

Dr.Ahmed Fathy Zaki

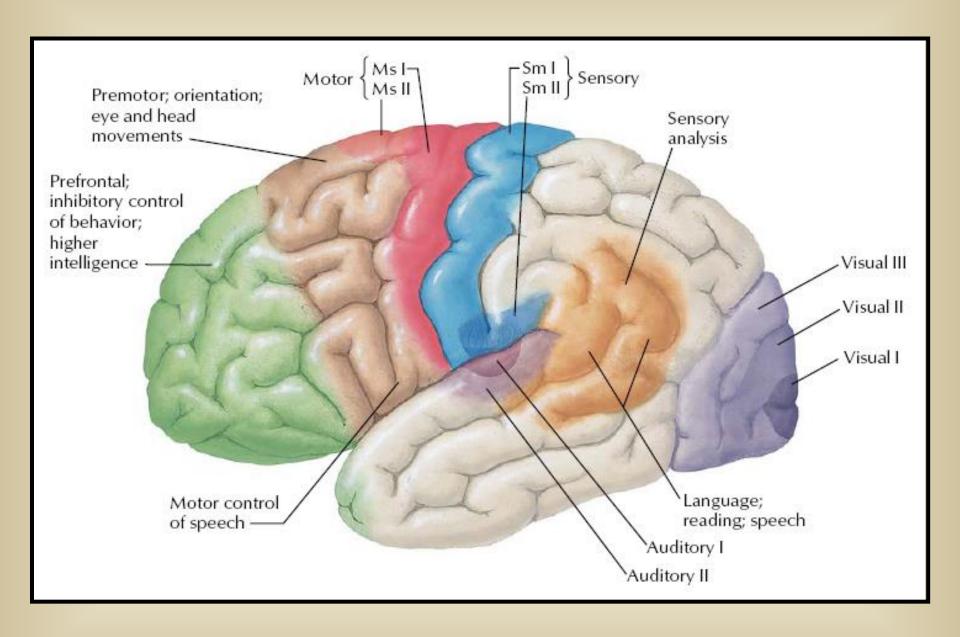
Definition

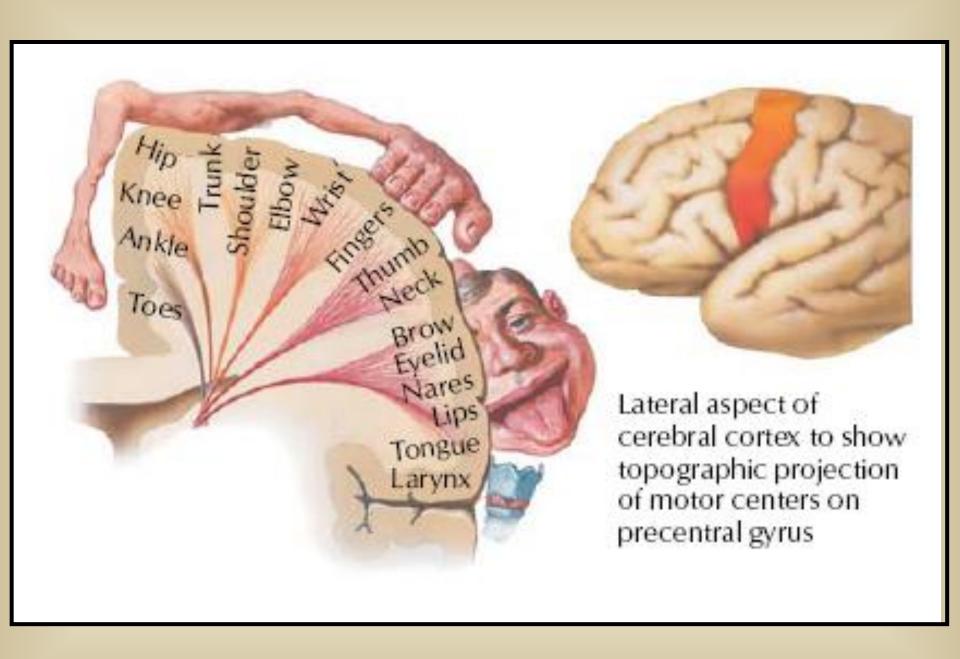
Stroke:-

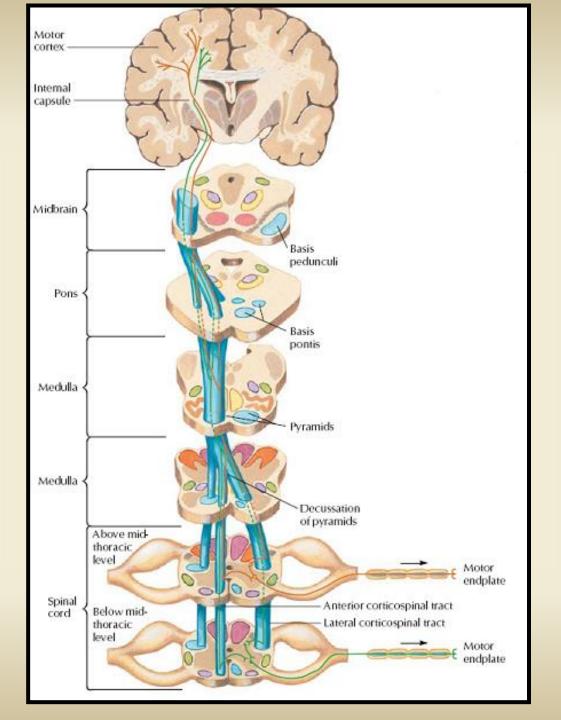
Is rapidly developing clinical symptoms and or signs of focal or global loss of cerebral function, lasting more than 24 hours or leading to death, with no apparent cause other than that of vascular origin.

Transient ischemic attacks (TIA):

Transient focal neurological deficits lasting for less than 24 hours, usually minutes, of vascular ischemic origin with complete recovery.







Risk factors for stroke

- 1) Age: is the strongest and inventible risk factor of stroke, stroke in people aged 75-84 ys is 25times the risk in people aged 45-54 ys.
- Sex: there is small male excess of strokes mainly in middle age most probably due to prophylactic role of endogenous sex hormone in female (estrogen).
- Hypertension: either elevated diastolic or systolic blood pressure is associated with increased risk, with each 7.5 mmhg increase in diastolic blood pressure stroke risk doubles.
- -Hypertension causes stroke (infarction) through its continuous friction power with increasing the extent and severity of atheroma, or with sudden rise → rupture of vascular malformations causing hemorrhage.

Risk factors for stroke

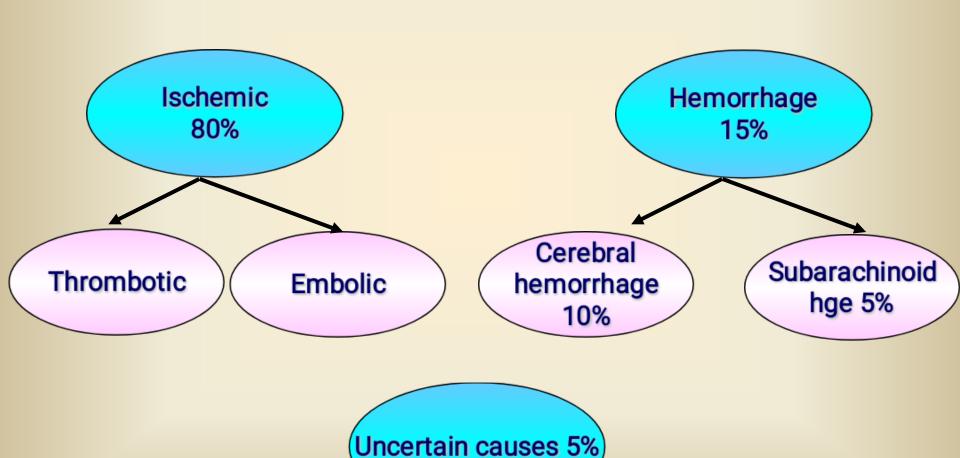
- 4. Diabetes mellitus.
- 5. Cardiac diseases: e.g. coronary heart diseases, heart failure, AF, valvular disease, arrhythmia → embolic infarction.
- 6. Dyslipidemia: high levels of LDL and low levels of HDL.
- 7. Hyper-coagulability: increase plasma fibrinogen, increase haematocrite, polycythemia (cigarette smoking).
- 8. Alcohol consumption: increase the blood pressure & blood lipids and is usually associated with AF and cardiomypathy.
- 9. Coffee consumption: small hyper-lipaedemic effect.
- 10. Cigarettes smoking: doubles stroke.
- 11. Dietary habits: diet rich in fish, with moderate salt intake, high k, and rich in fresh fruits, and vegetables, vitamin E, C, folic acid & selenium have a protective value.

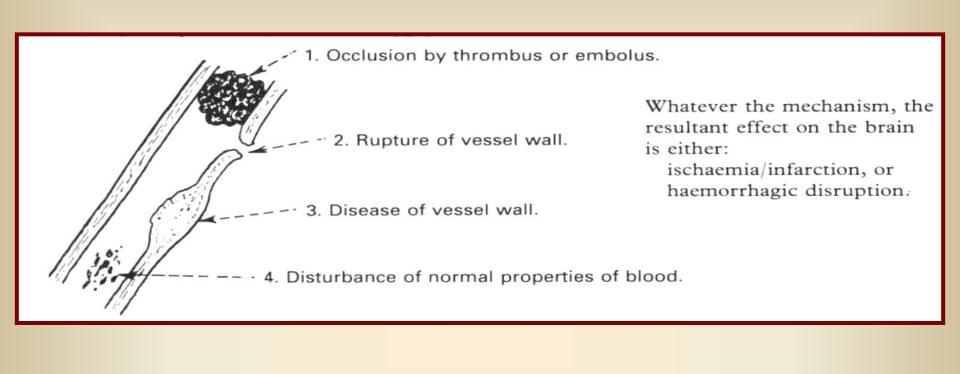
Risk factors for stroke

- 12) Drug induced stroke:
- Hypertensive drugs.
- Pills, cytotoxic drugs → thromboembolic strokes.
- -Aspirin, anticoagulants, thrombolytic therapy → *Hge*.
- 13) Contraceptive pills: with high doses of estrogen >50 ug → triple the risk of stroke while lower doses of estrogen lowers the risk, therefore post menopausal estrogen replacement may have some protective effect.
- 14) Past history of TIA or mild stroke.
- 15) Family history of stroke.
- 16) Lower social class.
- 17) Body built.
- 18) Physical inactivity.
- 19) Chronic stress.

The causes of stroke







Cerebral ischemia (infarction)

Etiology

- a) Arterial wall diseases:
- Atherothromboembolism → 50 % of causes
- Intracranial small vessel disease → 25 % of causes
- Trauma
- -Dissection
- Fibromusclar dysplesia
- Inflammatory vascular diseases
- Irradiation
- Infections
- Binswauger's disease

Etiology

- b) Embolism from the heart (20%)
- 1-Paradoxical embolism : Atrial septal defect
 - Ventricular septal defect
 - Patent foremen oval

- 2- Left atrium: AF
- 3- Mitral valve: -Rheumatic stenosis or incompetence
 - Infective endocarditis Prosthetic valve
 - MV prolapse MV calcification.
- 4- Left ventricle: Myocardial infarction.
 - Cardiomypathy. Aneurysm.
- 5- Aortic valve:
- 6- Congenital cardiac diseases.
- 7- Cardiac surgery, catheterization, angiography.

Etiology

c) Hematological disorders:-

- Polycythemia.
- Thrombocytopenia.
- Leukemia.
- Sickle cell disease.
- Iron deficiency anemia.
- Thrombotic thrombocytopenic purpura.
- DIC.
- Hypercoagulability.

Intracranial hemorrhage

This may be:-

- Subdural, extradural → mostly traumatic.
- 2. Subarachinoid hemorrhage.
- 3. Intraventricular hemorrhage.
- 4. Cerebral hemorrhage.
- 5. Cerebellar hemorrhage.

Causes of spontaneous intraventricular hge

- 1. Hypertension.
- 2. Vascular malformation.
- 3. Aneurysms.
- 4. Hemorrhagic blood diseases.
- 5. Vascular tumors.
- 6. Anti platelets, anticoagulant & thrombolytic therapy.
- 7. Intracranial venous thrombosis.
- 8. Drug abuse.

NB: the vascular causes are the most common in hemiplegia.

Other causes of hemiplegia

- 1. Space occupying lesion: *abscess*, *tumors*.
- 2. Infection: encephalitis, meningoencephalitis.
- 3. Demyelinating: *MS, disseminated encephalomyelitis*.
- 4. Congenital cerebral palsy.
- 5. Hysterical.

Clinical presentation and localization of a case of hemiplegia.

The clinical presentation of hemiplegia varies much according to-

- The site of the lesions in the nervous system.
- Etiology of hemiplegia.

General clinical features:-

- Onset: sudden in embolic and hemorrhagic, acute in thrombosis, gradual in neoplasm, intermittent in MS.
- 2) Course: may be regressive in inflammatory, vascular, traumatic causes, Progressive in neoplasm.
- 3) Weakness: is usually affecting one half of the body UL, LL in equal degree or one may be affected than the other, more affecting fine movements, more in distal muscles, more in progravity muscles.

Clinical presentation and localization of a case of hemiplegia.

1) Muscles tone:

- In acute lesions there is a shock stage (2-6 weeks) → complete loss of tone →tone gradually returns and spasticity appears
- In gradual lesions: spasticity develops from the start and affects the antigravity muscles more.

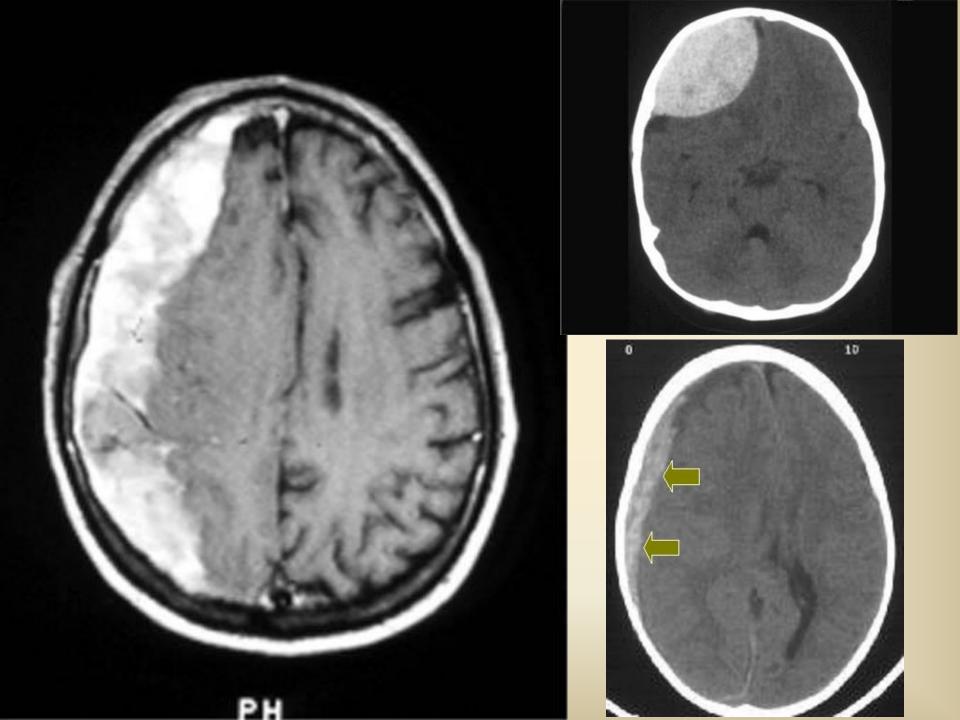
2) Reflexes:

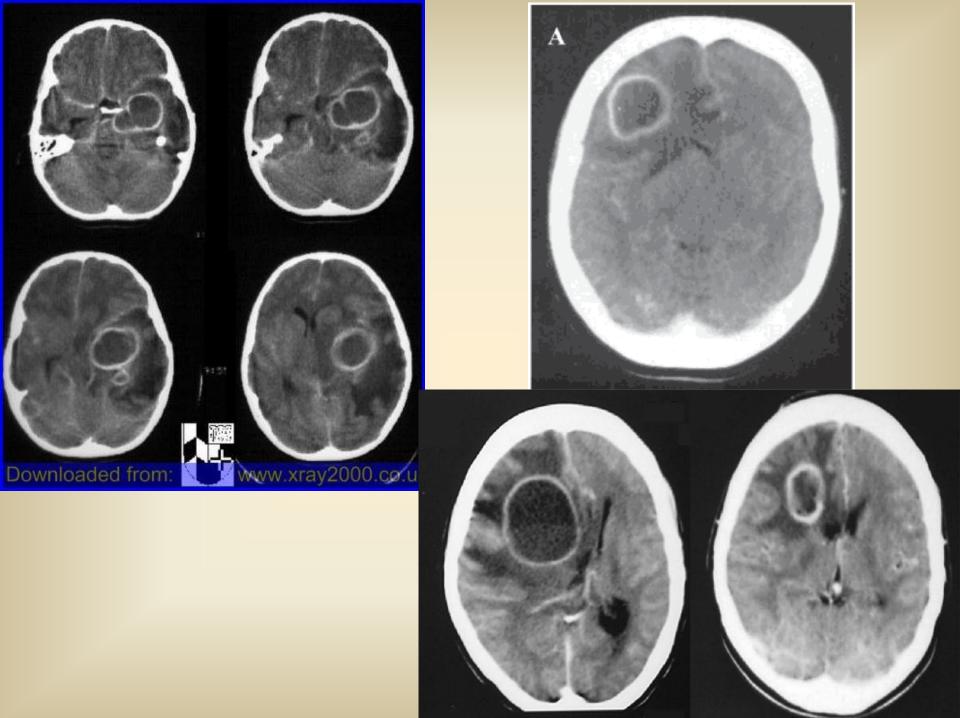
- Deep tendon reflexes in the affected limb are exaggerated, pathological reflexes and clonus may be elicited.
- Superficial reflexes:-

+ve babinski sign.

lost abdominal and cremasteric on the affected side.

- 3) Gait: circumduction.
- 4) Other features: sensory impairment, cranial nerves affection dependant on site of the lesions.

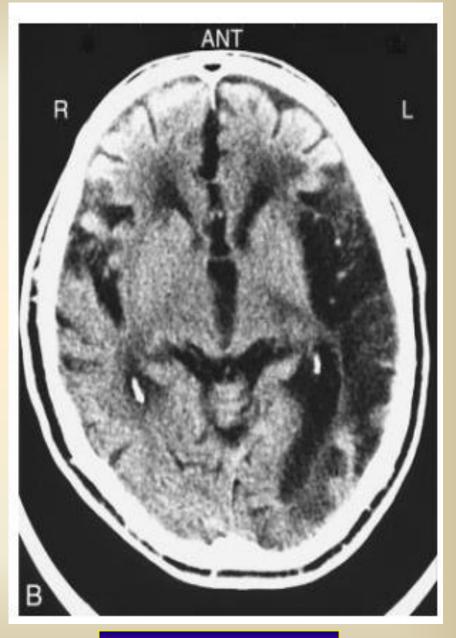




Investigation of stroke

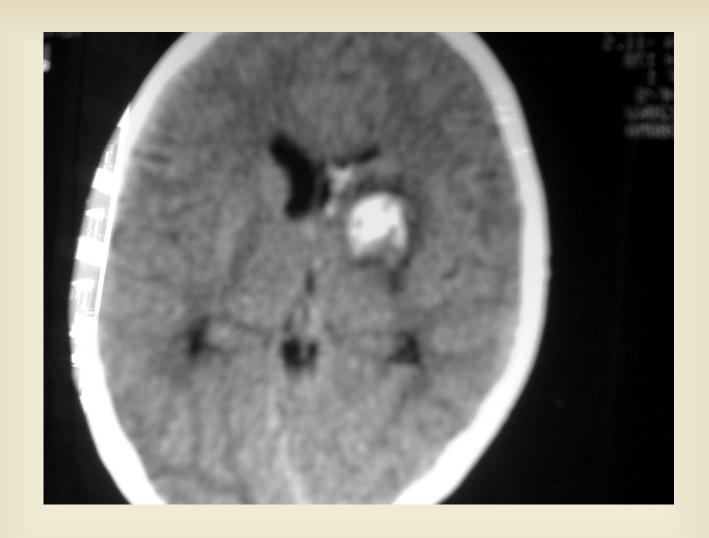
- A) Specific investigations:-
- 1. C.T scanning:
- The next step after clinical diagnosis Diagnose the type and site of stroke
- Hemorrhage appears once as welldemarcated hyperdense area (white)
 - •
- Infarction → hypodense area.



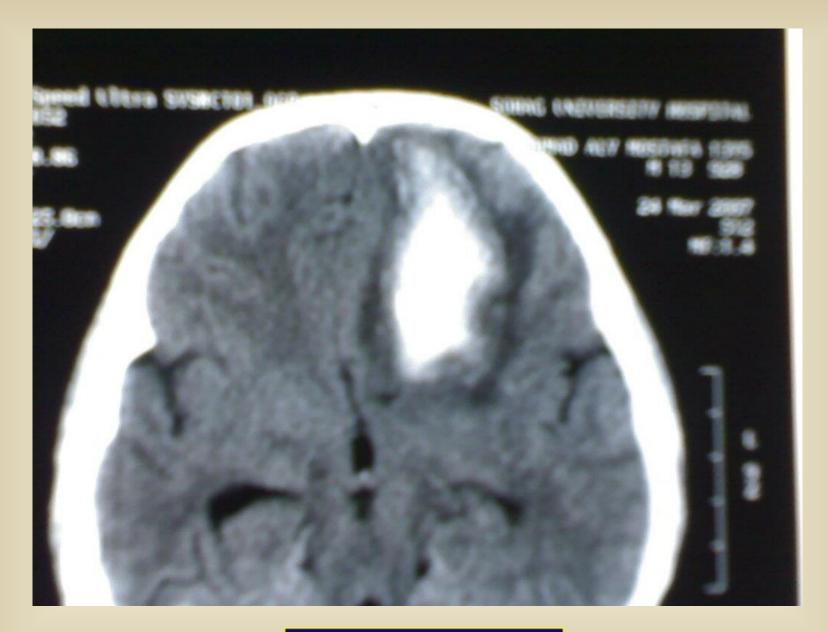


Cerebral hemorrhage

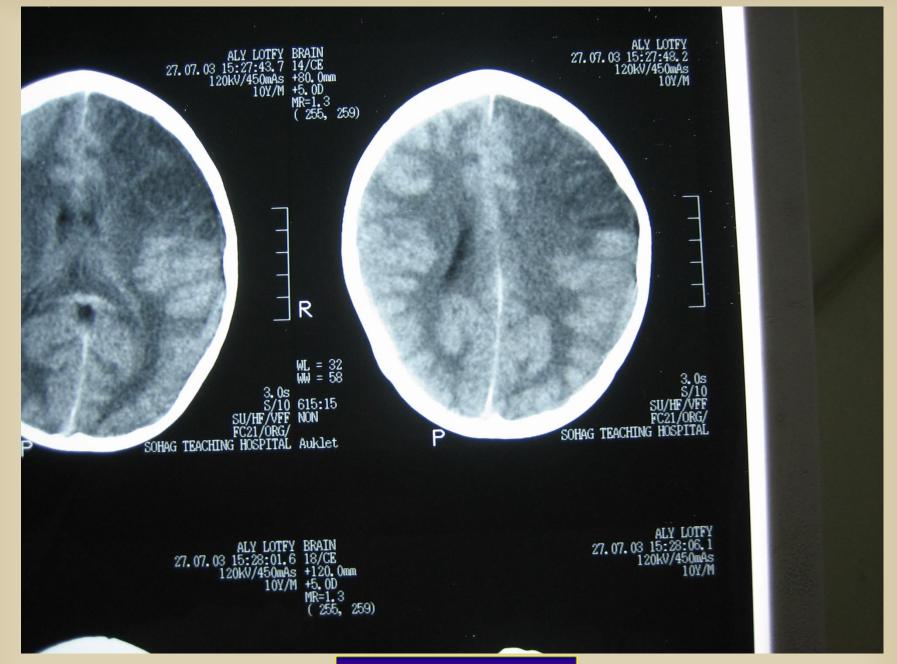
Cerebral infarction



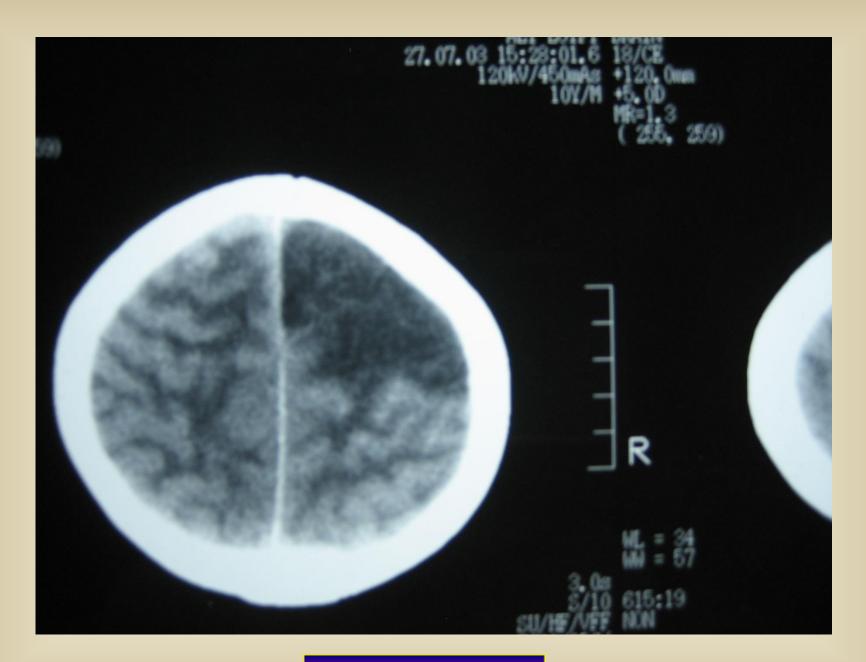
Cerebral hemorrhage



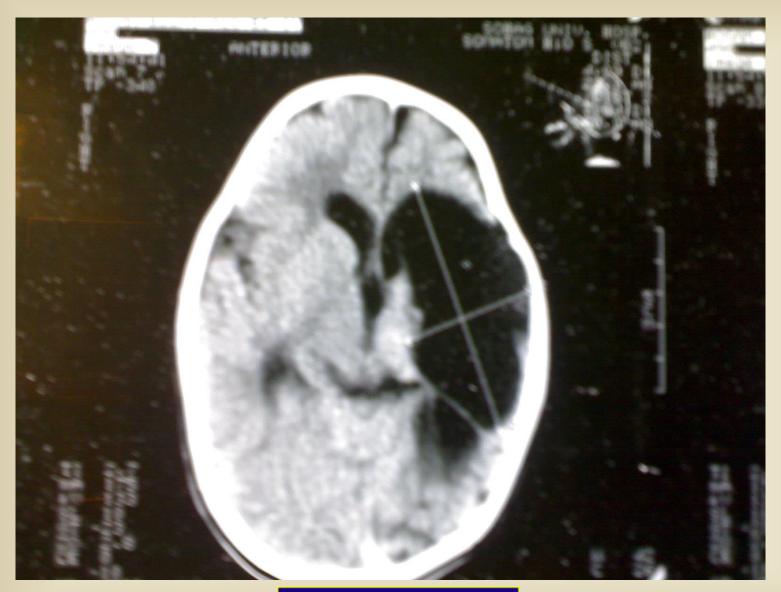
Cerebral haemorrhge



Cerebral infarction



Cerebral infarction



Cerebral infarction

Investigation of stroke

- 2. MRI:
- More sensitive, can show early infarctions and small or deeply seated infarction.
- 3. Angiography:
- Imaging the cerebral circulation → stenotic, dissecting Vs, AVM, MRA is now more sensitive.
- 4. EEG:
- Ipsilateral slow wave, not specific or sensitive.

Investigation of stroke

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B) General investigations:-
ECG 24hours monitoring → IHD & arrhythmia.
Renal function test → renal impairment in
   hypertension
Lipid profile → atherosclerosis
Blood sugar → diabetes mellitus
Blood electrolytes → electrolyte imbalance
Echocardiography → cardiac source of emboli.
CBC & coagulation profiles → hematological
   disorders.
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A- General measures:

- 1- Maintenance of vital signs:
- a- Pulmonary functions→ open air way, blood gases, O2 supply if there is hypoxia.
- b- Cardiovascular:
- -blood pressure (normal arm) not deal up to 160/100, if > 160/100→ gradual decrease is needed, urgent deal only in hypertensive encephalopathy
- Heart rate. -ECG
- c- Blood electrolytes and fluid intake reserve output.
- d- Temperature (normal axilla)
- e- Blood glucose.

2- Nursing care :-

- Turn the patient regularly at least every 2 hrs.
- Frequent massage & washing the body with alcohol & talc powder.
- Catheterization if there is retention or incontinence.
- If the patient is comatose or there is chocking, nasogasteric tube must be inserted for oral feed.

Routine monitoring in acute stroke:

Vital signs:-

Respiratory rate, rhythm, blood gases.

Heart rate, rhythm, ± ECG monitors..

Blood pressure.

Temperature.

- Neurological:-
- Conscious level. Weakness.
- Pupils. Seizures
- General:-
- Fluid intake. Electrolytes & urea.
- Blood glucose.

2-Specific measures:

- A) Dealing with the occluded vessels (in infarction only):
- 1. Anticoagulants (heparin) usually of value in :
- TIA, progressive stroke.
- Small infarction.
- Cardiac source of embolism.
- Young age patient.

Of doubtful value (better to be avoided) in:-

- Hypertensive patient.
- Elderly patient.
- History of peptic ulcer.
- Hemorrhagic tendencies.
- Full heparinization 20000-30000 IU/day, replace with oral anticoagulants or antiplatelets.

- 2. Antiplatelets:-
- Aspirin 300mg once for 2wks, maintaince 75-150 mg/ day for ever.
- 3. Thrombolytic therapy:-
- -Streptokinase, urokinase → of doubtful effect.
- -Tissues plasminogen activator (now is widely used).
- B) Improve cerebral blood flow.
- C) Dehydrating measures to relieve cerebral edema :-
- Manitol 1.5 2 gm/kg once, then 30 50 gm/3hours.
- Glycerol 30 ml /4-6 hours, for up to one weak.
- Corticosteroids 4-6mg/4-6hours, gradual withdrawal.

D) Neuronal protection:-

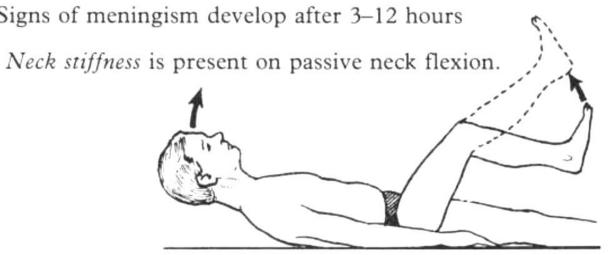
- Barbiturates.
- Phenytoin.
- Hypothermia.
- Ca+ channel blocker.
- Vit E, C, selenium.
- Free radical scavengers.
- Hyperpanic O2 atmosphere.

- E) Dealing with systemic complications which may occur and cause neurological deterioration as:-
- 1. Pneumonia 7- Pulmonary embolism.
- 2. Cardiac failure. 8 Cardiac arrhythmia.
- 3. Dehydration. 9 Electrolyte imbalance.
- 4. Bedsores. 10 Urinary tract infection.
- Bleeding stress ulcer.11 Septicemia.
- 6. DVT.

- F) Dealing with neurological causes of deterioration as:-
- 1. Hemorrhagic transformation of infarction.
- 2. Cerebral edema.
- Brain shift.
- Recurrent embolism.
- 5. Recurrent hemorrhage.
- 6. Propagation of the thrombus.
- Seizures.
- Rehabilitation after the 1st few days by the physiotherapist.

G) Starting prevention:

- Control of the risk factors mainly DM, hypertension, cardiac causes, hyperlipedemia, smoking, etc.
- Anticoagulants , in selected cases.
- Antiplatelets are given mainly to those:
- With TIA.
- Mild ischemic stroke.
- With high vascular risk factors.
- Carotid endarterectomy in symptomatic patients (TIA, mild ischemic stroke) with internal carotid artery stenosis.



Kernig's sign: stretching nerve roots by extending the knee causes pain.

