The Psychological Impact of Stroke

OLD / IDEAL ME
before stroke

Before stroke I knew….
- who I was,
- what I did and could do,
- what I wanted to be and do

STROKE

ME after stroke

SENSORY / PHYSICAL CHANGES e.g. Hemiparesis? Loss of sensation? Mobility difficulties? Pain? Fatigue? Balance difficulties? Seizures?

CHANGES IN THINKING SKILLS, e.g.
Slower?
Harder to focus?
Harder to remember?
Do things before thinking?
Can't plan as well?

CHANGES IN COMMUNICATION, e.g. Dysarthria? Dysphasia? Dyslexia? Dysgraphia? Interrupt more? Can't read others as well? Struggle with humour, sarcasm?

CHANGES IN MOOD, e.g. Apathy? Emotionalism? Emotion dysregulation?

CHANGES IN ACTIVITY, e.g. Difficulties with previous roles (work, family, social, hobbies) and social integration

Common Psychological Difficulties after Stroke

Normal reaction to an abnormal event

- Sad feelings / Low mood
- Sleep problems – not sleeping through the night, restless sleep
- Anxiety – fear of falling, looking out for stroke signs
- Agitation & Frustration
- Worrying about the future – “how will I cope”, “what if it happens again”

This reaction can become persistent, which can lead to:

- Disengaging with therapy sessions
- Feelings of hopelessness
- Preoccupation with lost skills or roles
- Unable to adjust to loss / changed sense of identity
- Striving for an unrealistic degree of recovery
- Repeated failure leading to loss of confidence
- Strained family relationships

Tyerman (2008)

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Stroke Threatens Mental Health

- From the John Hopkins Medicine website (http://www.hopkinsmedicine.org/gec/series/neuropsych_stroke.html#anxiety)
  - Depression : 35%
  - Anxiety disorder : 25%
  - Apathy : 20%
  - Pathologic affect : 20%
  - Catastrophic reaction : 20%

  ("an outburst of emotion, such as anxiety, agitation, or crying, that occurs when unable to perform simple tasks that were possible before")
  - Mania : rare
  - Bipolar disorder : rare
  - Psychosis : rare

About 1/3 develop a diagnosable mental health condition

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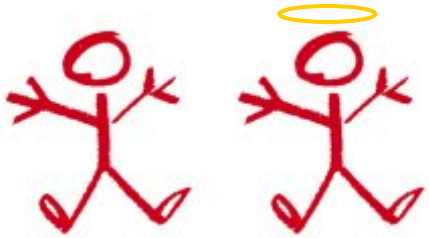
OLD / IDEAL ME
before stroke



STROKE

ME after stroke

WHO AM I NOW?
What can I do?
Are my roles / goals still possible?
Do I still like / accept myself?
Will others accept me?

EXISTING COPING STYLE

- Task-focussed
- Emotion-focussed
- Avoidant

But may not work as well as before and may not be adaptive.

DIFFERENT EMOTIONS

- Sadness
- Anger and Frustration
- Anxiety, fear, worry

DIFFERENT BEHAVIOURS

- Withdrawal
- Aggression
- Denial

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Sahyadri Hospitals

Post Stroke Psychiatric Manifestations

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Dr Sanjay K Pawar

Stroke Physician

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Introduction

- Globally, approximately 16million people have their first stroke each year of which approximately 5.7million people die and 5million are left with long-term disabilities.
- Neuropsychiatric symptoms following stroke occur in at least 30% of stroke survivors and are a major predictor of poor outcome and the particular combination of stroke and psychosis is considered to be among the most serious complications

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CHANGES IN MOOD, e.g.
Apathy? Emotionalism?
Emotion dysregulation?

CHANGES IN ACTIVITY, e.g. Difficulties with previous roles (work, family, social, hobbies) and social integration

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- The most common psychiatric disturbances seen after stroke include cognitive impairment and dementia, depression, mania, anxiety disorders, and pathological laughing and crying - now referred to as involuntary emotion expression disorder or IEDD
- Left hemisphere strokes frequently cause dysphasia, whereas right - hemisphere strokes are associated with anosognosia, inattention, impaired spatial reasoning, and neglect syndromes
- Motivation, memory, judgment, and impulse control may be affected after frontal stroke

Mental Health Disorders after Stroke

Post-Stroke Depression

- Feeling low, Lacking Interest, Changes in appetite, sleep, motor activity and energy, Concentration Difficulties, Thoughts about Worthlessness and Death.
- Affects 33% (Hackett et al. 2005)
- Associated with poor rehab participation and gains & increased mortality

Mental Health Disorders after Stroke

Generalised Anxiety Disorder

- Excessive anxiety and worry, difficulty controlling anxiety and worry, feeling tense and restless, concentration problems, muscular tension, fatigue and sleep difficulties
- Affects 28% in acute stage. Often becomes chronic. (Astrom, 1996).
- Likely to affect rehabilitation. Limits ability to recover from depression.

Mental Health Disorders after Stroke

Agoraphobia

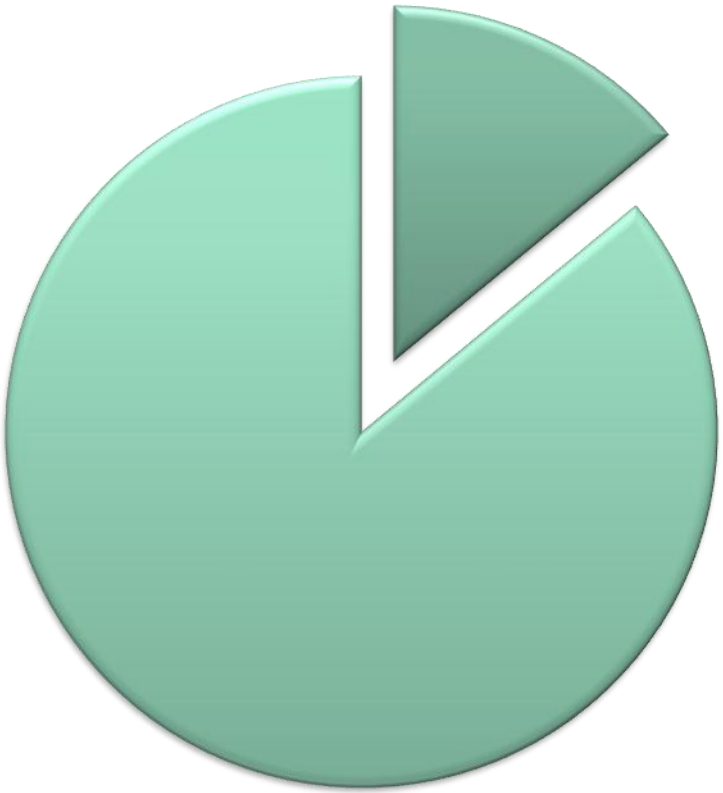
- Avoidance of situations triggering panic attacks.
- Generalised Anxiety Disorder and agoraphobia most common anxiety disorders after stroke (Burvill et al. 1995).
- Likely to interfere with or limit rehabilitation.

Mental Health Disorders after Stroke

Post-Traumatic Stress Disorder

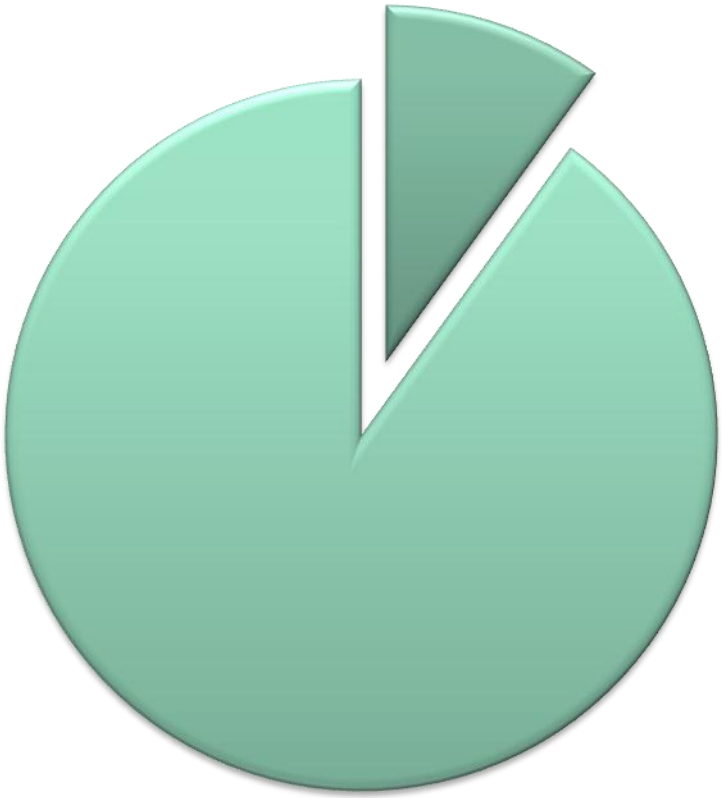
- Reliving stroke, avoidance, numbing, loss of interest, feeling detached from others, being keyed up and jumpy, irritability, sleep and concentration difficulties.
- 31% (Bruggiman et al, 2006).
- Likely to interfere with or limit rehabilitation.

Suicidal Thoughts after Stroke



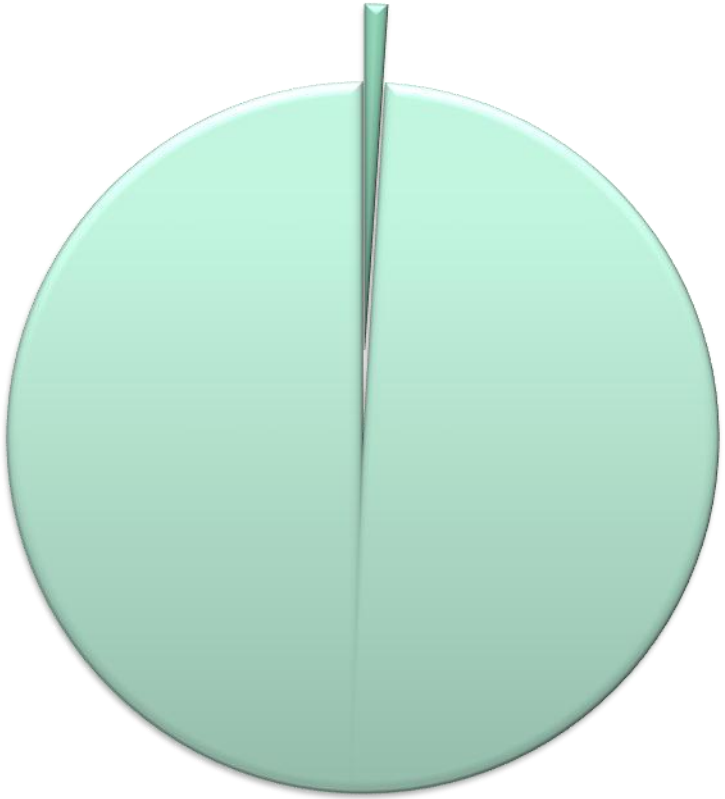
- 7-14%
- 6-17% in stroke survivors not screened as depressed
  (Hackett et al. 2010)

Suicide Plans after Stroke



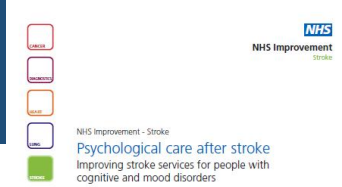
- 6-11%
  (Kishi, Robinson & Kosier, 1996)

Suicide Deaths after Stroke



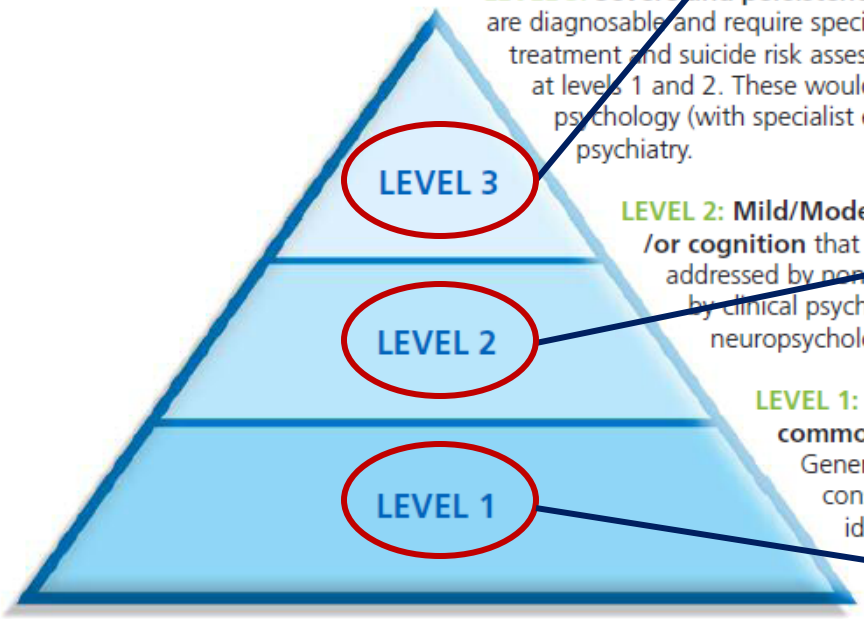
- < 1%
- Risk of suicide death doubles (Teasdale & Engberg, 2001).
- Risk greatest in first 5 years.

The Stepped Care Model of Psychological Support after Stroke



Clinical Psychology or Psychiatry required for severe, persistent or complex disorders

Figure 1: Stepped care model for psychological interventions after stroke.
Adapted from IAPT model with input from Professor Allan House and Dr Posy Knights



LEVEL 3: **Severe and persistent disorders of mood and/or cognition** that are diagnosable and require specialised intervention, pharmacological treatment and suicide risk assessment and have proved resistant to treatment at levels 1 and 2. These would require the intervention psychology (with specialist expertise in stroke) or neu psychiatry.

LEVEL 2: **Mild/Moderate symptoms of imp /or cognition** that interfere with rehabilitatio addressed by non-psychology stroke specia by clinical psychologists (with special expe neuropsychologists.

LEVEL 1: **'Sub-threshold problems' at a level common to many or most people with stroke.** General difficulties coping and perceived consequences for the person's lifestyle and identity. Mild and transitory and/or cognitive disorders attitude to the outcome which have little impa in rehabilitation. Su provided by peers specialist staff.

Stroke specialists with additional training under supervision could provide psychological interventions for mild to moderate difficulties

Any stroke specialist in the MDT could provide support for general difficulties coping

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Frontosubcortical Networks

- Five frontosubcortical circuits subserve cognition, behavior, and movement
- Neurotransmitters involved - Dopamine (DA), Glutamate, γ-aminobutyric Acid (GABA), Acetylcholine, Norepinephrine & Serotonin

Frontosubcortical Networks

- Motor circuit
- Oculomotor circuit
- Dorsolateral prefrontal circuit
- Orbitofrontal circuit
- Anterior cingulate circuit

Motor circuit

- Subserves somatic motor function
- Originating in the supplementary motor area

Oculomotor circuit

- Originating in the frontal eye fields
- Subserves oculomotor function

Dorsolateral Prefrontal Circuit

Executive functions, including

- Ability to plan and maintain attention
- Problem solve
- Learn, retrieve remote memories
- Sequence the temporal order of events
- Shift cognitive and behavioral sets
- Generate motor programs

Deficits

- Slowed information processing
- Memory retrieval deficits
- Mood and behavioral changes
- Gait disturbance
- Dysarthria
- Other motor impairments

- Involved in subcortical dementias

Orbitofrontal Circuit

- Connects frontal monitoring functions to the limbic system
- Governs appropriate responses to social cues, empathy, social judgment, and interpersonal sensitivity
- Pairs thoughts, memories, and experiences with corresponding visceral and emotional states
- Heavily involved in the process of decision making and evaluating the costs and benefits of specific behavioral responses to the environment

Orbitofrontal Circuit

Medial OFC → Evaluates reward

Monitors and decodes punishment ← **Lateral OFC**

Anterior OFC

Reward value for more abstract and complex secondary reinforcing factors such as money

Posterior OFC

Reward value for more concrete factors such as touch and taste

Evaluating the emotional significance of stimuli

Orbitofrontal Circuit

Dysfunction

- Disinhibition
- Irritability
- Aggressive outbursts
- Inappropriate social responses
- Impulsive decision making

Anterior Cingulate Circuit

- Nucleus accumbens with both afferent and efferent connections to the dorsolateral prefrontal cortex (DLPFC) and amygdale.
- Involved in motivated behavior.

Lesions

- Apathy, abulia, and akinetic mutism.

Prevalence of Post Stroke Neuropsychiatric Disorders

- Depression: 35%
- Mania: rare
- Bipolar disorder: rare
- Anxiety disorder: 25%
  - Apathy : 20%
- Psychosis: rare
- Pathologic affect 20%
- Catastrophic reaction: 20%

Most common are: depression, anxiety, emotional incontinence and catastrophic reactions

Neuropsychiatric consequences of stroke depend on

- Location and size of the stroke
- Preexisting brain pathology
- Baseline intellectual capacity and functioning
- Age
- Premorbid psychiatric history

Post Stroke Neuropsychiatric Disorders

- Period of high risk for psychiatric complications is first 6 months following a stroke
- In general, interruption of bilateral frontotemporal lobe function is associated with an increased risk of depressive and psychotic symptoms

Post Stroke Depression

- In 30% to 40% of patients within the first year most develop within the first month (Ballard and O'Brien, 2002)
- 30% to 50% of survivors at 1 year
- About half will meet criteria for a major depressive episode

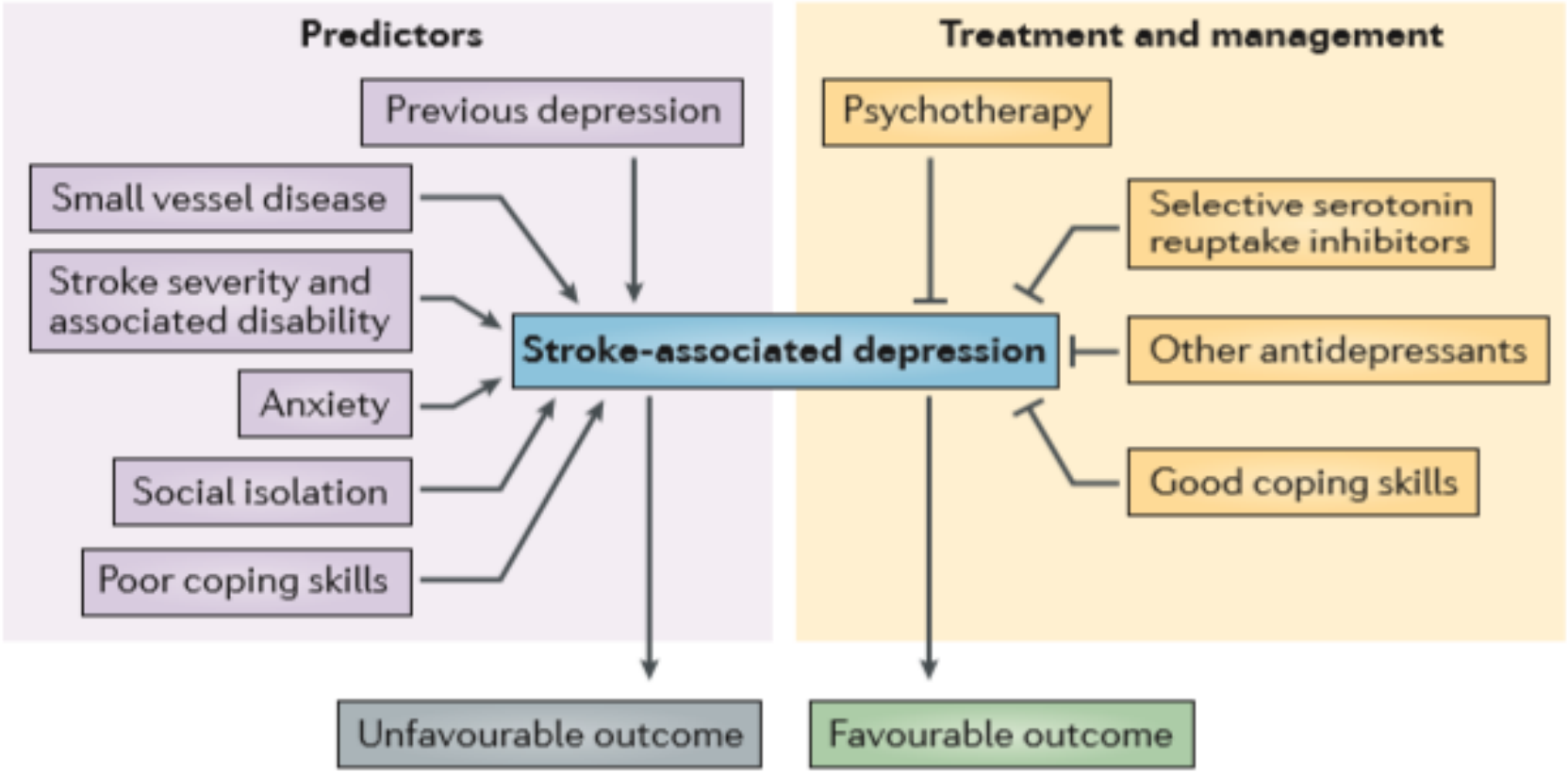


Figure 1 | **Major psychosocial and cerebrovascular predictors of stroke-associated depression.** Anxiety and poor coping-strategies, among other factors, have a negative influence on the course of depression. In stroke patients, depression is associated with unfavorable outcomes, including death. Treatment and management strategies such as the administration of antidepressants and good coping skills have beneficial effects on stroke-associated depression.

Post Stroke Depression

- Prevalence varies over time with an apparent peak at 3-6 months after stroke and a subsequent decline to about 50% of initial rates at 1 year
- Duration of major PSD is about 9 months to 1 year whereas the duration of minor depression is several years
- Spontaneous remission can occur 1-2 year post-stroke. (Robinson et al, 1987)

Risk factors for post stroke depression

Consistent	Controversial
Past Psych history	Age
Dysphasia	Gender
Poor Social Support	Impaired ADLs
	Lesion Location
	Lesion Volume

- Premorbid diagnosis of depression (5 to 6 times more likely) (Ried et al., 2010)

Diagnosis of PSD is difficult sometimes because of

- Language disorders - Difficulty in expressing or comprehending
- Cognitive impairment - Anosognosia or lack of insight or lack of awareness of depressive symptoms
- Overlap between symptoms of depression & medical condition - Several symptoms of depression such as loss of energy, decreased appetite, insomnia are found among non- depressed stroke patients secondary to hospital environment, use of medications, other medical conditions and stroke itself

Causes of PSD

Probably due to a combination of biological, psychological and social factors.

- Biological: disruption of neural circuits & neurochemicals. Genetic cause.
- Psychological: presence of poor coping skills
- Social: disability, limited social support, loss of independence may overwhelm coping skills

Psychological stressors can overwhelm anyone, and in the setting of biological vulnerability, can cause depression

Biology of Depression

Dysfunction in

- Orbitofrontal–basal ganglia– thalamic circuit
- Basotemporal limbic circuit that links the orbitofrontal cortex and the anterior temporal cortex by the uncinate fasciculus

Four interconnected functional compartments

- Mood regulation (medial frontal, medial orbital-frontal and pregenu anterior cingulate cortex)
- Mood monitoring (ventral striatum-caudate, amygdale, dorsomedial thalamus, midbrain-ventral tegmental area)
- Interoception (subcallosal cingulated, ventral-anterior hippocampus, anterior insula, brain stem, hypothalamus)
- Exteroception (prefrontal, premotor, parietal, mid- cingulate and posterior cingulate cortices with dorsal- posterior hippocampus)

Consequences of post stroke depression

- Longer hospital stays – affect rehabilitation
- Poorer recovery of activities of daily living
- Increased morbidity (more with presence of executive dysfunction)
- Poorer quality of life, even when neurological symptoms and disability are held constant

Depression and Lesion Location

- More with left anterior lesions ESP nearer the left frontal pole or left caudate nucleus More recent review articles have not supported a relationship between lesion location and depression in poststroke patients (Bhogal et al., 2004)

Depression and Lesion Location

- With left sided lesions in hospital samples, whereas
- With right-sided lesions in community samples
- With left-sided lesions in first month following stroke
- With right-sided lesions in more than 6 months after the stroke

(Bhogal et al., 2004)

Depression and Lesion Location

Left prefrontal lesions are more apt to be associated with acute depression and may be complicated by aphasia, resulting in the patient's inability to expressthe symptoms

Screening for depression

SIGECAPS

- Sleep, Interest level, Guilt, Energy level,
- Concentration, Appetite, Psychomotor activity level, and Suicidal thoughts
- Presence of 5 or more of these symptoms (one of which must be depressed mood or decreased interest level) for 2 weeks is the threshold for a diagnosis of major depression

Quantifing depressive symptoms

- Self-administered Beck Depression Inventory (BDI) and clinician-administered Hamilton Rating Scale for Depression (HDS)
- Clinician-administered Post-Stroke Depression Rating Scale (PSDRS) addresses the "major" and "minor" forms of PSD

Treatment of PSD

- Supportive psychotherapy and pharmacotherapy
- Antidepressants are well tolerated
- 60% respond to medications

Psychopharmacologic treatment

- Tricyclic antidepressants (TCAs)
- Selective serotonin reuptake inhibitors (SSRIs
- Psychostimulants (eg, methylphenidate)

No particular class has an advantage over the other

SSRIs

- First-line treatment In the acute phase
- Cause fewer serious side effects, such as delirium and sedation, than do TCAs
- May increase bleeding risk in some patients because of their effects on platelet function
- Recent major review found no causal relationship between SSRIs and bleeding in post-stroke patients

Dosing of Antidepressants

- “Start low and go slow,"
- Consider starting at half the typical adult starting dose
- Allow 1 to 2 weeks between dose increases
- Best to conduct an adequate trial: minimally, a trial of 6 weeks' duration at the usual adult therapeutic dose

Follow up at least monthly repeating the cognitive examination and depression inventory to monitor treatment response

Clinical remission

Continue treatment for up to 12 months at the full effective dose

No clinical response despite demonstrated adherence

Initial antidepressant is poorly tolerated

Switch to a different antidepressant class and/or augment the therapy with a psychostimulant (eg, methylphenidate, dextroamphetamine)

Psychostimulants

- Safe, well tolerated and efficacious
- No definitive conclusions can be made given lack of randomization
- Risk of seizure and/or cardiac side effects
- If history of seizures, consideration of antiseizure medication, along with psychostimulants, appears reasonable

Consider electroconvulsive therapy for

- Depression-related emergencies, such as repeated
- Suicide attempts and severe melancholic PSD
- Refractory to maximal medication management
- Complex psychopharmacologic regimens causing intolerable side effects

Prophylactic treatment with an antidepressant

- Prior episodes of depression
- Left-sided lesions
- History of other psychiatric illness
- Strong family history of psychiatric illness

Prophylactic treatment with an antidepressant

Advantages

- Antidepressants, neurotropic, stabilize the chemical imbalance;
- Increased compliance with vascular disease preventing regimens;
- They may have an effect on serotonin mediated platelet activation.

Antidepressants have side-effects such as falls, increased bleeding, seizures, and sedation

Pathological Laughing and Crying

Other names: Emotional Incontinence; post-stroke emotionalism

- Between 11% and 35% after stroke (Parvizi et al., 2009).
- Associated with brainstem and cerebellar lesions
- Sudden paroxysms of either laughter or crying, irrespective of the ambient mood state
- Can be triggered by nonspecific stimuli or by a low-threshold emotive stimulus

Distinguishing Types of Crying

- Pathological crying linked to infarct in basis of pontis and corticobulbar pathways and occurs in response to mood incongruent cues.
- Emotionalism is crying that is congruent with mood (sadness) but patient is unable to control crying as they would have before stroke.
- Catastrophic reaction is crying or withdrawal reaction triggered by a task made difficult or impossible by a neurologic deficit (e.g. moving a hemiplegic arm)

Pathological Laughing and Crying

Management

- Tricyclic and SSRI antidepressants
- Lithium and anticonvulsants are alternatives

Post-Stroke Mania

- Rare
- Associated with right-sided stroke
- Expansive and/or irritable mood, decreased need for sleep, increased goal-directed activity, recklessness, disregard for social constraints, talkativeness, racing thoughts, excessive laughter or giggling, and poor judgment

Post-Stroke Mania

Management

- Mood stabilizer and/or an atypical antipsychotic
- Observation for downward cycling of mood into an episode of PSD, using mood screening questions and/or depression inventories and clinical observation, is necessary

Post-Stroke Anxiety Disorders

- Risks of 26% and 39% in men and women respectively
- More common in cortical than subcortical stroke
- Discrete episodes of panic, tonic levels of increased anxiety, excessive sweating, worrying, and decreased sleep

Post-Stroke Anxiety Disorders

- Majority also having PSD
- Anxiety Depression (AD) was associated with left cortical lesions and anxiety alone with right hemisphere lesions
- Comorbidity of PSD and AD produced longer duration of PSD than PSD alone and this prolonged depression might lead to poorer physical and social outcomes

Post-Stroke Anxiety Disorders

Management

- Respond well to antidepressants (SSRIs)
- Avoidance of benzodiazepines is important; these agents may cause cognitive decline, verging on PSDem
- Follow-up should be done in 1 month to assess response
- If symptoms are incompletely responsive to antidepressant(s), consider buspirone, either with an antidepressant or as monotherapy

Post-Stroke Catastrophic Reactions

- Outburst of emotion, such as anxiety, agitation, or crying, that occurs when unable to perform simple tasks that were possible before
- Associated with PSD & Basal Ganglia lesions
- may be a release phenomenon due to subcortical damage
- Often associated with expressive aphasia.
- Treatment consists of prophylactic and supportive measures

Poststroke psychosis

- Rare complication
- Include paranoia, delusions, hallucinations (which may affect various sensory modalities; auditory and visual hallucinations are the most common), ideas of reference, thought disorganization, and regressed motor behavior

Poststroke psychosis

- More prone to have comorbid epilepsy
- Psychotic episodes can also be a manifestation of complex partial seizures secondary to stroke
- Correlate with right-sided lesions and cortical/subcortical atrophy

Paranoia

Associated with lesions in

- Left temporal strokes that result in Wernicke aphasia
- Right temporoparietal region and the caudate nuclei

Visual hallucinations and delusions

- Right hemispheric lesions

Peduncular hallucinosis

- Well-formed and complex visual hallucinations
- Lesions or infarcts of the ventral midbrain

Treatment of Poststroke psychosis

- Atypical antipsychotic, such as risperidone or olanzapine
- Close follow-up every 2 weeks and titration of antipsychotic dose to effect is recommended
- Reassessment for reemergence of psychosis, repeated cognitive examination, and depression inventory at each visit are recommended

Reduplicative paramnesia

- Patients claim that they are simultaneously in two or more locations
- Due to
  - Combined lesions of frontal and right temporal lobe
  - Remporal limbic- frontal dysfunction

Capgras syndrome

- False belief that someone familiar, usually a family member or close friend, has been replaced by an identical appearing imposter
- Right temporal-limbic-frontal disconnection - disturbance in recognizing familiar people and places

Fregoli syndrome

- Patient believes a persecutor is able to take on a variety of faces, like an actor
- Injury to the right frontal and left temporo-parietal areas

Obsessive-compulsive features

- Due to dysfunction in the orbitofrontal-subcortical circuitry (Saxena et al., 1998)

Poststroke Apathy Syndrome

Robinson, 1997

- Apathy is the lack of feeling, emotion or interest in one's surroundings or activities
- Is seen as the only neuropsychiatric symptom in as many as 11% of stroke patients
- Is often misdiagnosed as PSD
- Typically a result of deep posterior subcortical lesion
- Responds well to psychostimulants

Apathy

- Presents with profound lack of initiative without tearfulness, sleep/appetite disturbance, hopelessness, or suicidality
- In the absence of depression may be difficult to appreciate
- Treated with antidepressants and/or psychostimulants

Post-stroke fatigue

- Managed with Antidepressants and psychostimulants, particularly those with effects on noradrenergic and/or dopaminergic activity (eg, bupropion, venlafaxine, and mirtazapine)
- Follow-up within 1 month is needed

Aggression

- Associated with increased motor dysfunction and dysarthria
- Lesions in the area supplied by the subcortical middle cerebral artery → inability to control anger or aggression
- Lesions nearer to the frontal pole → irritability and aggression

Aggression

Management

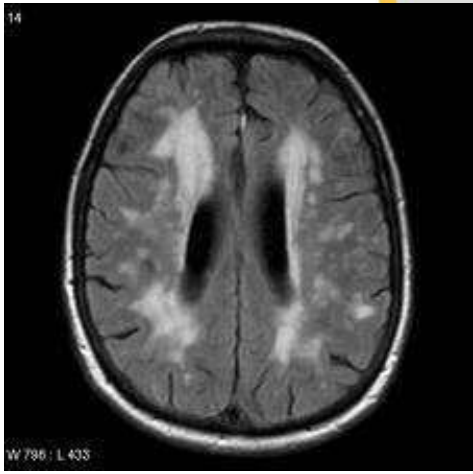
- Fluoxetine reduce levels of poststroke anger (Choi-Kwon et al., 2006)
- Measures to reduce depression (Chan et al., 2006)

Categories of Vascular Dementia

Category	Clinical presentation
Lacunar infarctions	Progressive dementia, focal deficits, or apathetic, frontal-lobe-like syndrome, may have no stroke history
Single strategic infarctions	Sudden onset aphasia, agnosia, anterograde amnesia, frontal lobe syndrome
Multiple infarctions	Step-wise appearance of cognitive & motor deficits
Mixed AD-VaD	Progressive dementia with remote or concurrent history of stroke
White matter infarctions (Binswanger's disease)	Dementia, apathy, agitation, bilateral cortico-spinal/bulbar signs

Post-Stroke Dementia

- A temporal relationship between a stroke and the onset of dementia
- Stepwise progression of cognitive decline
- Evidence of cerebrovascular disease on examination
- Neuroimaging findings



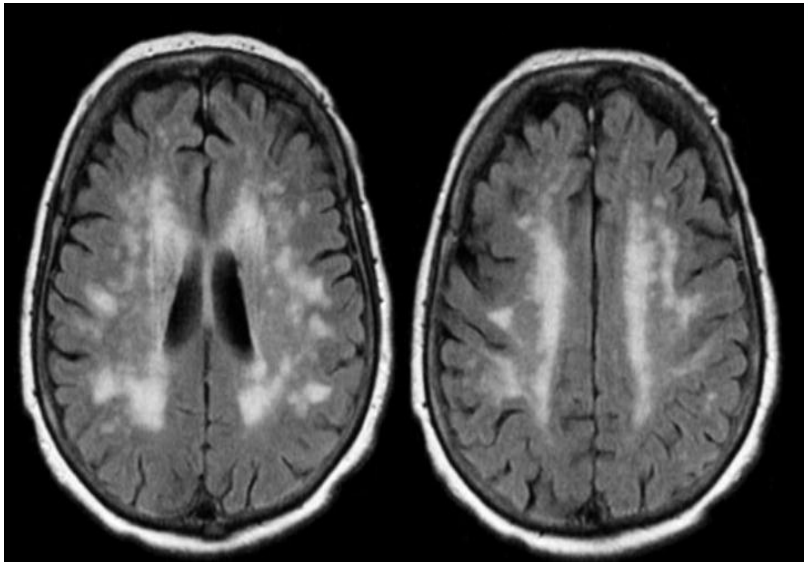
No specific neuroimaging profile exists that is diagnostic for pure cerebrovascular disease-related dementia.

Post-Stroke Dementia

- Small vessel disease is the most frequently observed vascular pathology
- Series of deep white matter infarcts
- Present with prominent cortical, subcortical, or mixed features

Binswanger disease

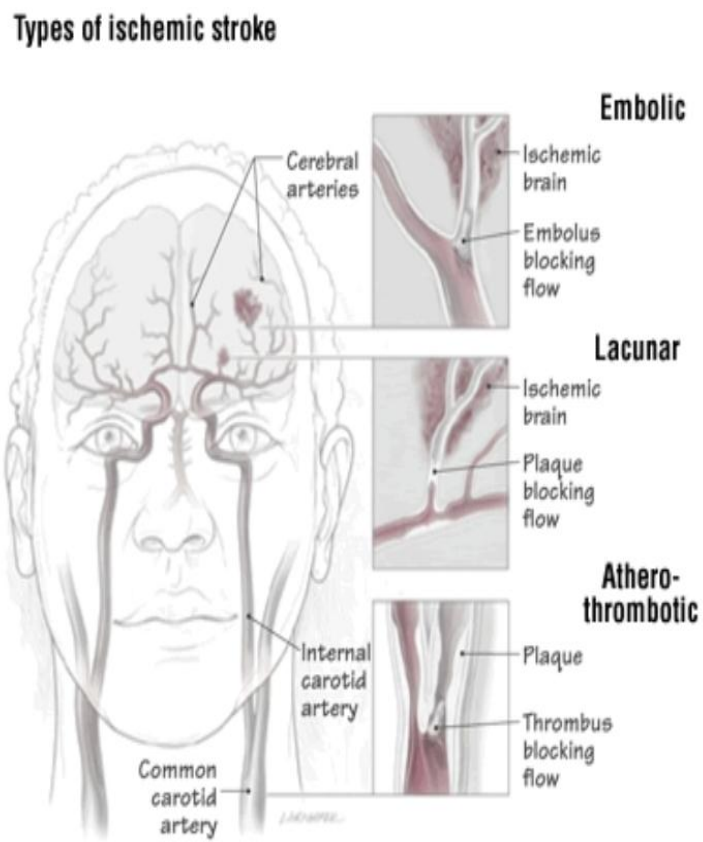
- Primarily caused by CVA or impaired blood flow and falls within spectrum of vascular cognitive impairment (VCI)
- 25 to 50% cases of dementia
- Risk factors:
  - Advanced age
  - DM Hypertension
  - Seizures
  - Recurrent small vessel stroke

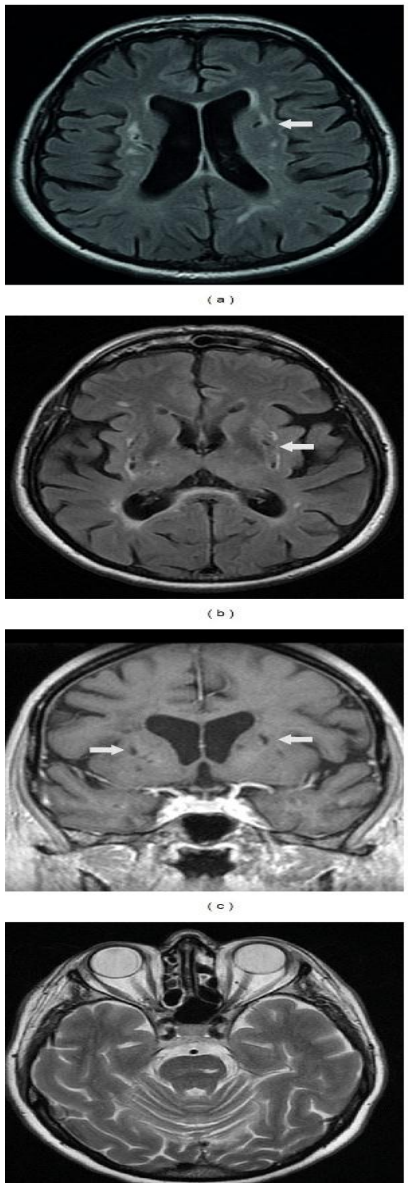


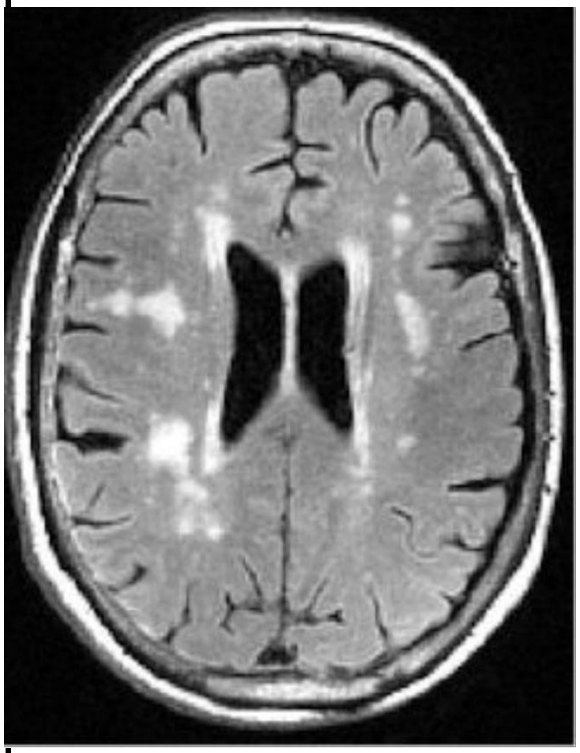
Subcortical vascular dementia

- Affects fronto subcortical circuitry
- Resulting in executive dysfunction, cognitive slowing, difficulties with abstraction, apathy, memory problems (recognition and cue recognition relatively intact), working memory impairment, and decreased ability to perform activities of daily living

Lacunar state







Lacunar state

- Pseudo bulbar disorder
- Appearance of small smooth walled cavities in brain tissue
- Small strokes (2-20mm) with arterial hypertension and arteriosclerosis in deep cerebral white matter, basal ganglia or pons from occlusion of small penetrating branches
- Apraxia, gait and memory impairment, parkinsonism

Cortical vascular dementia

- Unilateral sensorimotor dysfunction
- Abrupt onset of cognitive dysfunction and aphasia
- Difficulties with planning, goal formation, organization, and abstraction

Post-Stroke Dementia

- Some cases of dementia diagnosed in the post-stroke period may represent previously unrecognized cases of AD
- Memory difficulties tend to be less severe than in AD

Post-Stroke Dementia

- Changes in instrumental activities of daily living that require complex organizational and problem-solving skills (e.g., managing finances, following directions, “figuring things out”) are likely more prominent in a patient with VaD compared to one with AD
- Apathy is a hallmark symptom

Monitoring Post-Stroke Dementia

- Folstein Mini-Mental State Examination or the cognitive portion of the Cambridge Examination for Mental Disorders of the Elderly
- Repeated serially to monitor progression and/or treatment response

Treatment for post-stroke dementia

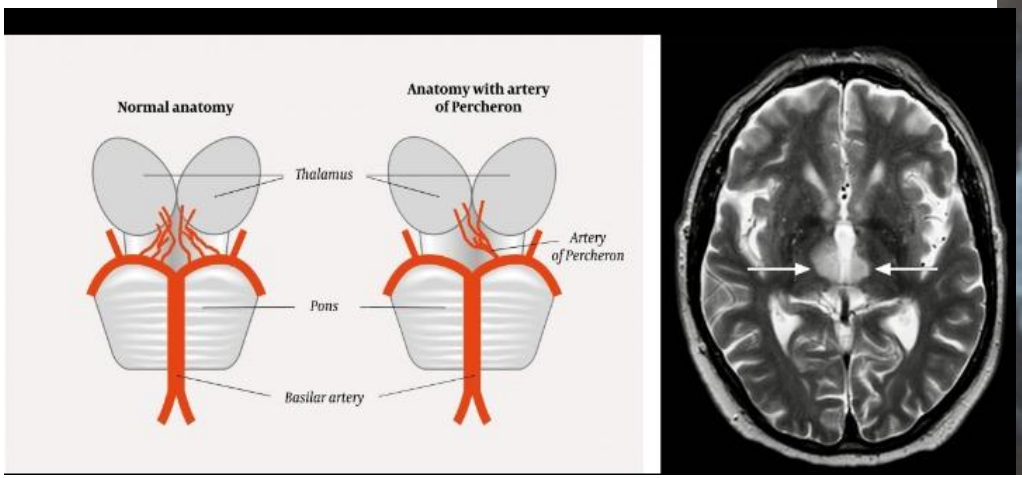
- Low threshold for psychiatry referral for agitated behavior, persistent confusion, or cognitive inability to participate in treatment
- An additional workup (vitamin B12, folate, and TSH analysis; toxicology screening; and rapid plasma reagent and HIV testing) for reversible causes of dementia should also be accomplished

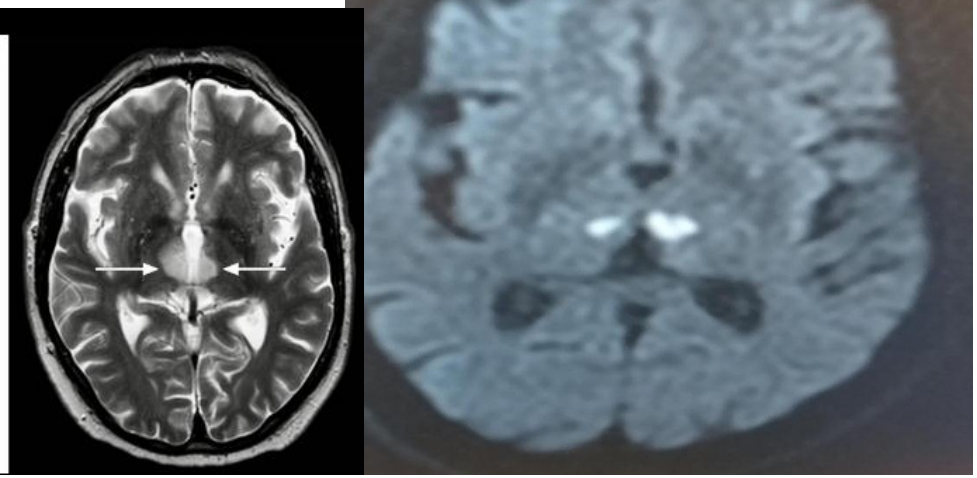
Treatment for post-stroke dementia

- May benefit from pharmacotherapy for AD (cholinesterase inhibitors and memantine)
- Dose increased at monthly intervals according to response
- Initiate atypical antipsychotics and/or antidepressants for agitated behavior
- Followed up monthly, with reassessment of cognitive examination, repeated depression inventory, and screening for psychotic symptoms

Artery of Percheron Infarct

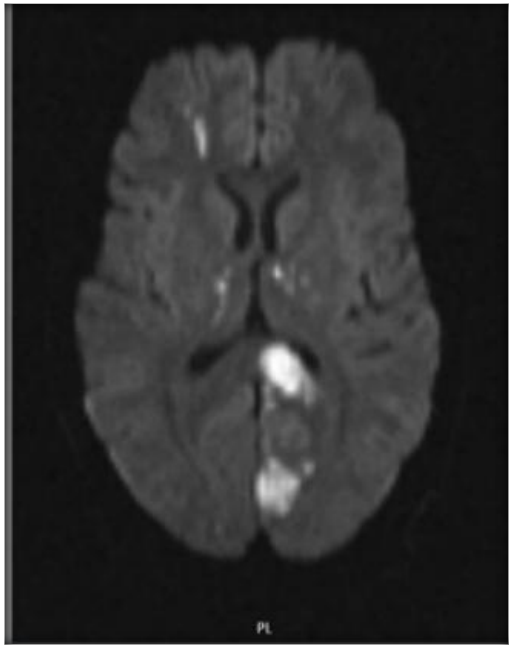
- Bilateral thalamic or midbrain infarcts causing acute altered mental status
- Hypersomnolence, memory impairment, psychosis, aphasia, dysarthria





Alexia without Agraphia

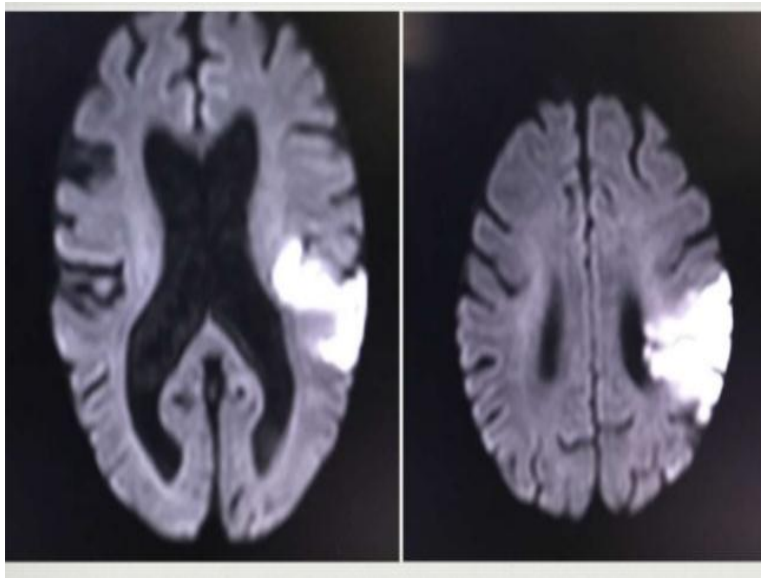
- Can write but not read
- Able to understand and produce speech
- Lesion is in left occipital lobe with extension into splenium of corpus callosum
- Visual information reaches the left visual field but pathways that allow interpretation of written language are interrupted



MISSING LINK

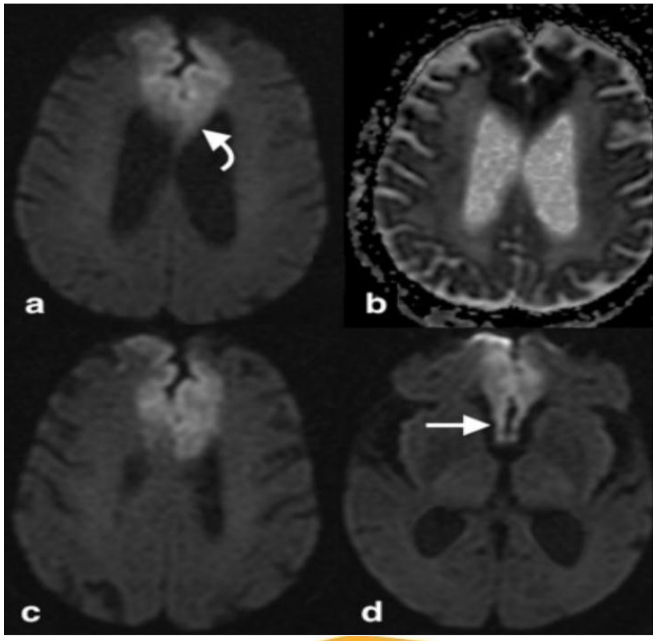
Wernickes aphasia

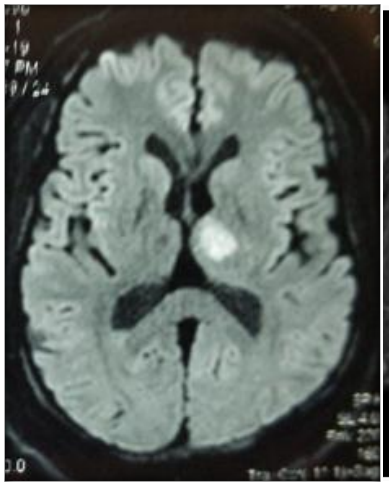
- Fluent aphasia with markedly impaired comprehension
- Speech is voluminous but meaningless
- Paraphasic errors and neologisms
- Lesion: posterior superior temporal gyrus

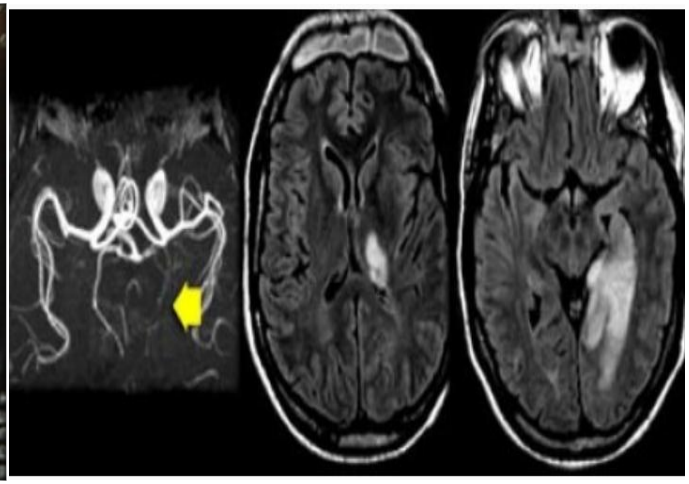


Single vessel dementia

- Anterior thalamic infarction: isolated memory impairment
- Medial frontal lobe: ACA territory
- Language cortices, thalamus and medial temporal lobes

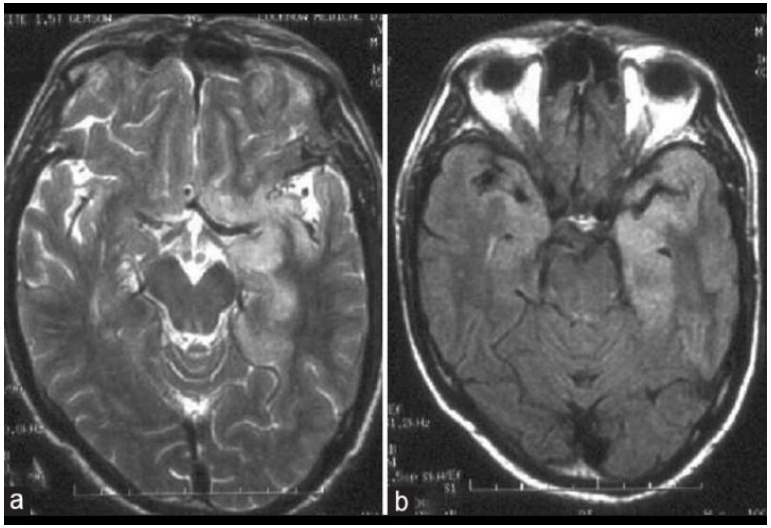


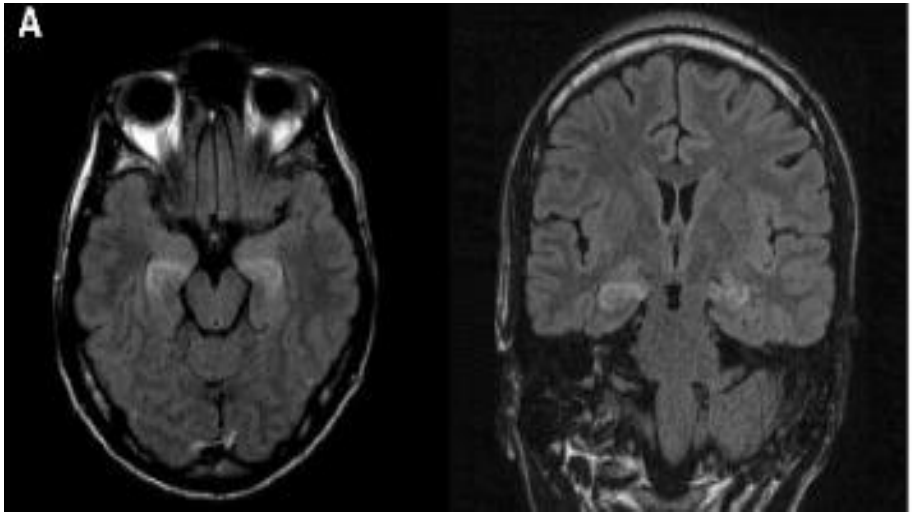




Kluver bucy syndrome

- Apathy, visual agnosia, increased sexual activity, compulsive eating and increased oral behaviour
- Bilateral medial temporal lobe including amygdaloid nucleus





Summary

- Depression & anxiety are the 2 most common post-stroke syndromes.
- Both depression and anxiety increase morbidity and delay rehabilitation.
- Treatment of neuropsychiatric post-stroke disorders have the greatest potential for improving outcome and quality of life.

To Sum-Up….

- This is no health without mental health
  - Mental health after stroke is everyone's business
- Stroke threatens identity, self-esteem and mental health
- It is associated with high rates of depression, anxiety disorders and emotionalism
  - These are treatable
  - Depression is not an inevitable consequence of stroke
- Suicide rates double after stroke
  - It is important to screen mood in all stroke survivors using a validated tool that includes a question about suicidality
- Psychological care after stroke is improving – we can help!

There is No Health Without Mental Health

"Mental health is ***everyone's business*** …. Good mental health and resilience are fundamental to our physical health, our relationships, our education, our training, our work and to achieving our potential."

Parkinson's disease and Psychiatry

- Depression, anxiety, and psychosis are common complications of Parkinson's disease (PD) and of the medications used in antiparkinsonian treatment
- Impair patients' functioning throughout the course of the chronic degenerative disease
- While motor signs dominate the presentation, cognitive symptoms such as shortened attention span, visuospatial impairment, personality changes, and dementia are also frequently present

Drug treatment side effects

- Treatment emphasizes dopamine replacement, dopamine receptor stimulation, or prevention of enzymatic breakdown of dopamine in the synaptic cleft
- Hallucinations and psychosis
- Dopamine levels are increased in an attempt to smooth the motor response.

Medications commonly used in managing parkinson's disease

Medication class	Example	Indication for use
MAO-B inhibitor	Selegiline	Neuroprotection
Anticholinergic agents	Trihexypheridyl, benztropine, biperiden, hyoscyamine, diphenhydramine	Tremor
Dopamine agonist	Pramipexole, pergolide, ropinirole	Neuroprotection Treatment of movement disorder
Dopamine replacement	Carbidopa-levodopa	Treatment of movement disorder
Catechol-O-methyltransferase inhibitor	Entacapone, tolcapone	Smooth motor fluctuations

Depression and PD

- The stress of anticipating and coping with a relentless degenerative disease helps to trigger depression and anxiety in patients with PD
- Most common psychiatric syndrome, with prevalence in PD as high as 42%
- History of depression are at particular risk
- Those with recent deterioration or advancing severity of PD, akinesia, history of falls, or cognitive impairment are also at increased risk for depression

- Diminished affect and psychomotor slowing may be secondary to the motor features of parkinsonism
- Diminished concentration may be secondary to cognitive decline rather than depression
- Diminished energy or fatigue that should trigger further investigation into other depressive symptoms
- Depression in PD predicts impaired social, physical, and role functioning
- Also results in higher distress for caregivers.

Anxiety

- 33 to 40%
- Presents with symptoms of panic disorder, generalized anxiety disorder, or social phobia
- Comorbid with depression in up to 92% of cases and— like depression—frequently predates the onset of motor symptoms
- Can also be an adverse effect of many of the antiparkinsonian medications
- Both anxiety and depression have been associated with an increased risk for falls

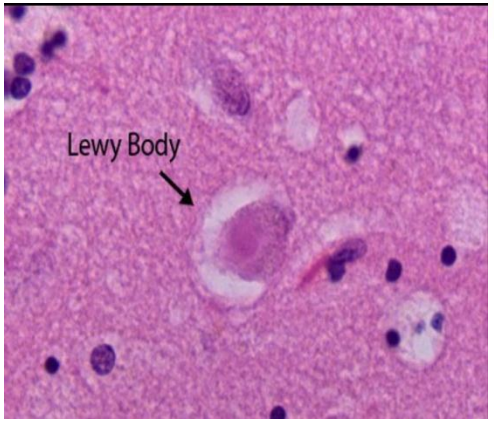
Psychosis

- Up to 25% of PD patients experience delusions or hallucinations
- Risk factors include dementia, sleep disturbance and most commonly the use of dopaminergic agents
- All known classes of antiparkinsonian medications has been associated with drug-induced psychosis
- Paranoid delusions, delusions of spousal infidelity, and visual hallucinations are common

- Goals for psychiatric treatment of depression, anxiety, and psychosis associated with PD seem relatively straightforward:
  - Improvement or remission of psychiatric symptoms
  - Restoration of optimal patient functioning
- Without causing sedation, orthostatic hypotension, or exacerbating motor symptoms

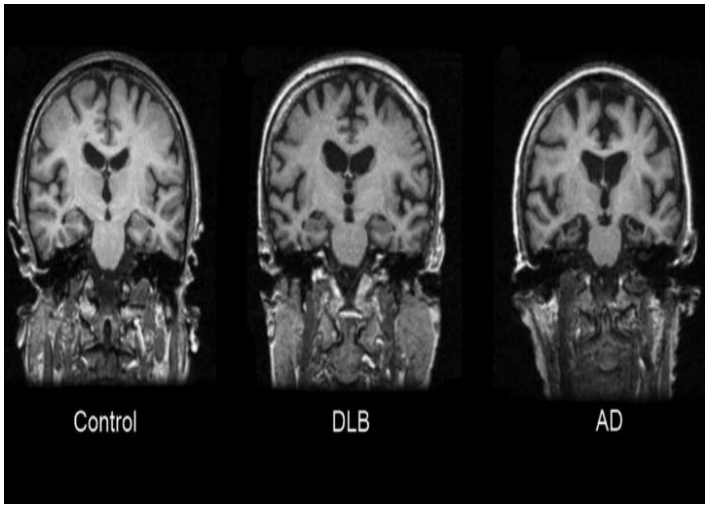
Lewy body dementia

- PD with dementia and Lewy bodies
- Gradual progressive cognitive decline with gait impairment
- Affects legs more than arms (lower body parkinsonism)



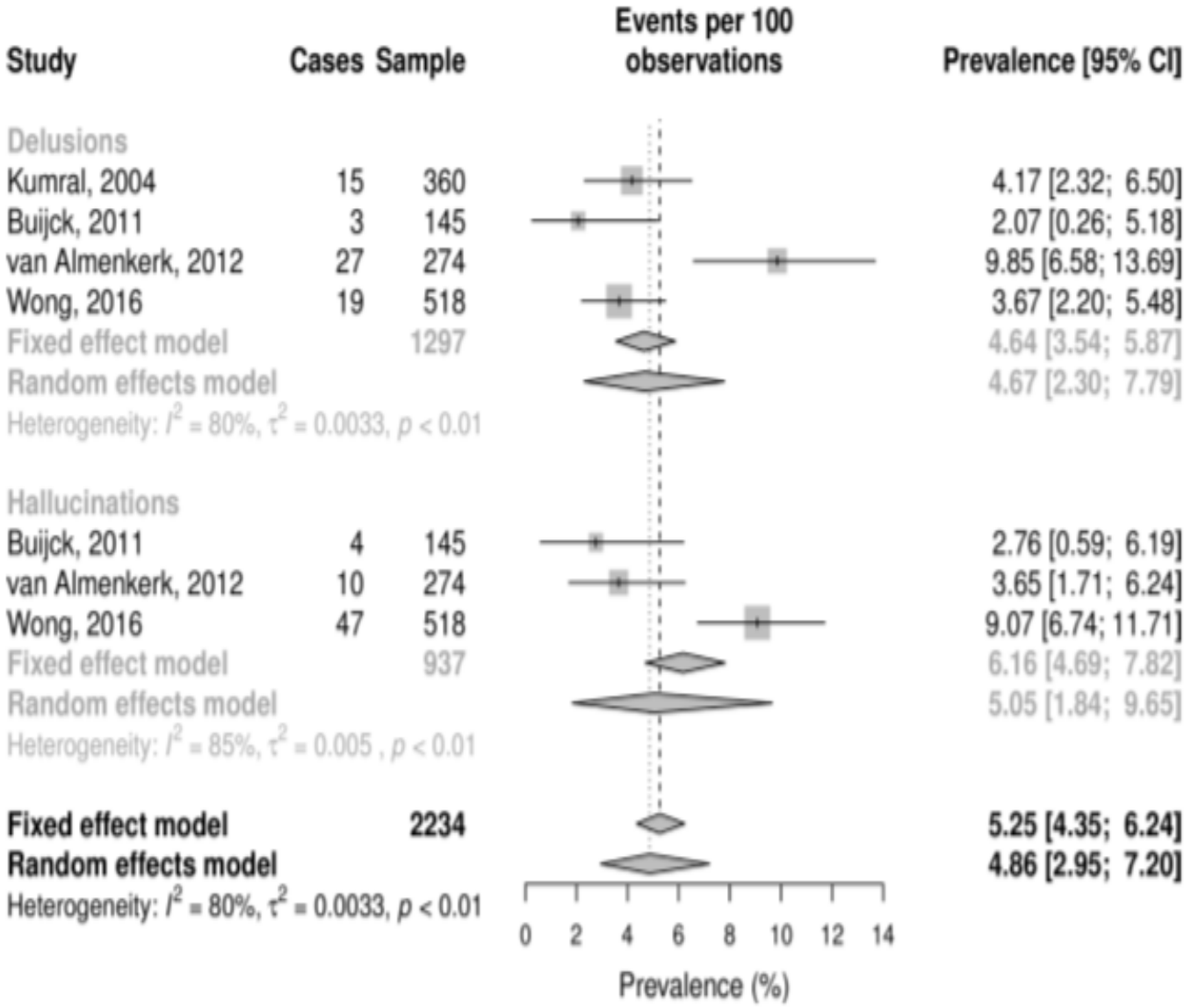
Must have at least 2 of following:

- Cognitive fluctuations
- Visual hallucinations
- Rapid eye movement (REM) sleep behaviour disorder
- Parkinsonism




Thank you

Neuropsychiatry



Pseudobulbar affect (emotional incontinence)

- Uncontrollable Excessive crying or laughing
- Widely dispersed neural network involving frontal, parietal and brainstem region

Neuropsychiatric Symptoms and Corresponding Neuroanatomy

Symptoms	Neuroanatomical Region
Depression	Frontal lobes, left anterior frontal cortex, anterior cingulate gyrus, subgenu of the corpus callosum, basal ganglia, left caudate
Mania	Inferomedial and ventromedial pfrontal cortex, right inferomedial pfrontal cortex, anterior cingulate, caudate nucleus, thalamus, and temporothalamic projections

Neuropsychiatric Symptoms and Corresponding Neuroanatomy

Symptoms	Neuroanatomical Region
Apathy	Anterior cingulate gyrus, nucleus accumbens, globus pallidus, thalamus
OCD	Orbital or medial frontal cortex, caudae nucleus, globus pallidus
Disinhibition	Orbitofrontal cortex, hypothalamus, septum
Psychosis	Frontal lobes, left temporal cortex

Neuropsychiatric Symptoms and Corresponding Neuroanatomy

Symptoms	Neuroanatomical Region
Paraphilia	Mediotemporal cortex, hypothalamus, septum, rostral brainstem
Hallucinations	Unimodal association cortex, orbitofrontal, paralimbic, limbic cortices, striatum, thalamus, midbrain
Delusions	Orbitofrontal cortex, amygdala, striatum, thalamus