Learning Objectives

- Acute Ischemic Stroke (AIS) and Transient Ischemic Attack (TIA)
- Introduction & Epidemiology
- Mechanisms of ischemia and stroke classification
- Lacunar infarction syndromes
- Amaurosis fugax “monocular blindness”
- Collateral blood flow
- Diagnosis and treatment of AIS/TIA
- Management of carotid artery disease
- Antithrombotic options for AIS
- Management of elevated intracranial pressure (ICP) due to stroke
- Signs of intracranial hemorrhage (ICH)
- Causes of ICH
- Subarachnoid hemorrhage

Stroke

Stroke: a focal neurological deficit of sudden onset explained by a vascular cause

The two broad categories of stroke, hemorrhage and ischemia, are diametrically opposite conditions: hemorrhage is characterized by too much blood within the closed cranial cavity, while ischemia is characterized by too little blood to supply an adequate amount of oxygen and nutrients to a part of the brain.

Stroke is classified into two major types:
- Brain ischemia due to thrombosis, embolism, or systemic hypoperfusion
- Brain hemorrhage due to intracerebral hemorrhage (ICH) or subarachnoid hemorrhage (SAH)

A stroke is the acute neurologic injury that occurs as a result of one of these pathologic processes. Approximately 80 percent of strokes are due to ischemic cerebral infarction and 20 percent to brain hemorrhage.
Brain Ischemia

There are three main subtypes of brain ischemia:

- **Thrombosis**: generally refers to local in situ obstruction of an artery. The obstruction may be due to disease of the arterial wall, such as arteriosclerosis, dissection, or fibromuscular dysplasia; there may or may not be superimposed thrombosis.

- **Embolism**: particles of debris originating elsewhere that block arterial access to a particular brain region. Since the process is not local (as with thrombosis), local therapy only temporarily solves the problem; further events may occur if the source of embolism is not identified and treated.

- **Systemic hypoperfusion**: a more general circulatory problem, manifesting itself in the brain and perhaps other organs.

- **Blood disorders** are an uncommon primary cause of stroke.

Mechanisms of Stroke

![Mechanisms of Stroke Diagram]

- Cryptogenic: 36%
- Other: 9%
- Atherosclerosis: 16%
- Carotid, arteriovenous: 29%
- Small vessel disease: 25%
- Isthmic stroke: 88%
Thrombosis

Thrombosis — Thrombotic strokes are those in which the pathologic process giving rise to thrombus formation in an artery produces a stroke either by reduced blood flow distally (low flow) or by an embolic fragment that breaks off and travels to a more distant vessel (artery-to-artery embolism).

Large Vessel Ischemic Stroke

Pathologies affecting large extracranial vessels include:
- Atherosclerosis
- Dissection
- Takayasu arteritis
- Giant cell arteritis
- Fibromuscular dysplasia

Pathologies affecting large intracranial vessels include:
- Atherosclerosis
- Dissection
- Arteritis/vasculitis
- Noninflammatory vasculopathy
- Moyamoya syndrome
- Vasoconstriction
Risk Factors for Atherosclerotic Cerebrovascular Disease

<table>
<thead>
<tr>
<th>Major Risk Factor</th>
<th>Minor Risk Factors</th>
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<tbody>
<tr>
<td>A) constitutional</td>
<td>Environmental influence</td>
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<tr>
<td></td>
<td>Obesity</td>
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<tr>
<td></td>
<td>Hormone estrogen def</td>
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<td>Physical inactivity</td>
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<tr>
<td>B) Acquired</td>
<td>Stress</td>
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<tr>
<td></td>
<td>Infection(C. pneumonia</td>
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<tr>
<td></td>
<td>OMI)</td>
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<td></td>
<td>Homocystin urea</td>
</tr>
<tr>
<td>Hyperlipidemia</td>
<td>Alcohol</td>
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<tr>
<td>Hypertension</td>
<td></td>
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<tr>
<td>Cigarette smoking</td>
<td></td>
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<tr>
<td>Diabetes mellitus</td>
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Common Sites for Atherosclerotic Disease
Atherosclerotic changes predominate at bifurcation points of large, major cervical (e.g., origin of internal carotid artery) or intracranial arteries.

Atheromas (arterial plaques) form from subintimal lipid deposition, smooth muscle proliferation & fibrosis. Bulging atheromas or plaques can narrow or occlude the arterial lumen (thrombois), or ulcerate and travel downstream (embolize).
Smaller perforator or lenticulostriate arteries supply deep, critical structures (internal capsule, basal ganglia, thalamus) and may also be affected by vascular disease.

**Small Vessel Disease**

Small vessel disease affects the intracerebral arterial system, specifically penetrating arteries that arise from the distal vertebral artery, the basilar artery, the middle cerebral artery stem, and the arteries of the circle of Willis. These arteries thrombose due to:

- Lipohyalinosis (a lipid hyaline build-up distally secondary to hypertension) and fibrinoid degeneration
- Aneurysm formation at their origin or in the parent large artery

The most common cause of obstruction of the smaller arteries and arterioles that penetrate at right angles to supply the deeper structures within the brain (e.g., basal ganglia, internal capsule, thalamus, pons) is lipohyalinosis.

A stroke due to obstruction of these vessels is referred to as a lacunar stroke. Lipohyalinosis is most often related to hypertension, but aging may play a role.

### Lacunar Stroke Syndromes

<table>
<thead>
<tr>
<th>Lacunar Syndrome</th>
<th>Location</th>
<th>Clinical Findings</th>
</tr>
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<tbody>
<tr>
<td>Pure motor syndromes</td>
<td>Internal capsule, corona radiata, basal ganglia, medial nuclei</td>
<td>Hemiparesis of face, arm, and leg, no sensory signs, ataxia, and paresis may be present</td>
</tr>
<tr>
<td>Pure sensory syndromes</td>
<td>Thalamic, pontine tegmentum, corona radiata</td>
<td>Isolated numbness of face, arm, and leg without motor deficit</td>
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<tr>
<td>Ataxic hemiparesis</td>
<td>Internal capsule-corona radiata, basal pons, thalamus</td>
<td>Isolated ataxia and limb dysmetria</td>
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<tr>
<td>Gait disturbance syndromes</td>
<td>Thalamocaudate, thalamic pons, or lateral medullary</td>
<td>Hemiparesis or hemiplegia of face, arm, and leg with ataxia of sensory movement</td>
</tr>
<tr>
<td>Ocular/mid-brain lacunar syndrome</td>
<td>Basal pons, internal capsule, corona radiata</td>
<td>Isolated facial weakness, diplopia, and dysphagia, with mild hemiparesis and ataxia</td>
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</table>
Amaurosis fugax: cholesterol emboli in retinal arterioles with occlusion of inferior temporal arteriole.

Embolism

Embolic strokes are divided into four categories:

- Those with a known source that is cardiac
- Those with a possible cardiac or aortic source based upon transthoracic and/or transesophageal echocardiographic findings
- Those with an arterial source (artery to artery embolism)
- Those with a truly unknown source in which tests for embolic sources are negative
Collateral Blood Flow

- If thrombosis (occlusion) of a cerebral artery occurs slowly, other collateral arteries may detour adequate blood to the endangered area of brain, limiting or even preventing an ischemic infarction
- A congenitally “complete” circle of Willis is beneficial in this regard
- Common collaterals:
  - Basilar artery (via PCA and post. communicating arteries) supplies MCA and ACA
  - External carotid artery (via retrograde flow in ophthalmic artery) can supply the intracranial internal carotid artery

Initial Evaluation of Acute Stroke

All patients with suspected stroke should have the following studies urgently as part of the acute stroke evaluation:
- Non-contrast brain CT or brain MRI
- Finger stick blood glucose
- Oxygen saturation

Other immediate tests for the evaluation of ischemic and hemorrhagic stroke include the following:
- Electrocardiogram
- Complete blood count including platelets
- Troponin
- Coagulation Studies
Transient Ischemic Attack

Transient ischemic attack (TIA) is defined clinically by the temporary nature of the associated neurologic symptoms, which last less than 24 hours by the classic definition. The definition is changing with recognition that transient neurologic symptoms are frequently associated with permanent brain tissue injury.
### ABCD² Criteria

<table>
<thead>
<tr>
<th>ABCD² Criteria</th>
<th>Points</th>
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<tbody>
<tr>
<td>Age ≥ 60 years</td>
<td>1</td>
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<tr>
<td>BP ≥ 140/90 mmHg at initial evaluation</td>
<td>1</td>
</tr>
<tr>
<td>Clinical Features of the TIA:</td>
<td></td>
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<tr>
<td>• Speech Disturbance without Weakness, or</td>
<td>1</td>
</tr>
<tr>
<td>• Unilateral weakness</td>
<td>2</td>
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<tr>
<td>Duration of Symptoms:</td>
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<tr>
<td>• 10-59 minutes, or</td>
<td>1</td>
</tr>
<tr>
<td>• ≥ 60 minutes</td>
<td>2</td>
</tr>
<tr>
<td>Diabetes Mellitus in Patient’s History</td>
<td>1</td>
</tr>
</tbody>
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### Total Risk Scores

<table>
<thead>
<tr>
<th>Total Risk</th>
<th>Scores</th>
<th>2 days</th>
<th>7 days</th>
<th>90 days</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low</td>
<td>0–3</td>
<td>1.0</td>
<td>1.2</td>
<td>3.1</td>
</tr>
<tr>
<td>Moderate</td>
<td>4–5</td>
<td>4.1</td>
<td>5.9</td>
<td>9.8</td>
</tr>
<tr>
<td>High</td>
<td>6–7</td>
<td>8.1</td>
<td>12</td>
<td>18</td>
</tr>
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</table>
Risk of Intracerebral Hemorrhage with IV t-PA 0-3 hrs

<table>
<thead>
<tr>
<th>NIHSS</th>
<th>Risk of ICH</th>
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<tbody>
<tr>
<td>0-10</td>
<td>2-3%</td>
</tr>
<tr>
<td>11-20</td>
<td>4-5%</td>
</tr>
<tr>
<td>&gt;20</td>
<td>17%</td>
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Endovascular Treatment
### Post Acute Management of Stroke and TIA

- Start antithrombotic agent: ASA, clopidogrel, ASA/dipyridamole, anticoagulation
- Start high-intensity statin (moderate intensity in age >75 yrs)
- Vascular/Carotid Imaging (CTA, MRA, Carotid Duplex)
- Consider Transthoracic echocardiogram
- Consider 30-day ambulatory cardiac monitor
- Encourage smoking cessation, weight loss, BP control
- Physical Therapy and Occupational Therapy
- Speech Language Pathology Evaluation

### Prevention of future TIA or ischemic infarction

- Control of atherosclerotic risk factors
  - hypertension, heart disease, diabetes, high cholesterol, smoking, obesity, lack of exercise
- Carotid endarterectomy
  - for symptomatic flow-limiting cervical internal carotid artery (ICA) stenosis of 70% to 99%
  - smaller benefit for symptomatic ICA stenosis 50-69%, or asymptomatic ICA stenosis 60-99%
  - carotid angioplasty or stenting (endovascular) options
- Anticoagulants (heparin, warfarin)
  - prevention of cardiac emboli (e.g., atrial fibrillation)
- Antiplatelet drugs (aspirin, clopidogrel, dipyridamole)
  - for TIA, small or large artery ischemic infarcts
Cerebral angiogram: high grade stenosis at origin of internal carotid artery. Carotid endarterectomy is a viable option here.

Management of Elevated Intracranial Pressure
Intracerebral hemorrhage (ICH) is the second most common cause of stroke (10-20%), trailing only ischemic stroke in frequency. Hypertensive vasculopathy is the most common etiology of spontaneous ICH. Cerebral amyloid angiopathy is the most common cause of nontraumatic lobar ICH in older adults, vascular malformations are the most common cause of ICH in children, but may also lead to ICH in adults.

RISK FACTORS: Major risk factors for spontaneous ICH are hypertension, older age, the presence of cerebral amyloid angiopathy (a primary cause of lobar ICH), and the use of anticoagulant therapy. Other risk factors for ICH include high alcohol intake, black race, lower cholesterol and low density lipoprotein (LDL) levels, and genetic variation.

Spontaneous Intracerebral Hemorrhage

- The signs and symptoms of ICH vary according to the location and size of the hemorrhage.
- Patients typically present with an acute onset of a focal neurologic deficit that corresponds to the part of the brain affected.
- The neurologic symptoms and signs usually increase gradually over minutes or a few hours. Headache, vomiting, and a decreased level of consciousness develop if the hemorrhage becomes sufficiently large.
- Seizures complicate 15 percent of patients, particularly if the hemorrhage is more superficial than deep.
• ICH is a neurologic and medical emergency because it is associated with a high risk of ongoing bleeding, progressive neurologic deterioration, permanent disability, and death.

• Neuroimaging with head CT or MRI is mandatory to confirm the diagnosis of ICH and to exclude ischemic stroke and stroke mimics as possible causes.

LABS:
• CBC, BMP, glucose, coagulation factors, troponin

Arteriovenous malformation (AVM) of the posterior cerebral artery

Axial CT head (non contrast): Right basal ganglia hemorrhage with extension in the ventricles complicated by acute hydrocephalus
**Subarachnoid Hemorrhage**

*Traumatic—most common cause overall of SAH*

*Ruptured congenital berry aneurysm—most common nontraumatic cause*

- Thinned outpouchings at intracranial arterial bifurcations, especially anterior circle of Willis may enlarge over time, with increased risk of rupture

SAH may also occur with cerebral hemorrhage or a systemic bleeding disorder

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- Aneurysmal SAH occurs at an estimated rate of 3 to 25 per 100,000 population; the incidence appears to vary geographically. Most aneurysmal SAH occur in individuals between 40 and 60 years of age; however young children and older adults can be affected. There is a slight preponderance of aneurysmal SAH in women.
- Cigarette smoking appears to be the most important preventable risk factor for SAH. Family history, hypertension, moderate to heavy alcohol consumption, and sympathomimetic drug use are other risk factors.
- Most aneurysms do not rupture. Aneurysm size and location influence the risk of aneurysmal SAH
- Physical exertion may trigger some aneurysmal ruptures, perhaps by precipitating an acute rise in blood pressure. Most aneurysmal SAH occur without an identifiable trigger.
- Rupture of an aneurysm releases blood directly into the cerebrospinal fluid (CSF) under arterial pressure. Rebleeding is common, especially within the first 24 hours. Blood spreads throughout the CSF space and leads to secondary complications of increased intracranial pressure, vasospasm, and hydrocephalus.