



Stroke/Transient ischemic attack

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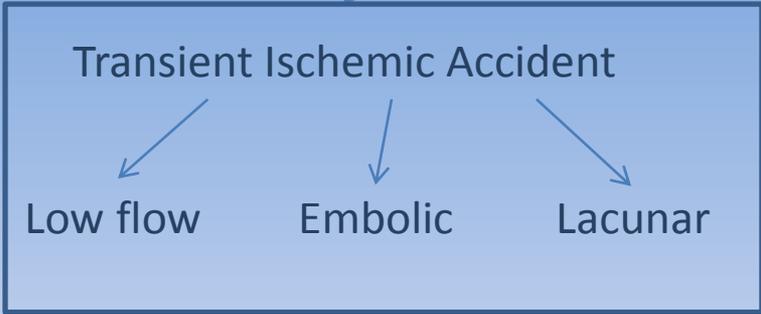
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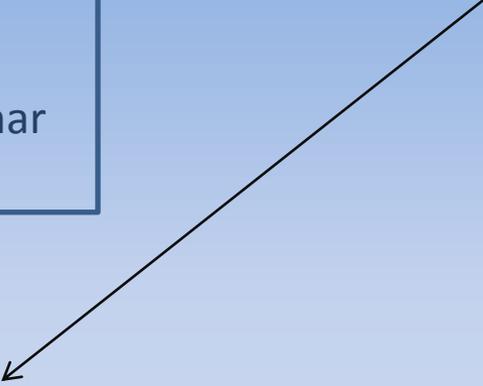
Ischemia



Infarction



Stroke



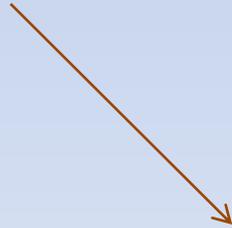
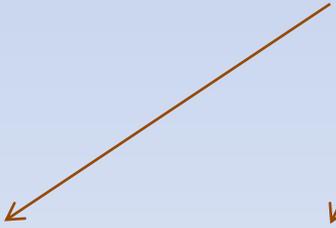
Ischemic Stroke

Hemorrhagic Stroke



Intracerebral hemorrhage

Subarachnoid hemorrhage



Thrombosis

Embolism

Systemic Hypoperfusion

Transient Ischemic Attack (TIA)

TIA

- Sometimes called a mini-stroke
- Stroke symptoms usually lasting less than 24 hours and not leaving infarcted brain tissue

Pathophysiology of TIA

- A transient ischemic attack (TIA) is a syndrome. These syndromes are divided into three pathophysiologic mechanisms:
 - Large artery low-flow TIA (true TIA)
 - Embolic TIA, which may be artery-to-artery, or due to a cardioaortic or unknown source
 - Lacunar or small penetrating vessel TIA

Large artery low-flow TIA (true TIA)

- They are often associated with a tightly stenotic atherosclerotic lesion at the internal carotid artery at its origin or in the intracranial portion.
 - Or:
 - Atherosclerotic stenotic lesions in the middle cerebral artery stem or at the junction of the vertebral and basilar artery.
 - But essentially any obstructive vascular process in the extracranial or intracranial arteries can cause a low-flow TIA syndrome
 - with collateral flow to the potentially ischemic brain also is impaired.

Clinical presentation of Low-flow TIA

- Low-flow TIAs usually are **short-lived** (minutes) and often **recurrent**. They may occur as little as several times per year but typically occur more often (once per week or up to several times per day).
- Low-flow TIAs are generally stereotyped.
 - Symptoms due to ischemia from these lesions often include **hand, arm, leg, face, tongue, or cheek numbness or weakness together, or a combination of one or more**.
 - Dominant hemisphere: recurrent aphasic syndromes appear when there is focal ischemia
 - Nondominant hemisphere: recurrent neglect

Embolic TIA

- The embolus may arise from a pathologic process in an artery, usually extracranial, or from the heart (eg, atrial fibrillation or left ventricular thrombus)
- If the primary pathologic process is thought to be embolic, a diligent search for its source is necessary before therapy to prevent future stroke can be initiated.

Clinical presentation of Embolic TIA

- Embolic TIAs typically last **hours** rather than minutes as in low-flow TIAs. They may be **infrequent** since they are the result of emboli from a specific source (eg, a one, two, or three-time phenomenon).
- When the source of the embolus is in a proximal vessel, recurrent emboli can lodge in different branches of the parent vessel giving different symptoms.
- Symptoms depend upon the size of the embolic fragment in relation to the size of the artery occluded and location of emboli.

Lacunar or small penetrating vessel TIA

- Lacunar or penetrating or small vessel TIAs are due to transient cerebral ischemia induced by stenosis of one of the intracerebral penetrating vessels.
- Occlusion of these small intracerebral penetrating vessels usually is due to lipohyalinosis from hypertension, but also may arise because of atheromatous disease at their origin.
- Recurrent stereotyped TIAs may occur here as well (as in low-flow)

Clinical presentation of lacunar TIA

- These small vessel TIAs cause symptoms that are similar to the lacunar strokes that are likely to follow.
 - **Face, arm, and leg weakness or numbness**
- Lacunar infarcts may be preceded by lacunar TIAs consisting of brief repetitive stereotyped clinical symptoms and signs.

What a TIA means....

- A substantial proportion of patients with classically defined TIA have corresponding small ischemic lesions on MRI (diffusion or perfusion weighted).
- A classically defined TIA with symptoms lasting for as little as a few minutes can be associated with infarction, whereas a spell lasting for many hours may cause no signal changes on diffusion-weighted MRI.
- While TIAs generally do not cause permanent brain damage, they are a serious warning sign of future strokes
 - 40 percent of all people who have experienced a TIA will go on to have an actual stroke.
 - Studies show that almost half of all strokes occur within the first 2 days after a TIA.
 - Within 2 days after a TIA, 5 percent of people will have a stroke.
 - Within 3 months after a TIA, 10 to 15 percent of people will have a stroke

Management of TIA

- Basic laboratory studies that are suggested by the history and physical examination.
 - ruling out metabolic and hematologic causes of neurologic symptoms, including hypoglycemia, hyponatremia, and thrombocytosis
- Electrocardiogram
- Brain imaging
 - CT or MRI is indicated in all patients with suspected TIA or minor stroke as soon as possible
- Neurovascular imaging
- Antiplatelet therapy for secondary prevention of stroke is recommended in many TIA patients

Stroke

Epidemiology

- Stroke is the fourth leading cause of death in America and a leading cause of adult disability.
 - Kills over 130,000 people per year in US
 - There are around 7 million stroke survivors in US over age 20.
 - 2010, over \$73 billion in direct and indirect costs from stroke
- African-Americans almost twice as likely to have stroke than whites.
- More common in males
 - Females may become more ill however
- Approximately 80% of strokes are preventable.

Risk Factors you can not change

- **Risk Factors You Cannot Change**
 - Age
 - Gender
 - Men have a higher risk of getting heart disease than women except in older adults.
 - African-Americans, Mexican Americans, American Indians, Hawaiians, and some Asian Americans also have a higher risk for heart problems.
 - Diseases such as cancer, chronic kidney disease, and some types of arthritis.
 - Aneurysms
 - Pregnancy-- both during and in the weeks right after the pregnancy
 - Congenital heart defects
 - Heart arrhythmias

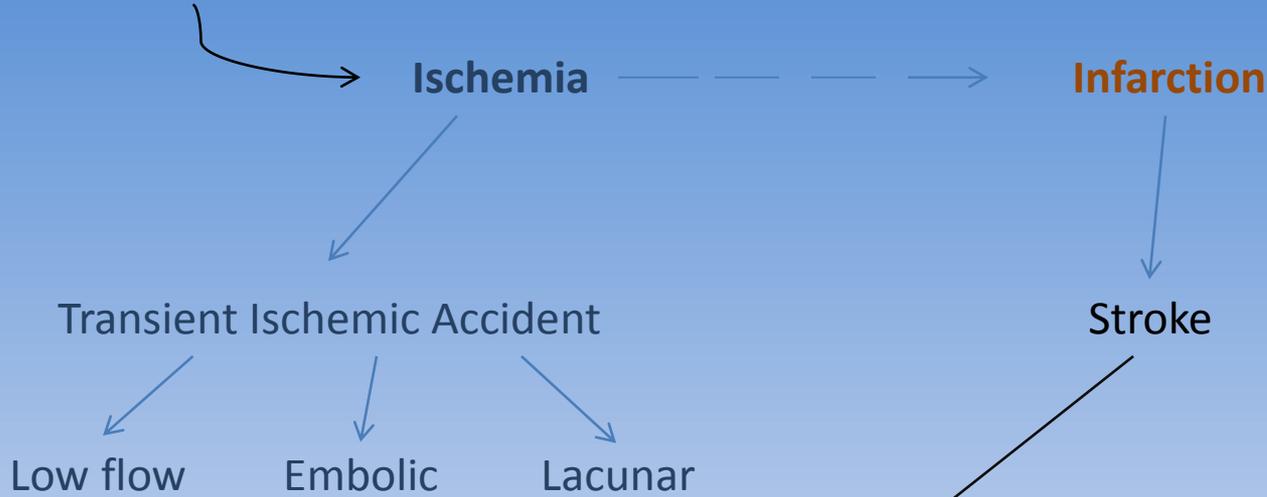
Modifiable Risk Factors

- **Modifiable Risk Factors**
 - Smoking
 - Cholesterol
 - Hypertension
 - Obesity/Diabetes
 - Alcoholism
 - Cocaine
 - Blood thinners and falls

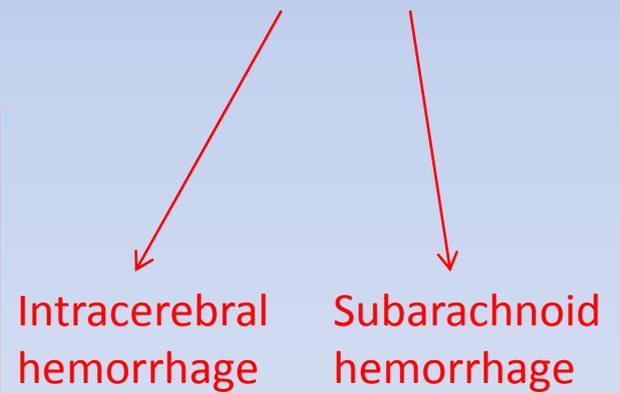
Types of Stroke

- Ischemic
 - Thrombosis
 - Embolism
 - Systemic Hypoperfussion
- Hemorrhagic
 - Intracerebral hemorrhage
 - Subarachnoid hemorrhage

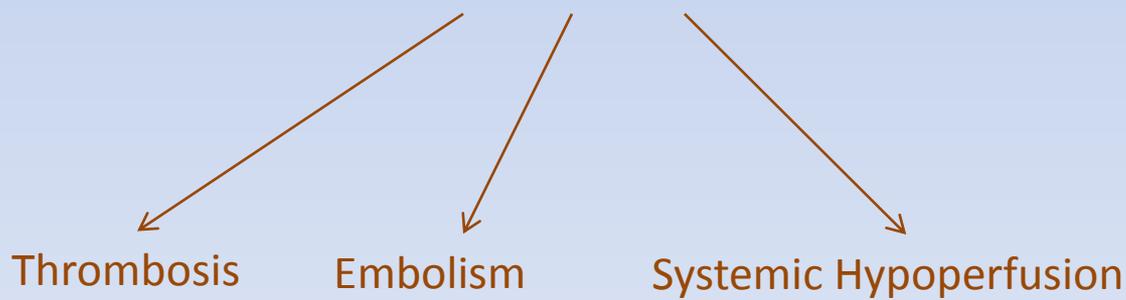
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Hemorrhagic Stroke



Ischemic Stroke



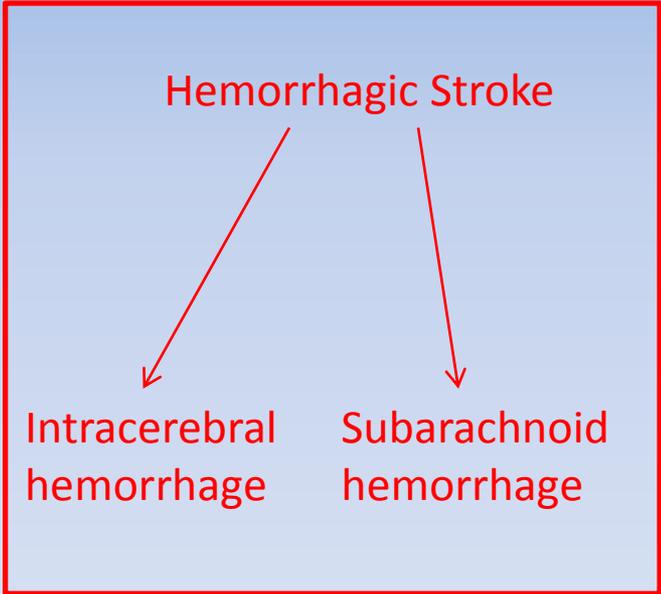
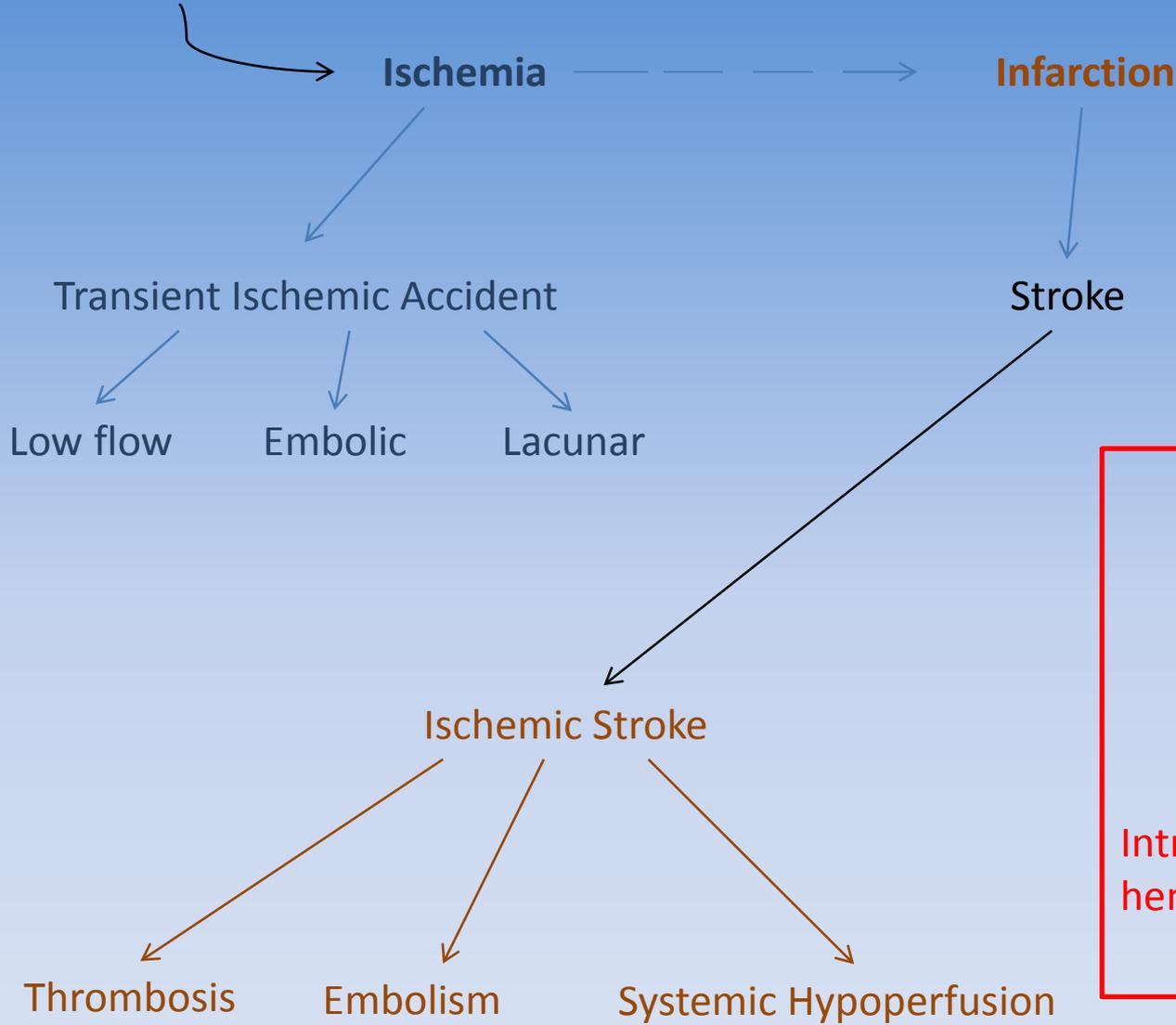
Brain Ischemic Stroke

- **Thrombosis**- local in situ obstruction of an artery. The obstruction may be due to disease of the arterial wall(arteriosclerosis, dissection, or fibromuscular dysplasia) or due to a superimposed thrombosis.
 - Patients **varying** between normal and abnormal or progressing in a stepwise or fashion with periods of improvement
 - Anemic infarct
- **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; continued emboli can occur if the source of embolism is not found and treated.
 - **Abrupt** and **maximal** at onset with rapid recovery
 - Hemorrhagic infarct
- **Systemic hypoperfusion** is a more general circulatory problem, manifesting itself in the brain and perhaps other organs.
 - Usually tissue was at risk prior to hypoperfusion (arterial disease or watershed areas).

TOAST classification scheme

- Large-artery atherosclerosis
- Cardioembolism
- Small-vessel occlusion
- Stroke of other determined etiology
- Stroke of undetermined etiology
 - Two or more causes identified
 - Negative evaluation
 - Incomplete evaluation

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Hemorrhagic Stroke

- *Intracerebral hemorrhage* refers to bleeding directly into the brain parenchyma.
- *Subarachnoid hemorrhage* refers to bleeding into the cerebrospinal fluid within the subarachnoid space that surrounds the brain.

Intracerebral hemorrhage

- Bleeding in intracerebral hemorrhage (ICH) is usually derived from arterioles or small arteries. The bleeding is directly into the brain, forming a localized hematoma that spreads along white matter pathways. Accumulation of blood occurs over **minutes or hours**.
- The most common causes of ICH are hypertension, trauma, bleeding diatheses, amyloid angiopathy, illicit drug use (mostly amphetamines and cocaine), and vascular malformations.
- The neurologic symptoms usually increase **gradually over minutes or a few hours**.
 - In contrast to brain embolism and SAH, the neurologic symptoms related to ICH may not begin abruptly and are not maximal at onset.
- ICH destroys brain tissue as it enlarges. The pressure created by blood and surrounding brain edema is life-threatening; large hematomas have a high mortality and morbidity. The goal of treatment is to contain and limit the bleeding.

Subarachnoid hemorrhage

- The two major causes of SAH are rupture of arterial aneurysms and bleeding from vascular malformations.
- Rupture of an aneurysm releases blood directly into the cerebrospinal fluid (CSF) under arterial pressure. The blood spreads quickly within the CSF, **rapidly** increasing intracranial pressure. The bleeding usually lasts only a **few seconds** but rebleeding is very common.
- Symptoms of SAH begin abruptly in contrast to the more gradual onset of ICH. The sudden increase in pressure causes a cessation of activity. This is the stereotyped “thunderclap headache”.
- Vomiting occurs soon after onset.
- The goal of treatment of SAH is to identify the cause and quickly treat it to prevent rebleeding. The other goal of treatment is to prevent brain damage due to delayed ischemia related to vasoconstriction of intracranial arteries; blood within the CSF induces vasoconstriction, which can be intense and severe.
 - bedrest, analgesia, prophylactic treatment for deep venous thrombosis, and discontinuation of antithrombotics.
 - Complications worsen outcome and should be prevented and promptly treated.
 - Antithrombotic therapy can be started or resumed after definitive treatment of the aneurysm.
 - Ventriculostomy is place in patients with elevated intracranial pressure

Symptoms

- All stroke and TIA focal deficits are dependent on anatomical location of ischemia/infarct.

Anterior Cerebral Artery

- Cognitive/confusion/behavioral disturbances
- Contralateral weakness in arms
- Sensory loss in legs > arms
- Incontinence

MCA

- Contralateral hemiplegia
- Hemisensory loss and homonymous hemianopia
 - Eyes deviate toward lesion
- Dominant-aphasia
- Non Dominant- comprehension with confusion, apraxia

Penetrating branches of PCA

- Weber syndrome
 - Contralateral hemiplegia
 - CN III palsy
- Benedikt
 - Contralateral ataxia or athetosis
 - CN III palsy

PICA

- Wallenberg
 - Ipsilateral facial sensory loss
 - Contralateral body sensory loss
 - Vertigo
 - Ataxia
 - Dysarthria
 - Dysphagia
 - Horner's syndrome

Management of patient presenting with signs of stroke

- Patient presenting with sudden loss of focal brain function
 - Ischemic stroke?
- Differential Dx
 - Migraine*
 - Intracerebral hemorrhage
 - Head trauma
 - Brain tumor
 - Todd's Palsy
 - Systemic infx
 - Toxic-metabolic disturbances (hypoglycemia, acute renal failure, hepatic insufficiency, exogenous drug intoxication)

Management cont'd

- Diagnosing an intracerebral hemorrhage (ICH) or subarachnoid hemorrhage (SAH) as soon as possible with brain imaging (CT preferred)
 - Ensure stable patient first
- All suspected stroke patients need following tests immediately
 - Noncontrast brain CT or brain MRI
 - Electrocardiogram
 - Complete blood count including platelets
 - Cardiac enzymes and troponin
 - Electrolytes, urea nitrogen, creatinine
 - Serum glucose
 - Prothrombin time and international normalized ratio (INR)
 - Partial thromboplastin time
 - Oxygen saturation
 - Lipid profile

Interventions

- **Intravenous thrombolysis with alteplase, Tissue plasminogen activator (tPA) within 3 HOURS....kind of**
- Aspirin initiated within 48 hours of stroke onset
 - Already on Aspirin...either switch to Clopidogrel or add Dipyridamole
- Prophylaxis for deep venous thrombosis and pulmonary embolism
- Antithrombotic therapy at discharge
- Lipid lowering therapy initiation
- Blood pressure reduction, once the acute phase of ischemic stroke has passed
- Smoking cessation along with management of obesity, diabetes, and metabolic syndrome are unproven but generally recommended as well

Treating beyond 3 hours?

- History of using tPA in ischemic stroke:
 - Dec 14th 1995 in NEJM
 - ECASS showed no benefit
 - NINDS showed increased functional recovery but also increased symptomatic brain bleed chances
 - FDA approves tPA for within 3 hours
- Atlantis JAMA 1999 showed no benefit for 3-5 hrs with increased symptomatic bleed rate
- ECASS 3 in NEJM 2008 showed benefit but with higher number needed to treat than <3 hrs for treating patients 3-4.5 hrs
 - Also showed increased bleed rate
 - American heart association gives go for tPA after 3 hours but no FDA approval yet

Thrombolysis under 50

- Recent article in *Neurology* showed that outcomes were better among patients aged 18 to 50 than among those aged 51 to 80.
 - In functional recovery at 3 months.
 - Also decreased risk of rebleeding.

The future

- Study used a stroke model in monkeys to test a drug that may alleviate the neurotoxicity caused by ischemia.
- Neurological function — not just infarct size — was substantially improved by an agent that may lessen the neurotoxicity caused by ischemia.
 - Could be delivered up to 3 hours after stroke so fits in current treatment protocol.

OMT and stroke

- While an acute management with OMT is not used for stroke patients there is obviously a place for an osteopathic approach in a general sense.
- Strokes and TIAs may present with only neuromuscular complaints that need to be recognized.
- Helping to retrain muscles during recovery.
 - With PT and OT
- Full body approach in mind, body and spirit recovery.

Sources

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 - Antiplatelet therapy for secondary prevention of stroke
 - Clinical diagnosis of stroke subtypes
 - Initial evaluation and management of transient ischemic attack and minor stroke
 - Clinical diagnosis of stroke subtypes