NEUROPSYCHIATRIC ASPECTS OF CEREBROVASCULAR DISORDERS

OUTLINE

- INTRODUCTION
- HISTORY
- STROKE
- NEUROPSYCHIATRIC CORRELATES OF STROKE
- OTHER RELATED CONDITIONS

INTRODUCTION

- Cerebrovascular disorders consist of a large number of disorders that disrupt the vascular supply to the brain.
- Based on aetiology, these disorders are divided into:

  * Ischemic (absence of blood flow)

  * Hemorrhagic (bleeding in or around covering of brain)
- Can be classified according to the size of affected blood vessel

  (Large and Small Artery Diseases)
- Diseases of the vascular system of the brain contribute greatly to the psychiatric disability, chiefly in the elderly population and mainly result of stroke.

INTRODUCTION (CONT.)

- The vast majority of studies have focused on patients with stroke. Neuropsychiatric correlates of stroke includes (Post-Stroke) : [1]

  - Vascular Neurocognitive Disorder
  - Depression
  - Mania
  - Anxiety
  - Psychosis
  - Apathy
  - Catastrophic Reaction
  - Pathological Laughter and Crying
  - Anosognosia

- The psychiatric components of some illness such as SLE and other forms of vasculitis appear to be attributable in part to the involvement of cerebral vasculature.[2]

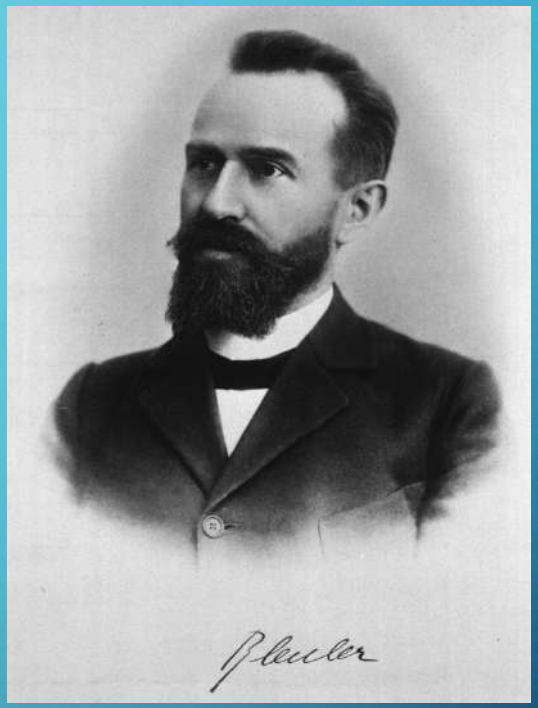
HISTORY

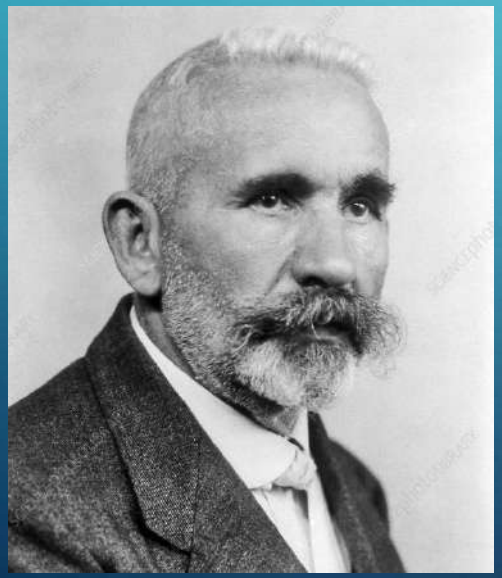
- Early reports of depression after brain damage (usually caused by cerebrovascular disease) were made by neurologists and psychiatrists in case descriptions.

- In the early 1900s, **Adolf Meyer (1866-1950)** identified that several clinical syndromes such as delirium, dementia and aphasia were the direct result of brain injury.

HISTORY (CONT.)

- **Eugen Bleuler (1857-1939)** described "melancholic moods lasting for months and sometimes longer appear frequently" in Patients after strokes.



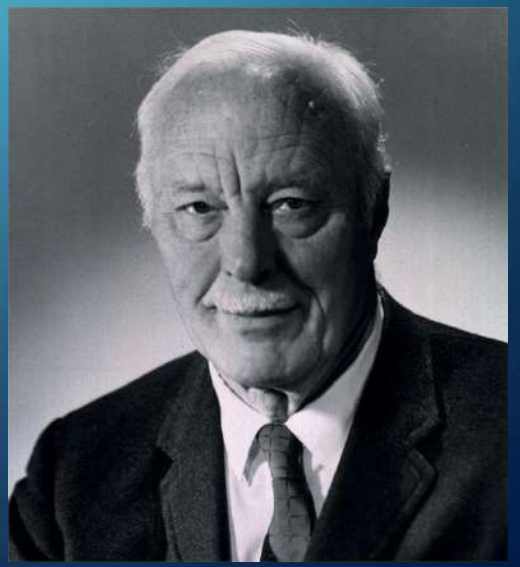


- **Emil Kraepelin (1856-1926)** recognized an association between manic-depressive insanity and cerebrovascular disease.

HISTORY (CONT.)

- **Redvers Ironside (in 1956)** described the clinical manifestations of a condition, Pathological laughter and crying (PLAC), emotional disorder that has only been associated with brain injuries, including cerebral infarction, and represents one of the differential diagnoses for depression in stroke patients.

- Another emotional abnormality, also thought to be characteristic of brain injury, is the indifference reaction described by **Derek Denny-Brown (in 1952)**.



HISTORY (CONT.)

- **Leonore Welt (In the late 19th century)** first described euphoria and loquaciousness associated with orbital frontal injury.
- **Hermann Oppenheim (1858-1919)** used the term "witzelsucht" to refer to the inappropriate humour in these patients.
- While **Karl Kleist (1879-1960)** stated that the orbital frontal cortex was the centre of emotional life and the dorsal lateral frontal cortex was the source of psychomotor and intellectual activity.

HISTORY (CONT.)

- **Kurt Goldstein (1878-1965)** described the catastrophic reaction, another neuropsychiatric disorder historically associated with brain injury such as stroke.

- Anosognosia is a term introduced by **Joseph Jules François Félix Babinski (1857-1932)** to indicate a patient's lack of awareness of his or her hemiplegia.

STROKE

- A stroke is defined as ‘the sudden loss of blood supply to the brain leading to permanent tissue damage.’
- The World Health Organization (WHO) definition of stroke is: “rapidly developing clinical signs of focal (or global) disturbance of cerebral function, with symptoms lasting 24 hours or longer or leading to death, with no apparent cause other than of vascular origin” . [3]
- By applying this definition transient ischemic attack (TIA), which is defined to last less than 24 hours, and patients with stroke symptoms caused by subdural hemorrhage, tumors, poisoning, or trauma are excluded.

STROKE (CONT.)

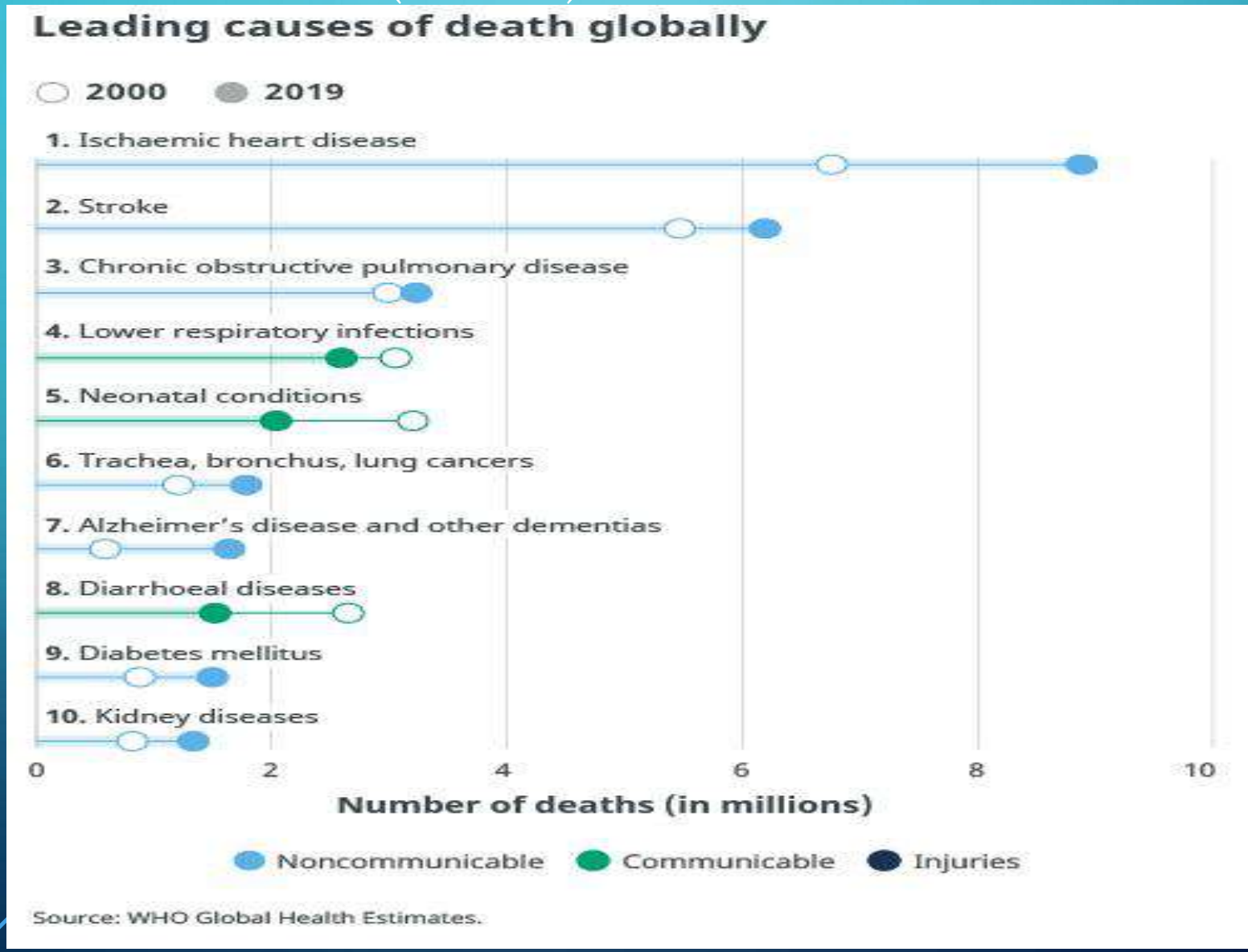
- Two essential pathological processes underlying stroke:

  -Infarction (ischemia)

  -Haemorrhage

- 90 % of strokes are due to ischemia
- 10 % are caused by hemorrhagic events; less common, more severe and greater mortality than ischemic strokes.
- In adults, stroke incidence increases considerably with age, and the incidence of first stroke is greatest in those 85 years or older.

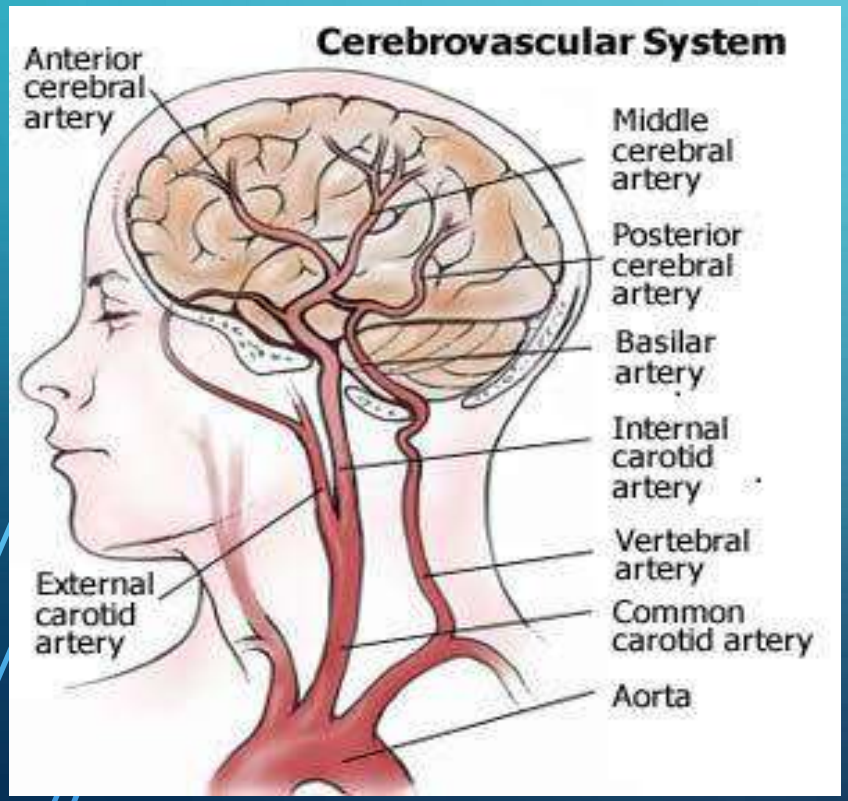
STROKE (CONT.)

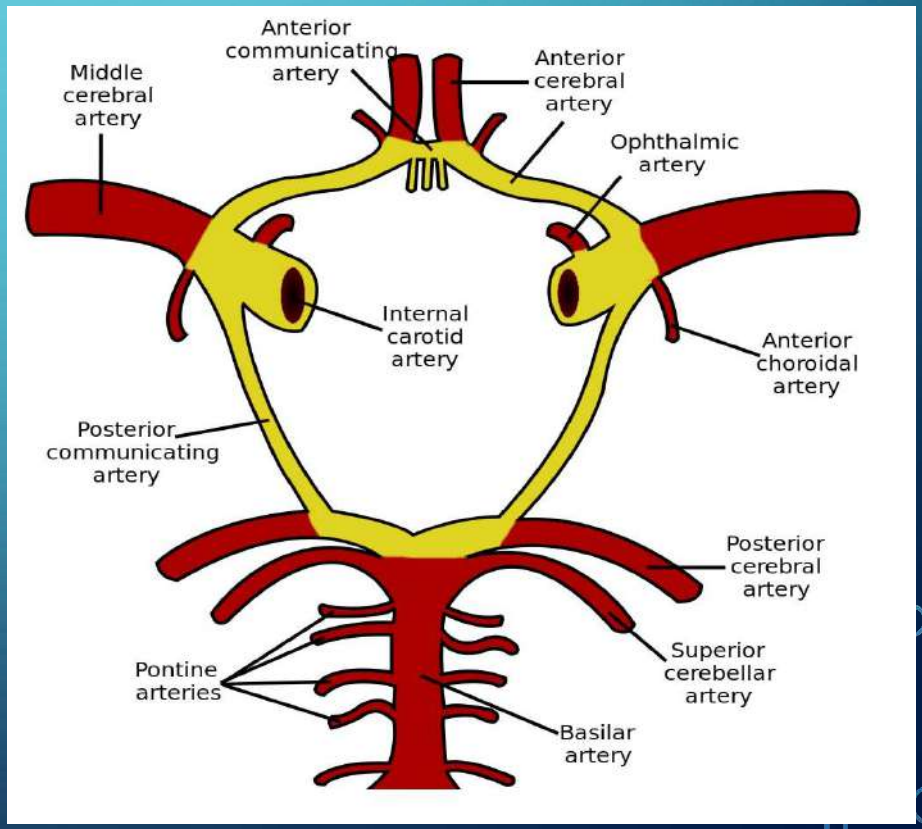

Leading causes of death globally
2000
2019
1. Ischaemic heart disease
2. Stroke
3. Chronic obstructive pulmonary disease
4. Lower respiratory infections
5. Neonatal conditions
6. Trachea, bronchus, lung cancers
7. Alzheimer's disease and other dementias
8. Diarrhoeal diseases
9. Diabetes mellitus
10. Kidney diseases
0
2
4
6
8
10
Number of deaths (in millions)
Noncommunicable
Communicable
Injuries
Source: WHO Global Health Estimates.

STROKE (CONT.)

- Cerebral Blood Flow-

The entire cerebrum is supplied by anterior, middle and posterior cerebral arteries.





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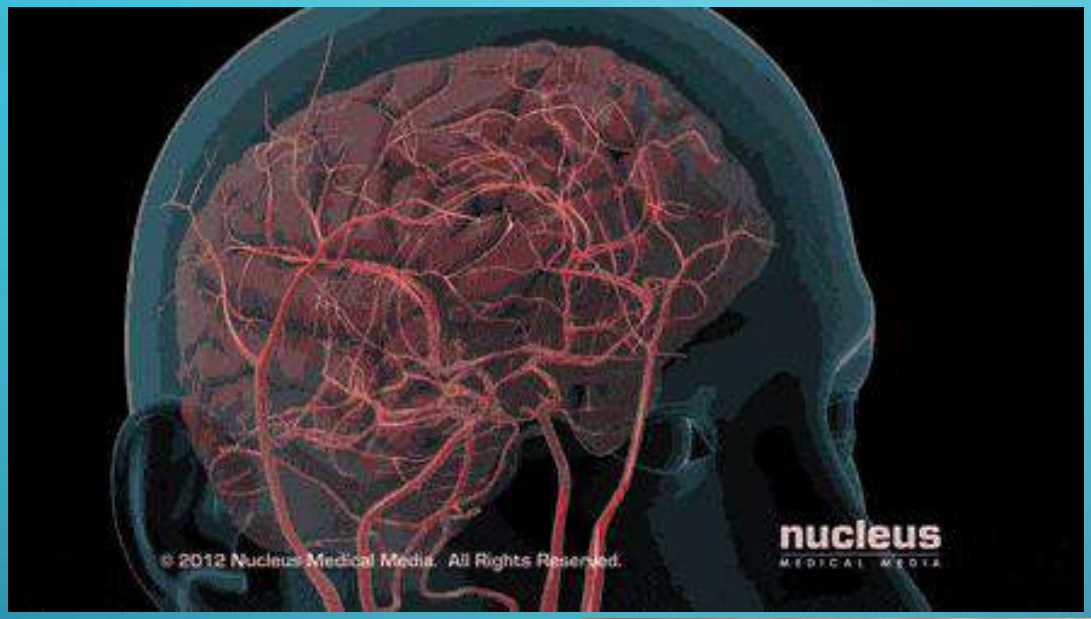
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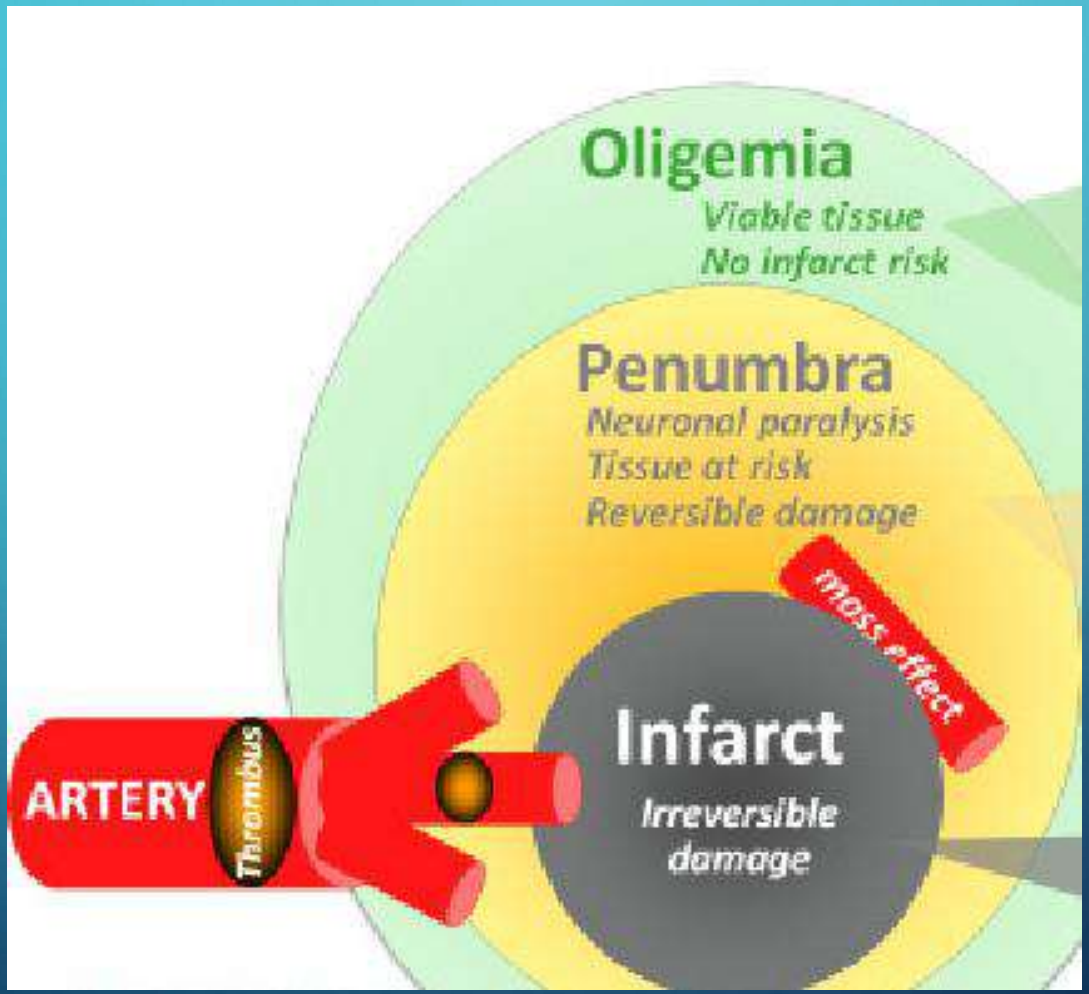
STROKE (CONT.)


Oligemia
Viable tissue
No infarct risk
Penumbra
Neuronal paralysis
Tissue at risk
Reversible damage
moss effect
Infarct
Irreversible damage
ARTERY
Thrombus

STROKE (CONT.)

VASCULAR TERRITORIES

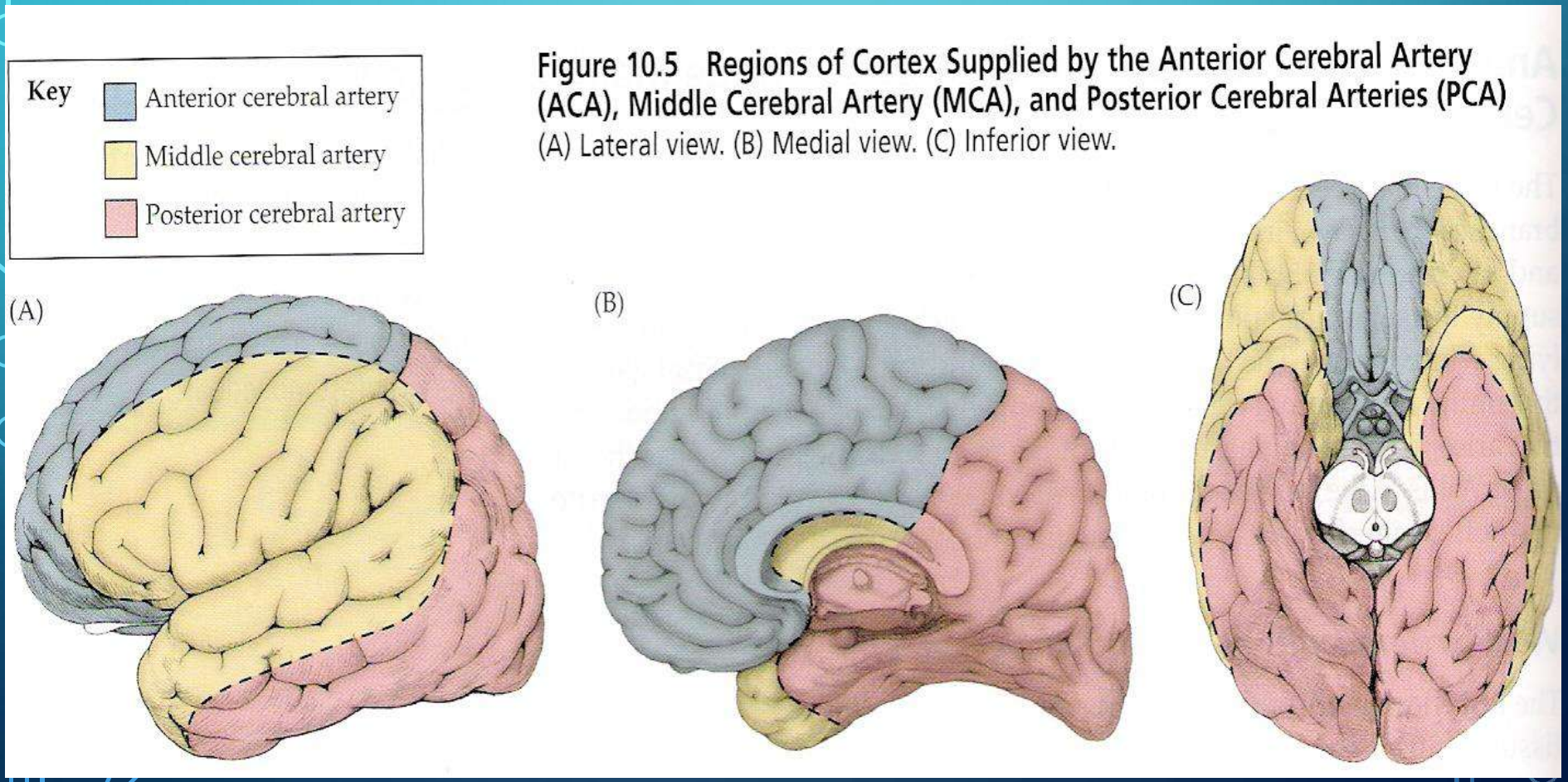


Figure 10.5 Regions of Cortex Supplied by the Anterior Cerebral Artery (ACA), Middle Cerebral Artery (MCA), and Posterior Cerebral Arteries (PCA)
(A) Lateral view. (B) Medial view. (C) Inferior view.

STROKE (CONT.)

- **ANTERIOR CEREBRAL ARTERY:** A branch of the internal carotid artery which supplies entire medial surface of cerebral hemispheres and the medial aspects of the frontal lobe and the parietal lobe; this includes the cortical areas responsible for sensory-motor function of the lower limb.

Signs and symptoms	Structures involved
Contralateral hemiparesis affecting leg more than arm	Motor leg area
Grasp reflex in hand	Medial surface of the posterior frontal lobe
Cortical sensory loss over toes, foot, and leg	Sensory area for leg and foot
Urinary incontinence	Posteromedial part of superior frontal gyrus (bilateral)

STROKE (CONT.)

- **MIDDLE CEREBRAL ARTERY:** Supplies the entire lateral surface of cerebrum including lateral half of orbital surface except Frontal pole and a strip of cortex for about 2cm along superolateral surface of frontal lobe, Medial half of orbital surface and Lower temporal and occipital lobe

Signs and symptoms	Structures involved
Paralysis of the contralateral face, arm, and leg	Somatic motor area for face and arm
Sensory impairment over the contralateral face, arm, and leg	Somatosensory area for face and arm and thalamoparietal projections
aphasia, anomia, alexia, agraphia, acalculia, finger agnosia, right–left confusion	parietooccipital cortex of the *dominant* hemisphere
Ataxia of contralateral limb(s)	Parietal lobe

STROKE (CONT.)

- **POSTERIOR CEREBRAL ARTERY:** A branch of the vertebro-basilar system which supplies the medial surface of temporal and occipital lobes and the visual areas of the cortex; also supplies medulla, pons, midbrain, subthalamus and thalamus.

Signs and symptoms	Structures involved
Thalamic syndrome: sensory loss (all modalities), spontaneous pain and dysesthesias, choreoathetosis, intention tremor, spasms of hand, mild hemiparesis	Ventral posterolateral nucleus of thalamus Involvement of the adjacent subthalamic nucleus
Alexia without agraphia	Dominant occipital lobe along with splenium of corpus callosum
Bilateral or unilateral pyramidal signs & a variety of ipsilateral cranial nerve palsies	Brainstem
Bilateral homonymous hemianopia, cortical blindness	Bilateral occipital lobe

STROKE (CONT.)

risk factors for stroke:

- Inherent biological traits such as age and sex
- physiological characteristics that predict future occurrence such as high blood pressure, serum cholesterol, fibrinogen;
- behaviours such as smoking, diet, alcohol consumption, physical inactivity;
- social characteristics such as education, social class and ethnicity;
- environmental factors that may be physical (temperature, altitude), geographical, or psychosocial.
- In addition, medical factors including previous TIA or stroke, ischemic heart disease, atrial fibrillation, and glucose intolerance, all increase the risk of stroke.[4]

STROKE (CONT.)

- **CRITERIA** for diagnosing stroke-

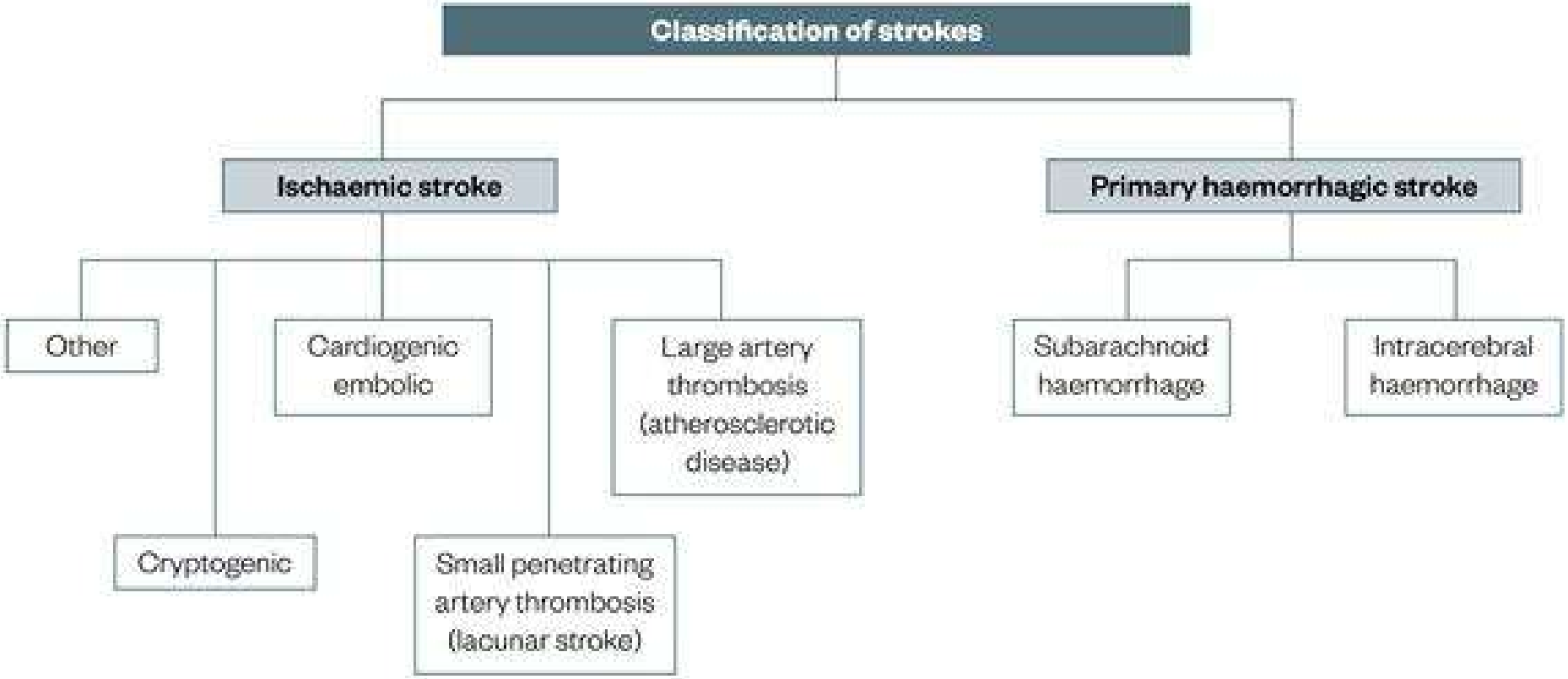
-Sudden onset of symptoms

-Focal signs and symptoms are present that can be explained by a single brain lesion

-Loss of function

-Symptoms and signs are maximal at onset, then remain stable or improve over time.

STROKE (CONT.)



Bamford stroke classification

- **Total anterior circulation stroke (TACS)**
- **Partial anterior circulation stroke (PACS)**
- **Lacunar syndrome (LACS)**
- **Posterior circulation syndrome (POCS)**

NEUROPSYCHIATRIC CORRELATES OF STROKE

- Following are neuropsychiatric correlates of stroke:

-Vascular Neurocognitive Disorder
- Depression
- Mania
- Anxiety
- Psychosis
- Apathy
- Catastrophic Reaction
- Pathological Laughter and Crying
- Anosognosia

VASCULAR NEUROCOGNITIVE DISORDER

- The only DSM-5 disorders that are specific for cerebrovascular disease are the major or minor vascular neurocognitive disorders. DSM-5 identifies two forms of cognitive impairment due to vascular disease: major and minor vascular neurocognitive disorders.
- 'Major neurocognitive disorder due to vascular disease' replaces 'Vascular dementia' used in DSM-IV.
- 'Minor vascular neurocognitive disorder' replaces 'Mild cognitive impairment'.
- Prevalence vary widely from 6-32%. Increases with advancing age.
- Vascular dementia: 2nd most common type of dementia

  - 15 to 20% of all cases of dementia.

VASCULAR NEUROCOGNITIVE DISORDER (CONT.)
DSM V CRITERIA

- 1. Criteria are met for major neurocognitive disorder (i.e., the presence of acquired impairment in one or more cognitive domains and significant impairment in activities of daily living not better explained by another medical condition);
- 2. The clinical features are consistent with a vascular aetiology as suggested by either

(a) cognitive deficits that are temporally related to one or more cerebrovascular events or

(b) presence of prominent executive dysfunction (e.g., inattentiveness or slow processing speed);

- 3. The patient's history, physical examination, and/or neuroimaging findings are consistent with cerebrovascular disease of sufficient severity to account for patient's cognitive impairment.

VASCULAR NEUROCOGNITIVE DISORDER (CONT.)
COURSE AND PROGNOSIS

- characterized by a close Temporal relationship between stroke occurrence and deterioration of cognitive function.
- Probability of recurrent stroke ~ 7 % per year.
- Framingham Heart Study (2014) [5] reported after prospective follow-up of 132 new-onset, dementia-free stroke patients. At 6 months after stroke, significantly greater impairment was seen in the following cognitive domains : immediate recall of logical and visual memory, verbal learning, naming, executive function, visuospatial and motor skills; deficits independent of prestroke cognitive function and vascular risk factors.
- Hypertension- most common modifiable risk factor for stroke worldwide.

VASCULAR NEUROCOGNITIVE DISORDER (CONT.)
TREATMENT

- Treatment with antiplatelet aggregate drugs has reduced the number of repeated ischemic vascular episodes in patients with TIAs.
- Acetylsalicylic acid (ASA) and other antiplatelet drugs have been shown to be effective in the secondary prevention of stroke.
- The United Kingdom-TIA Aspirin Trial [6], with 2,435 patients using two different dosages of ASA, found that there were 21.7 and 25.1 % reductions, based on dosage, in the risk of nonfatal strokes, myocardial infarction, or death compared with placebo treatment.

VASCULAR NEUROCOGNITIVE DISORDER (CONT.)
TREATMENT

- Main strategy is to modify risk factor for further chance of CVA.
- Use of antihypertensives , lipid lowering agents such as statins, smoking cessation, and prevention or careful management of diabetes mellitus.
- Treatment may also include antidepressant agents, cholinergic agonists (e.g., donepezil), antiplatelet aggregation agents, statins, and neurotrophic factors.

POST-STROKE DEPRESSION

- DSM-5 defines post stroke psychotic, mood, and anxiety disorders as disorders due to another medical condition, stroke.
- EPIDEMIOLOGY
- In the most recent studies including 20,293 patients found the pooled prevalence of depression observed at any time point was 29% (Ayerbe L et al. 2013) [7]
- Various other studies prevalence varies from 9-33%.
- The cumulative incidence of PSD was 39 to 52 % within 5 years of stroke.

POST-STROKE DEPRESSION
ETIOLOGY

- A number of hypotheses have been postulated.
- Numerous studies have found abnormalities in biomarkers linked to mood dysregulation such as the serotonin transporter gene and certain inflammatory cytokines (interleukin 6 and 18).
- Elevated levels of glucocorticoids, which affect glutamate transmission, and stroke location, which results in the interruption of biogenic amine transmission, are additional findings.
- In studies performed 3 to 12 months after stroke, the prevalence of PSD is similar in those with left- and right-hemispheric lesions.

POST-STROKE DEPRESSION
ETIOLOGY

- In the first few months after stroke, lesion location does appear to increase the risk of PSD.

  Left anterior strokes > Left posterior strokes

  Left anterior strokes > Right anterior strokes

- Disability after stroke showed a positive and significant association with PSD
- Cognitive deterioration is maximum in left hemispheric stroke
- Personal history or a family history of psychiatric disorders and poor social support are important risk factor for PSD

POST-STROKE DEPRESSION
DIAGNOSIS AND CLINICAL FEATURES

- According to DSM-5, two types of poststroke depressive disorders are recognized:

    1. Depressive disorder due to stroke with major depression-like episode
    2. Depressive disorder due to stroke with depressive symptoms (i.e., sub-syndromal depression).

- The diagnostic criteria, however, require the clinician to determine whether he or she believes that the mood disorder is the direct physiological consequence of the stroke.
- If this judgment is made, then the patient is diagnosed with "depression due to stroke with major depressive-like episode."

POST-STROKE DEPRESSION
COURSE AND PROGNOSIS

- A significant number of patients who develop either major or minor depression following stroke may experience persistent depression for 2 or more years after stroke.
- Major depression following acute stroke is associated with more severe cognitive impairment if the stroke occurred in the left hemisphere.
- This effect of right versus left hemispheric stroke is not seen among non-depressed patients with similar lesions.
- A recent study [8] of 976 patients with stroke found that patients with depression, assessed at 3 weeks poststroke using the Wakefield Self-Assessment Depression Inventory, had 50 % higher mortality at 1 year compared to nondepressed patients.

POST-STROKE DEPRESSION
TREATMENT

- In 1984, [9] the first randomized, blinded, placebo-controlled study in PSD found that 14 patients treated with nortriptyline (50 to 100 mg/day) had significantly greater reduction in Hamilton Depression Scores over 6 weeks than 20 patients given placebo.
- Prophylactic treatment with sertraline, citalopram, fluoxetine, and nortriptyline has been demonstrated to decrease the incidence of poststroke depression in the first 3 months after stroke.
- ECT is safe and effective; rTMS may also be effective.
- Group and Family therapy; Psychotherapy

STROKE AND SUICIDE

- The annual rate of suicide among persons with stoke was nearly twice that of the general population.
- Suicide risk was greatest among stroke patients 50 years or younger.
- Duration of hospitalization was inversely associated with suicide risk, being lowest among those hospitalized longer than three months post stroke and highest among those hospitalized less than two weeks post stroke.
- The risk for suicide appeared to be greatest in the first five years following stroke.

STROKE AND MANIA

- Compared to PSD, mania after stroke is a rare.
- Etiology of mania remains unknown.
- The frequency of right hemispheric lesions was significantly higher.
- Genetic predisposition to affective disorders and brain atrophy may be independent risk factors for post stroke mania (Santos C O et al. 2011) [10]
- DSM-5 classified PSM with specifier as mania with poststroke onset.
- The course of mania following stroke has not been systematically examined.

STROKE AND MANIA

- Data on individual patients with single or recurrent episodes of mania suggest that they respond to lithium, although some fail to respond to either lithium or carbamazepine.
- No randomized controlled studies of the treatment of poststroke mania have been conducted.
- One longitudinal study of 23 patients showed that most patients' mania remit within a few months.

STROKE AND ANXIETY

- Prevalence varies from 2-40%
- Phobic disorders and GAD were the most common
- More common in female
- Most have comorbid depression
- A 2013 study looked at long-term outcomes of post stroke anxiety in 220 patients followed longitudinally [11]

At 5 years post stroke, 64 (29%) of patients were deemed anxious and 73 (33 %) were depressed, with anxiety and depression comorbidity in 43 (20%).

STROKE AND ANXIETY

- Cortical right hemisphere lesions are most common associated with anxiety-only (Tang et al 2012)
- Both depression and anxiety are associated with poor recovery.
- Regular assessments for PSD and anxiety are warranted.
- Data from three randomized, double-blind treatment studies were merged.

Results showed that anxiety symptoms showed greater improvement in response to nortriptyline treatment compared with placebo.

STROKE AND ANXIETY

- A 2011 Cochrane Review of treatment of poststroke anxiety without placebo control, examining 175 patients in two trials of paroxetine or buspirone, concluded that there is insufficient evidence to guide the treatment of anxiety after stroke. [12]

STROKE AND PSYCHOSIS

- Although rare, case reports and empirical studies have documented that psychosis, primarily identified by the presence of delusions or hallucinations, may occur after stroke.
- Based on two hospital registry studies, Frequency of psychotic disorders was 0.4 and 3.1%.
- A right hemispheric lesion, seizures and subcortical brain atrophy appear to be important factors in the pathogenesis of post stroke psychosis.
- Psychosis may be in form of delusion
- Hallucination(visual, auditory)
- Majority patients showed agitation, confusion.

STROKE AND PSYCHOSIS

- There are no controlled treatment trials among patients with delusions or hallucinations following stroke.
- Anecdotal reports have suggested two basic approaches to treatment:

  - Utilization of Anticonvulsant therapy.

  - Antipsychotic medication.

- The use of anticonvulsants has its rationale in the frequent coexistence of seizures with psychotic disorders following stroke.

APATHY

- DSM-5 does not include diagnostic criteria for this condition.
- Apathy is typified by the absence or lack of feeling, emotion, interest, concern, or motivation.
- A recent meta-analysis of 2,706 patients from 24 cohorts found a mean prevalence of apathy of 34.6 % at a median of 120 days following stroke.
- Depression occurred up to 40 % of patients with apathy.
- Imaging studies have found that apathy is significantly associated with lesions of the pons, frontal cortex, basal ganglia, dorsal thalamus, posterior limb of the internal capsule, and temporal cortex.

APATHY

- A 2013 meta-analysis of 2,221 stroke patients found that apathy was associated with older age, depression and cognitive impairment. [13]
- The frequency of apathy did not differ with gender, stroke type (i.e., ischemic vs. hemorrhagic), and lesion location (i.e., left- vs. right-hemispheric lesions).
- Based on sample size of 237 patients with hemorrhagic or ischemic stroke, it was found that apathy was a stronger predictor of poor functional recovery than depression.

APATHY

Symptoms of post-stroke depression

- Loss of interest in activities, not improved when others encourage/facilitate participation
- Patient describes mood as low, sad, or down
- Frequent tearfulness
- Patient distressed by symptoms

Symptoms of post-stroke apathy

- Decreased initiative in undertaking activities, but may enjoy activities when others initiate/facilitate participation
- Patient denies low mood or sadness
- Mood generally unreactive
- Family more concerned than patient about symptoms

APATHY

- Apathy following stroke has been treated with nortriptyline, bromocriptine, methylphenidate, amantadine, selegiline, and tacrine with some success.
- Administration of antidepressant medication such as Escitalopram may prove helpful for the prevention of poststroke apathy [14]

CATASTROPHIC REACTION

- Characterized by a disruptive emotional outburst involving anxiety, tears, aggressive behavior, swearing, displacement, refusal, renouncement, and/or, sometimes, compensatory boasting.
- Prevalence varies from 19-35% (based on a survey of 62 patients and prospective study of 202 patients)
- Lesions involving the basal ganglia.
- Catastrophic reactions occurred predominantly in patients with major depression associated with anterior subcortical lesions.

CATASTROPHIC REACTION

- Currently, no diagnostic criteria are available for catastrophic reactions.
- Patients with catastrophic reactions were found to have a significantly higher frequency of familial and personal history of psychiatric disorders, mostly depression.
- Effective treatments have not been established for catastrophic reactions.
- Treatment consists of prophylactic and supportive measures.

PATHOLOGICAL LAUGHTER AND CRYING (PLAC)

- Characterized by the sudden onset of crying, or more rarely, laughing, which is out of proportion to the conversation or situation in which the emotional reaction occurred.
- The emotion may last from a few seconds to a few minutes with no residual feelings of sadness or happiness.
- This phenomenon has been given various names, including emotional incontinence, emotional lability, pseudobulbar affect, pathological emotionalism and involuntary emotional expression disorder.
- Prevalence varies from 6.3-17.9%.

PATHOLOGICAL LAUGHTER AND CRYING (PLAC)

- Pathological emotions may arise from partial destruction of raphe serotonergic neurons or their projections.
- Recent studies suggested that the critical lesions eliciting PLAC are located along fronto-ponto-cerebellar pathways.
- There are no established diagnostic criteria for PLAC.
- Antidepressant treatment, including TCA, SSRI and SNRIs significantly reduce the frequency and severity of PLAC.
- Case reports suggest that Bupropion, Mirtazapine, Venlafaxine and Lamotrigine may be effective for PLAC.
- FDA approved combination of dextromethorphan hydrobromide/quinidine sulfate in 2010.

ANOSOGNOSIA

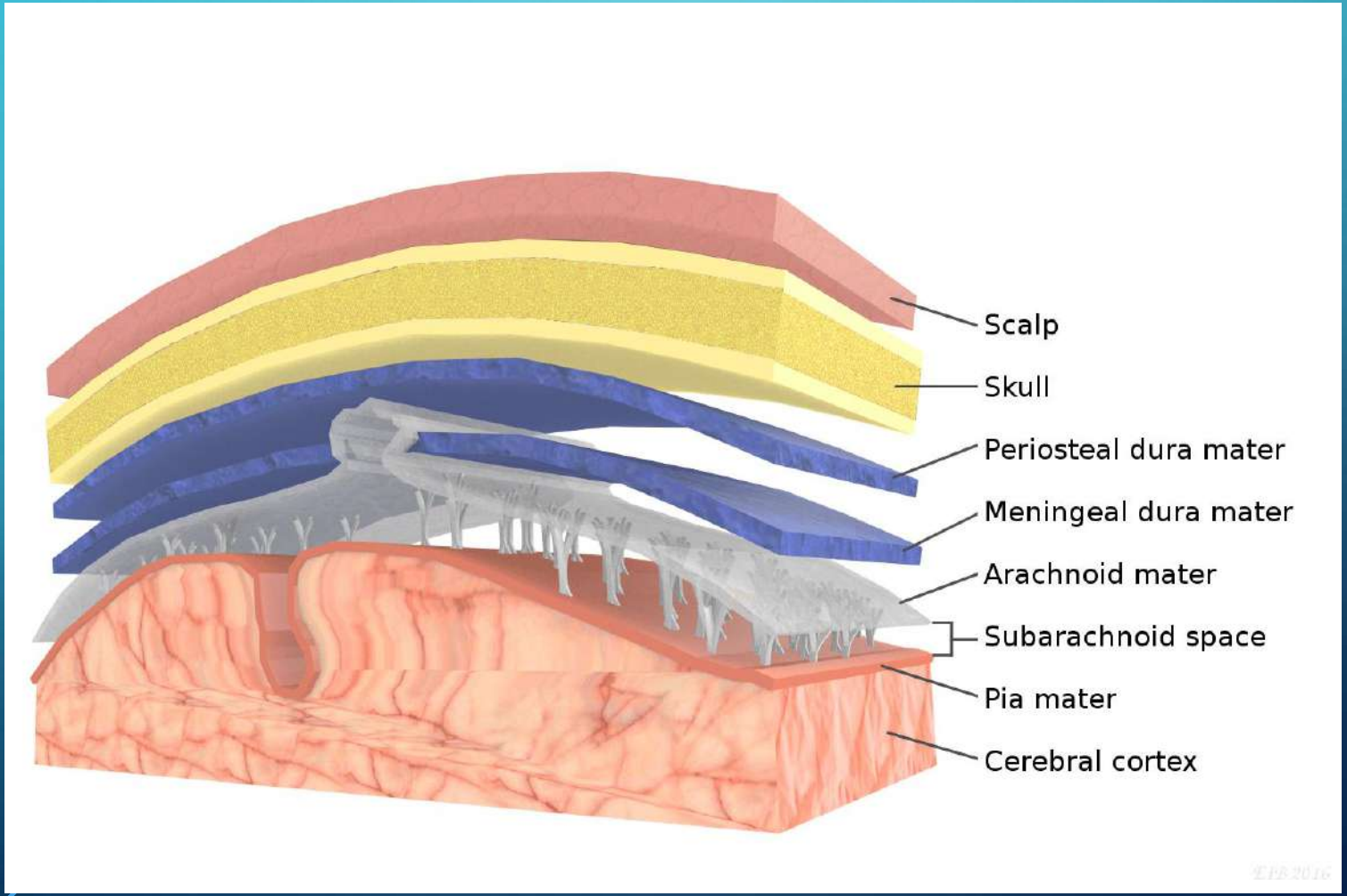
- Anosognosia is a term introduced by Joseph Jules François Félix Babinski to indicate a patient's lack of awareness of his or her hemiplegia.
- This term has been extended to unawareness of other deficits after stroke, such as cortical blindness, hemianopia, and amnesia.
- In the only meta-analysis to date, Lorenzo Pia and colleagues [14] identified 52 studies from 1938 to 2001 for study inclusion. They found the frequency of anosognosia for hemiplegia to range from 20 to 44 %
- Anosognosia is uncommon beyond 3 months poststroke.
- Effective treatments have not been established for anosognosia.

OTHER RELATED CONDITIONS

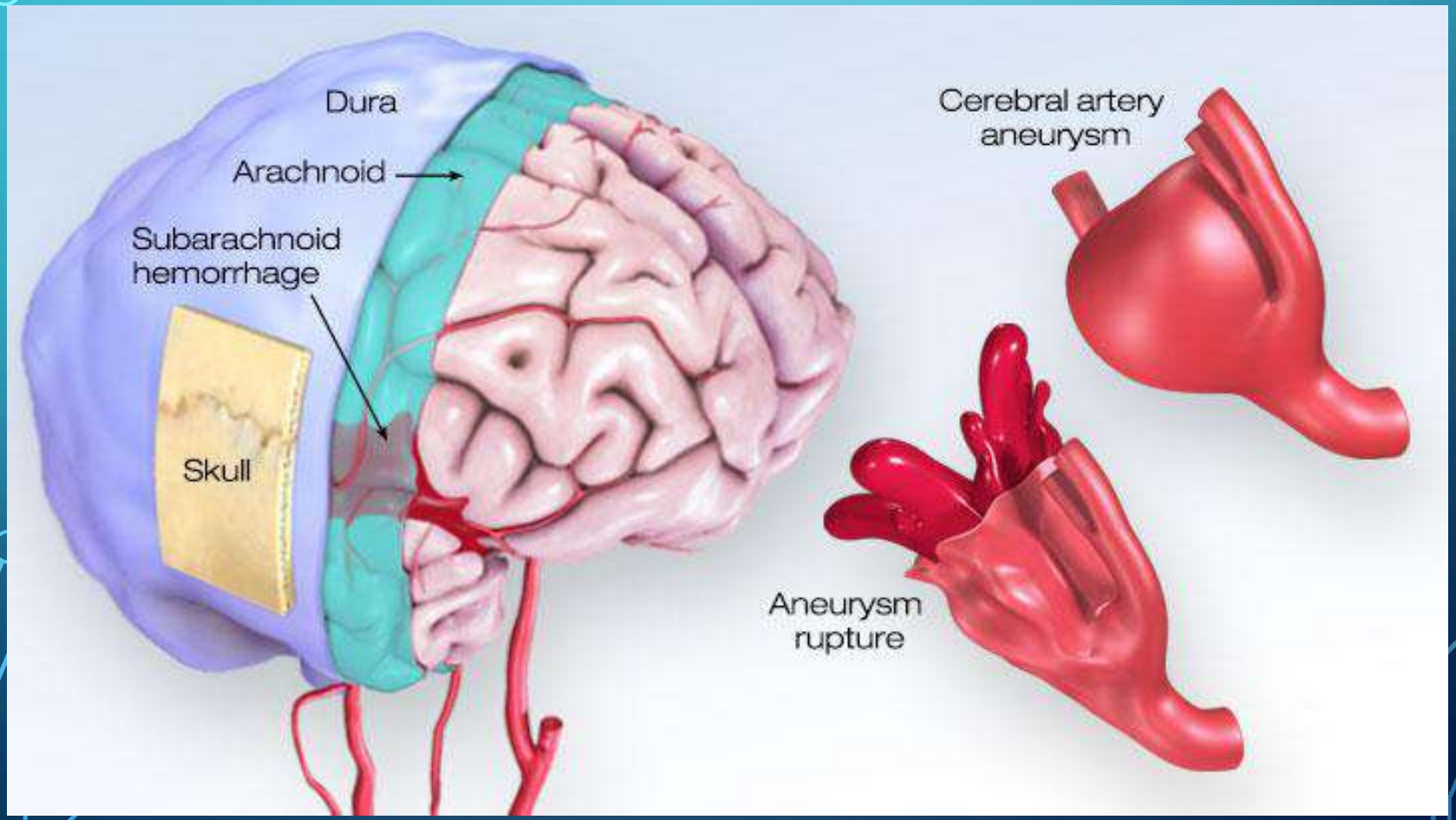
SUBARACHNOID HAEMORRHAGE

- Incidence- 6-7 per 1,00,000 per year
- Accounts for 8% of all strokes.
- Half of the pts of SAH are less than 55 years old.
- About 80% cases of SAH is the result of rupture of an intracranial aneurysm.

SUBARACHNOID HAEMORRHAGE (CONT.)



SUBARACHNOID HAEMORRHAGE (CONT.)



SUBARACHNOID HAEMORRHAGE (CONT.)

The most frequent location is the **anterior communicating artery** (35%), followed by the **internal carotid artery** (30%-including the **carotid artery** itself, the **posterior communicating artery**, and the ophthalmic artery), the **middle cerebral artery** (22%), and finally, the **posterior circulation** sites, most commonly the **basilar artery tip**.

- CLINICAL MANIFESTATIONS-

  -Headache -vomiting -altered sensorium -neck stiffness

  -III CN palsy (with aneurysm of PCA)

  -focal neurological deficit

SUBARACHNOID HAEMORRHAGE (CONT.)

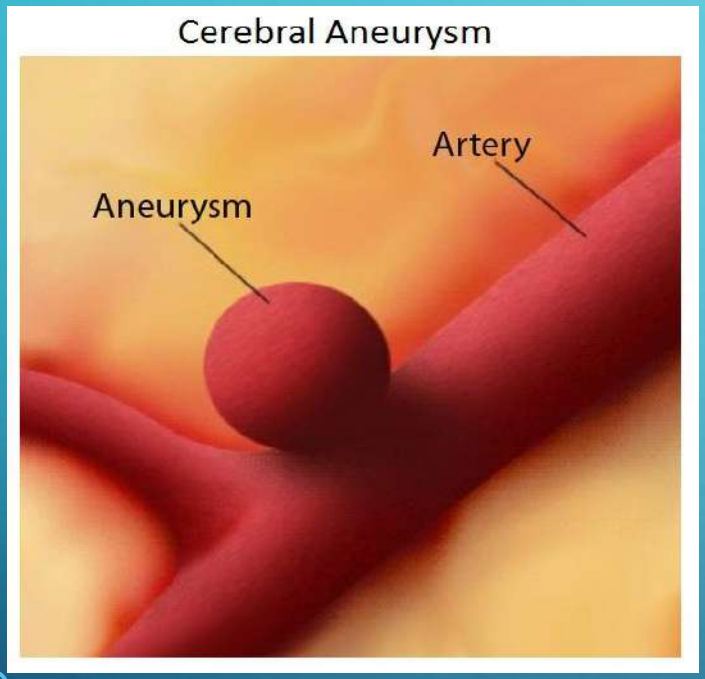
- ACA aneurysms are particularly associated with severe executive and memory impairment, producing a Korsakoff syndrome of amnesia, disorientation, poor insight and confabulation.
- Personality changes are most common after rupture of Middle cerebral aneurysms.
- Increased risk taking behaviour, possibly due to disruption of orbital prefrontal cortex.

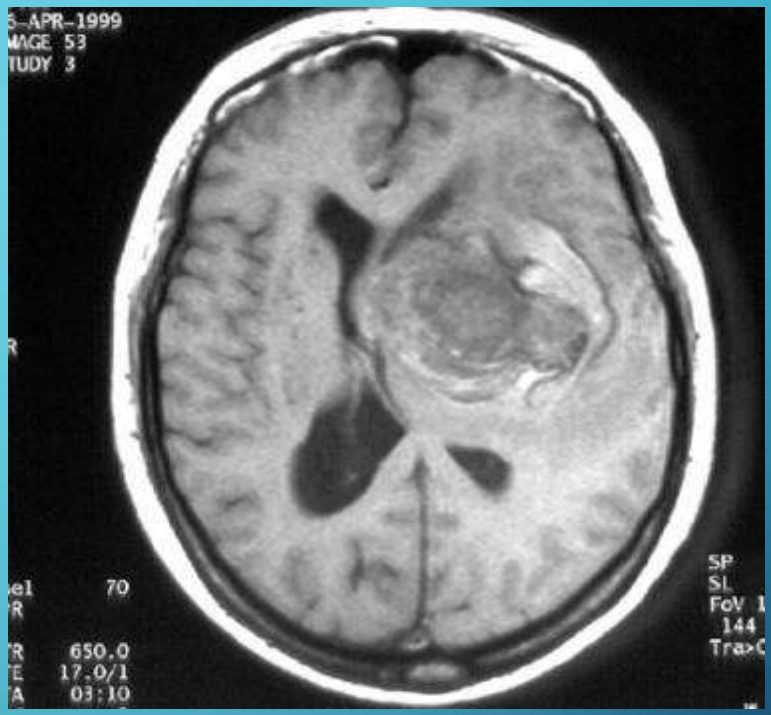
SUBARACHNOID HAEMORRHAGE (CONT.)

- In early stages of recovery, severe but usually transient confusion and states of akinetic mutism can be seen.
- Memory found to be often impaired than intelligence.
- Anxiety symptoms found to be associated in 27 % pts.
- Storey(1972) found symptoms of anxiety or depression in one quarter of patients, moderate or severe in 14%. Depression was commonest of all.
- PTSD is also a quite common sequelae. Psychosis appear to be rare.

GIANT CEREBRAL ANEURYSMS

- The word "aneurysm" comes from the Greek word *aneurysma* (*ana*, meaning across, and *eurys*, meaning broad) and denotes an abnormal dilatation of an artery.
- The great majority of aneurysms giving rise to SAH are small, rarely exceeding 1-2cm in diameter.
- Massive aneurysms are uncommon
- Morely (1967) found that patients with unruptured vertebrobasilar aneurysms, were having symptoms of depressive illness.
- Best known is intracavernous portion of ICA, which may compress surrounding nerves leading to ophthalmoplegia, sensory loss of trigeminal distribution, diplopia, pain in the face, eyes or head.





T1-WEIGHTED MAGNETIC RESONANCE IMAGE (MRI) OF A MIDDLE-AGED WOMAN WITH PROGRESSIVE HEADACHES, APHASIA, AND RIGHT-SIDED HEMIPARESIS. A LARGE INTRACEREBRAL MASS WITH A SIGNIFICANT AMOUNT OF SURROUNDING EDEMA IS DEPICTED. THE LESION IS A GIANT INTERNAL CAROTID ARTERY ANEURYSM.

MIGRAINE

- Previous hypothesis suggesting that migraine aura was caused by vasoconstriction with headache produced by subsequent dialation, have now been discarded.

- It is now being conceived as related to events in the cortex and adjacent meninges on one side and in the brainstem on the other side.

MIGRAINE

- Pts with migraine are at a increased risk of anxiety and depression.
- The increased risk of having an anxiety disorder is greater than that for having depression.
- The risk of suicide attempt is also increased in migraine.
- Lifetime prevalence of MDD found 40.7% in migraine(Breslau *et al.*2000)

MIGRAINE

- Sacks(1970) illustrated in his study that in pts with migraine, 23% showed obsessional trends, 22% were hyperactive and 13% showed anxiety symptoms.

- It is also found that periods of stress, or the anticipation of stress are associated with attacks of migraine.

MIGRAINE

- Anxiety and irritability are common early on with drowsiness and lethargy as the headache continues.

- Klee(1986) found that attacks were accompained by marked impairment of memory in 10%, delirium in 8%, anxiety in 8%, hallucinations in 6%, changes of body image in 6% and severe depression in 4%

SYSTEMIC LUPUS ERYTHEMATOSUS

- Multiorgan autoimmune connective tissue disorder.
- Antibodies against intracellular components are probably involved in the development of tissue lesions.
- Onset is usually insidious.
- **Cerebrovascular** disease may be directly attributed to the disease as a manifestation of **neuropsychiatric SLE.**
- Various pathological mechanisms like ischaemic and haemorrhagic events, white matter abnormalities due to aPLs, accelerated atherosclerosis, small vessel vasculopathy, and thromboembolic processes can all play a part in cerebral vasculitis in SLE.

SYSTEMIC LUPUS ERYTHEMATOSUS

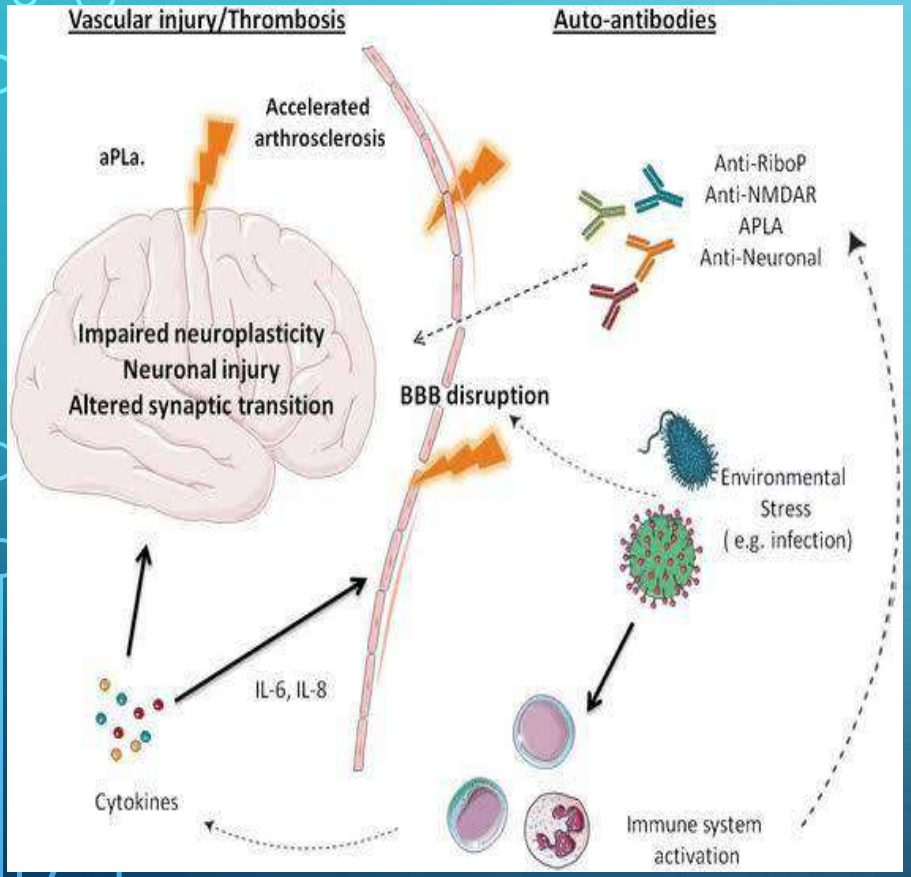


Table 1 – Neuropsychiatric syndromes in SLE as defined by ACR nomenclature

CNS	Peripheral nervous system
Acute confusional state	Autonomic neuropathy
Anxiety disorder	Cranial neuropathy
Aseptic meningitis	Guillain-Barré syndrome
Cerebrovascular disease	Mononeuropathy
Cognitive dysfunction	Myasthenia gravis
Demyelinating syndrome	Plexopathy
Headache	Polyneuropathy
Mood disorder	
Movement disorder	
Myelopathy	
Psychosis	
Seizure disorders	

SLE, systemic lupus erythematosus; ACR, American College of Rheumatology.

Adapted from American College of Rheumatology Ad Hoc Committee on Neuropsychiatric Lupus Nomenclature. *Arthritis Rheum*. 1999.[2]

SYSTEMIC LUPUS ERYTHEMATOSUS

- 60% patients with SLE have neuropsychiatric manifestations.
- Do not have any characteristic pattern.
- Anxiety, depression and sleep disturbances are common.
- In the past, delirium was most frequent. Cognitive impairment is usually mild.
- Headache is perhaps the most common of all neurological problems. Seizures, strokes, TIA, neuropathies are also common.

VASCULITIS OF CNS

- It is inflammation of blood vessel wall and is synonymous with angitis.
- Vasculitis of the CNS, in absence of systemic involvement is called as PRIMARY vasculitis of CNS.
- Psychiatric sequelae are related to encephalopathy, focal cortical lesions and psychological reaction to illness.

VASCULITIS OF CNS

- **Polyarteritis nodosa** (PAN) is a necrotizing vasculitis involving small and medium-sized arteries and it affects multiple organ systems in the body. Central nervous system (CNS) involvement appears less frequently, and usually develops after the disease is established.
- Small peripheral cerebral infarctions, consistent with an arteritis involving medium-sized and small arteries, were the most common finding.

- **Giant cell arteritis** (GCA) is the most frequent vasculitis in patients aged over 50 years old in Europe and North America. **Stroke** may occur in patients with GCA.
- In most cases, **stroke** is related to vasculitis of extracranial **cerebral** arteries causing vertebral or internal carotid arteries stenosis.

POLYCYTHAEMIA RUBRA VERA

- Polycythaemia rubra vera Is a rare haematological Disorder that has high risk of stroke. In polycythaemia rubra vera, neuropsychiatric symptoms are prominent.
- There have been multiple case reports.
- Murray and Hodgson(1991) reported a patient who developed a severe depressive illness with psychotic symptoms.
- Mania after an episode of delirium has also been reported (Chawla and Lindesay 1993)
- Acute psychosis in polycythaemia rubra vera n J. Pantela, J. Schrödera, J. W. Schmierb, S. Bachmanna

SUMMARY

- **Neuropsychiatric** syndromes are common in the setting of **cerebrovascular disease**.
- Stroke is 2nd leading cause of death globally.
- There are many neuropsychiatric correlates of stroke.
- The most frequent psychiatric syndrome after stroke is depression.
- Anxiety and apathy are also common.
- Other psychiatric conditions have also been seen.

SUMMARY

- These conditions causes increase in morbidity and delay in rehabilitation in post-stroke patients.
- There are very few treatment studies available.
- Treatment of neuropsychiatric post-stroke disorders have the greatest potential for improving outcome and quality of life.

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THANK YOU