• **Definition.**
  An acute onset of neurological dysfunction due to an abnormality in cerebral circulation with resultant signs & symptoms that correspond to involvement of focal areas in the brain...

• Used interchangeably with CVA that refer to the cerebrovascular conditions that accompany either ischemic or hemorrhagic lesions

• Variety of deficits are possible including changes in the level of consciousness, impairments of sensory, motor, cognitive, perceptual and language functions.

• Neurological deficits must persist at least 24 hours to be classified as stroke

• May result to Hemiplegia (paralysis) or hemiparesis (weakness)

• **Epidemiology**
  - 3rd leading cause of death in the US
  - Most common serious neurologic condition in clinical practice
  - In the US, about 500,000 strokes occur annually
  - Leading cause of serious neurologic disability
  - Common in people greater than 65 years
  - Double incidence among blacks
  - incidence increases dramatically with age, 2x every decade after 55 y.o.
  - pts. with intracerebral hemorrhage accounts for the largest no. of deaths
  - thromboembolic stroke survival rates are lessened with the presence of other medical comorbidities

• **Pathophysiology**
  - interruption of blood flow for only a few minutes sets in motion the series of pathoneurological events.
  - ISCHEMIC PRENUMBRA - the area surrounding the core of focal infarction
  - release of excess glutamate(excitatory neurotransmitter) causes changes in calcium distribution

• **Classification**
  **Etiological Categories**
  - Thrombosis
  - Embolus
  - Hemorrhage

• **Management Categories**
  - TIA’s
  - RIND
  - minor stroke
  - major stroke
  - deteriorating stroke
  - young stroke

• **Main Mechanism**
  - ISCHEMIC — result of a thrombus, embolism or conditions that produce low systemic perfusion pressures.
Cerebral Thrombosis – refers to the formation of blood clot or thrombus within the cerebral arteries or their branches

Lead to ischemia with resulting infarction or tissue Death (Atherothrombotic Brain Infarction[ABI])

Cerebral Embolus – are traveling bits of matter formed elsewhere that are released in the bloodstream and travel to the cerebral arteries where they lodge in a vessel producing occlusion and infarction.

most common cause comes from the HEART

HEMORRHAGIC – abnormal bleeding into the extravascular areas of the brain secondary to aneurysm or trauma

Intracranial Hemorrhage(HH)- rupture of a cerebral vessel with subsequent bleeding into the brain.

Primary cerebral Hemorrhage- typically occurs in small blood vessels weakened by atherosclerosis

Subarachnoid Hemorrhage – bleeding into the subarachnoid space

AV malformation

EARLY SIGNS OF STROKE

sudden , severe headaches with no known cause

sudden weakness or numbness of the face, arm or leg on one side of the body

loss of speech, or trouble or understanding speech

sudden dimness or loss of vision particularly in one eye

unexplained dizziness, unsteadiness or sudden falls

especially along with any of the symptoms

RISK FACTORS

Modifiable by Lifestyle Changes

Cigarette Smoking

Heavy – 1.5-2 packs per day

Light – less than 10 sticks per day

Hyperlipidemia/Hypercholesterolemia

Cholesterol: > 200 mg/dL

Low Density Lipoprotein (LDL): > 160 mg/dL

Obesity

Cardiac Disease

Modifiable by Medical Means

Diabetes Mellitus (DM)

Important sign of stroke risk

Approximately 35% of persons with TIA will have a stroke in 5 years

Considered a physical warning sign of stroke and require urgent clinical attention

Asymptomatic Carotid Bruit

Also a well-established risk factor of stroke

Presence of blood murmur which indicates turbulence

Erythrocytosis

Increase RBC

Transient Ischemic Attack (TIA)

Hypertension (HTN)

Non-Modifiable

Race: blacks

Age: > 65 years old;
Sex: males

Previous Stroke

**CEREBRAL BLOOD FLOW**

* Circle of Willis

* VASCULAR SYNDROMES

**ACA** – supplies the MEDIAL aspect of the brain (frontal and parietal lobes) and subcortical structures including the basal ganglia, ant. fornix and 4/5 of the corpus callosum

  - Most common characteristic is contralateral hemiparesis and sensory loss with greater involvement of the LOWER EXTREMITY

**MCA**

  - supplies the entire LATERAL aspect of the brain (frontal, parietal, temporal lobes)

Most common characteristics are contralateral spastic hemiparesis and sensory loss of the face, UE and LE with the face and the UE more involved.

Lesions of parieto-occipital cortex (dominant hemisphere) produce aphasia

Lesions of the non-dominant hemisphere of the same area produce perceptual deficits including unilateral neglect, anosognosia, Apraxia and spatial disorganization.

**MOST COMMON SITE OF OCCLUSION IN STROKE**

**PCA**

  - supplies the occipital lobe and medial and inferior temporal lobe; also supplies the upper brainstem, midbrain, and posterior diencephalon and most of the thalamus

  - Thalamic sensory syndrome (a persistent and unpleasant hemibody sensation)

  - Occipital infarction produces homonymous hemianopsia, visual agnosia, prosopagnosia or cortical blindness

**Vertebrobasilar Artery Syndrome**

  - vertebral artery — supply the cerebellum and medulla

  - basilar artery — supplies the pons and the internal ear and the cerebellum
Lacunar Syndrome

<table>
<thead>
<tr>
<th>FLARE MOTOR STROKE</th>
<th>- POSTERIOR LIMB of internal capsule, basis pontis, medullary pyramids</th>
</tr>
</thead>
<tbody>
<tr>
<td>FLARE SENSORY STROKE</td>
<td>- thalamus, thalamocortical projections</td>
</tr>
<tr>
<td>SENSORY-MOTOR STROKE</td>
<td>- junction of internal capsule and thalamus</td>
</tr>
<tr>
<td>DYSPHARIA-CLUMSY HAND</td>
<td>- ANTERIOR LIMB of internal capsule and pons</td>
</tr>
<tr>
<td>ATAXIC HEMIPARESIS</td>
<td>- corona radiata, internal capsule, pons, and cerebellum</td>
</tr>
<tr>
<td>HEMIBALLISMUS</td>
<td>- head of caudate nucleus, thalamus and subthalamic nucleus</td>
</tr>
</tbody>
</table>

- unmasking of previously latent functional pathways
- assumption of function by undamaged redundant neural pathways

OTHERS:
- reversibility from diachisis
- denervation supersensitivity
- regenerative proximal sprouting of transected neuronal axons

LANGUAGE & PERCEPTUAL FUNCTION:
- variable
- aphasia SLOWER THAN motor recovery
- starts between 3-6 mos.
- Non-fluent aphasia < favorable than fluent aphasia
- Compression EARLIER return than expression
- Perceptual deficits may show improvement 20 wks-1 yr post-stroke

NEUROLOGIC RECOVERY

1ST 3-6 MOS. (1st mechanism)
- resolution of local brain edema
- resorption of local toxins
- improved circulation
- recovery of partially damaged ischemic neurons

2nd mechanism – NEUROPLASTICITY
- 2 TYPES:
  - collateral sprouting of new synaptic connections

CLAUDE'S SYNDROME
- crossed cerebellar ataxia

BRACHIAL SYNDROME
- embolic occlusion of MCA = hand/arm & hand weakness alone

FRONTAL OPERCULAR SYNDROME
- facial weakness
- motor aphasia
- wrist/arm weakness

BALTZ'S SYNDROME
- unilateral ataxia

GERSHMAN
- ataxia
- finger agnosia
- F-A confusion

THALAMIC SYNDROME OF DEJEANS & BLOOM
- contra hemisensory loss of both superficial (P/T) & deep sensation (U/M)

OTHERS:
- crossed hemiplegia
- bilateral hemiparesis
- bilateral cranial nerve palsies (CN 3, 4, 6, 7)
- bilateral hemifacial spasm
- bilateral oculomotor palsies
- bilateral oculomotor palsies
- bilateral oculomotor palsies
PATTERNS OF RECOVERY:
- at onset, complete, flaccid paralysis with areflexia
- within 48 hours have return of DTR
- Brunnstrom stages
- Limb synergies

POTENTIAL COMPLICATIONS:
- falls
- musculoskeletal probs
- bowel/bladder incontinence
- physiologic deconditioning
- common associated medical probs
- TYPICAL POST-STROKE PROBLEMS
- absent/weak ms strength
- joint contractures
- incoordination
- abnormal ms tone
- SOME SPECIFIC PROBLEMS IN STROKE
- shoulder subluxation
- brachial plexus and PNI
- shoulder-hand syndrome
  - stage 1 – painful stiff hand, edema, dec. temp.
  - stage 2 – dec. pain & edema, inc. stiffness, osteoporosis, trophic changes
  - stage 3 – atrophy
- cognitive aspects
- post-stroke depression – 6 mos-2 yrs
- communication/language probs

BEHAVIOR RELATED TO THE LEFT AND RIGHT HEMISPHERES

<table>
<thead>
<tr>
<th>BEHAVIOR</th>
<th>LEFT HEMISPHERE</th>
<th>RIGHT HEMISPHERE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cognitive Style</td>
<td>Processing information in a sequential, linear manner Observing and analyzing details</td>
<td>Processing information in a simultaneous, holistic, or gestalt manner Grouping overall organization or pattern</td>
</tr>
<tr>
<td>Perception/Cognition</td>
<td>Processing and producing language</td>
<td>Processing nonverbal stimuli (environmental sounds, speech information, complex shapes, and designs) Visual-Spatial perception Drawing Inferences, synthesizing information</td>
</tr>
<tr>
<td>Academic Skills</td>
<td>Reading: sound-symbol relationships, word recognition, reading comprehension Performing mathematical calculations</td>
<td>Mathematical reasoning and judgment Alignment of numerals in calculations</td>
</tr>
<tr>
<td>Motor</td>
<td>Sequencing movements Performing movements and gestures to command</td>
<td>Stabilizing a movement or posture</td>
</tr>
<tr>
<td>Emotions</td>
<td>Expression of positive emotions</td>
<td>Expression of negative emotions Perception of emotion</td>
</tr>
</tbody>
</table>

HISTORY AND EXAMINATION
- of importance are the pattern of onset & the course of neurological symptoms
- abrupt onset with rapid coma --- suggestive of cerebral hge
- severe headache precedes LOC
- embolus – occurs rapidly with no warning and is frequently assoc. with heart disease
- thrombosis – more variable and uneven onset with pt.’s history of TIA’s presence of minor or major risk factors, etc.
- Physical exam includes both the general medical and neurological examination

NEUROVASCULAR TESTS INCLUDE
- neck flexion
- palpation of arteries
- auscultation of heart and blood vessels
- ophthalmic pressures
- DIAGNOSTIC TESTS
- Urinalysis
- Blood analysis
- Blood sugar level
- Blood chemistry
• Etc.

More modern imaging techniques

• CT scan - most commonly used imaging
• used in acute stroke to rule out other brain lesions such as brain tumors
• it is important to remember that the extent of CT lesion does not necessarily correlate with clinical signs
• MRI - greater resolution of the brain and its structural detail is obtained than the CT scan
• Positron Emission Tomography (PET)
• Ultrasound Transcranial Doppler
• Cerebral Angiography
• DIRECT IMPAIRMENTS

SOMATOSENSORY DEFICIT
• most common distribution is face-UE-LE pattern (55%)
• followed by face-UE pattern (29%)
• UE-LE pattern (7%)

PAIN
• lesions in the PCA involving the ventral posterior-lateral thalamus can lead to THALAMIC SENSORY SYNDROME
• delayed in onset triggered by simple stroking of the skin, pinprick loud, noises, etc...
• little relief in analgesics

VISUAL DEFICITS

MOTOR DEFICITS

EARLY STAGES OF STROKE -- flaccidity
• usually followed by spasticity

Sequential Recovery stages
• mass patterns of movement - synergies
• general pattern of recovery was described by Twitchell & Brunnstrom into six stages (see box 17-1)
• Bobath collapsed the sequence into three main recovery stages:
  • initial flaccid stage
  • stage of spasticity
  • stage of relative recovery

Alterations in Tone

• flaccidity - present immediately after stroke and is due to CEREBRAL SHOCK
• usually short-lived but some will persist in pts. with lesions restricted to the primary motor cortex or cerebellum.
• Spasticity - emerges in about 90% cases and occurs on one side opposite the lesion predominantly in the antigravity muscles
• Abnormal Synergy Patterns
• Certain muscles that are not affected or involved in either the synergy patterns are: (FLATS)
  • latissimus dorsi
  • teres major
  • serratus anterior
  • finger extensors
  • ankle evertors

Abnormal Reflexes

• initially – hyporeflexia
  • - middle stages of recovery - hypereflexia
• stretch reflexes are hyperactive and pts. demonstrate clonus, clasp-knife reflexes and (+)Babinski
• STNR-Symmetric Tonic Neck Reflex
• flexion of the neck produce flexion of the UE’s
• ATNR –Asymmetric Tonic Neck Reflex
• head rotation to the left may cause extension of the left UE’s and LE’s with flexion of the right UE’s & LE’s.

• STLR – Symmetric Tonic Labyrinthine Reflex

• supine positioning may produce an increase in extensor tone while prone may produce an increases in flexor tone.

• TLR – Tonic Lumbar Reflex

• rotation of the trunk to the hemiplegic side results in the flexion of the UE and extension of hemiplegic LE. Rotation to the uninvolved side produces opposite reactions.

Associated Reactions – are unintentional movements of an involved limb resulting from an intended action of another limb or reflex stimulation.

  – flexion evokes flexion in the UE
  – flexion of one LE evokes extension of the other LE
  – elevation of the hemiplegic UE with the elbow extended above the horizontal may elicit an abduction and extension of the fingers. (Souque’s phenomenon)
  – Resistance to abduction/adduction produce a similar response to the opposite limb in both UE & LE. (Raimiste’s phenomenon)
  – Mutual dependency between hemiplegic limbs (flexion of the UE elicits flexion of the LE on the hemiplegic limb. (Homolateral Limb Synkinesis)

PARESIS & ALTERED MUSCLE ACTIVATION

• paresis accounts for 80-90% of cases

• owing the high incidence of MCA strokes, the UE is more affected than the LE

• changes in muscle composition include greater muscle atrophy of type II fast-twitch fibers that results to difficulty with initiation and production of rapid, high-force movements.

MOTOR PROGRAMMING DEFICITS

• left hemisphere- primary role in sequencing movements thus if affected, pts. have APRAXIA

• ideomotor apraxia – where movement is not possible on command but may occur automatically

• ideational apraxia – purposeful movements are not possible either automatically or on command.

SPEECH AND LANGUAGE DISORDERS

• APHASIA – an acquired communication disorder caused by brain damage characterized by an impairment of language comprehension, formulation and use.

• major classification include : fluent, non-fluent and global

• Fluent aphasia/ Wernicke’s aphasia/ Receptive aphasia
  – speech flows smoothly with a variety of grammatical constructions and preserved melody of speech. Pt. demonstrates difficulty in understanding spoken language and in following commands.
  – Lesion is in the auditory association cortex in the left lateral temporal lobe

• Nonfluent Aphasia/ Broca’s/Expressive aphasia
  – flow of speech is slow and hesitant, vocabulary limited and syntax is impaired. Speech production is labored or absent.
  – Lesion is located in the premotor area of the left frontal lobe

• Global Aphasia – severe aphasia characterized by marked impairments of both production and comprehension.
• **DYSPHAGIA** – swallowing difficulty; occurs in pts. with lesion affecting the medullary brainstem (C.N. 9&10), large vessel pontine lesions as well as acute hemispheric lesions.

**Types of aphasia**

<table>
<thead>
<tr>
<th>Type</th>
<th>Comprehension</th>
<th>Repetition of Spoken Language</th>
<th>Naming</th>
<th>Fluency</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wernicke's</td>
<td>Impaired</td>
<td>Impaired</td>
<td>Impaired</td>
<td>Preserved or increased</td>
</tr>
<tr>
<td>Broca's</td>
<td>Preserved (except grammar)</td>
<td>Impaired</td>
<td>Impaired</td>
<td>Decreased</td>
</tr>
<tr>
<td>Global</td>
<td>Impaired</td>
<td>Impaired</td>
<td>Impaired</td>
<td>Decreased</td>
</tr>
<tr>
<td>Conduction</td>
<td>Preserved</td>
<td>Impaired</td>
<td>Impaired</td>
<td>Preserved</td>
</tr>
<tr>
<td>Nonfluent (motor)</td>
<td>Preserved</td>
<td>Preserved</td>
<td>Impaired</td>
<td>Impaired</td>
</tr>
<tr>
<td>Fluent (sensory)</td>
<td>Impaired</td>
<td>Preserved</td>
<td>Impaired</td>
<td>Preserved</td>
</tr>
<tr>
<td>Isolation</td>
<td>Impaired</td>
<td>Echolalia</td>
<td>Impaired</td>
<td>No purposeful speech</td>
</tr>
<tr>
<td>Anomic</td>
<td>Preserved</td>
<td>Preserved</td>
<td>Impaired</td>
<td>Preserved except for word-finding pauses</td>
</tr>
<tr>
<td>Pure word deafness</td>
<td>Impaired only for spoken language</td>
<td>Impaired</td>
<td>Preserved</td>
<td>Preserved</td>
</tr>
<tr>
<td>Pure alexia</td>
<td>Impaired only for reading</td>
<td>Preserved</td>
<td>Preserved</td>
<td>Preserved</td>
</tr>
</tbody>
</table>

**COGNITIVE DYSFUNCTION**

**PERCEPTUAL DYSFUNCTION**

- Occurs mostly in right sided lesions and left hemiparesis, these include body scheme d/o & body image d/o, spatial relation d/o, agnosias & apraxia.
  - BODY SCHEME – refers to the postural model of the body including the relationship of the parts to each other and the relationship of the body to the environment.
  - BODY IMAGE – visual & mental of the body that include feelings about one’s body.
  - Spatial Relations Syndrome refers to the constellation of impairments that have in common a difficulty in perceiving the relationship between the self and two or more objects in the environment.
  - - IPSILATERAL PUSHING SYNDROME/PUSHER SYNDROME
    - unusual motor behavior characterized by the pt.’s strong lateral lean toward the hemiplegic side.

- Attention Disorders – includes impairment in sustained attention, selective attention, divided attention or alternating attention.
  - Memory d/o – include impairments in immediate recall and short-term or long term memory.
  - immediate and short term recall are most common deficits
  - Confabulation – memory gaps that are filled with inappropriate words or fabricated stories
  - Perseveration – continued repetition of words, thoughts or acts not related to current text.
Executive Function—those capacities that enable a person, to engage in a purposeful behaviors that includes volition, planning, purposeful action.

AFFECTIVE DISORDERS

- Lesion to the right hemisphere impair emotional functioning.
- Pt. may demonstrate emotional dysregulation syndrome termed EMOTIONAL LABIALITY. Characterized by pathological laughing and weeping in which the patient changes quickly from laughing to crying with only slight provocation.

- DEPRESSION—extremely common, occurring in about 1/3 of the cases, with an average time of 7-8 months.
  - 6 months-2 years after CVA is most likely time
  - Pts. with lesions in the left hemisphere may experience more frequent and more severe depression than the right hemisphere.

BEHAVIORAL HEMISPHERIC DIFFERENCES

- Left hemisphere damage (right hemiplegia)
- Demonstrates difficulties in communication and in processing information in a sequential manner.
- Cautious, anxious and disorganized, more hesitant in trying new tasks, however tend to be realistic in their problems.
- Right hemisphere damage (left hemiplegia)
- Difficulty in spatial-perceptual tasks and in grasping the whole idea of the task.
- Quick and impulsive
- Safety is the greater issue

SEIZURES

BLADDER & BLADDER DYSFUNCTION

- Indirect impairments

VENOUS THROMBOSIS

- Potential complications for all immobilized patients.

SKIN BREAKDOWN

DECREASED FLEXIBILITY

SHOULDER SUBLUXATION AND PAIN

REFLEX SYMPATHETIC DYSTROPHY

DECONDITIONING

- Rehabilitation Management
  - Begun early in the acute stage optimizes the pt.’s potential for functional recovery.
  - Can begin as soon as the patient is medically stabilized, typically 72 hours after.
- Assessment Methods
  - Determined by the therapists based on the patient unique needs, impairments and disabilities. Vary by severity, stage of recovery, phase of rehabilitation.

LEVELS OF CONSCIOUSNESS

- Use Glasgow Coma scale, Rancho Levels of Cognitive Functioning Scale.
- Speech and language deficits
  - Close collaboration with the speech pathologist is important in making an accurate determination of the patient’s communication deficits.

STANDARDIZED STROKE ASSESSMENT INSTRUMENTS

- FUGL-MEYER ASSESSMENT OF PHYSICAL PERFORMANCE (FMA) developed by Signe Brunnstrom
- Motor Assessment Scale (MAS) developed by Carr & Shepherd.