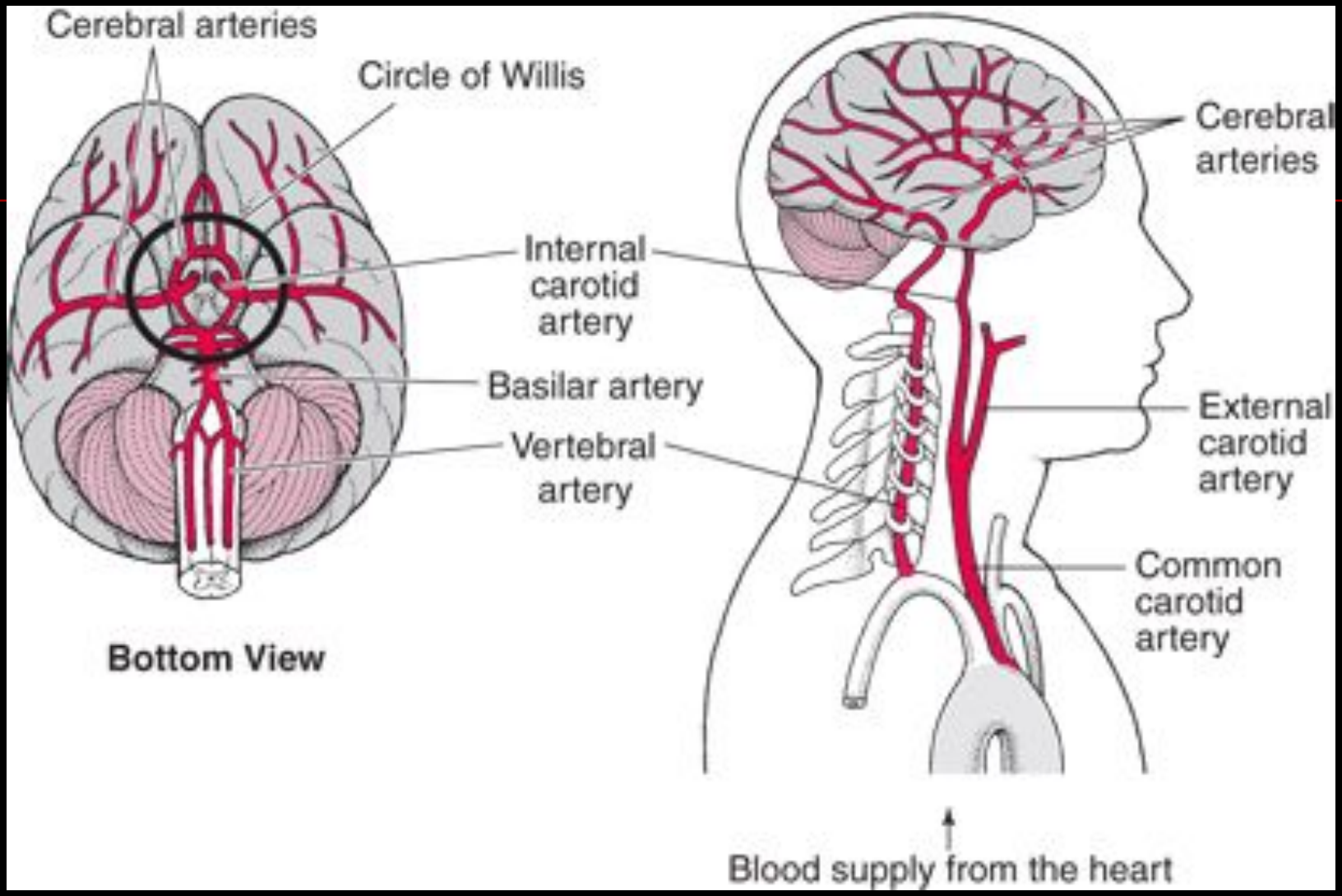
Overview of Cerebral Circulation

- There are four major cerebral arteries that supply the brain
  - Two internal carotid arteries (ICAs) that supply the anterior circulation, and
  - Two vertebral arteries (VAs) that supply the posterior circulation



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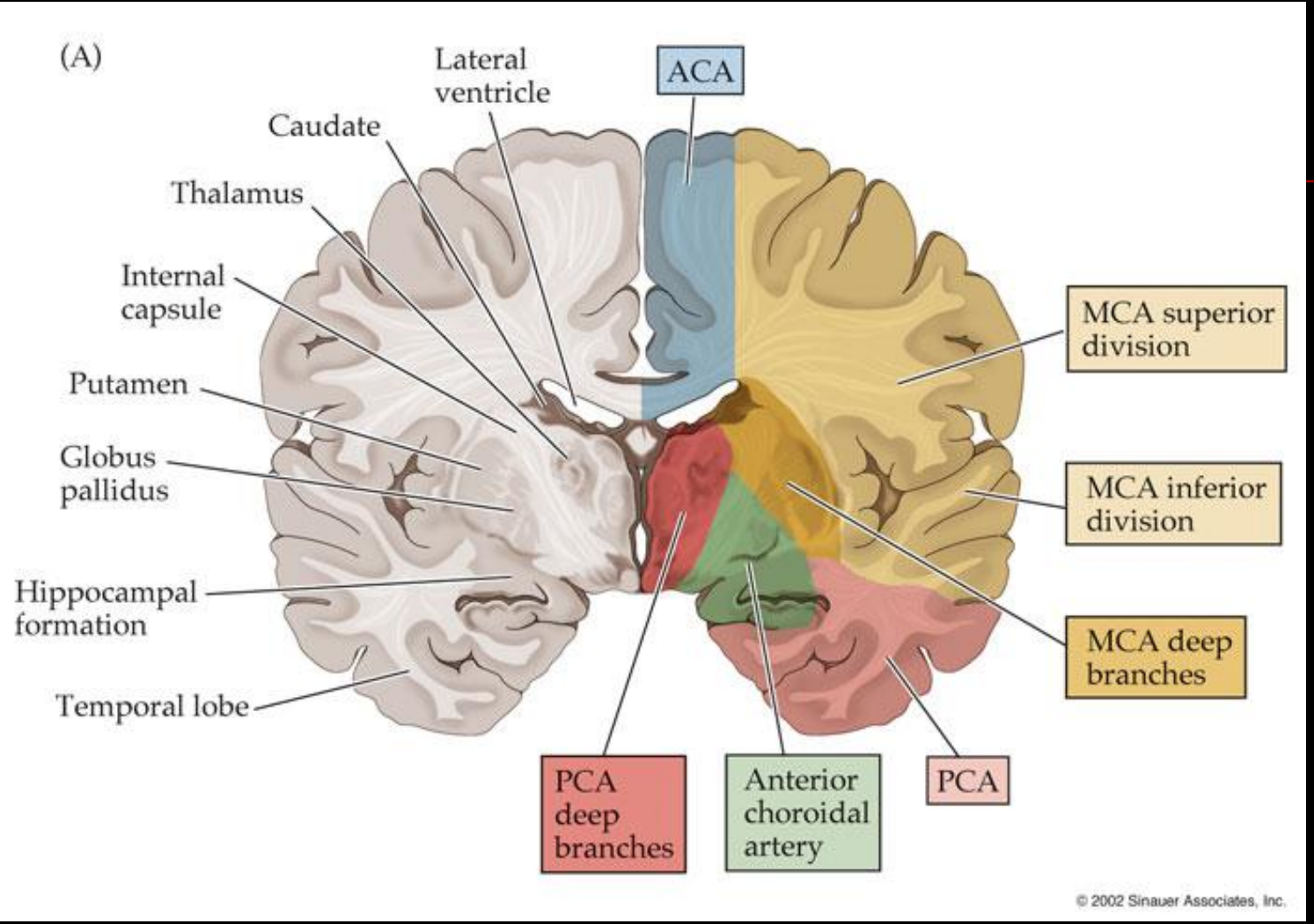
From the ICAs

- Middle Cerebral Artery (MCA) – provides blood supply to the lateral portions of the brain in the frontal, parietal, and occipital lobes of the brain
- Anterior Cerebral Artery (ACA) – provides blood supply to the medial portion of the brain
- Anterior Communicating Artery (ACoA) – connects the two ACAs
- Posterior Communicating Artery (PCoA) – connects the carotid circulation with the vertebrobasilar circulation

This figure illustrates the areas of the brain supplied by the major arteries



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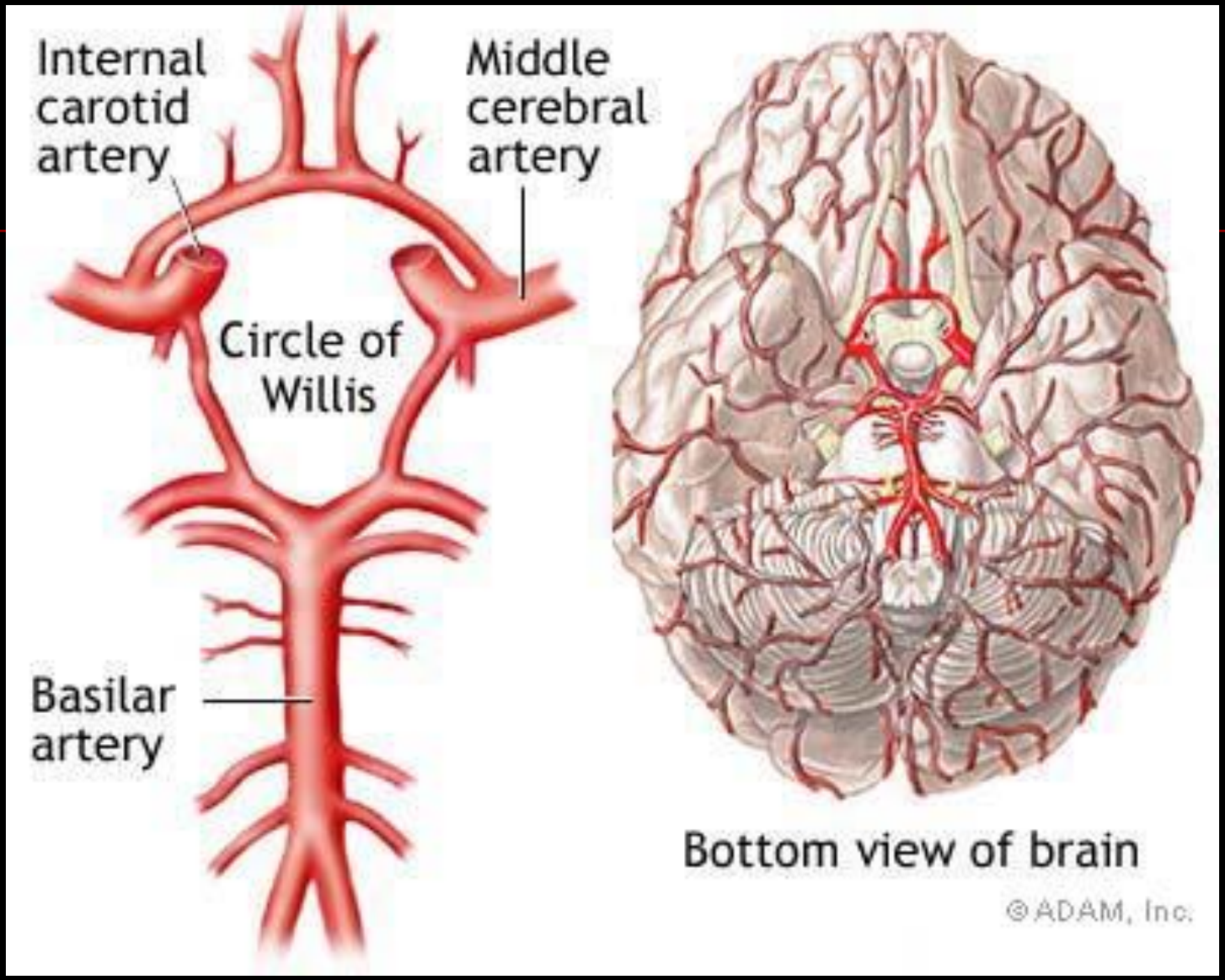
The Vertebral Arteries

- Unite to form the basilar artery (BA)
- The BA divides to form the Posterior Cerebral Arteries (PCA)
- The PCAs supply blood to the occipital lobe and portions of the temporal lobe



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Lesson 1 Review

- Hemorrhagic stroke is more common in men than in women, except in older adults.
- Hemorrhagic stroke kills more women than men.
- Younger African-Americans are two to three times more likely to experience a stroke and four times more likely to die from stroke.

Lesson 1 Review

- Two Types of Hemorrhagic Stroke:
  - ICH: Intracranial Hemorrhage – Bleeding into brain tissue as a result of bleeding or rupture of a small, deep cortical artery that is damaged by chronic hypertension
  - SAH: Subarachnoid Hemorrhage – The result of bleeding into the subarachnoid space, most often in relation to a ruptured aneurysm or AVM (arteriovenous malformation)

Lesson 1 Review

- Risk factors for hemorrhagic stroke include:
  - Hypertension
  - Smoking
  - Diabetes
  - Obesity
  - Illicit drug use
  - Stress
  - Sickle cell anemia

Lesson 1 Review

- There are four major cerebral arteries that supply the brain
  - Two internal carotid arteries (ICAs) that supply the anterior circulation, and
  - Two vertebral arteries (VAs) that supply the posterior circulation
- The divisions and deep branches of the Middle Cerebral Artery (MCA) supply the largest territory of brain

Lesson 2: Intracranial Hemorrhage

- Identify classic symptoms (presentation) of a patient experiencing intracranial hemorrhage (ICH)
- Review common sites of ICH
- Discuss possible causes of ICH

Intracranial Hemorrhage (ICH)

- ICH that is massive or near vital brain centers constitutes a medical emergency that requires immediate diagnosis and intervention to prevent death
- However, ICH in small penetrating vessels can mimic the evolution of an ischemic stroke, which complicates diagnosis and treatment

Presentation

- Symptoms vary depending on the area of the brain affected and the extent of the bleeding
- Headache, vomiting, altered level of consciousness (LOC), seizure at onset (or within 24 hours)
- May present as new onset of seizure
- Hypertension is commonly a prominent finding

Presentation Is:

- Characterized by sudden loss of consciousness that usually occurs without warning and while the person is active
- Typical situation: an older person with a long history of poorly controlled hypertension straining at stool develops a severe headache, decreased LOC, hemiplegia, nausea/vomiting, and possibly a focal seizure

ICH Symptoms and Syndromes

- In addition to altered LOC, headache, nausea/vomiting, nuchal rigidity, hypertension and bradycardia that occur related to increased intracranial pressure (ICP), there are ICH syndromes.
- Depending on the location of the ICH, each syndrome has distinguishing characteristics

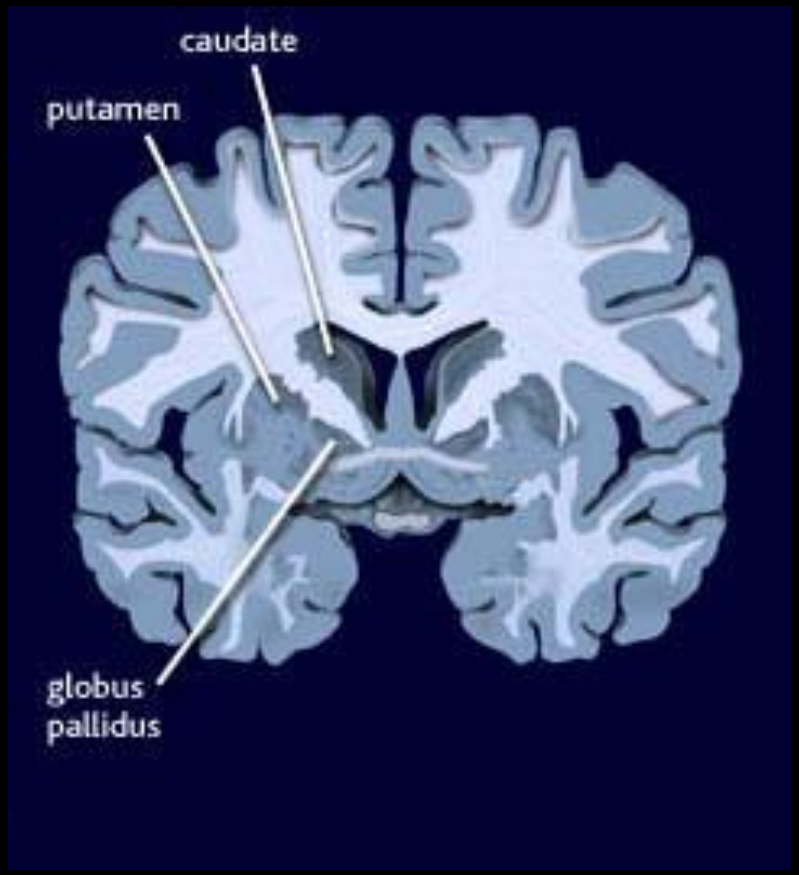
Intracranial Hemorrhage

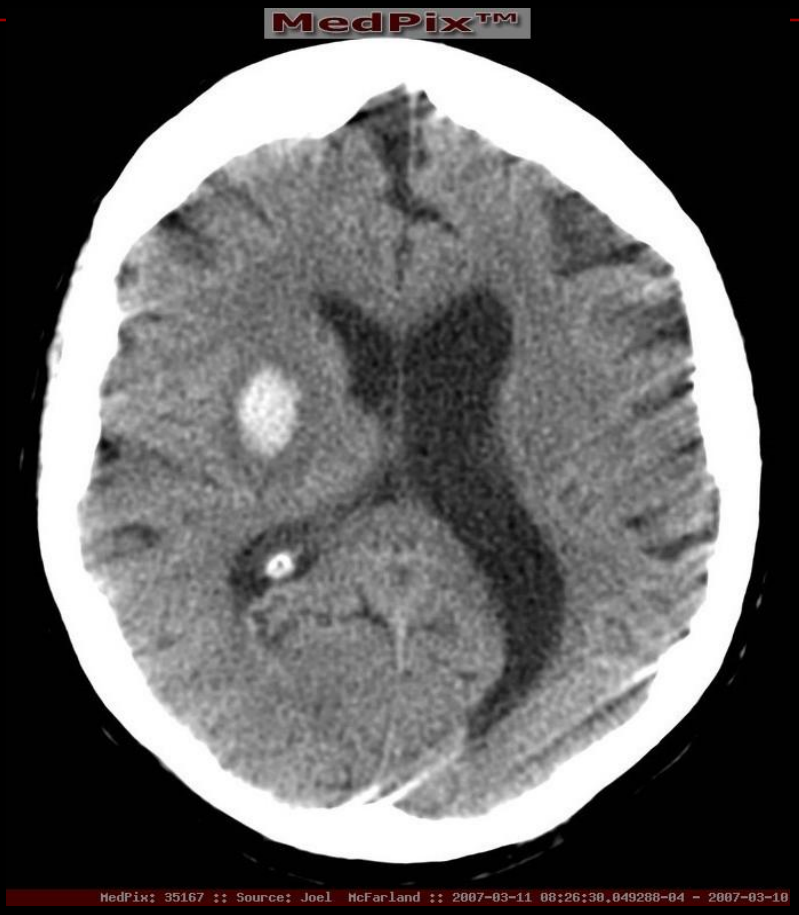
- With ICH, the usual hemorrhage sites are small, deep cortical arteries
- The most common sites are:
  - Putamen (50%)
  - Thalamus (30%)
  - Cerebellum (10%)
  - Pons (10%)

ICH: Deep Cortical Syndromes

- **The Putamen** is involved in sensorimotor integration and motor control.
- **Putamen hemorrhage** accounts for 50% of ICH
- Symptoms include:
  - Contralateral hemiplegia
  - Contralateral hemisensory deficits
  - Hemianopsia
  - Slurred speech

Putamen and Putamen Hemorrhage

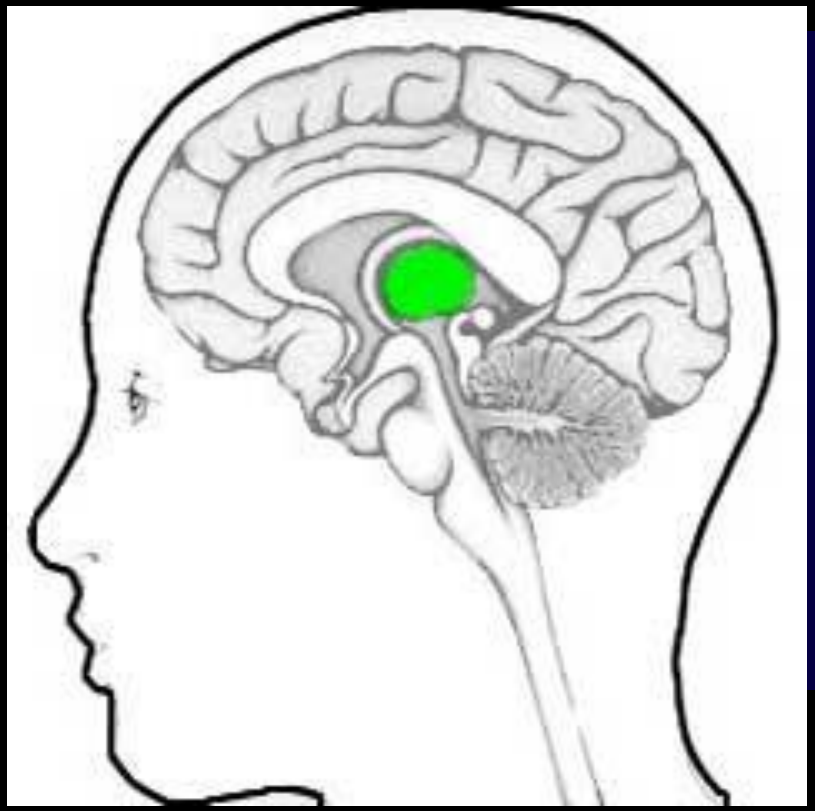


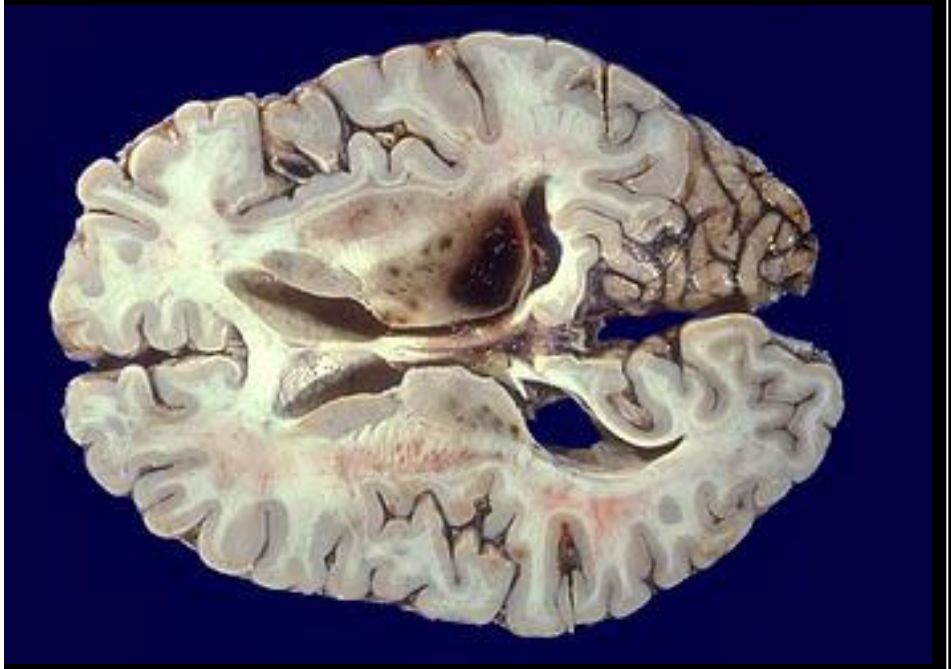


ICH: Deep Cortical Syndromes

- **The Thalamus** relays sensory information to various parts of the cerebral cortex
- **Thalamic Hemorrhage** accounts for 30% of ICH
- This syndrome is characterized by:
  - Contralateral hemiplegia
  - Contralateral hemisensory deficits
  - Deficit of vertical and lateral gaze

Thalamus and Thalamic Hemorrhage





Thalamic Hemorrhage

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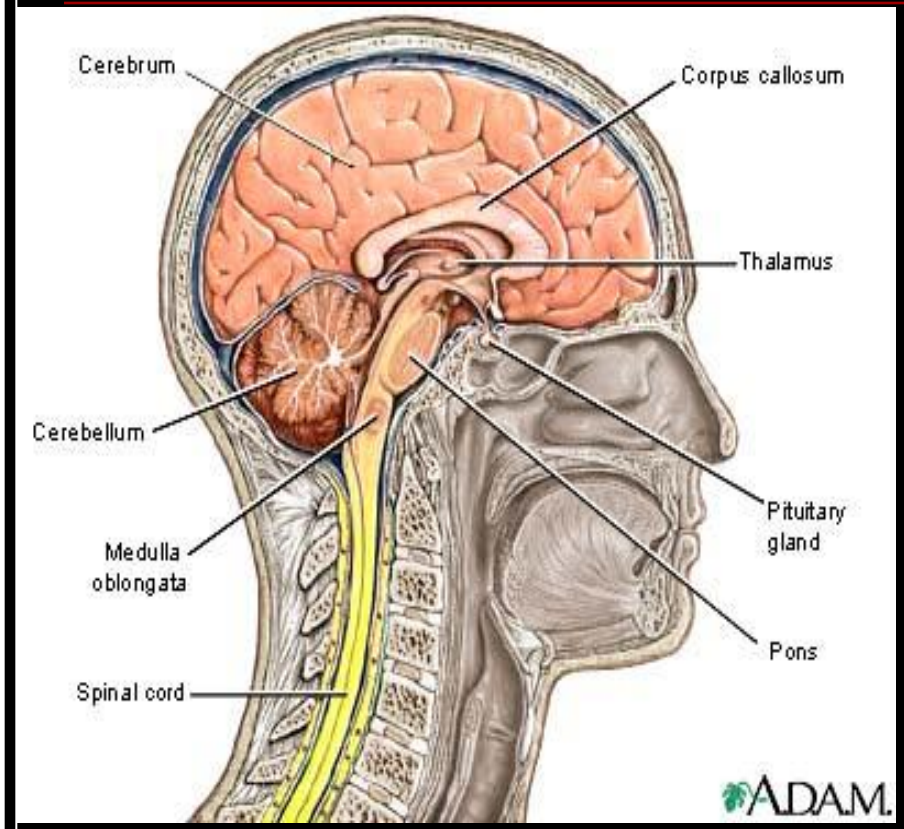
ICH: Deep Cortical Syndromes

- **The Pons** relays sensory info between the cerebellum and cerebrum, controls arousal, and regulates respiration
- **Pontine hemorrhage** accounts for 10% of ICH
- It is characterized by:
  - “Locked in” syndrome
  - Deficits in lateral eye movements

The Pons and Pontine Hemorrhage

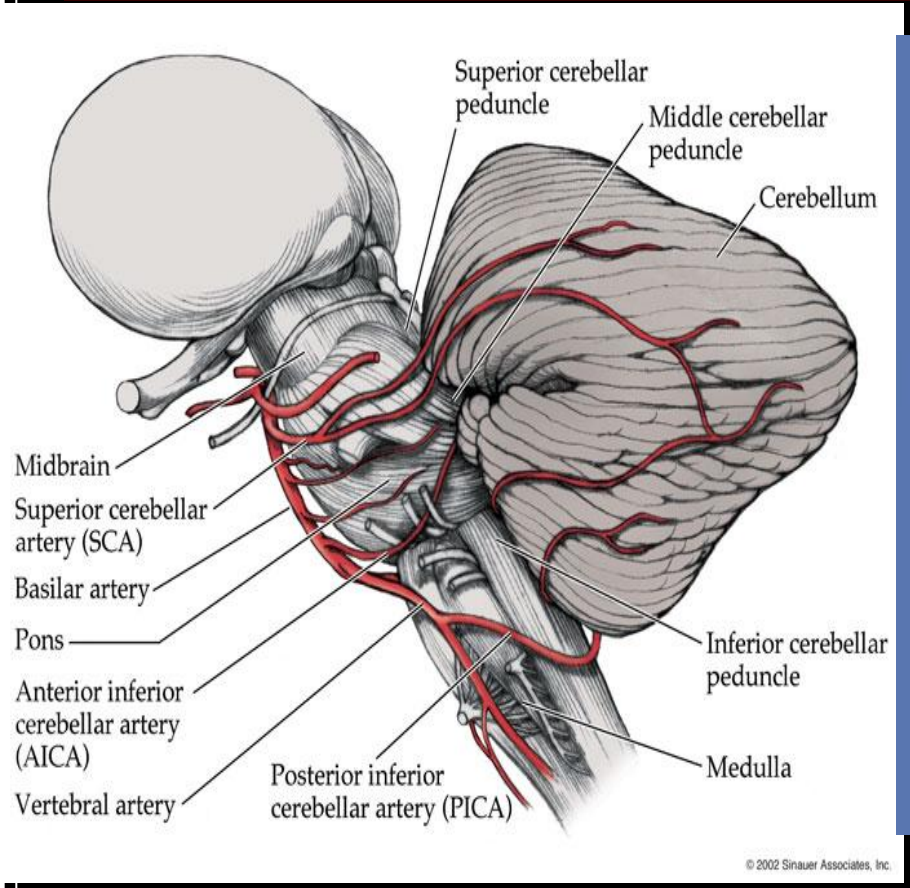


http://neuropathology.neoucom.edu/chapter2/images2/2-16l.jpg
http://www.holistic-massage-relief.com/images/Pons_corpus.jpg

ICH: Deep Cortical Syndromes

- **The Cerebellum** is primarily used for coordination and motor control
- **Cerebellar Hemorrhage** accounts for 10% of ICH
- Symptoms include:
  - Occipital headache
  - Dizziness
  - Ataxia
  - Vertigo

Cerebellum and Cerebellar Hemorrhage



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Causes of ICH

- Hypertension
- Aneurysm
- Arteriovenous Malformation (AVM)
- Trauma
- Coagulopathies
- Cocaine use

Cocaine Abuse

- Creates a high risk for hemorrhagic stroke as a result of a sudden rise in blood pressure, heart rate, and contractions of the left ventricle of the heart
- Cocaine also tightly constricts coronary and cerebral arteries

Lesson 2 Review

- Symptoms of hemorrhagic stroke vary depending on the area of the brain affected, but can include:
  - Headache
  - Nausea/vomiting
  - Altered level of consciousness (LOC)
  - Seizure at onset (or within 24 hours)
  - Hypertension

Lesson 2 Review

- Possible Causes of ICH include:
  - Hypertension
  - Aneurysm
  - Arteriovenous Malformation (AVM)
  - Trauma
  - Coagulopathies
  - Cocaine use

Lesson 2 Review

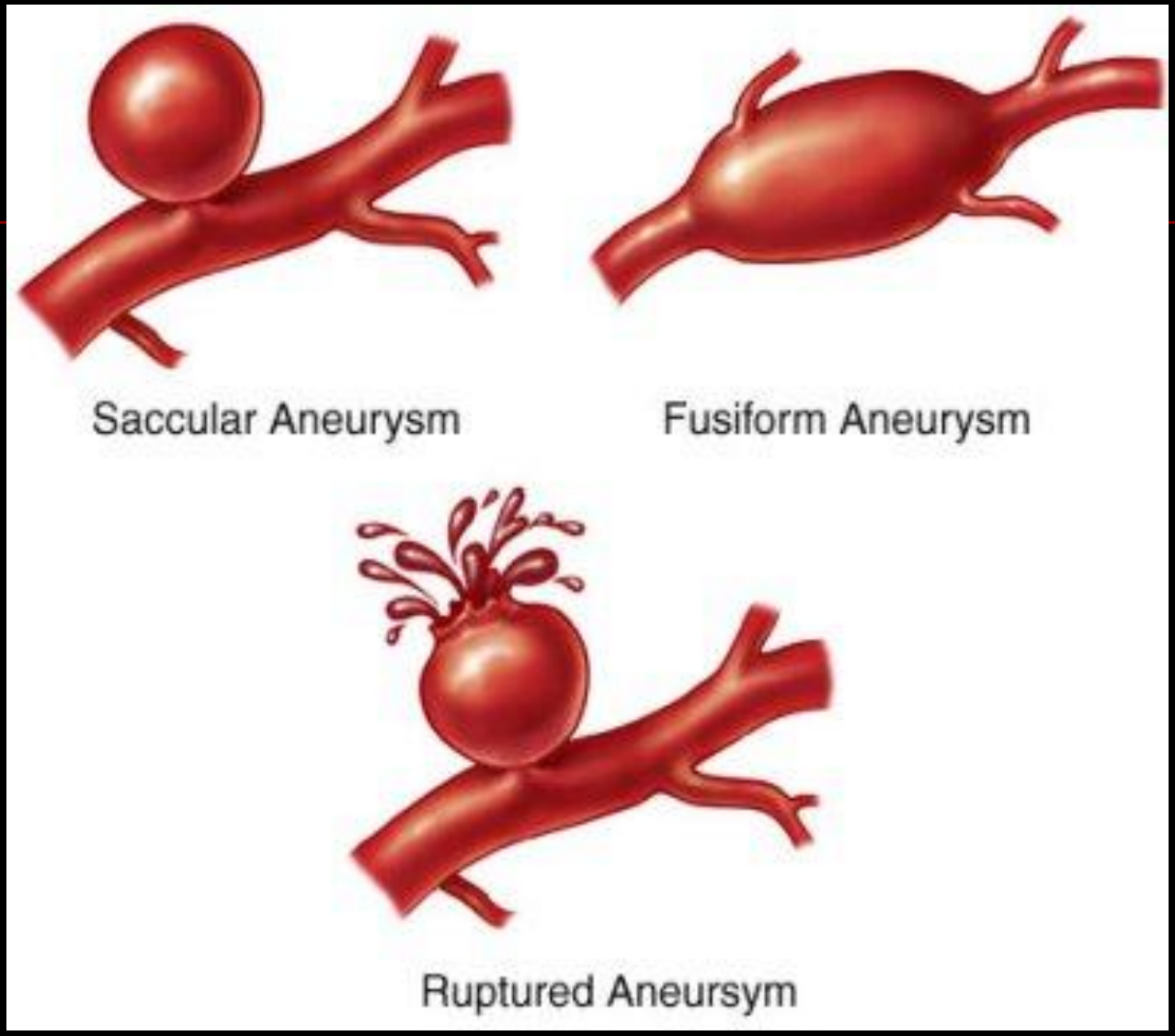
- With ICH, the usual hemorrhage sites are small, deep cortical arteries; most commonly:
  - Putamen (50%)
  - Thalamus (30%)
  - Cerebellum (10%)
  - Pons (10%)

Lesson 3: Cerebral Aneurysm

- Define cerebral aneurysm
- Identify warning signs of aneurysm rupture
- Discuss two treatment options for cerebral aneurysm
- Review patient care strategies and potential complications in cerebral aneurysm

Cerebral Aneurysm

- A **cerebral aneurysm** is a saccular outpouching of a cerebral artery
- **Aneurysm rupture** is the most common cause of non-traumatic subarachnoid hemorrhage (SAH)
- Aneurysm usually occurs at arterial bifurcations in the Circle of Willis



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Incidence of Cerebral Aneurysm

- There are 30,000 new cases of SAH secondary to aneurysm rupture each year
- It is most prevalent in 35 – 60 year olds
- Occurs more often in women at a 3:2 ratio
- Only one third recover without major disability

Incidence Cont'd

- Autopsy studies show that the overall frequency of un-ruptured aneurysms in the general population is about 5%
- It is estimated that 10 -15 million Americans have a cerebral aneurysm (most of them are small, though, and do not bleed throughout life)
- Although advanced imaging techniques have allowed for noninvasive detection of an aneurysm, **most are found following rupture**

The Chance of Aneurysm Rupture

- Influenced by:
  - History of smoking
  - Age
  - Gender
  - Hypertension
  - Number of existing aneurysms
  - Aneurysm size > 3 – 5 cm

Aneurysm Rupture

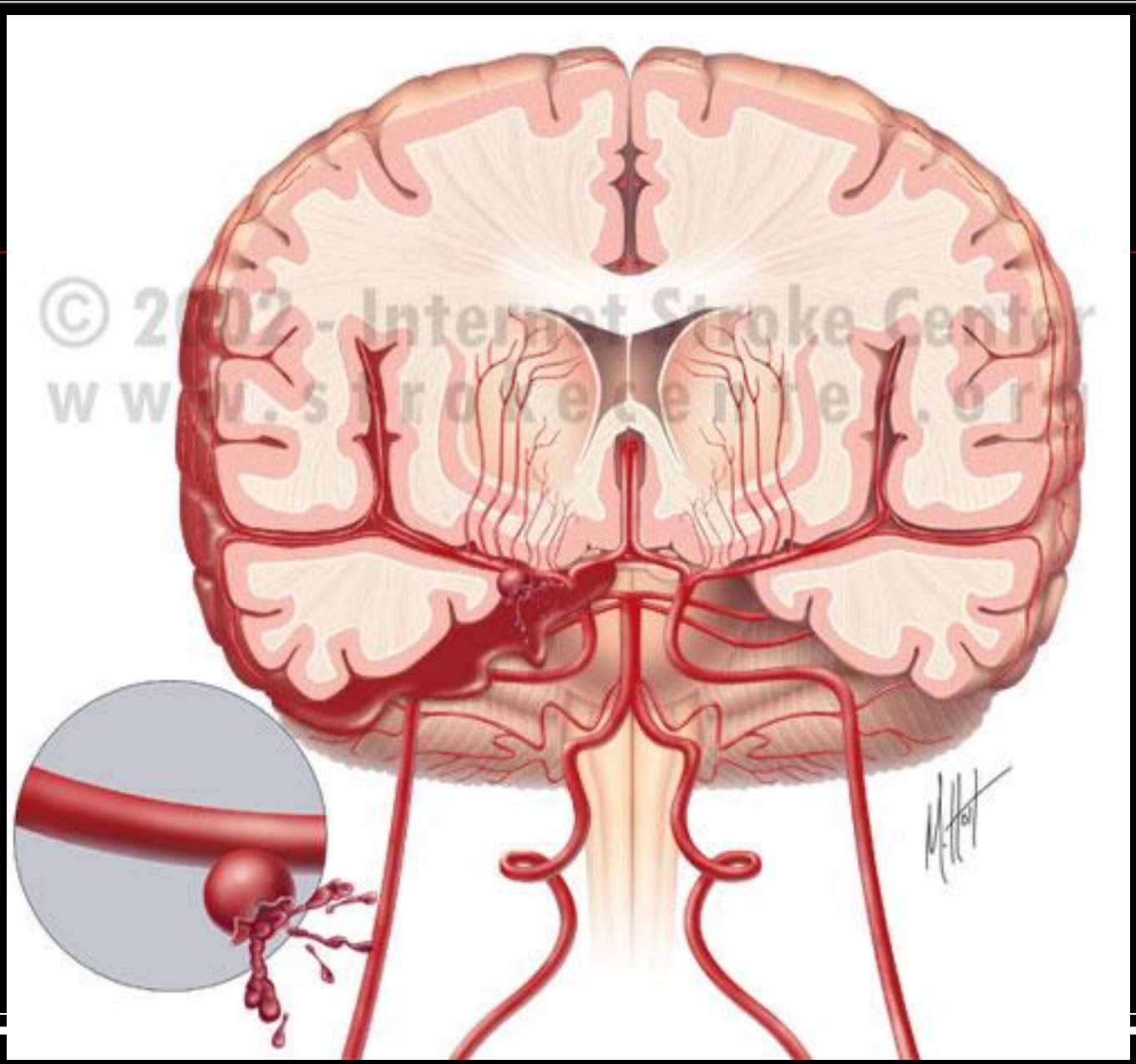
- The occurrence, growth and rupture of aneurysms can be directly related to the effect of hemodynamic forces
  - Blood pressure
  - Stress on the arterial wall
  - Impingement forces

Prodromal Signs present 10 – 20 days prior to rupture

- 50% experience warning signs that are either ignored or attributed to other causes
  - Headache (48%)
  - Dizziness (10%)
  - Pain above & behind the eye (7%)
  - Sensory or motor disturbance (6%)
  - Photophobia, diplopia, or vision loss (4%)
  - Bruits (3%)
  - Impaired extra ocular movements (EOMs) or ptosis

Pathophysiology of Aneurysm Rupture

- At the time of rupture, blood under high pressure is forced into the subarachnoid (SA) space
- Pressure in the surrounding tissue actually controls or stops the bleeding
- Fibrin, platelets and fluid form a plug that seals off the site of bleeding
- The resulting clot can occlude the area & interfere with CSF absorption, leading to hydrocephalus
- The blood released irritates the brain tissue, setting up an inflammatory response & cerebral edema (increased ICP and decreased cerebral perfusion)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Presentation

- The patient experiences a characteristic, intense, unrelenting headache of sudden onset often described as ***"the worst headache of my life!"***
- Patients with history of headaches including migraines typically describe SAH headache as different, being more severe and accompanied by a feeling of impending doom
- Transient loss of or altered consciousness

Intracranial Pressure (ICP) Increases

- Signs of elevated ICP:
  - Cushing's Triad:
    - Hypertension
    - Bradycardia
    - Widening pulse pressure
  - Seizures
  - Nausea and vomiting

Diagnosis

- CT scan
  - 90% sensitivity to bleeding in the brain
- Lumbar puncture
  - For negative CT with clinical signs of SAH
- Angiography
  - Identifies location/characteristics of the aneurysm
  - Helps with treatment decision
  - Detects vasospasm

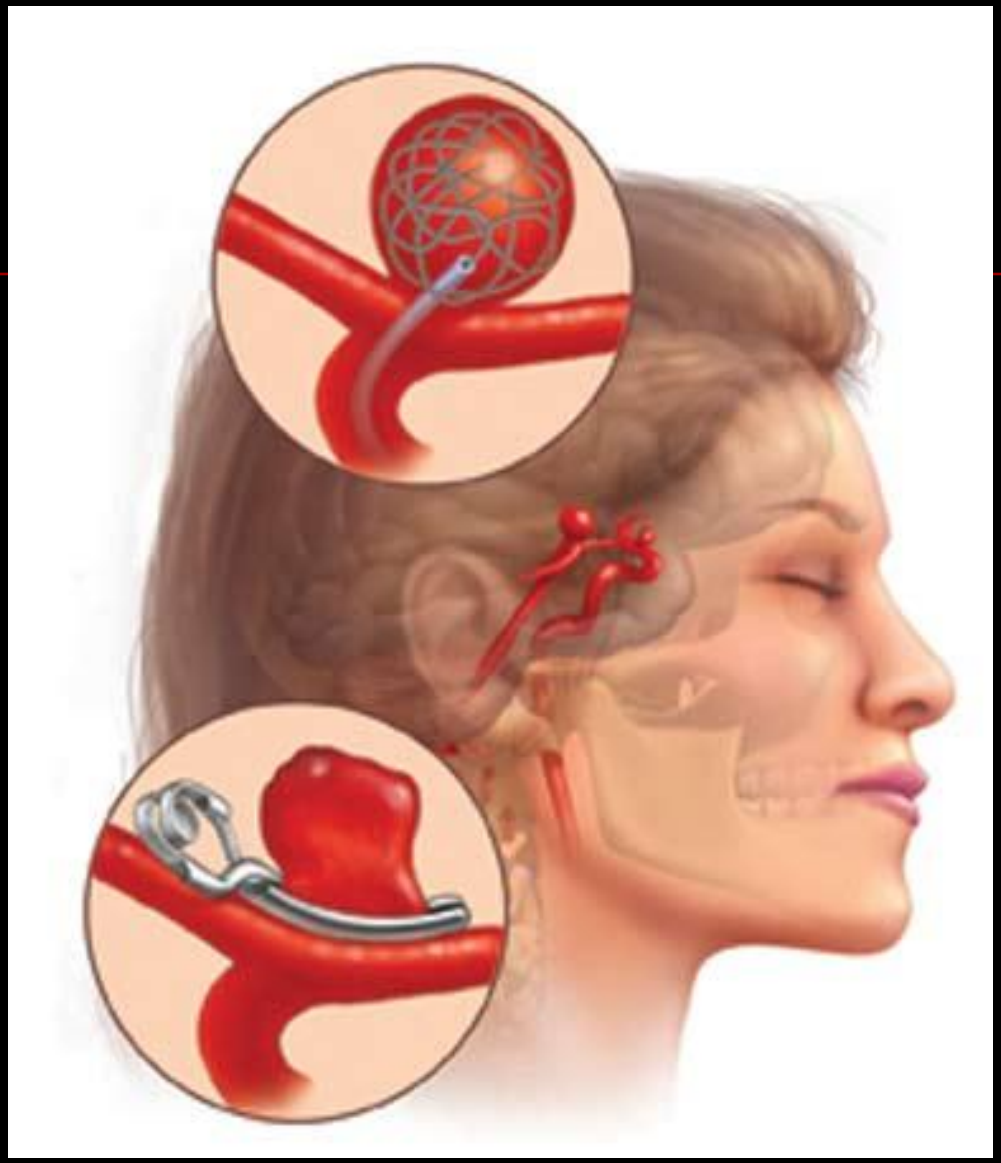
Treatment Options for Aneurysm

- Craniotomy with aneurysm neck **clipping**
- Endovascular embolization, also called **coiling**
- The stroke team considers several factors when deciding which treatment option is best for a particular patient. These include:
  - Size of Aneurysm
  - Location of Aneurysm
  - Type of Aneurysm
  - Condition of Patient
  - Medical History

Coiling

vs.

Clipping



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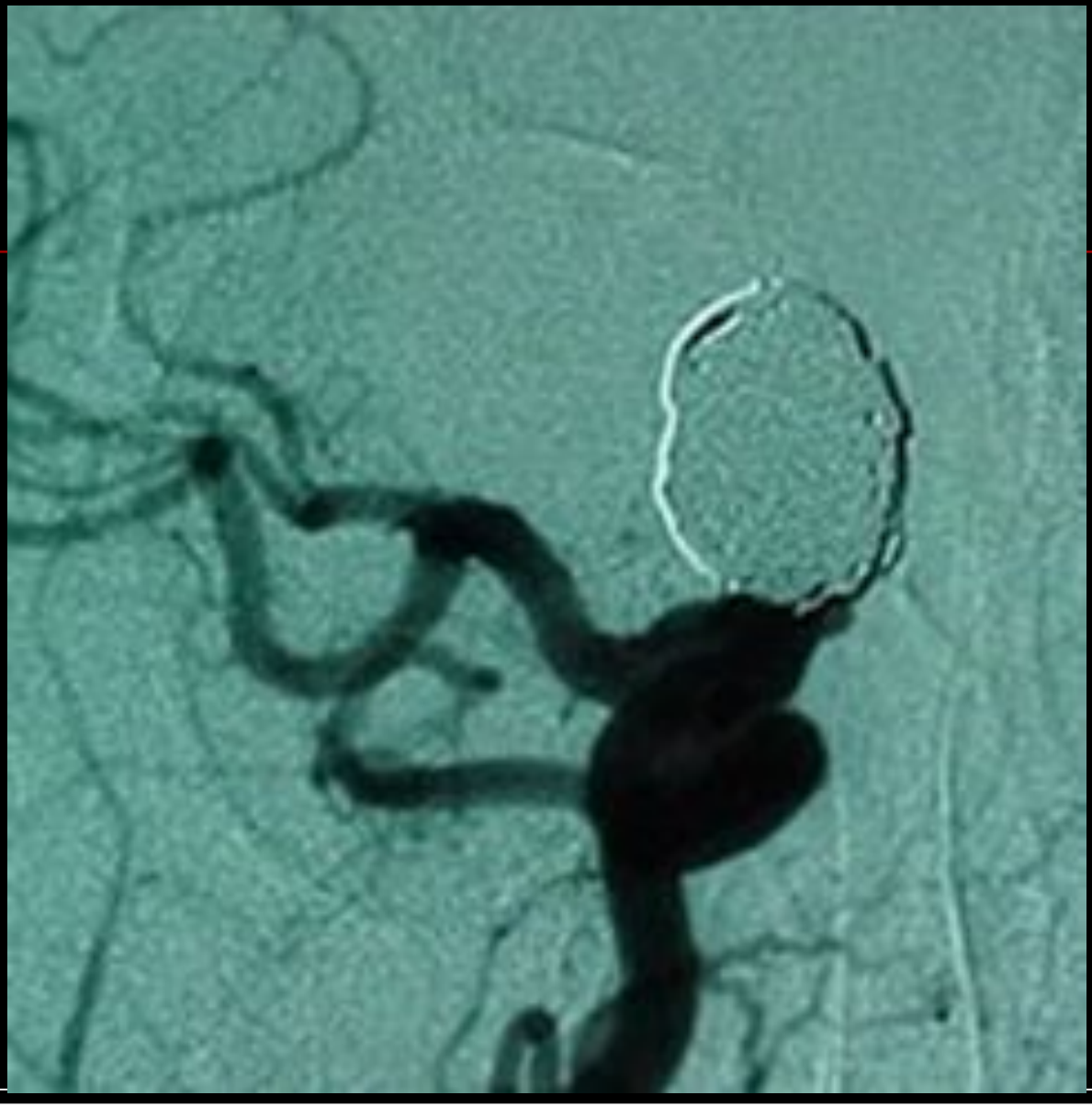
Treatment Options for Aneurysm

- Craniotomy with aneurysm neck **clipping**
  - Requires incision and removal of skull bone
  - The parietal & temporal lobe are separated
  - Surgical clip is applied to prevent blood from re-entering the aneurysm & bleeding
  - The dome of the aneurysm is punctured
  - Risks include: infection, cerebral edema, pneumocephalus and anesthesia risks

Treatment Options for Aneurysm

- Endovascular embolization (**coiling**)
  - Approved in 1995 by the FDA
  - Coiling is associated with improved outcomes
  - Uses angio techniques to guide a catheter to the location of the aneurysm
  - Coils are packed into the aneurysm until filled
  - Blood can no longer enter the aneurysm and there is no further risk of re-bleed
  - Risks: perforation of vasculature; bleeding at site



Imaging of an aneurysm that had previously undergone coiling

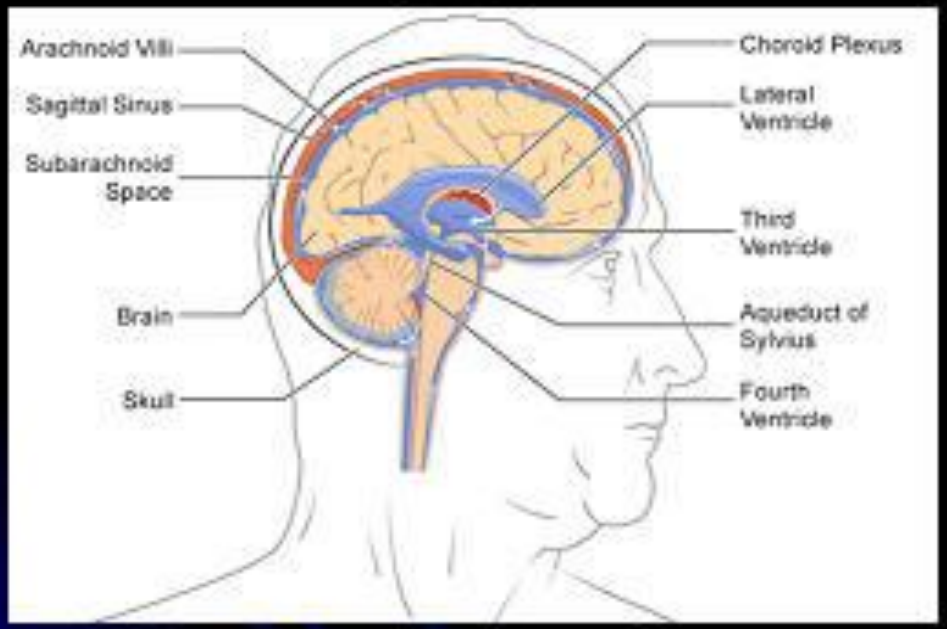
Potential Complications: RE-BLEEDING

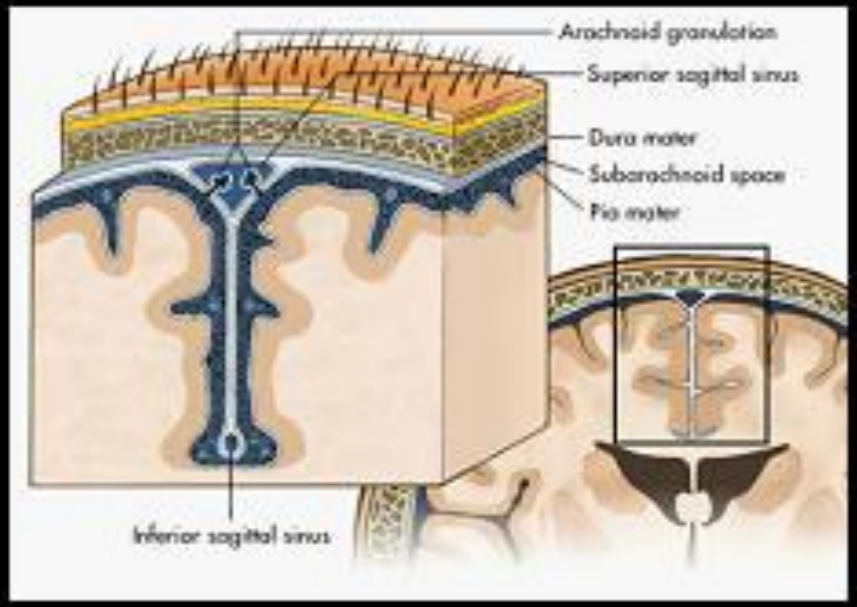
- Re-bleeding
  - Of the 18,000 victims that survive annually, 3,000 will die or be disabled from re-bleed
  - The highest incidence of re-bleeding is in the first two weeks
  - Symptoms are related to increased ICP – increase in headache, decrease in LOC & new onset of focal (stroke-like) symptoms

Potential Complications: HYDROCEPHALUS

- Hydrocephalus
  - Occurs in 65% of SAH patients
  - Can be life-threatening
  - Acute signs: drowsiness, abrupt mental status and behavioral changes
  - Treatment: Ventriculostomy, Lumbar drain and/or VP shunt

Anatomy of Hydrocephalus





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Potential Complications: SEIZURE

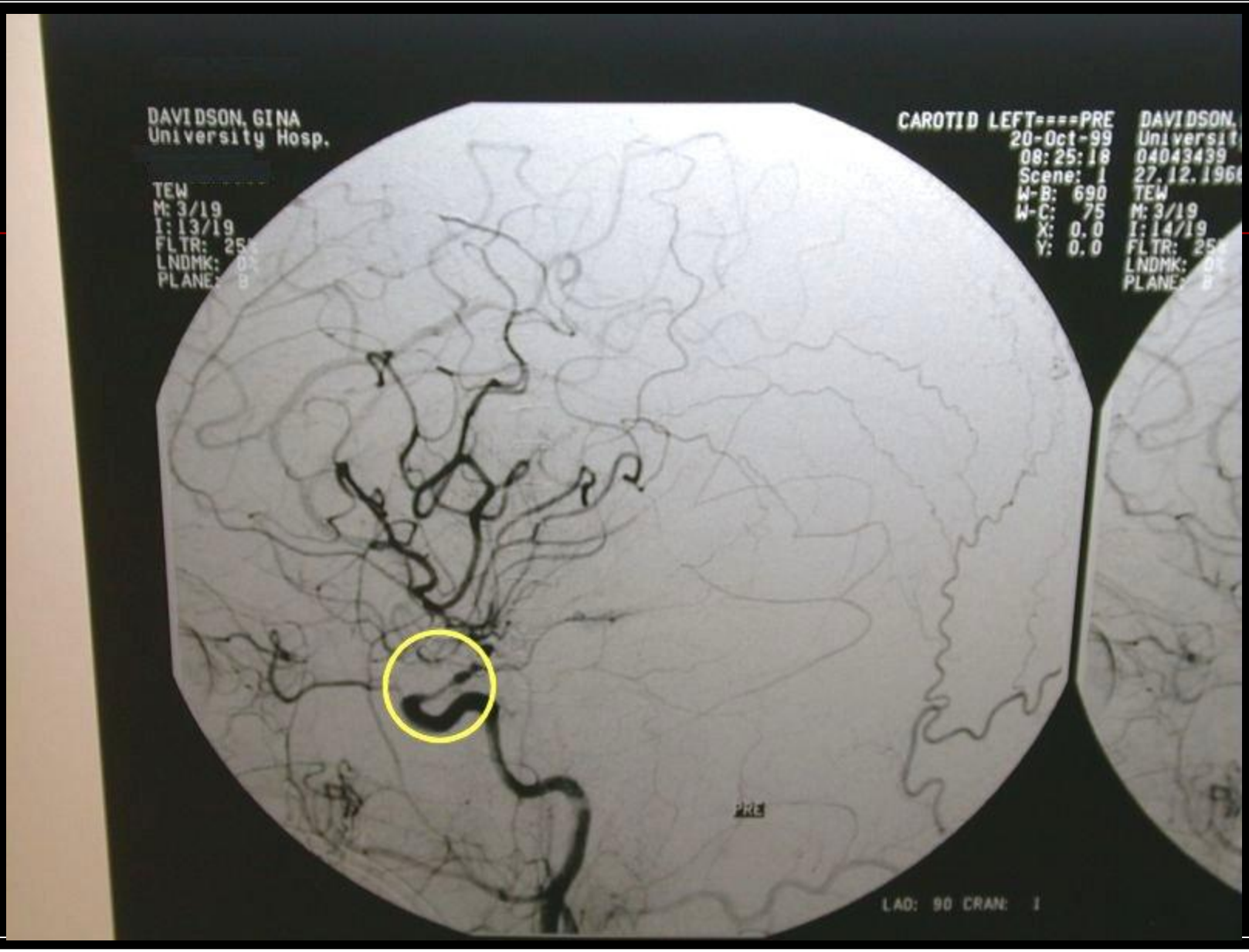
- **Seizures** can occur in as many as 25% of patients
- Most common after MCA ruptures
- Lead to increased cerebral blood flow, increased ICP and hypertension
- Escalate the risk of aneurysm re-bleed and neurologic deterioration

Potential Complications: FEVER and Secondary INFECTION

- Patients are at risk for developing both infectious and non-infectious fever and often do not respond to treatment
- Fever occurs in as many as 54% of patients recovering from SAH and is a predictor of poor prognosis
- Fever increases cerebral metabolic rate, releases excitatory neurotransmitters, increases production of free radicals and breakdown of the blood-brain barrier – all result in an increased risk for ischemia

Potential Complications: VASOSPASM

- Definition: the narrowing of a cerebral blood vessel causing decreased cerebral perfusion initially; leading to delayed ischemic deficit, infarction & brain damage if untreated
- 40% of patients will experience vasospasm
- Etiology of this phenomenon is unknown
- If patient's condition deteriorates after 3-14 days, vasospasm should be considered



Vasospasm – the narrowing of a cerebral blood vessel is depicted in this image

Available at: http://www.keithandgina.com/aneurysm/ang3.jpg

Vasospasm

- Signs:
  - Gradual neurological deterioration
  - Confusion
  - Decreased LOC
  - Mimics ischemic stroke as new focal neurologic deficits (hemiplegia, CN deficits, & aphasia)

Treatment of Vasospasm

- Aimed at increasing cerebral perfusion (Triple H Therapy)
  - Hypervolemia
  - Hypertension
  - Hemodilution
- Triple H Therapy helps to maintain a stable, slightly higher blood pressure, which increases and maintains cerebral perfusion pressure.

Nimodipine

- Nimodipine (Calcium Channel Blocker) has been used to treat vasospasm for >10 yrs
  - Believed to act at a cellular level as a neuronal protector
  - Shows improved neurological outcomes, but does not improve or stop the vasospasm
  - 30-60mg PO or NG every four hours for 21 days
- Nimodipine is used as long as the ability to treat with hypertension is not compromised

Potential Complications: HYPONATREMIA

- Low sodium levels create a high risk for vasospasm when combined with hypovolemia
- Hyponatremia can be caused by SIADH (**S**yndrome of **I**n-**A**ppropriate **D**iuretic **H**ormone) or Cerebral Salt Wasting (CSW).
- The cause must be distinguished because the treatment is so different:
  - If caused by true SIADH – fluid restriction
  - If caused by salt wasting – fluid replacement
- Fluid restriction in a patient with salt wasting places them at high risk for vasospasm and ischemia

SIADH vs. Cerebral Salt Wasting

SIADH

- Hyponatremia (dilutional)
- Increased ECF
- Increased plasma volume
- Increased body weight
- Low BUN
- Low serum osmolality
- Not necessarily negative salt balance

Salt Wasting

- Hyponatremia (primary)
- Decreased ECF
- Decreased plasma volume
- Decreased body weight
- High BUN
- High urine sodium
- Negative salt balance (primary loss of sodium)

Patient Care in Aneurysm & SAH

- Frequent neuro assessments
  - Report changes in neuro status immediately
  - May detect vasospasm, which occurs suddenly
- Airway & oxygenation
- Blood Pressure management
  - SBP between 100 and 160 mmHg – maintain perfusion
  - Rapid drop is not recommended – risk of ischemia
- ICP monitoring
  - Routine cultures
  - Prophylactic antibiotics

Patient Care cont'd

- Fever management
  - Fever is associated with poorer recovery
  - Acetaminophen every 4 – 6 hours
- Labs
  - Chemistry studies and electrolytes
  - Coags and CBC
  - Fingersticks (80 – 120 mg/dl) w/ insulin coverage
- IV fluids
  - Normal saline 80 – 100 mL/hr

Patient Care cont'd

- Nutrition - NPO until speech & swallow evaluation
- Strict I&Os
- Monitor for SIADH or CSW
- Activity
- Limited to prevent increased ICP and re-bleed
- SAH precautions: quiet environment
- DVT prophylaxis
- SCDs – no heparin until aneurysm securement
- Risk of Heparin Induced Thrombocytopenia 15%

Medications

- Seizure prophylaxis
- Blood pressure management
- Stool softeners – should not strain at BM
- Pain management – headache
- Antiemetic – n/v increases ICP & re-bleed
- GI prophylaxis
- Nimodipine – prevention of vasospasm

Lesson 3 Review

- A cerebral aneurysm is a saccular outpouching of a cerebral artery
- Aneurysm rupture is the most common cause of non-traumatic subarachnoid hemorrhage (SAH)
- Although advanced imaging has allowed for noninvasive detection of an aneurysm, most are found following rupture

Lesson 3 Review

- 50% of patients experience prodromal (warning) signs 10-20 days prior to aneurysm rupture:
  - Headache (48%)
  - Dizziness (10%)
  - Pain above & behind the eye (7%)
  - Sensory or motor disturbance (6%)
  - Photophobia, diplopia, or vision loss (4%)
  - Bruits (3%)
  - Impaired extra ocular movements (EOMs) or ptosis

Lesson 3 Review

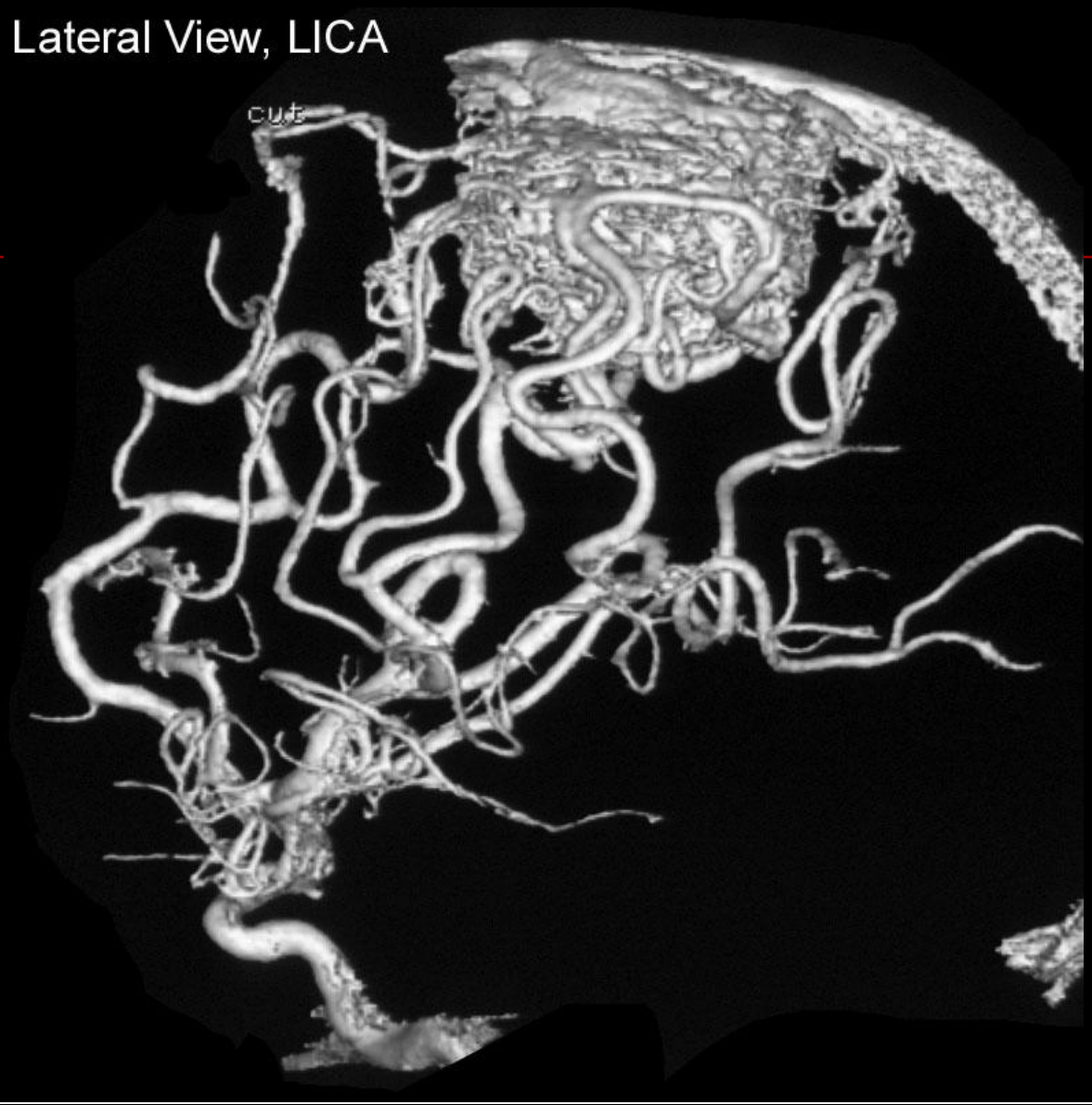
- Initial presentation with aneurysm rupture:
  - Intense, unrelenting headache of sudden onset, described as “the worst headache of my life!”
  - Transient loss of or altered consciousness
  - Feelings of impending doom
- Treatment options for aneurysm include:
  - Craniotomy with aneurysm neck **clipping**
  - Endovascular embolization, also called **coiling**

Lesson 4:
Arteriovenous Malformation (AVM)

- Define Arteriovenous Malformation (AVM) and associated terms
- List medical manifestations of AVM
- Discuss treatment options for AVMs

Arteriovenous Malformation (AVM)

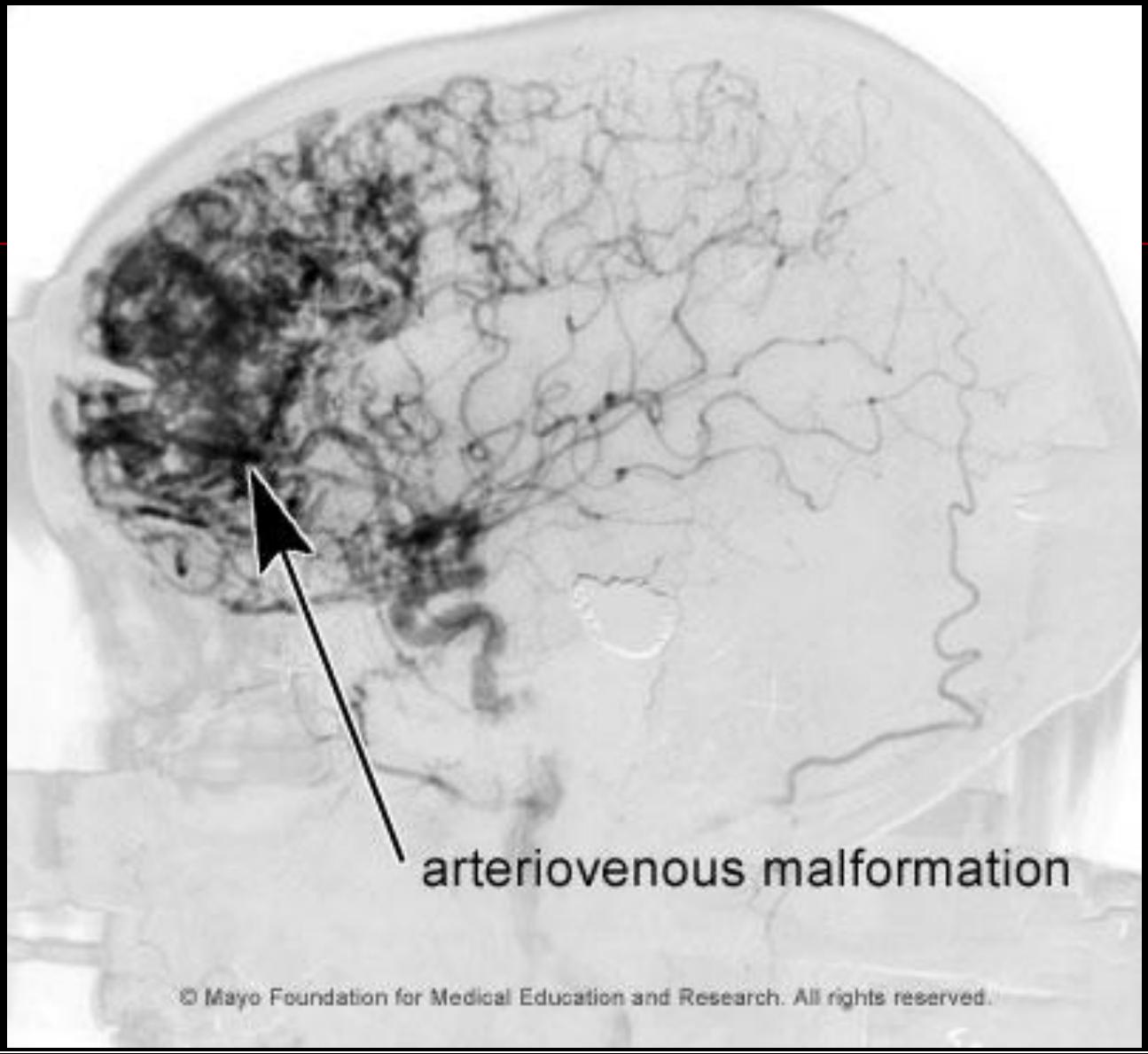
- Composed of a tightly tangled collection of abnormal, thin-walled, dilated blood vessels that directly shunt arterial blood into the venous system without the usual connecting capillary network
- Found in children and adults
- Believed to be congenital (embryonic vascular network did not develop properly)



Available at: http://scienceblogs.com/purepedantry/upload/2006/12/avm.jpg

AVMs

- The focus of the AVM is called the “nidus”, the tangle of abnormal vessels
- The arterial vessels are called “feeder arteries” and supply the AVM
- The lesion is drained by dilated veins
- As a result of the absence of the capillary network, blood flow is elevated within the fragile vessels of the AVM
- These conditions predispose the lesion to hemorrhage



Incidence of AVM

- Account for 8.6% of SAHs and 1% of strokes
- Clinically significant because AVM is the most common cerebrovascular lesion that causes symptoms
- 80% develop symptoms between 20-40 years of age

Vascular Steal Phenomenon

- There is an effect of impaired perfusion of the cerebral tissue surrounding the AVM
- The diversion of blood to the AVM is called “vascular steal” phenomenon
- Vascular steal is said to be the cause of progressive neuro deficits and psychiatric behaviors/manifestations seen in some AVM patients

Major Clinical Manifestions of AVM

- 50% present with hemorrhage
- 70% have a seizure at some point
- 15% have migraine-like headaches that do not respond to usual drug therapy
- Progressive neuro deficits – depend on the area of the brain with vascular steal
- Psychiatric manifestions/behaviors

Diagnosis of AVM

- CT scan
- MRI/MRA
- Angiography –
  - Definitive
  - Demonstrates the feeding arteries, the nidus and the draining veins

Treatment of AVMs

- Surgery – usually elective; delayed for 3 weeks after hemorrhage to allow the patient to be stabilized and for the brain to recover from the effects of hemorrhage
- Endovascular Tx – may be needed in multiple stages to reduce blood supply to the AVM and/or in combination with surgery

Treatment of AVMs cont'd

- Embolization – permanent occlusion of the nidus and feeding vessels
  - Surgically inaccessible lesions
  - Facilitate surgical excision
  - Promote thrombosis of the nidus
- Radiation (Gamma Knife)

Key Assessment Points

- Level of Consciousness
- Pupil checks
- Motor function
- Sensory function
- Meningeal irritation signs (headache, stiff neck, photophobia)
- CN deficits (blurred vision, diplopia, EOMs, ptosis, dysarthria, facial droop, etc)

Caution!
Alterations in assessment could be the result of :

Potential complications such as

- re-bleed,
- hydrocephalus,
- ischemia,
- vasospasm,
- hypoxia,
- focal seizures,
- increased ICP, or
- electrolyte imbalances

However,

- Alterations may also be caused by:
  - Effects of immobility (DVT, PE, constipation, atelectasis)
  - Sensory deprivation (depression, psychological effects/behaviors)
  - Infections (aspiration or bacterial pneumonia, UTIs)

Additional Points of Care

- Recognition of common complications
- Prevention of other complications
  - Monitoring blood pressure and lab values
  - Adequate oxygenation
  - Bowel and bladder management
  - Dysphagia/nutritional compromise
  - DVT prophylaxis
  - Aseptic technique
  - Skin breakdown
- Seizure precautions and prophylaxis
- Drug therapy (Nimodipine, pain control, etc)
- Patient Education (disease process & treatment)

Lesson 4 Review

- An AVM is:
  - composed of a tightly tangled collection of abnormal, thin-walled, dilated blood vessels that directly shunt arterial blood into the venous system without the usual connecting capillary network
  - believed to be congenital
  - most commonly found at 20-40 years of age

Lesson 4 Review

- Major medical manifestations of AVM
  - Hemorrhage
  - Seizures
  - Migraine-like headaches
  - Progressive neuro deficits
  - Psychiatric manifestions/behaviors

Lesson 4 Review

- Treatment options for AVM include:
  - Surgery
  - Embolization
  - Radiation (Gamma-knife)
  - Combinations of these

Summary

- Although hemorrhagic strokes constitute only a fraction of total strokes, the effects can be devastating or deadly
- New research and technical advances have led to less traumatic and safer methods of treatment for patients
- Recovery is often complicated by secondary injuries
- When a patient suffers hemorrhagic stroke, each team member plays an important role in patient monitoring, therapies and management of care to prevent secondary injury and complications
- Nursing can directly impact care, patient outcomes and quality of life with thorough assessment, early recognition of complications, patient education and advocacy

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