

Dementia 1.01

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First dementia case
described ever!

Sebastiano Ricci (1659-
1734)
Aurora and Tithonos (ca.
1705)

Content

- Learning objectives
- Introduction
- Neuro-anatomy
- Ageing & cognition
- Definition & criteria
- Types of dementia

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Learning objectives

Insight into different forms of dementia

Knowledge of relevant brain structures

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Dementia: of all times

- De + mens: “being outside one’s mind”
- Ptahhotep (2nd century BC, in hieroglyphs): describes an elderly person becoming each night more and more childish
- Plato (5th-4th century BC) describes an illness that gives way to forgetfulness and stupidity

Dementia: of all times

- Juvenalis (1st-2nd century AD) describes multiple diseases in the elderly, but

“worse than any other physical disease is the loss of the mind, causing him to forget the names of his slaves, causing him not to remember the face of a friend he ate with yesterday, nor the children he raised himself”

- Renaissance: witch burnings...

Pop
Quiz!



Memory

How many kinds of memory are there?

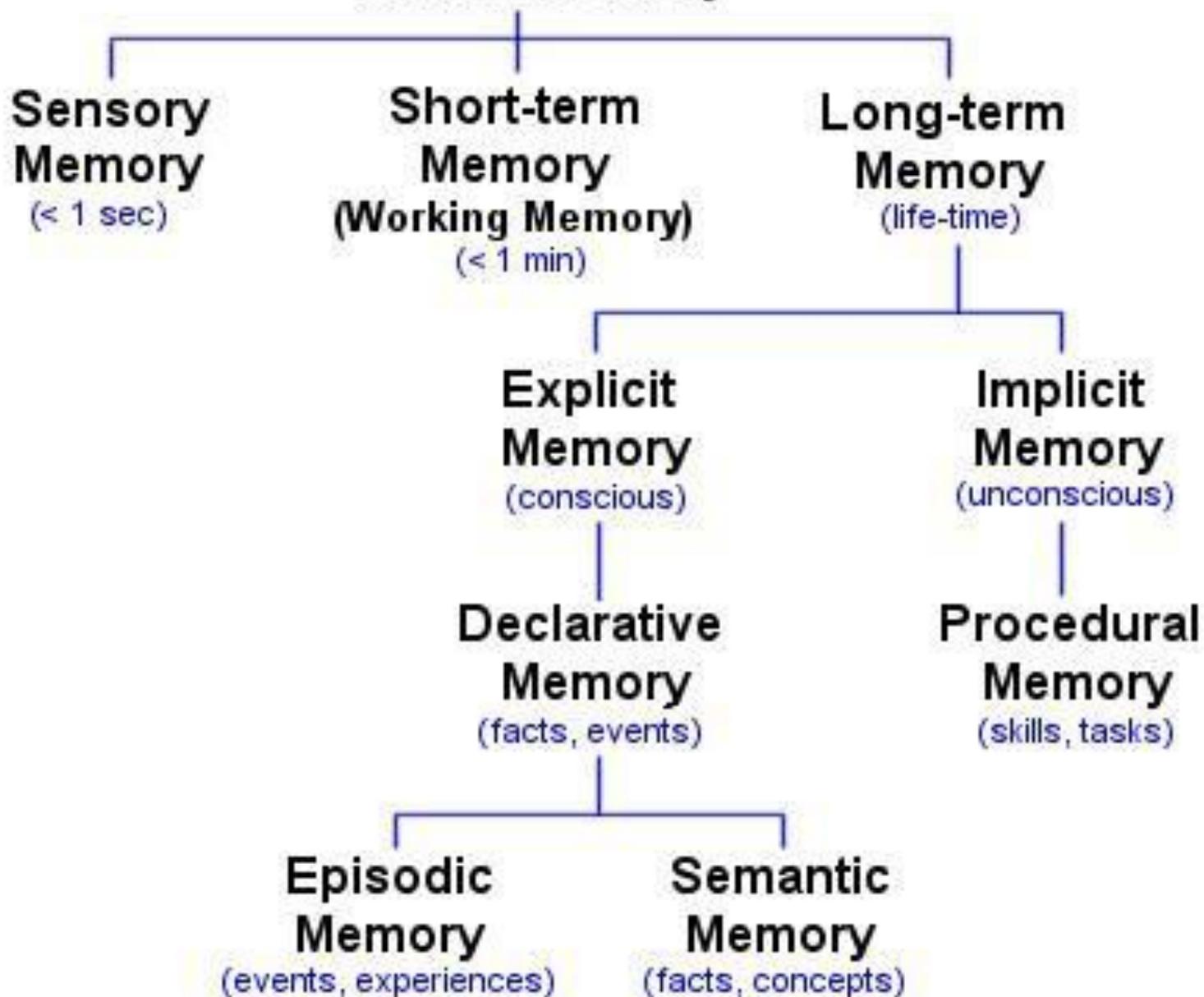
1.1

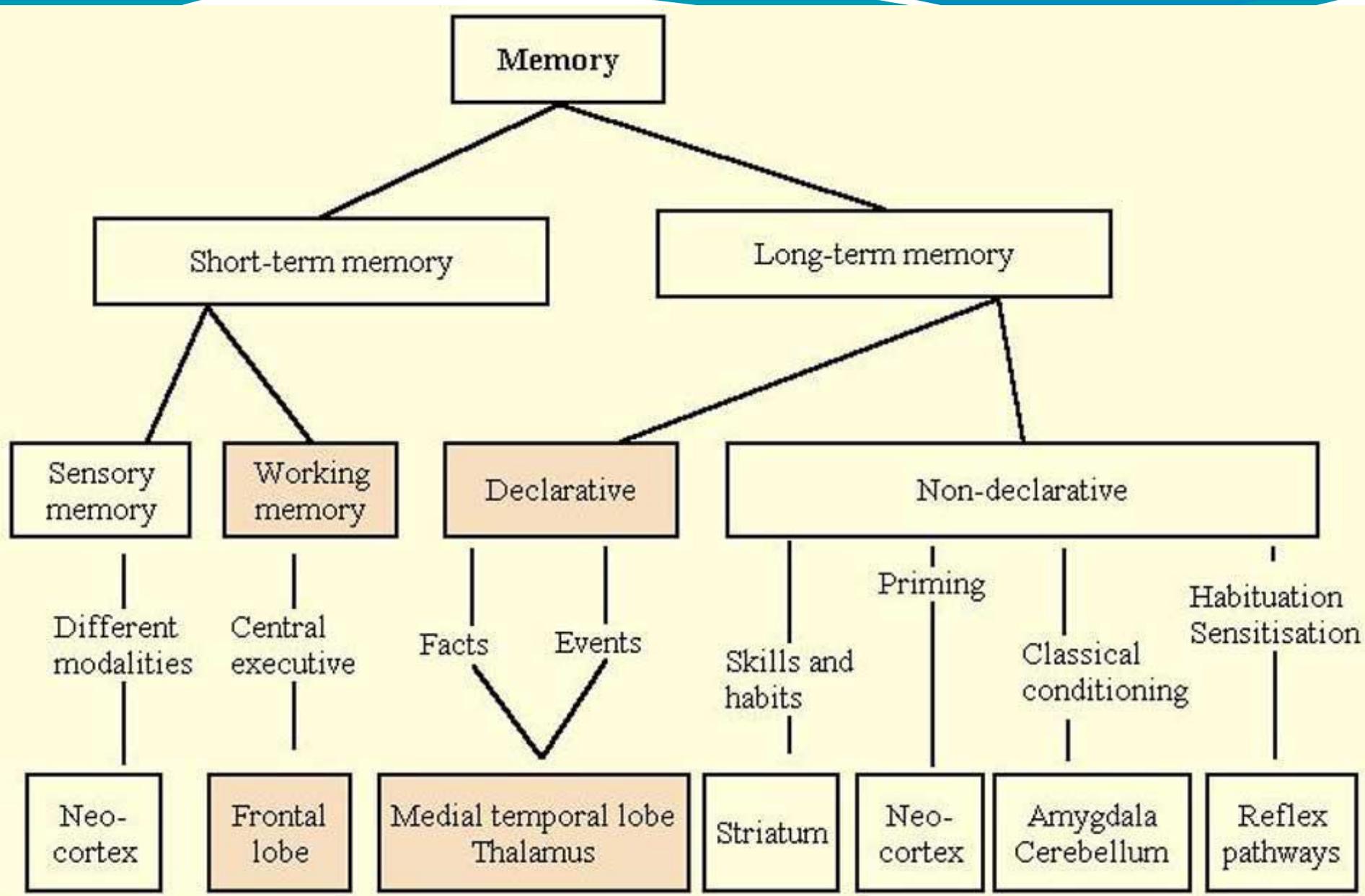
2.5

3.9

4.12

Human Memory





Memory

Short-term memory

Long-term memory

Sensory memory

Working memory

Declarative

Non-declarative

Different modalities

Central executive

Facts

Events

Skills and habits

Priming

Classical conditioning

Habituation Sensitisation

Neo-cortex

Frontal lobe

Medial temporal lobe
Thalamus

Striatum

Neo-cortex

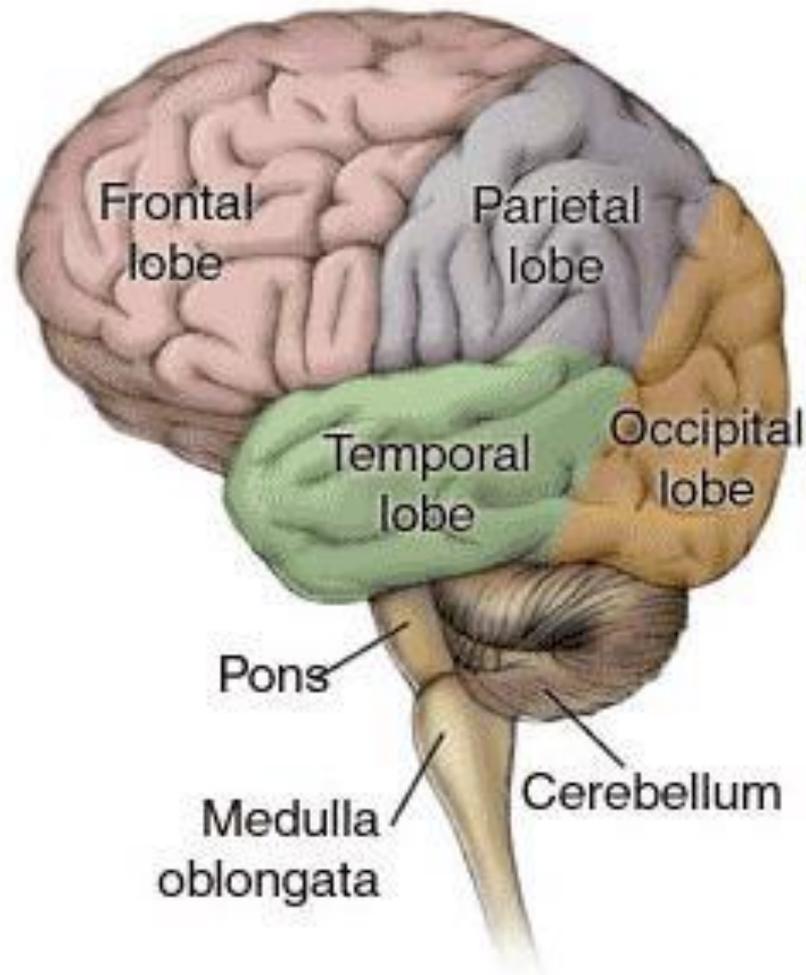
Amygdala
Cerebellum

Reflex pathways

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Gross anatomy of the brain



Frontal lobe

Handles executive/management function, such as planning, judgment, motivation, impetus and behavior

Damage results in becoming uninhibited, lethargic.

A person may become “stuck” on a situation or word, causing them to repeat the action.

Frontotemporal dementia Alzheimer's disease

Parietal lobe

Handles spatial relationships, perception and magnitude.

In addition, this part of the brain is responsible for the sense of one's body.

Damage results in for instance forgetting how to do common movements (such as those that are necessary to get dressed) and may not recognize objects, faces or surroundings.

Temporal lobe

Handles auditory processing (primary auditory cortex), high-level visual processing of complex stimuli such as faces and scenes, language processing (comprehension, verbal memory).

The medial temporal lobe is involved in long term memory / memory storage / new memory formation.

Damage leads to problems with language / memory encoding.

Occipital lobe

Helps interpret what the eyes are seeing.

Damage leads to misinterpreting the environment through illusions, misperceptions and misidentification.

It may also result in hallucinations.

Lewy body dementia, Parkinson's disease dementia,
Alzheimer's disease

Cerebellum

Linked to the coordination of voluntary movement, gait, posture, speech and motor functions.

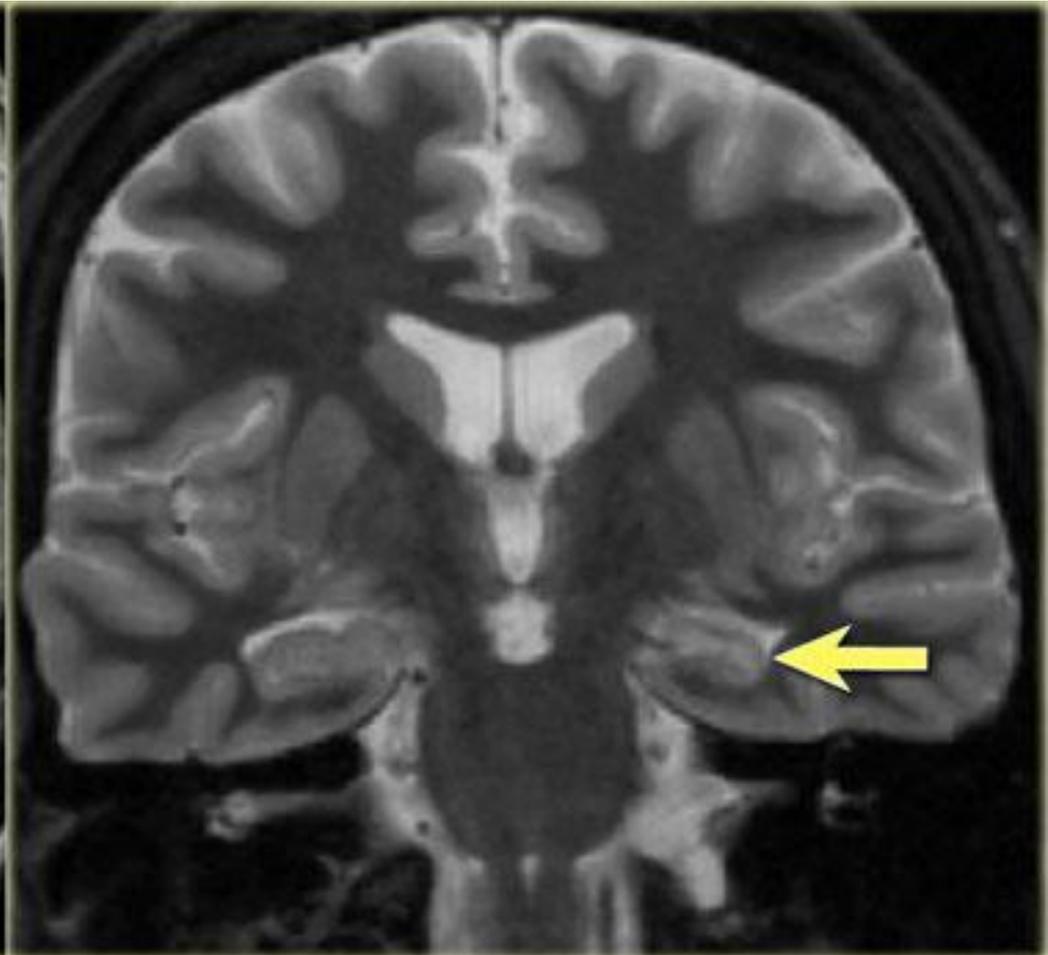
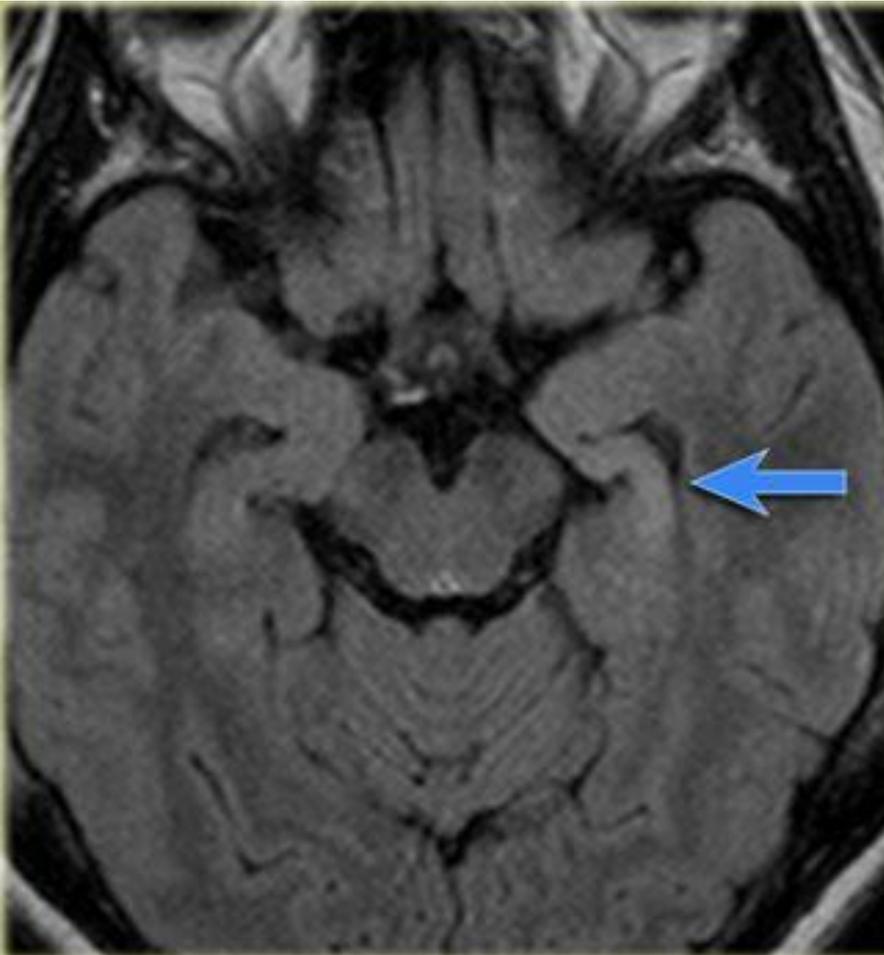
Believed to help with control of cognition and behavior.

Vascular dementia, others in later stages

Pop
Quiz!



What is this lobe called?



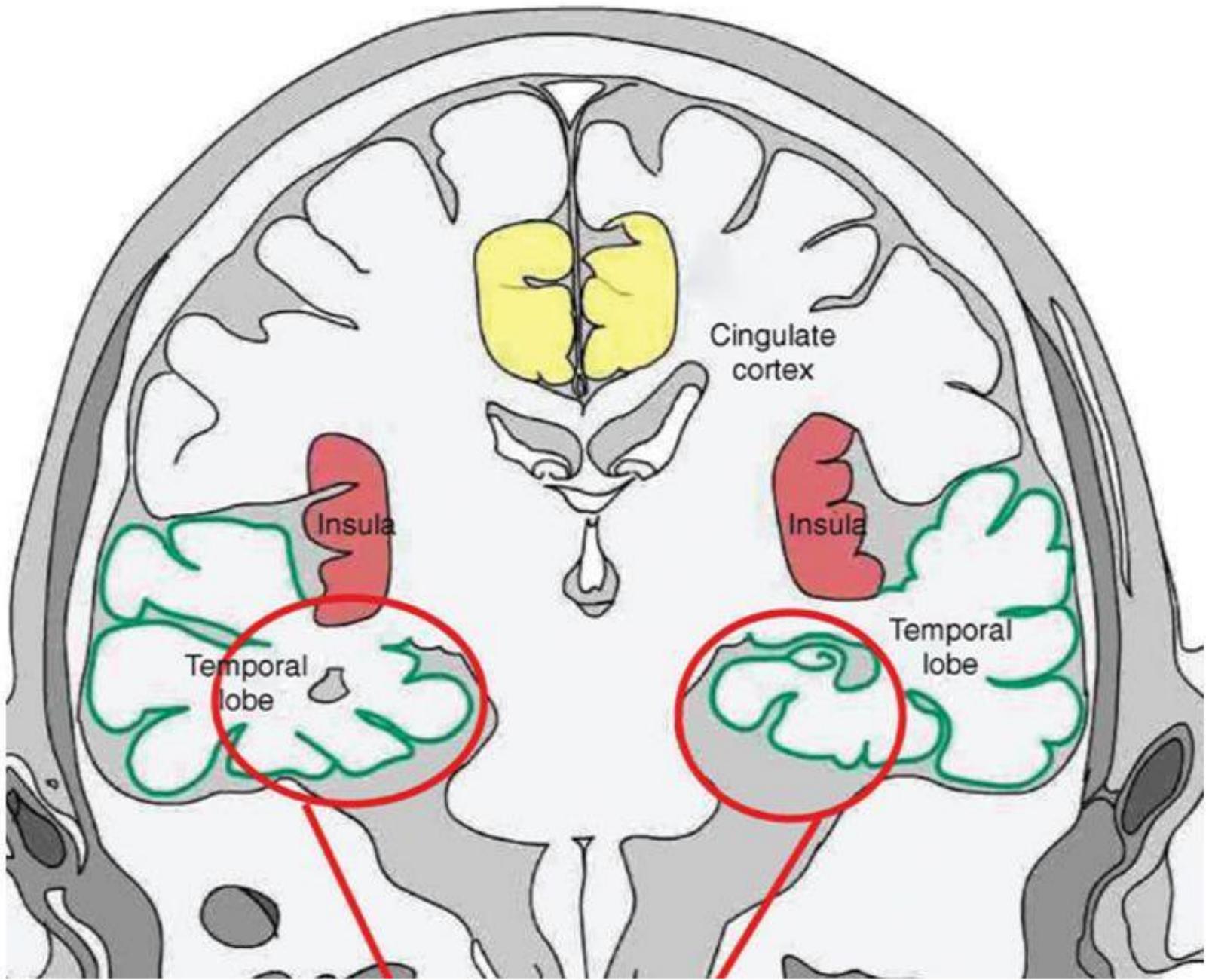
What is this lobe called?

1. The entorhinal lobe
2. The basofrontal lobe
3. The medial lobe
4. The mesotemporal lobe

In clinical practice, what is this medial (meso) temporal lobe most used for?

Screening for disorders in

1. Auditory processing (primary auditory cortex)
2. Visual processing (complex stimuli)
3. Language processing (comprehension, verbal memory)
4. Memory processing (long-term, new memories)



Cingulate cortex

Insula

Insula

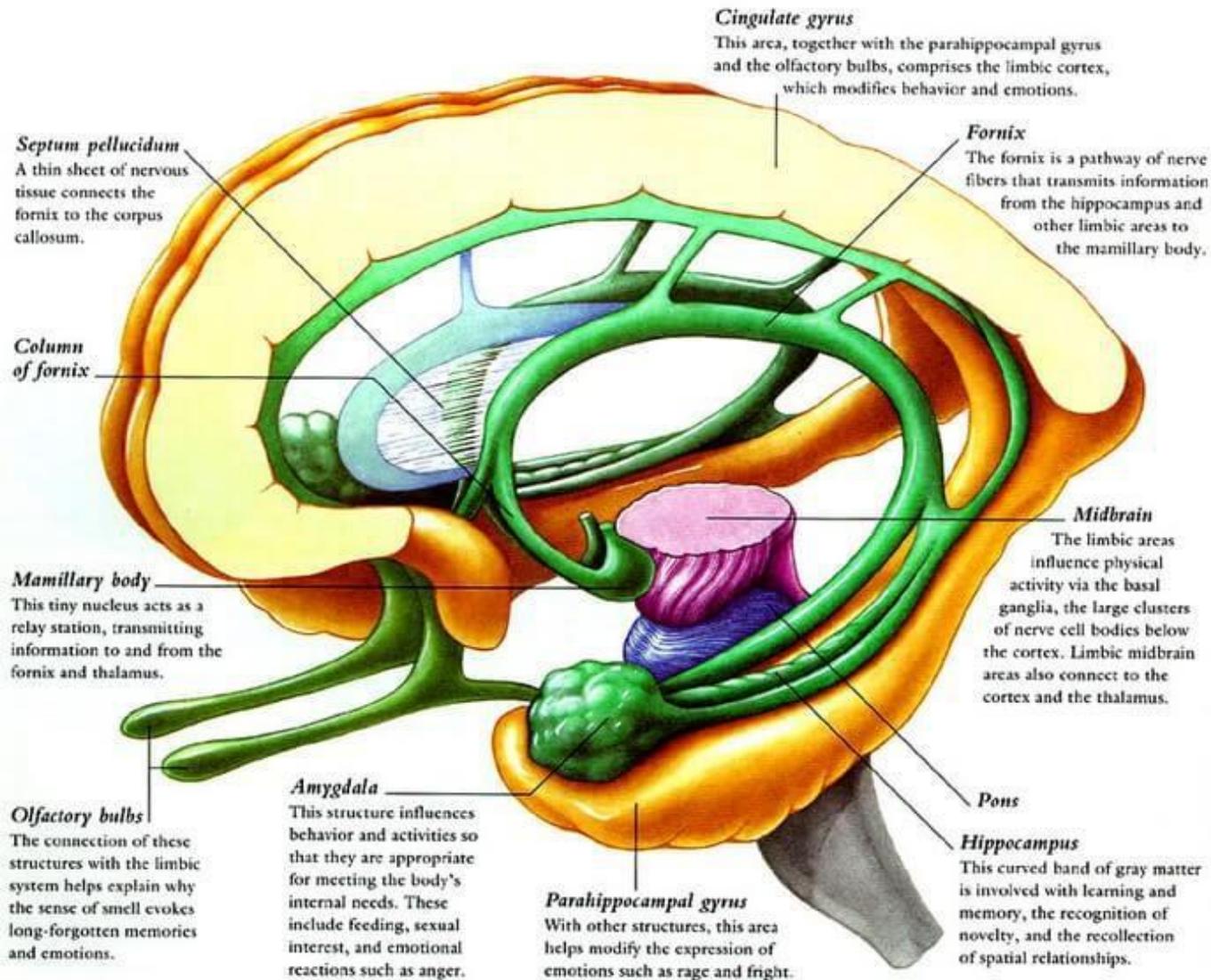
Temporal lobe

Temporal lobe

Which structure is no part of the medial temporal lobe?

1. Fornix
2. Amygdala
3. Dentate gyrus
4. Hippocampus / parahippocampal gyrus

Limbic system



Hippocampus

This brain region is responsible for forming new memories.

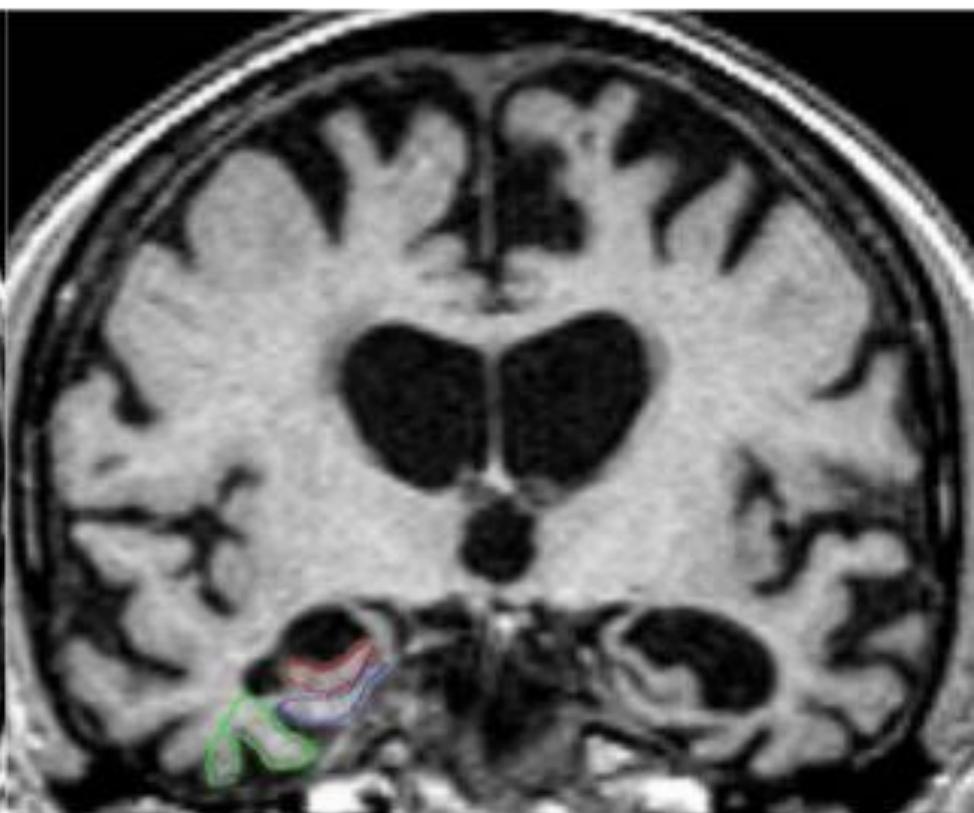
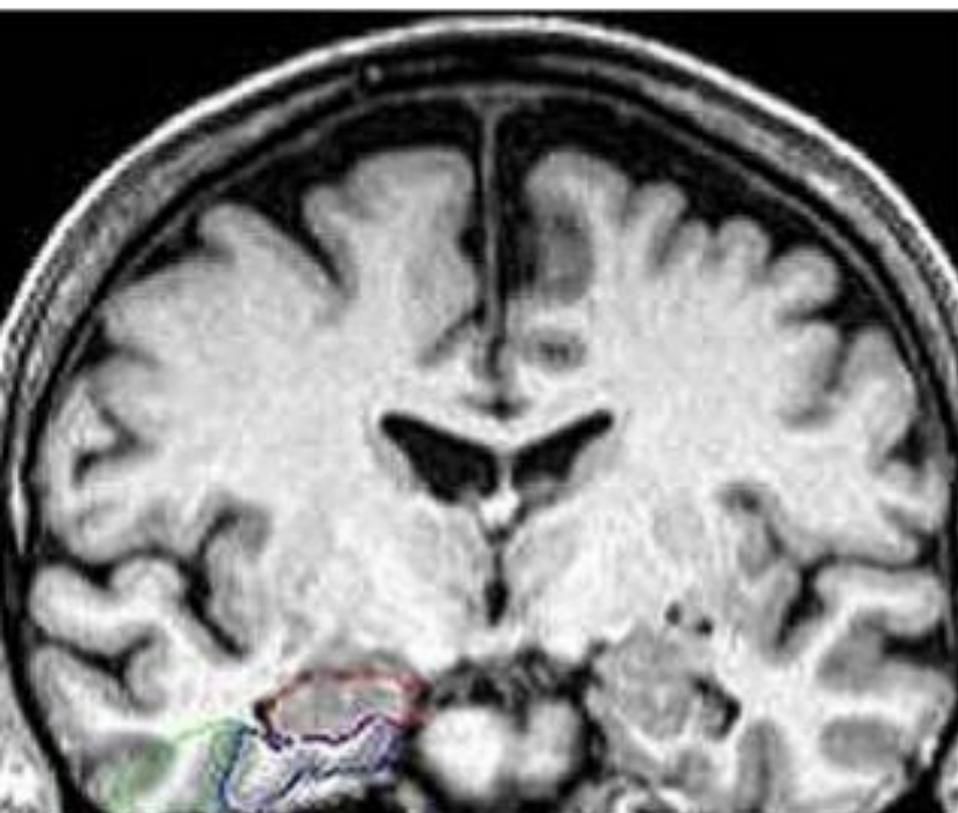
A study found that the volume and ratio of this region was reduced by 25% in Alzheimer's disease, 21% in mixed dementia, 11% in vascular dementia, and 5% in normal pressure hydrocephalus.

This part of the brain is where Alzheimer's begins and is seen in loss of short-term memory.

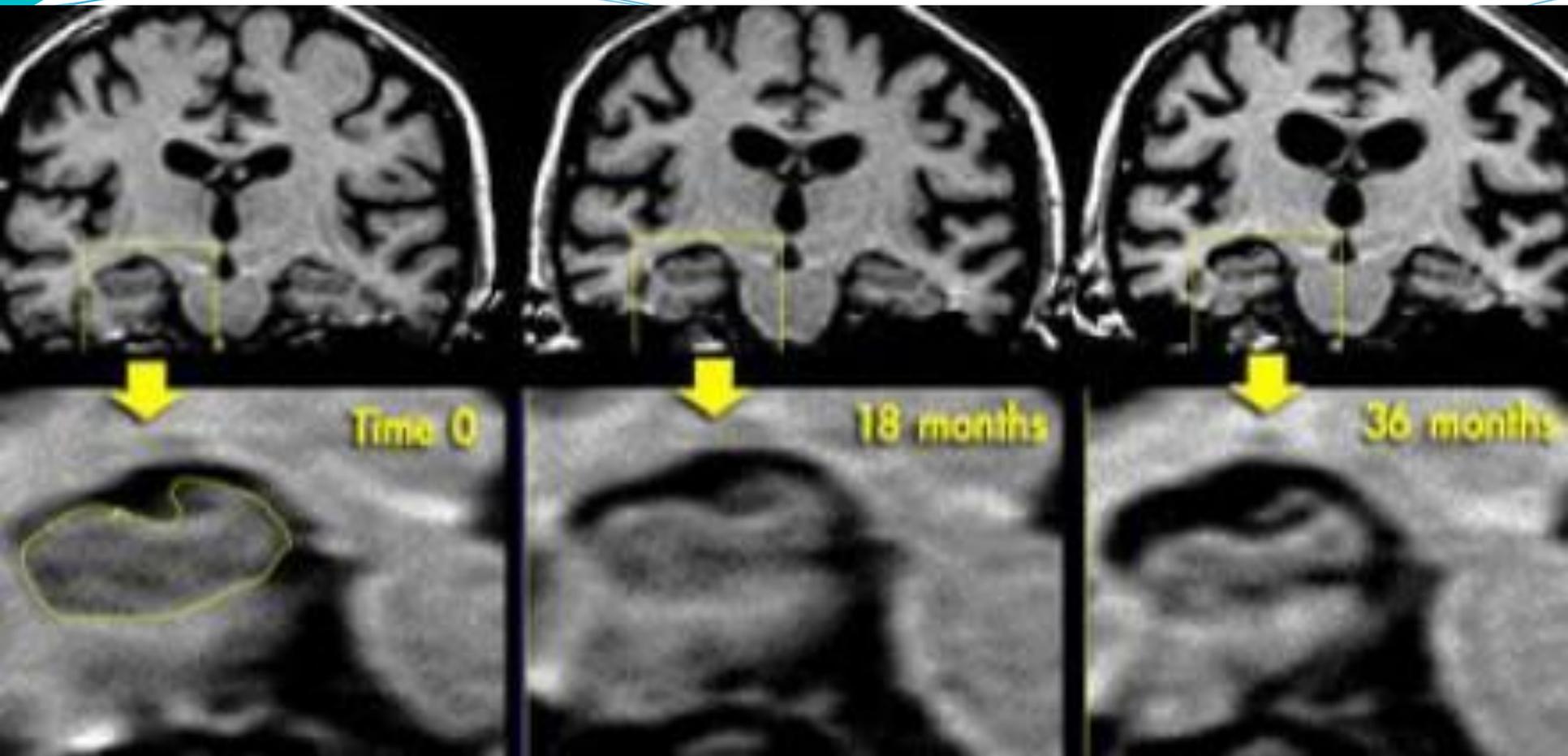
Biomarker: Hippocampal atrophy

Normal

Alzheimer's



T1 MRI coronal view

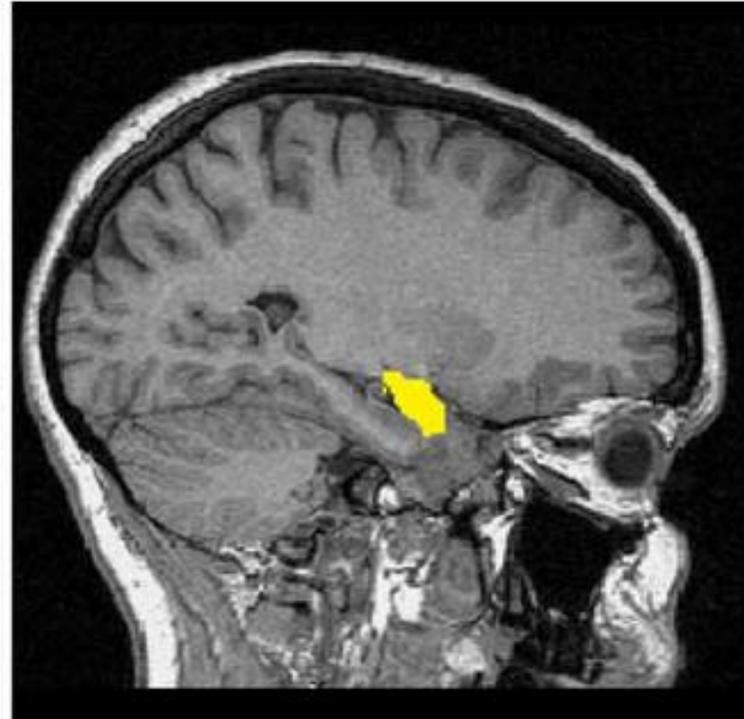
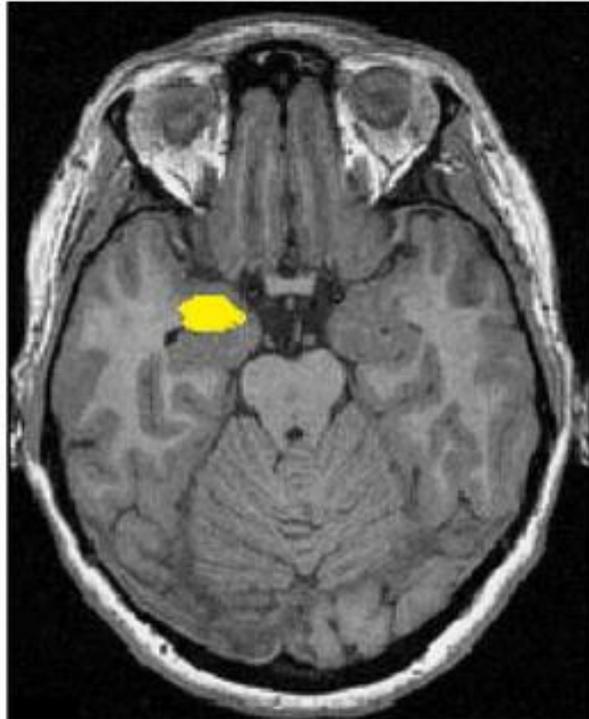
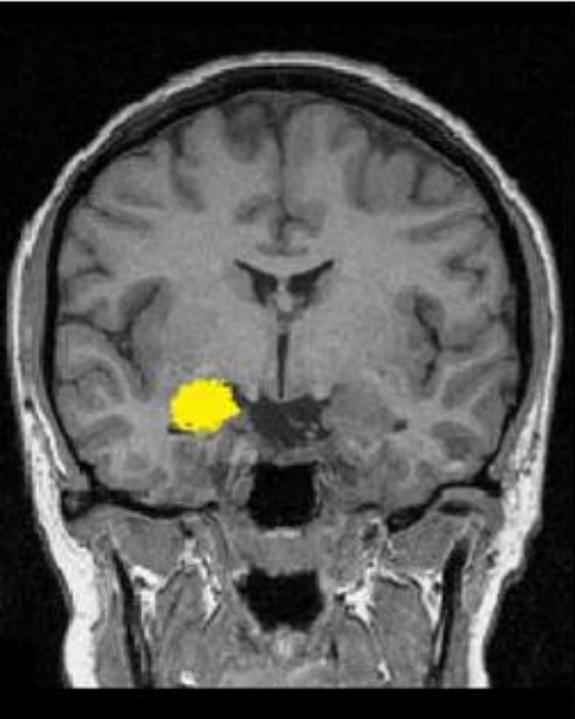


Amygdala

Responsible for the experience and expressions of emotion.

This area of the brain is linked to fear responses, pleasure, and sexual emotions. Depression is believed to be linked to abnormal functioning of the amygdala.

In Alzheimer's as well as frontotemporal dementia, this part of the brain is attacked, leading to personality changes.



Fornix

Collection of white matter tracts connecting different brain structures and main efferent system of the hippocampus

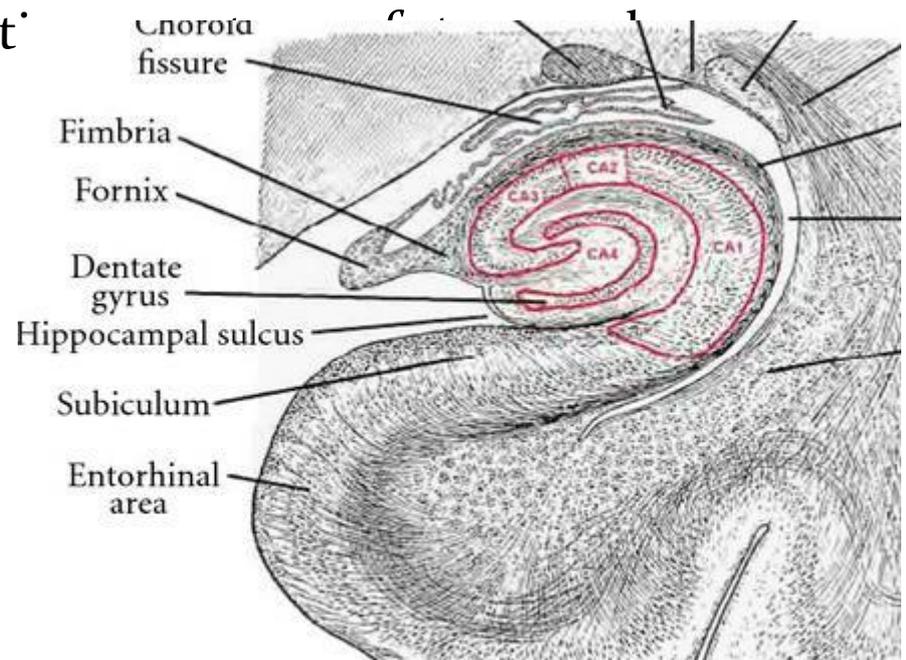
Connections

- the hippocampus to the mammillary bodies
- the hippocampus to the septal nuclei and the nuclei accumbens
- the mammillary bodies to the anterior nuclei of the

Dentate gyrus

Contribute to the formation of new episodic memories. Does pre-processing of very similar information into distinct and unique details. This prepares the relevant data for storage in the hippocampus.

Plays an important role in modulating depression.



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Forgetfulness of the elderly

- Almost everyone
- Not abnormal
- Sometimes difficult but not invalidating
- Only memory
- Details of the event are missing

• Dementia

- Illness, it is abnormal
- Invalidating
- More than only memory
- Event itself is lost
- No recollection afterwards
 - problem is imprinting

Normal ageing and cognition

- Language is moderately affected
- Understanding, vocabulary and syntax remain unaffected
- Mild decline in retrieval of words and verbal fluency
- Verbal intelligence is also unaffected
- Speed of information processing diminishes
- Executive functions for every day tasks are retained
- New tasks or when attention is divided: slower execution

Hard data

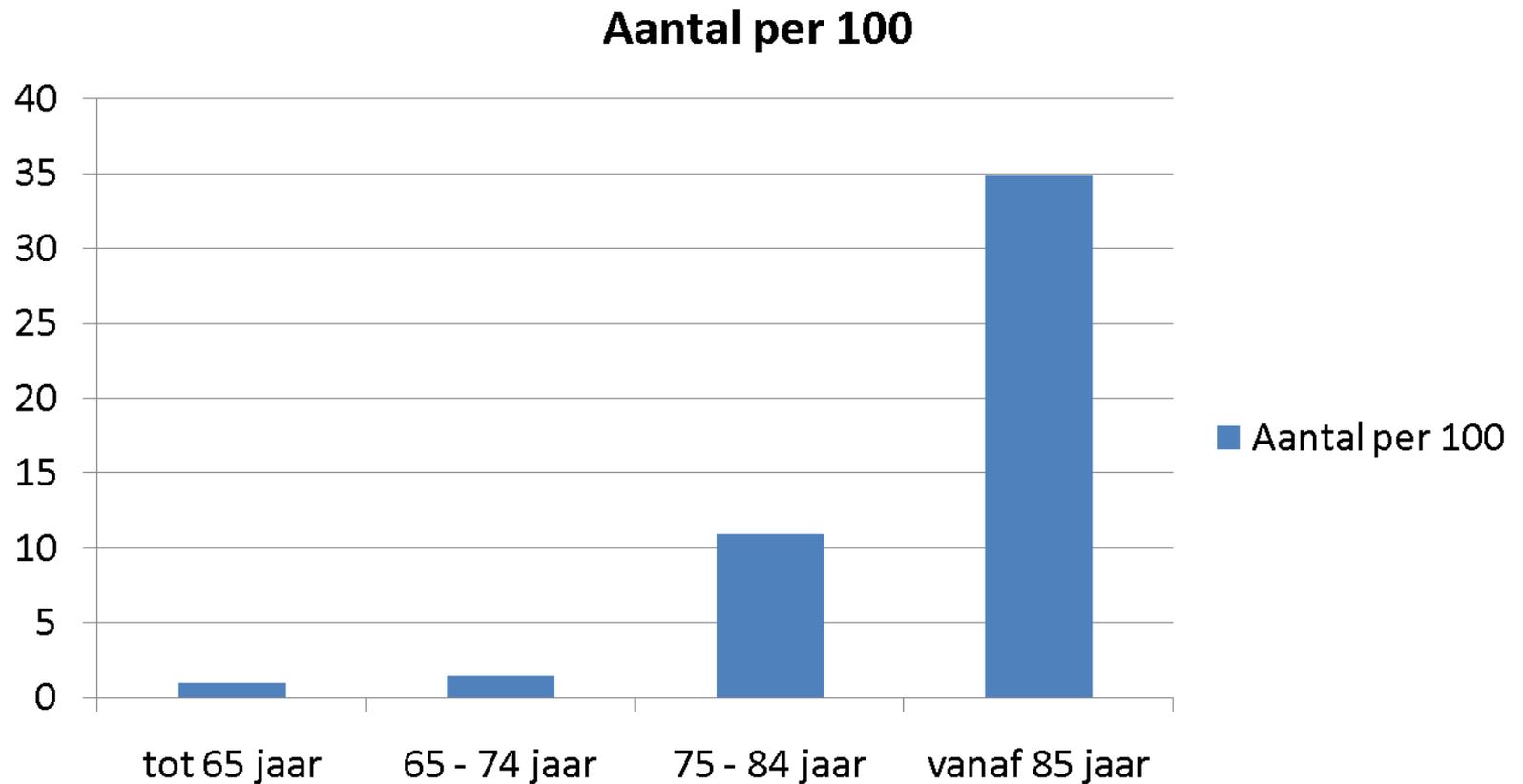
Persons living with dementia

- Doubles every 20 years
- 2015: 46.800.000 persons // 2030: 74.700.000 persons // 2050: 131.500.000 persons

Where?

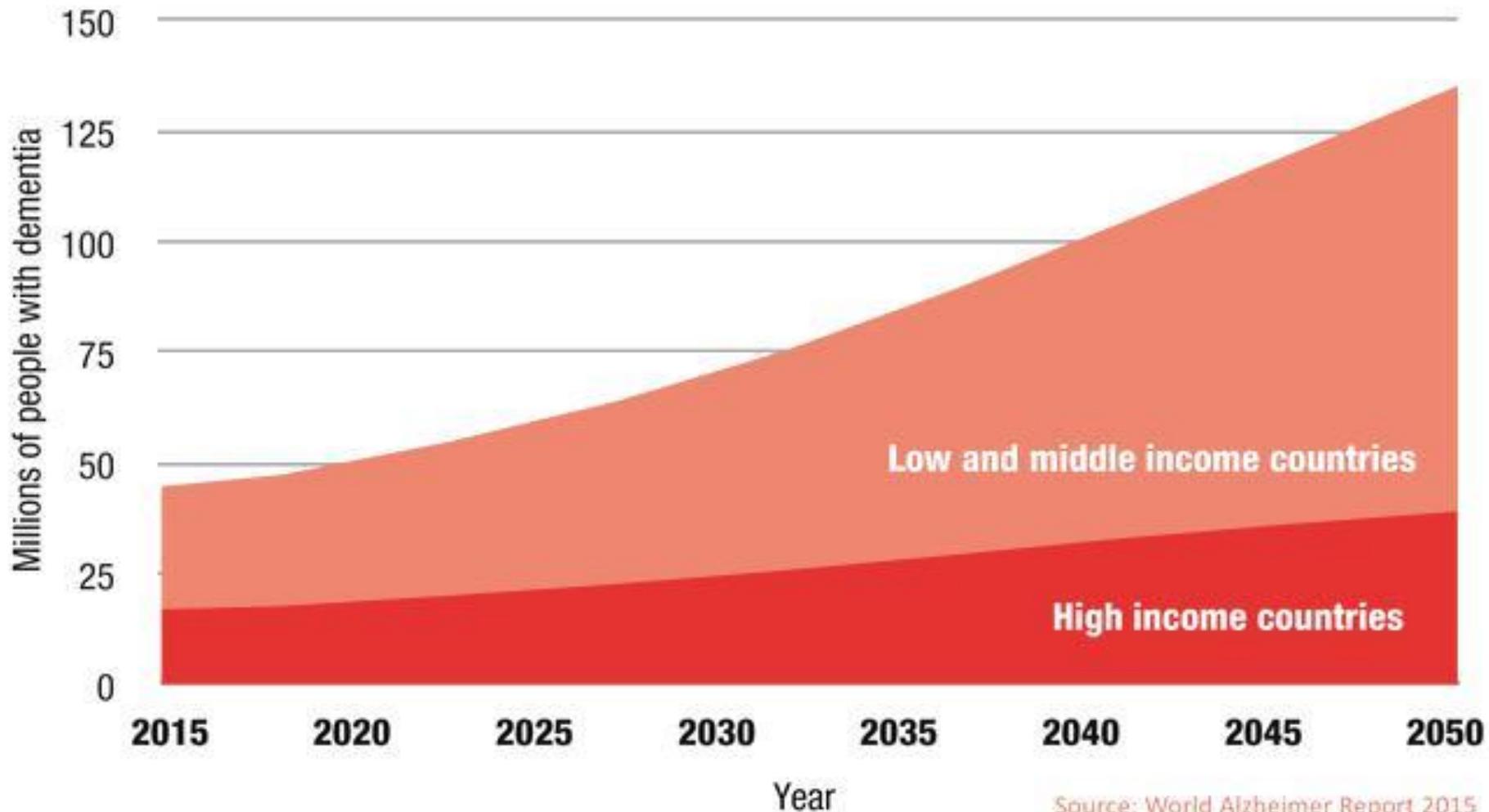
- 58% is living in low- and middle income countries, by 2050: 68%
- Fastest grow: China, India and their south-east Asia

Dementia and age



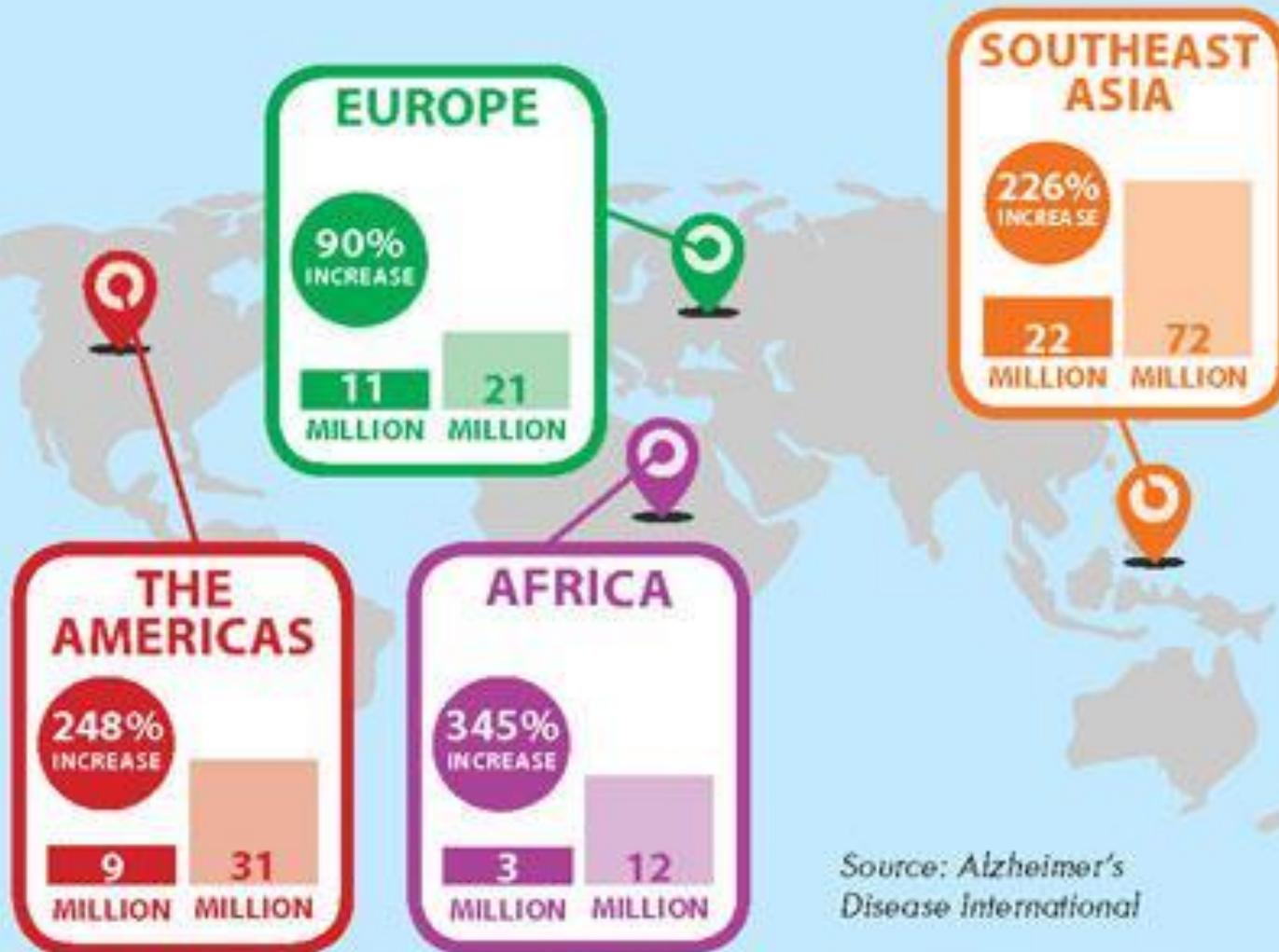
bron: www.nationaalkompas.nl (2005)

Number of people with dementia in low and middle income countries compared to high income countries



Source: World Alzheimer Report 2015

Growth in dementia cases by 2050



Source: Alzheimer's Disease International

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Dementia is a complex of symptoms

- ▶ Dementia is a brain disorder that creates cognitive disorders.
- ▶ Cognitive disorders are divided in :
 - Memory loss
 - Aphasia
 - Apraxia
 - Agnosia
 - Disorders in executive functions
 - Attention/concentration disorders
 - Disorders in mental processing speed
 - Disorders in visual-spatial skills

Dementia is a complex of symptoms

- ***Memory disorder***: a diminished ability to learn new information or remembering information that was already learned.
- ***Aphasia***: being unable to express oneself verbally or in writing and / or unable to understand spoken or written language.
- ***Apraxia***: not being able to execute deliberately planned actions.

Dementia criteria

(Diagnostic Statistical Manual of Mental Disorders, DSM-IV)

1. Memory impairment

1. One or more of following cognitive disorders:

- Aphasia
- Apraxia
- Agnosia
- Disorder in executive functions

1. The cognitive deficits in 1 and 2 each cause significant impairment in social or occupational functioning and

Other symptoms

Besides cognitive impairment there may be other symptoms:

“Behavioral and psychiatric symptoms in dementia”
(BPSD)

Early phase: irritability, agitation, depression, fear

Later phases:

Intermezzo: sleeping disturbances

Normal age-related changes

- Prolonged sleep latency
- Diminished sleep efficiency
- More arousals during the night
- Earlier waking up

Sleeping disorders in elderly?

Prevalence sleeping problems: 35%

Prevalence in depression: 70%

Insomnia ~ higher mortality

Sleeping disorders in dementia

- More frequent and longer arousals – awake for 40% of the night
- Being awake leads to wandering, unrest, shouting
- Daytime sleeping, changes in sleep-wake cycle
- ‘Sundowning’

Intermezzo: delusions and hallucinations

Delusion

- ▶ Disorder in the content of thinking
- ▶ Somebody is convinced of the accuracy of their own thought(s), even if they don't stroke with reality

Hallucination

- ▶ Experiences and observations
- ▶ For the person involved it is the reality, for the environment it is not

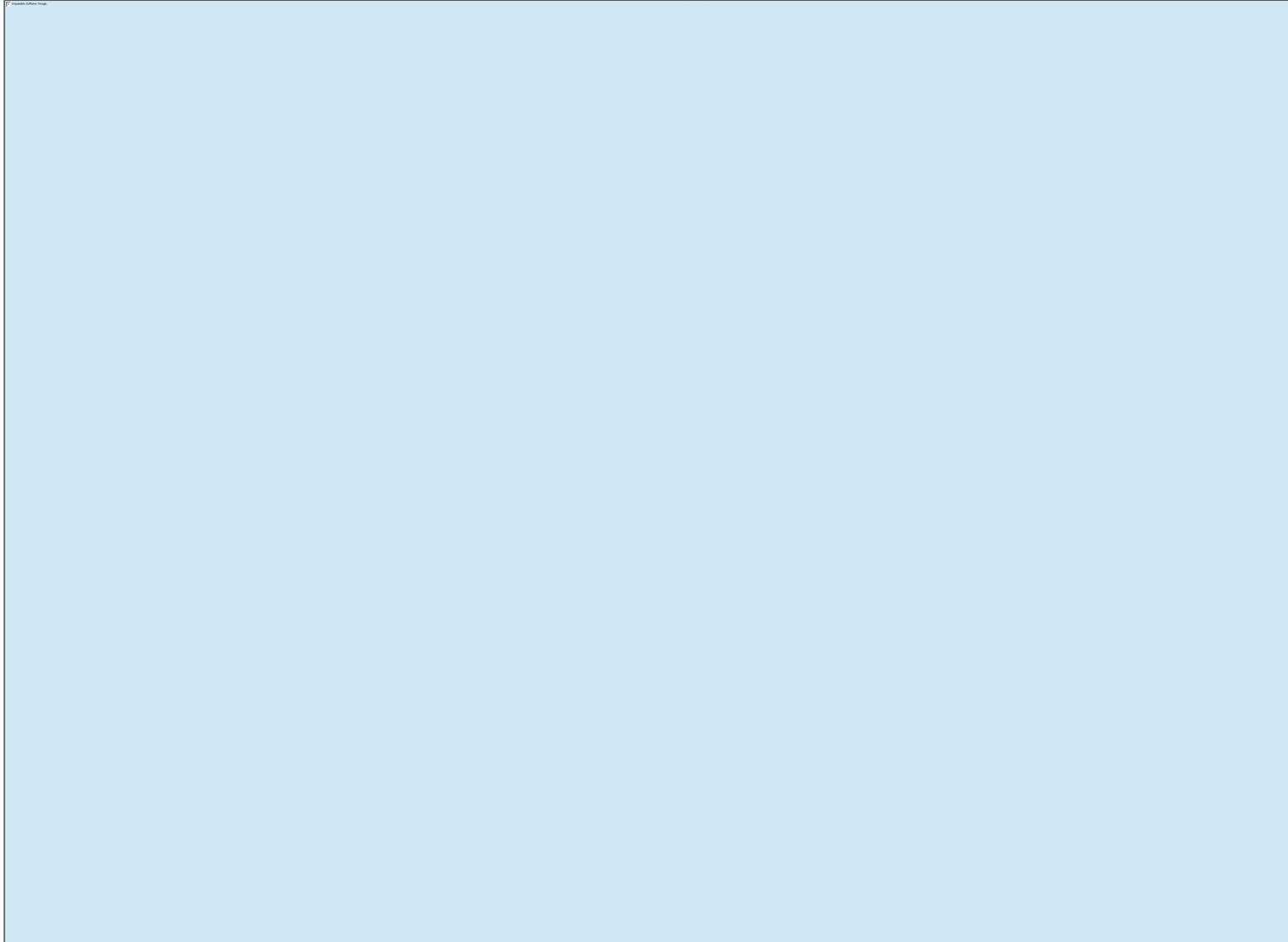
DSM-IV versus DSM-5

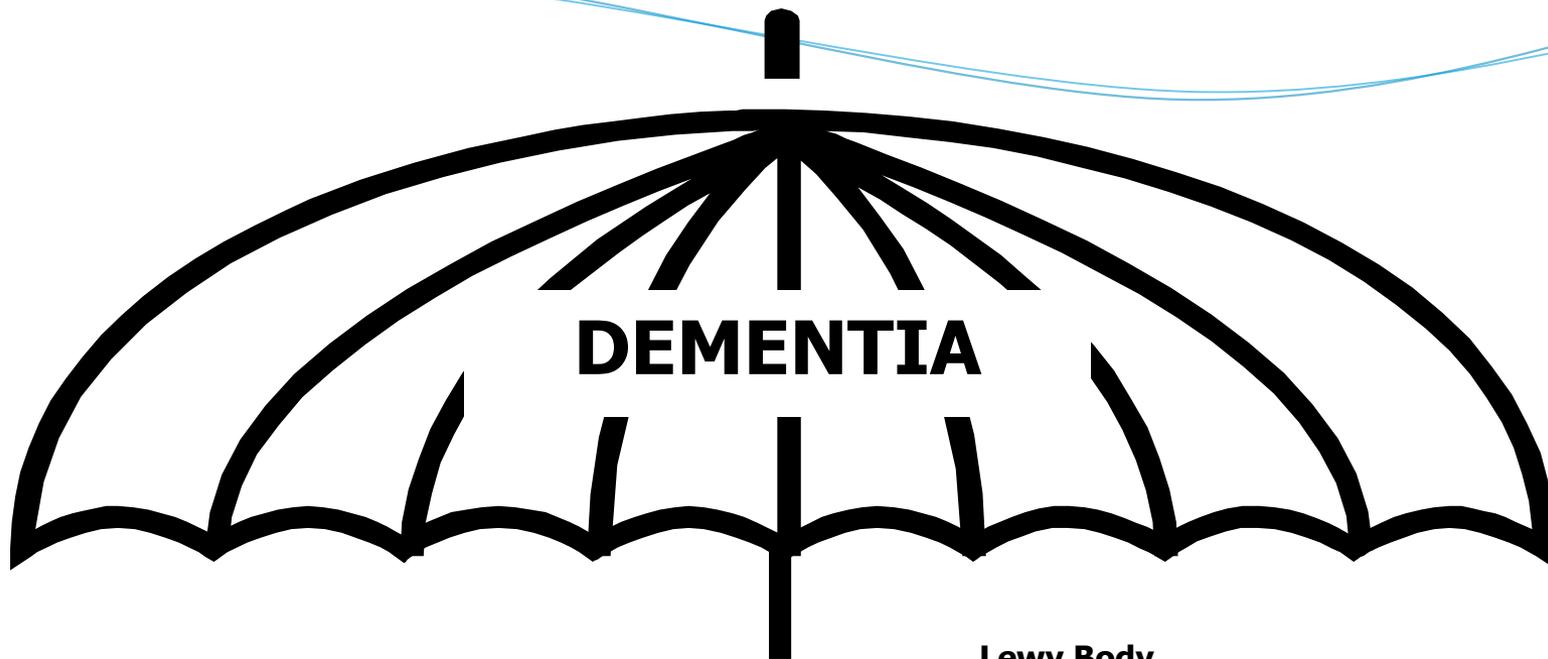
- ▶ DSM-5 (2013) has replaced the term 'dementia' by the term 'neurocognitive disorder'.
- ▶ In practice: still a lot of use of both DSM-IV and 'dementia'
- ▶ Important:
 - Memory impairment is no longer a criterium for having a 'neurocognitive disorder'
 - The memory is not always the first afflicted
 - E.g. primary progressive aphasia

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Dementie: DSM-IV versus DSM-5





DEMENTIA

Alzheimer's Disease

- .Early - Young Onset
- .Normal Onset

Vascular Dementias (Multi-infarct)

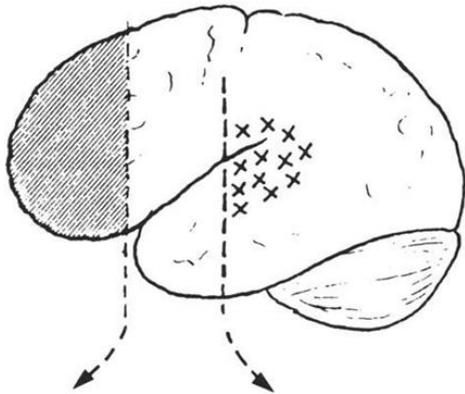
Lewy Body Dementia

Fronto-Temporal Lobe Dementias

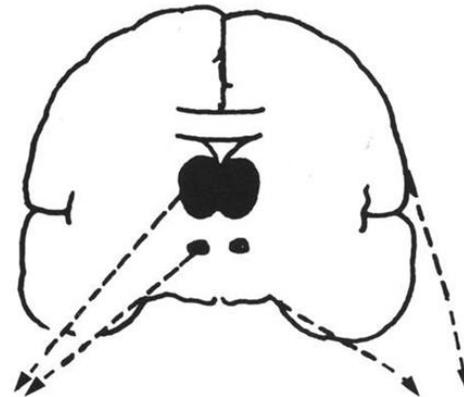
Other Dementias

- .Genetic syndromes
- .Metabolic pxs
- .ETOH related
- .Drugs/toxin exposure
- .White matter diseases
- .Mass effects
- .Depression(?) or Other Mental conditions
- .Infections – BBB cross
- .Parkinson's

Classification according to localization



or



Anterior

(Frontal premotor cortex)



Behavioural changes/loss of inhibition, antisocial behaviour, facile and irresponsible



e.g. Normal pressure Hydrocephalus
Huntington's chorea
Metabolic disease

Posterior

(Parietal and temporal lobes)



Disturbance of cognitive function (memory and language) without marked changes in behaviour



ALZHEIMER'S DISEASE

Subcortical

Apathetic
Forgetful and slow, poor ability to use knowledge
Associated with other neurological signs and movement disorders



e.g. **PARKINSON'S DISEASE**
AIDS DEMENTIA COMPLEX

Cortical

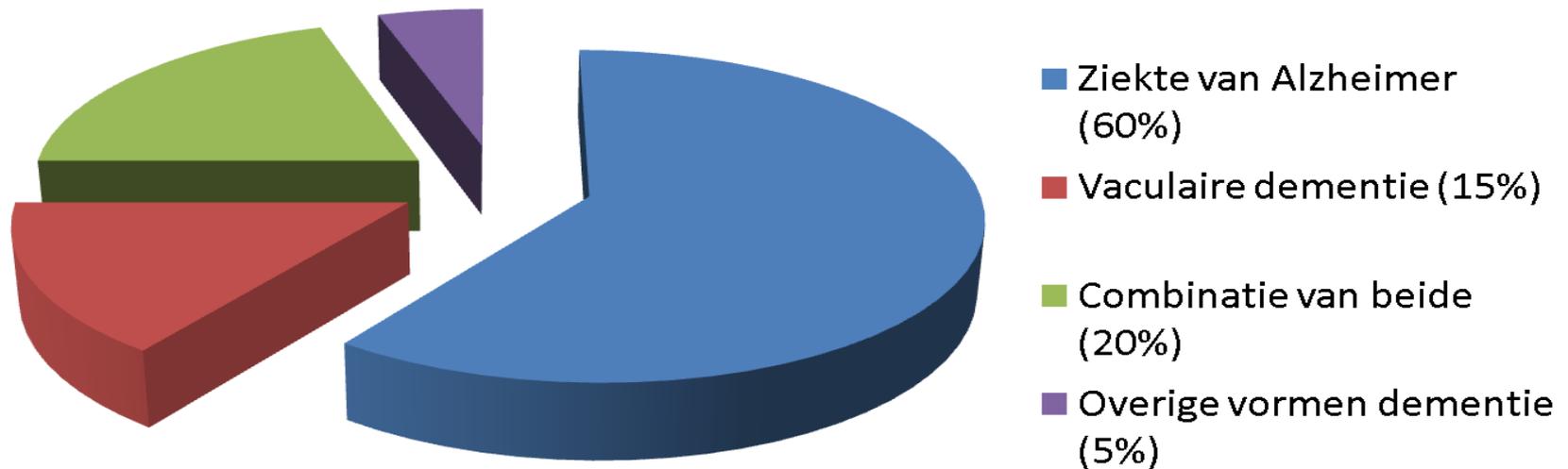
Higher cortical abnormalities
– dysphasia
– agnosia
– apraxia



e.g. **ALZHEIMER'S DISEASE**

Types of dementia

Verdeling



Better diagnostic techniques change the percentages!

Types of dementia

Most common – in order of appearance:

1. Alzheimer's
2. Vascular
3. Lewy body
4. Fronto-temporal

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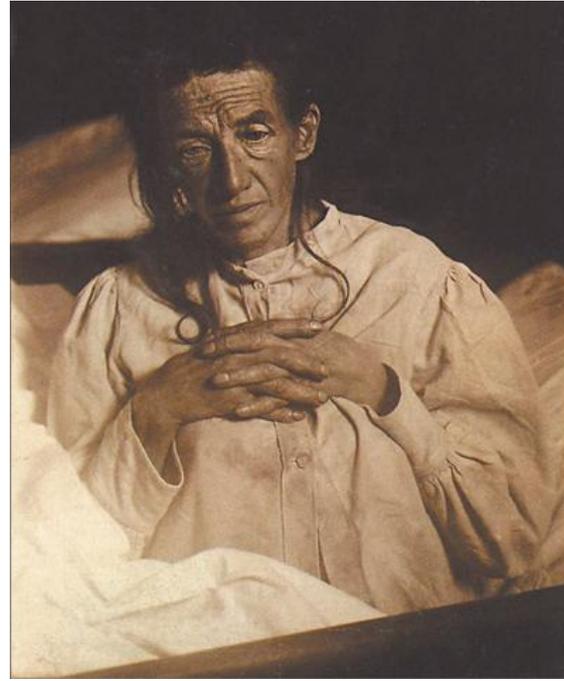
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Alzheimer's disease



Alois Alzheimer
1864-1915



Auguste Deter
1850-1906

Alzheimer's disease

Course of the disease:

- It is present for a long time before complaints arise
- Progressive
- 3 stages (mild – moderate – severe)

3 stages: stage 1

Mild

Forgetfulness (names/words, addresses, ...)

Most important deficit: recent memory

Deficits can be confirmed by neuropsychological testing

Sometimes patients are aware of their complaints, which can lead to fear, depression, denial

No clear physical disorder

3 stages: stage 2

Moderate

Important memory loss: close relatives – known routes/places

Changes in personality / behaviour

Self deprivation

Desorientation in time / space

Impossible to do simple tasks (getting dressed properly...)

3 stages: stage 3

Severe

Dysphagia with a disturbed speech – fragmented, unorganised

Agression, unrest, wandering

Hallucinations and delusions

Incontinence

7 stages - Reisberg

Course and stages of Alzheimer's/dementia

- Staging: Global Deterioration Scale//FAST Scale of Reisberg et al. (Am J Psychiat 1982;139:1136-1139, Psychopharm Bull 1988;24:653-659):
- 1: Normal (no cognitive decline)
- 2: Forgetfulness (very mild cognitive decline)
- 3: Forgetfulness (MCI; earliest clear cut deficits)
- 4: Late confusional (mild AD/moderate cognitive decline)
- 5: Early dementia (moderate AD/moderately severe decline)
- 6: Middle dementia (moderate to severe AD/severe cognitive decline)
- 7: Late dementia (severe AD/very severe cognitive decline).

FIRST STAGE

Normal adult.
No functional decline.



SECOND STAGE

Awareness of
some functional
decline



THIRD STAGE

Early Alzheimer's
Functions at about
12 years of age.



The Functional Assessment Staging (FAST) Scale

SEVENTH STAGE

Severe.
Functions at level
of a newborn.



SIXTH STAGE

Needs 24/7 care.
Functions at 2-4
years of age.



FIFTH STAGE

Needs help getting
dressed. Functions
at 5-7 years of age.



Mild Alzheimer's.
Requires some
assistance.

FOURTH STAGE

Alzheimer's Disease: Natural History

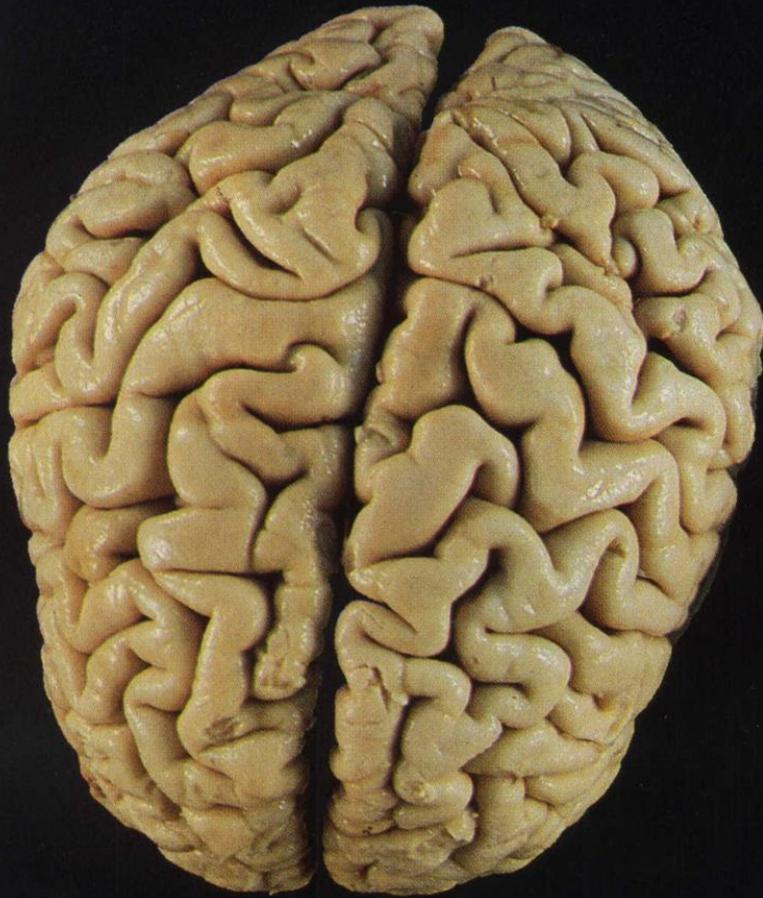


Adapted from Feldman H, Gracon S. Alzheimer's disease: symptomatic drugs under development. In: Gauthier S (ed). Clinical Diagnosis and Management of Alzheimer's Disease. Martin Dunitz: London, 1996:239_259.

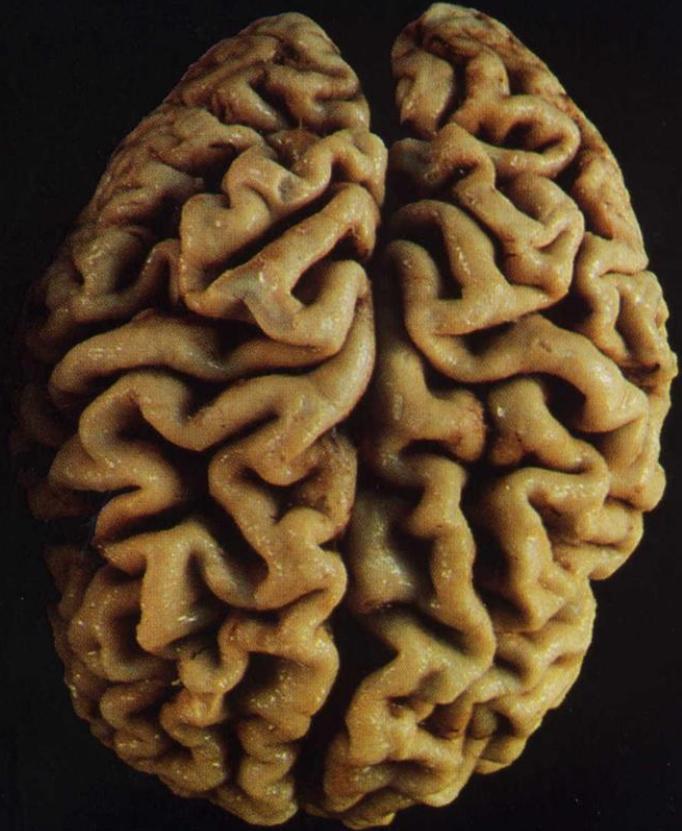
Alzheimer's

Pathology

- Beta-amyloid depositions (plaques)
- Tau-proteine depositions (tangles)
- Leads to neuron damage and neuron death

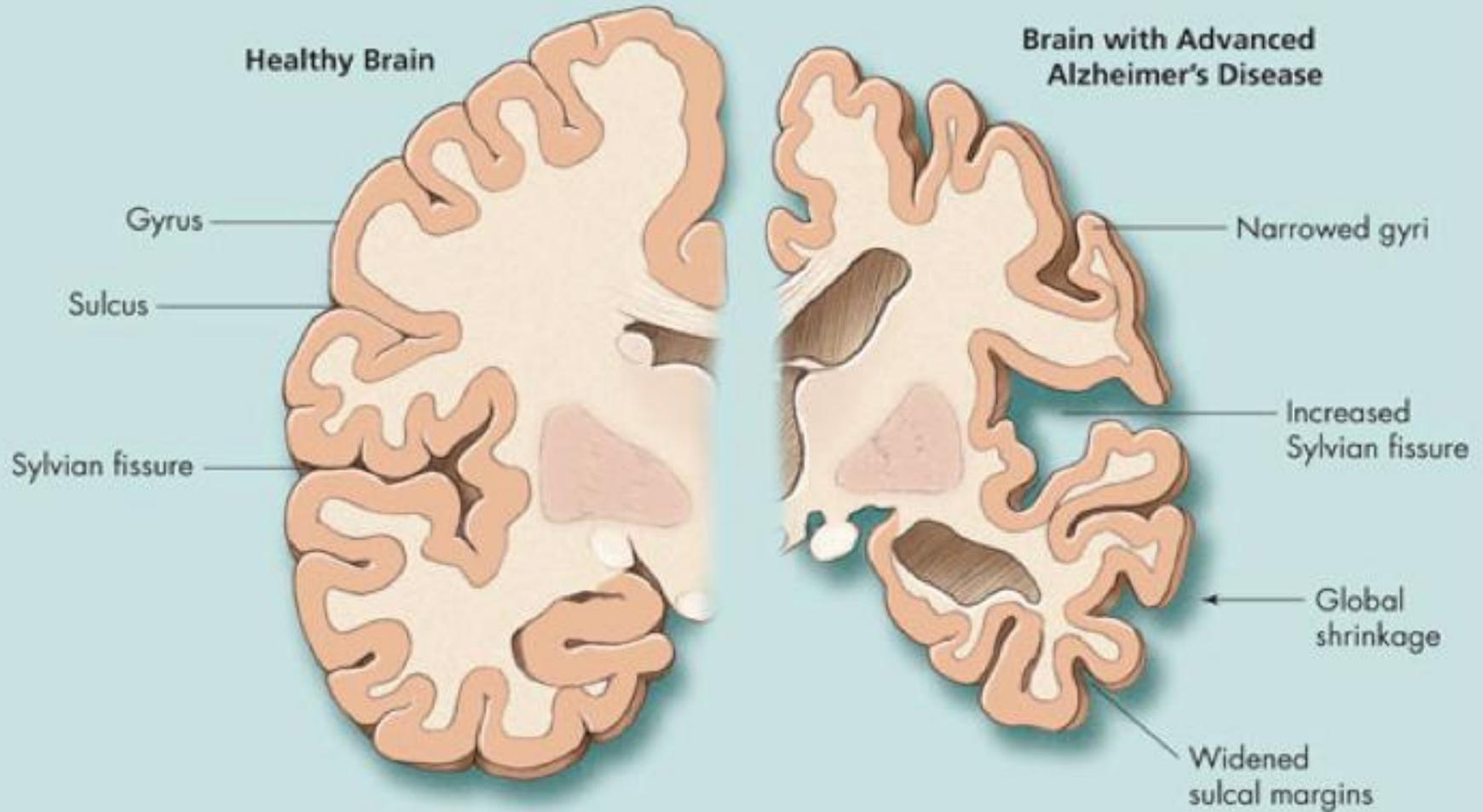


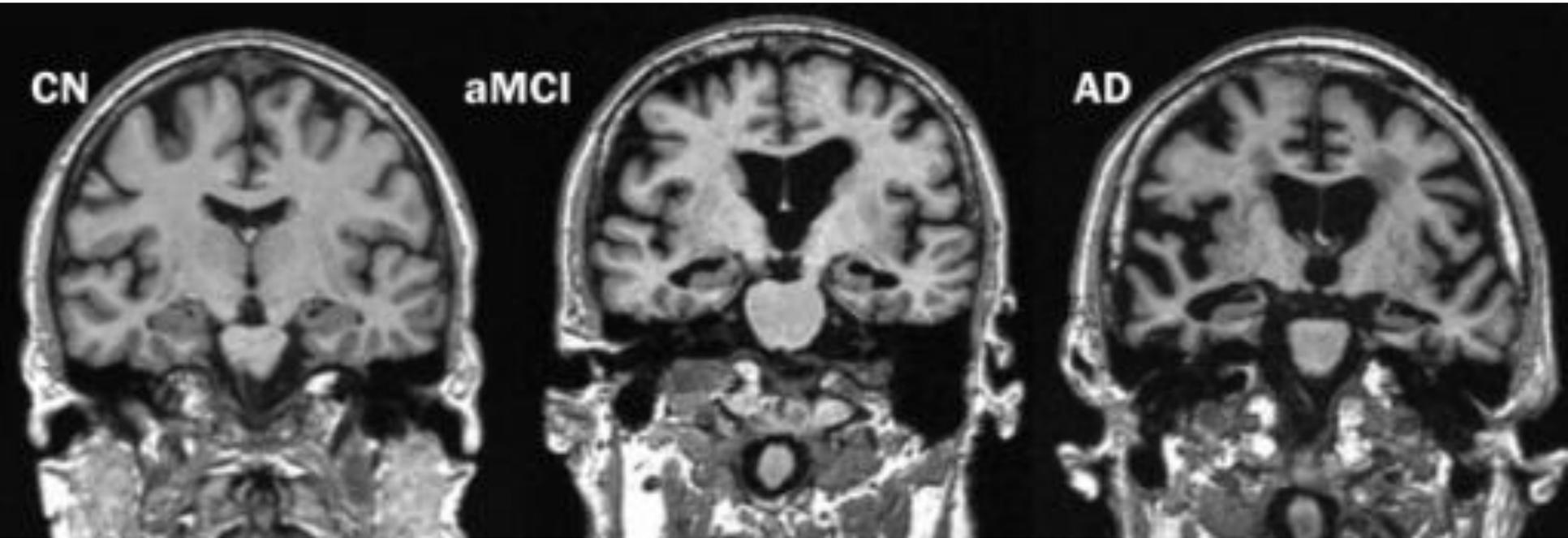
Normal

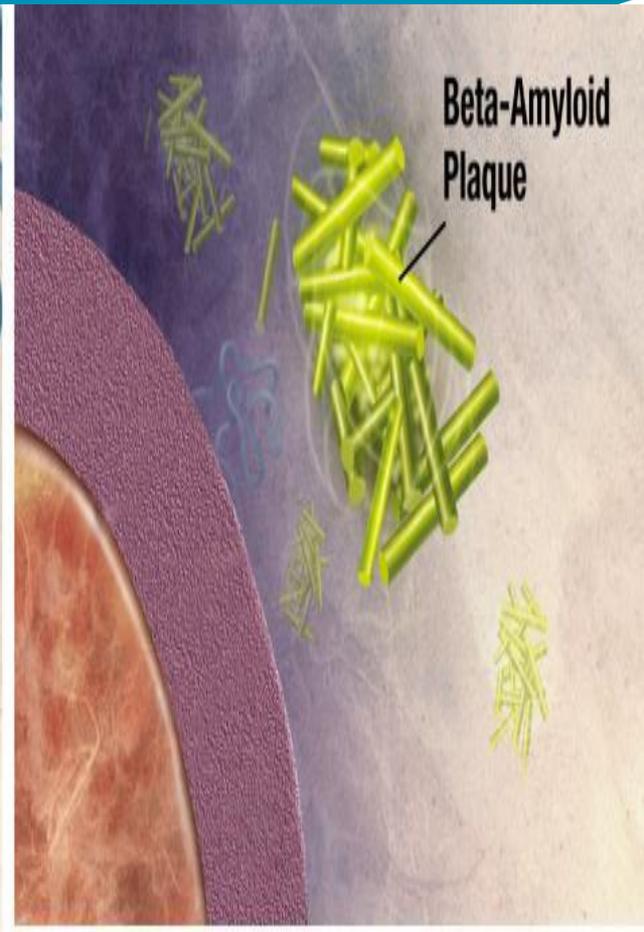
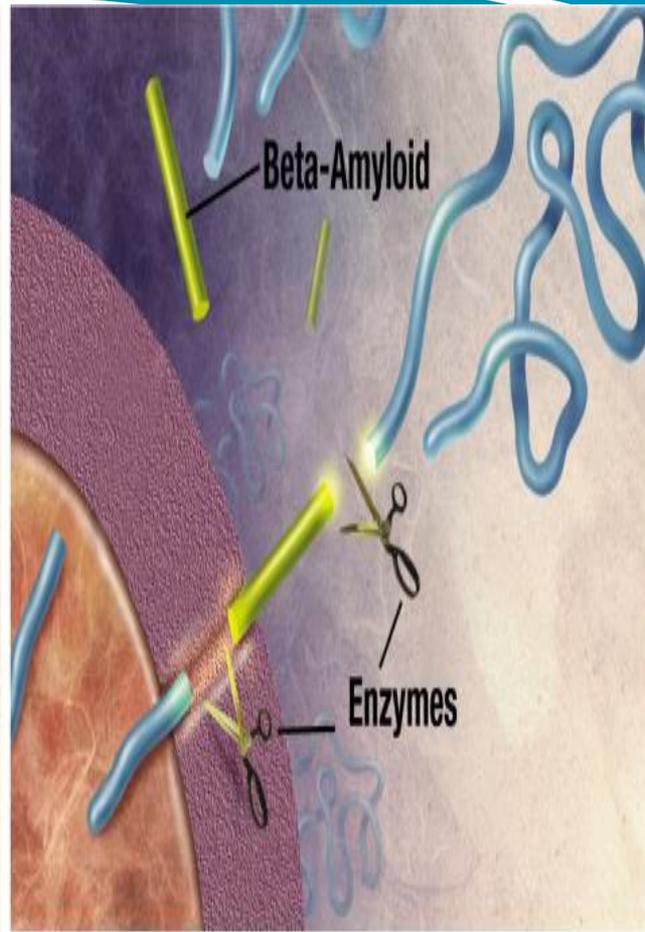
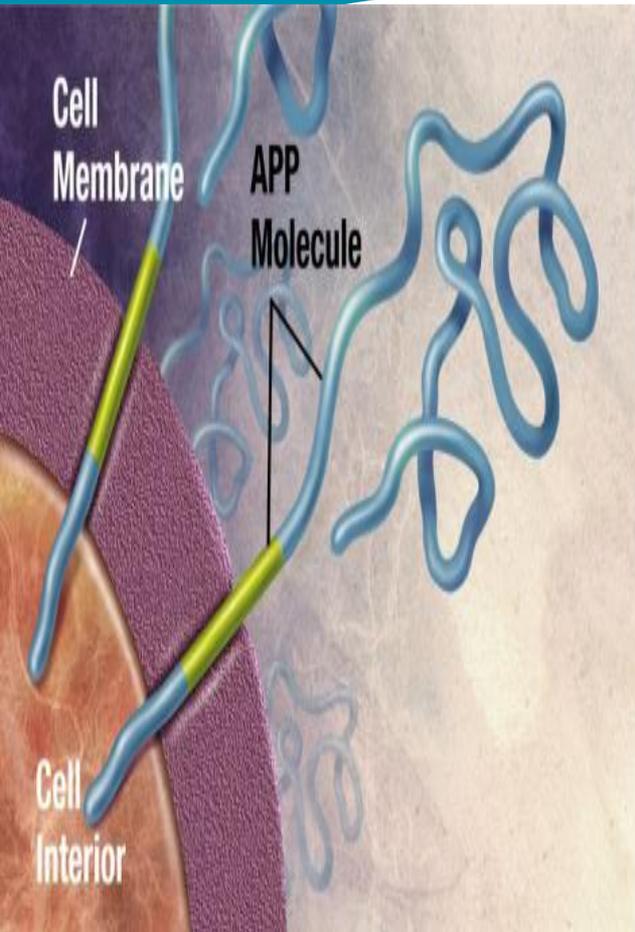


Alzheimer

Pathology of Alzheimer's Disease in the Brain



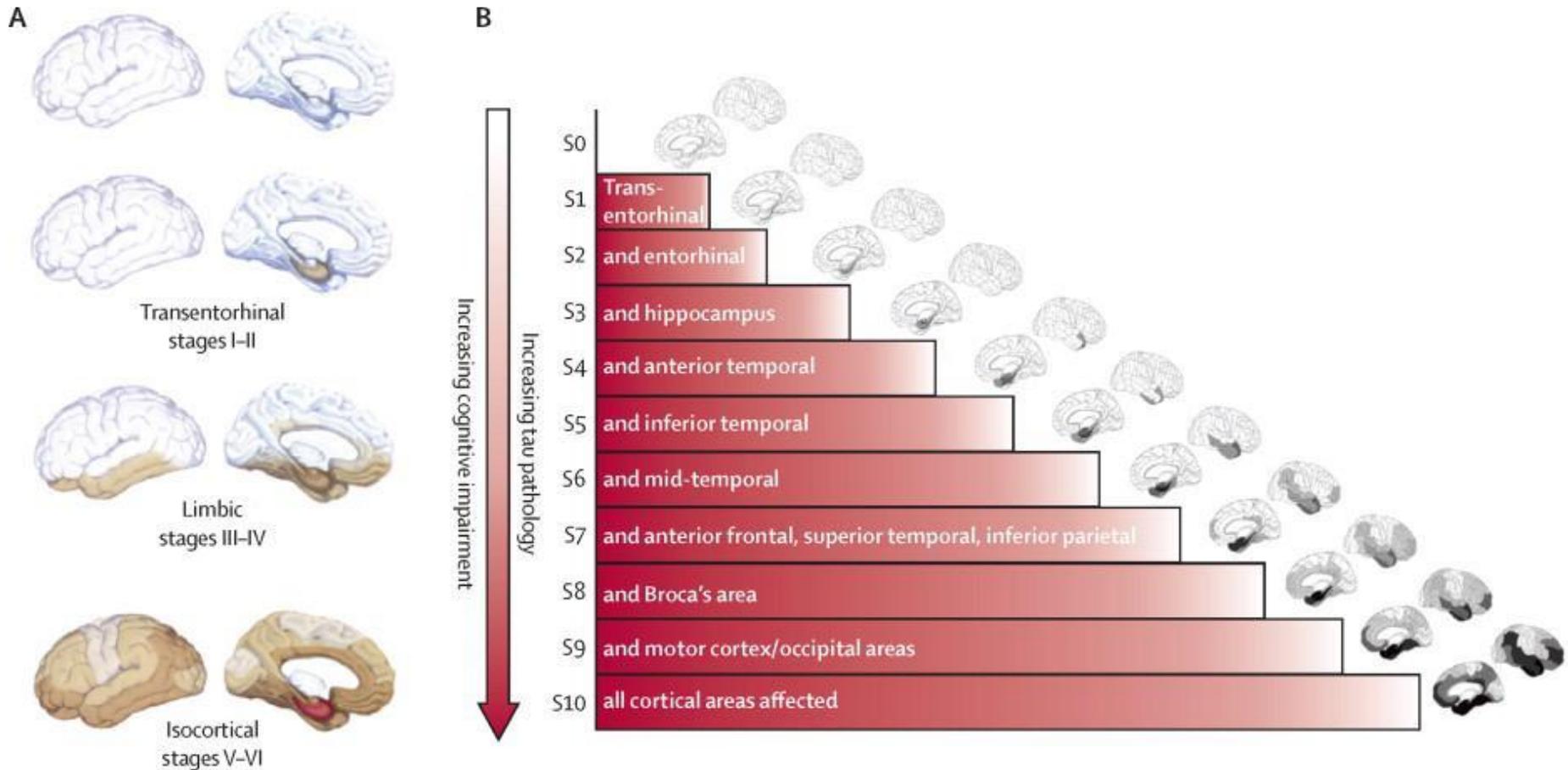




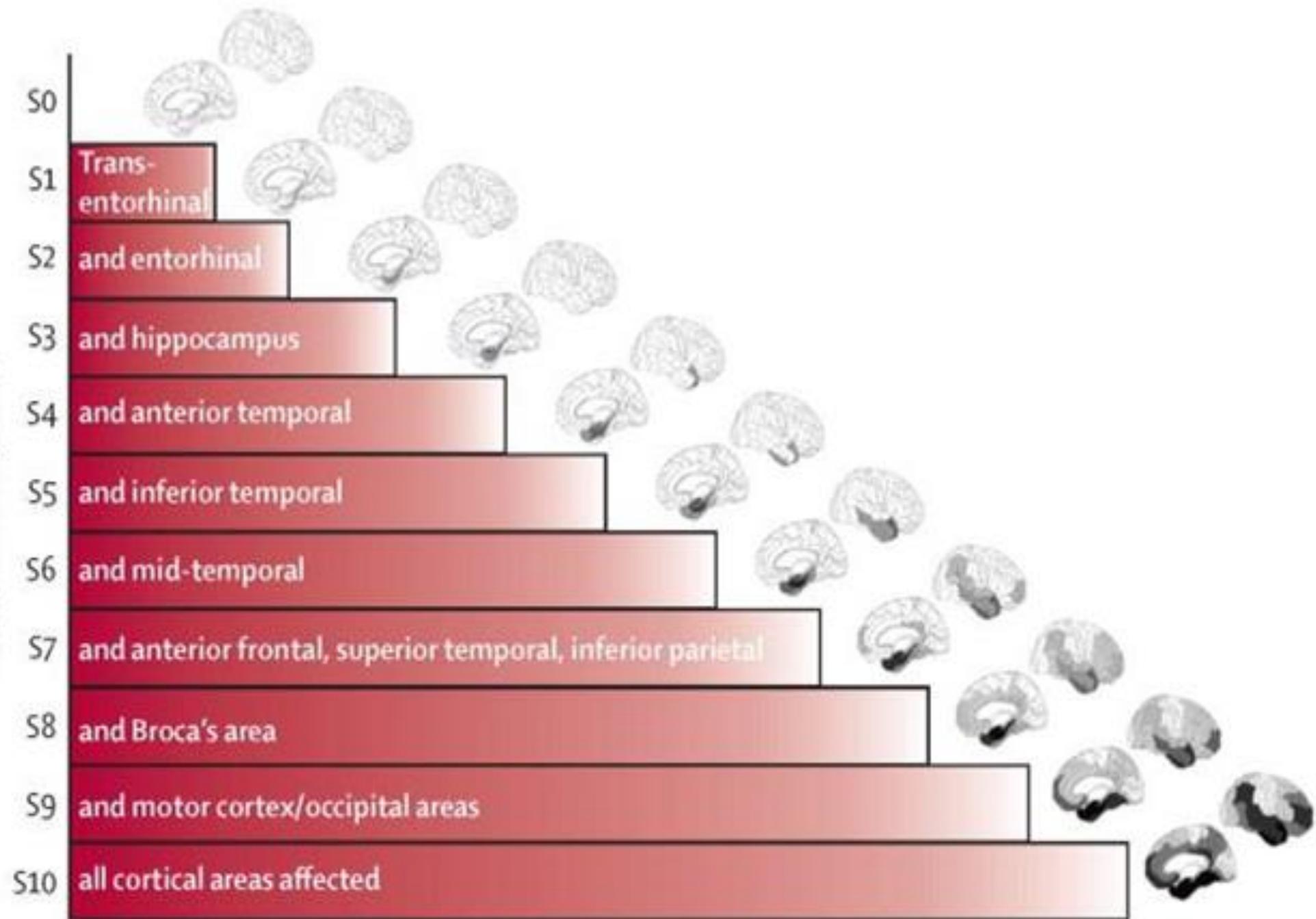
Amyloid precursor protein (APP) is necessary for cell growth, survival and repair after being damaged

Unclear why this is fragmented by enzymatic activity in Alzheimer's disease

Progression of tau deposition in AD

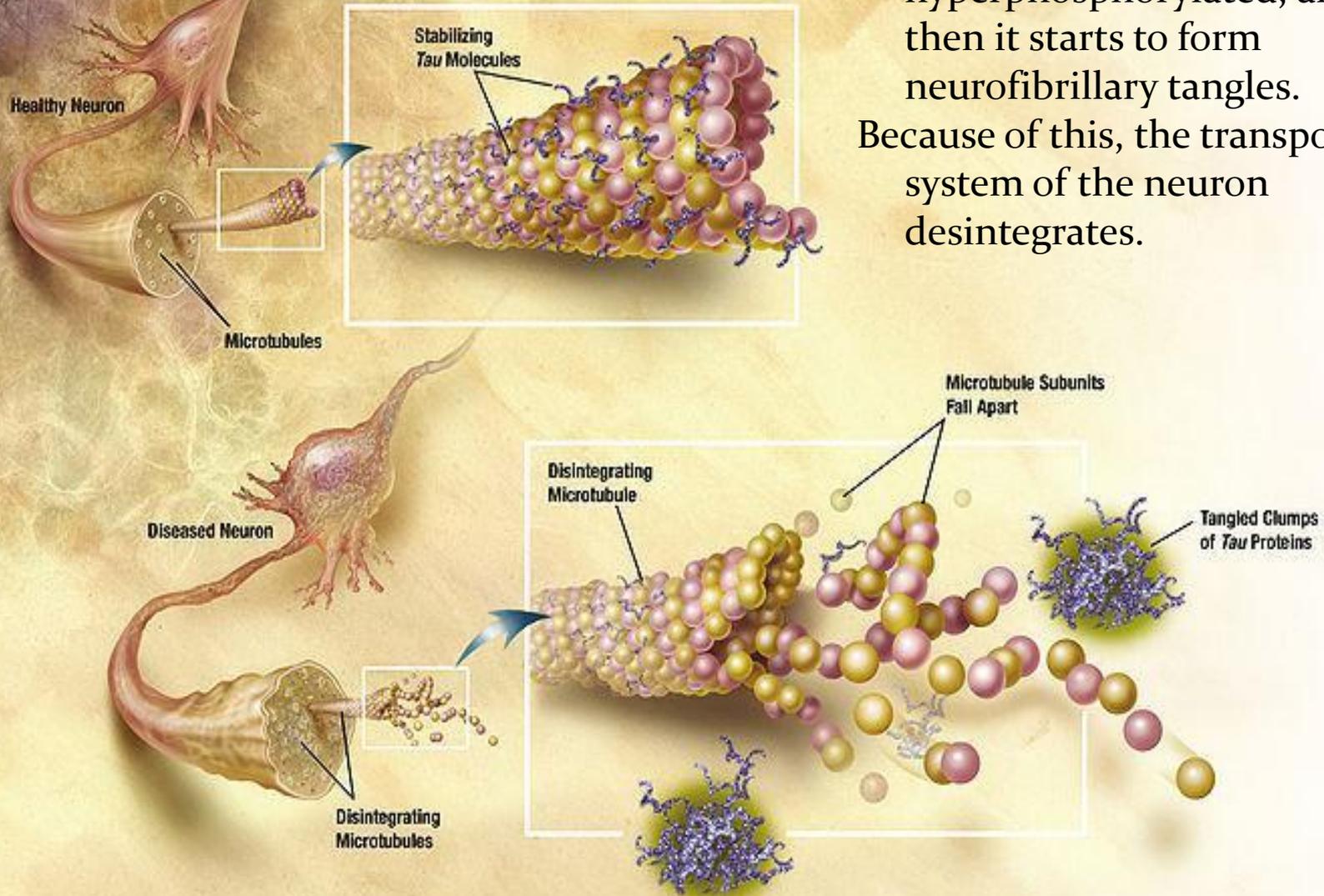


Braack and Braak (I-VI), and Delacourte staging (S1-10)

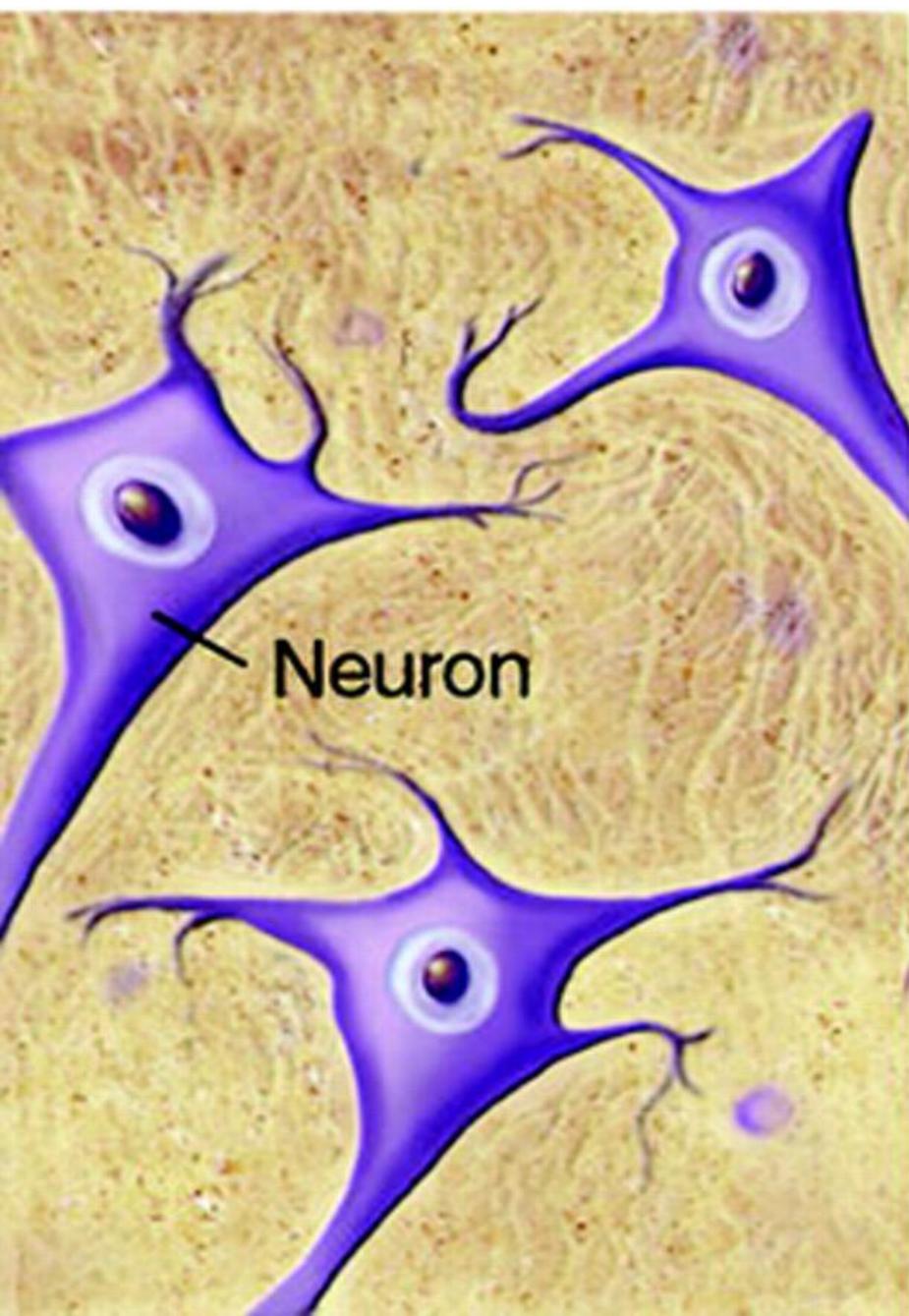


Tau protein stabilizes the microtubuli.

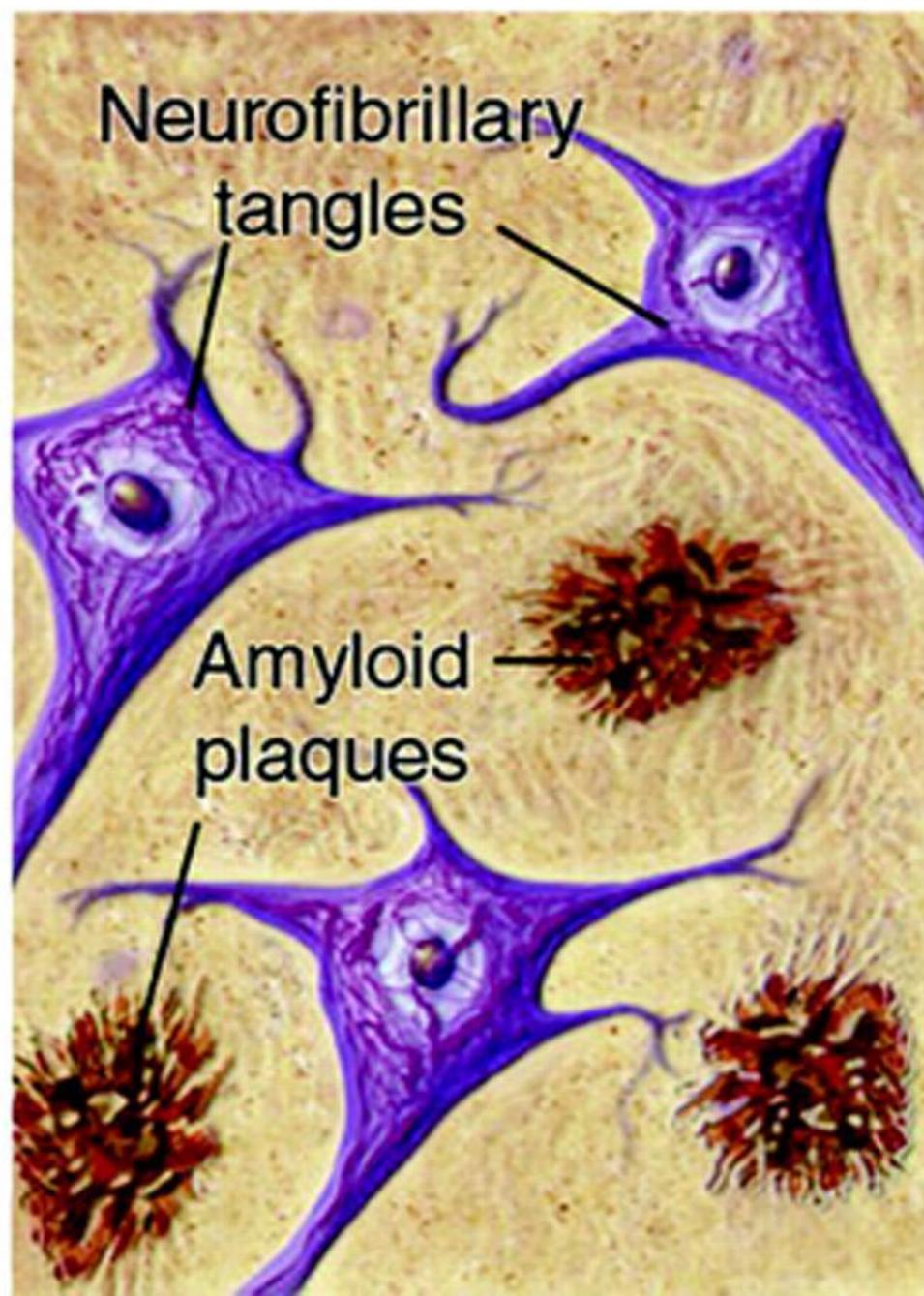
In AD, *tau* is hyperphosphorylated, and then it starts to form neurofibrillary tangles. Because of this, the transport system of the neuron desintegrates.

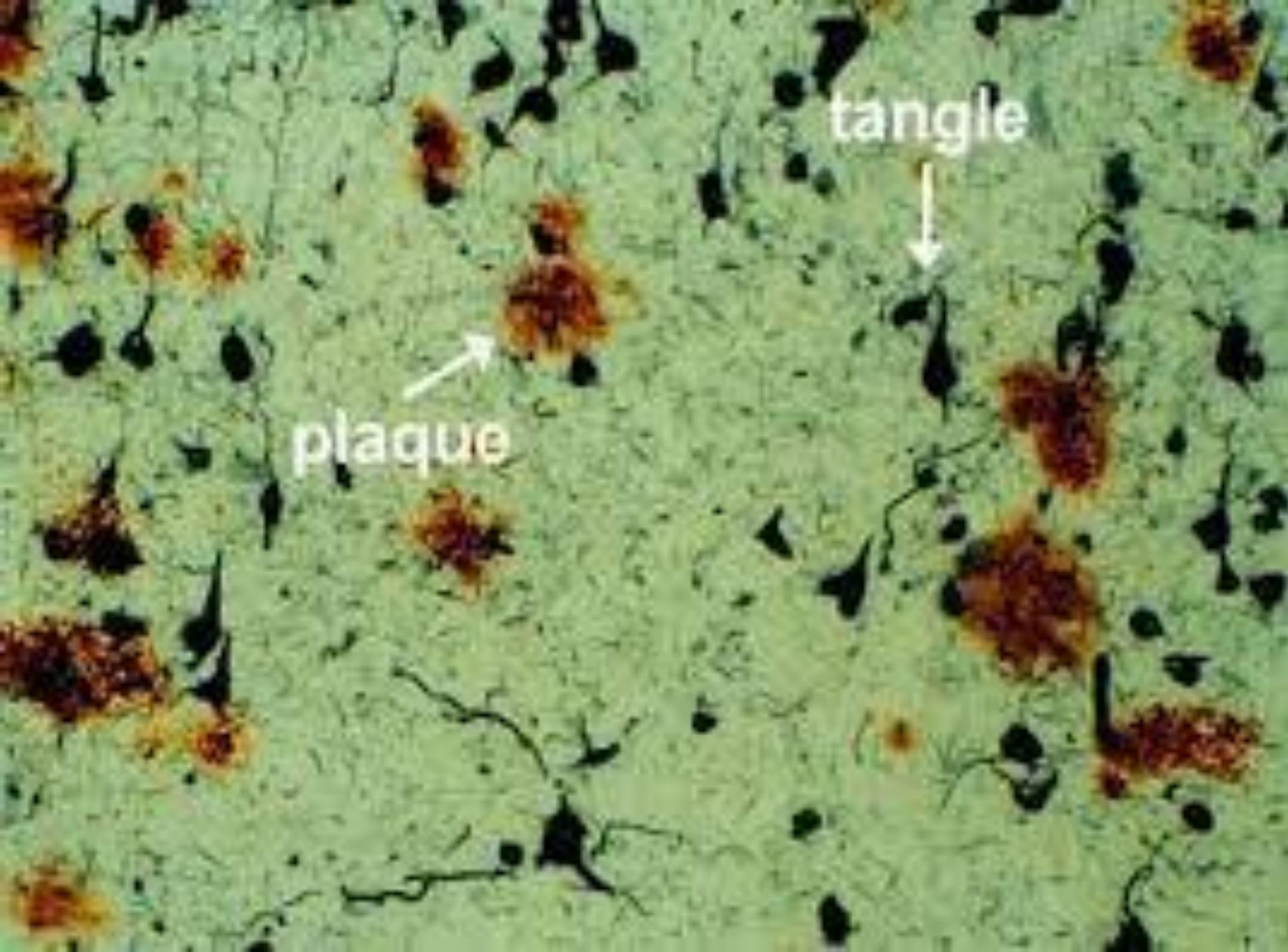


Normal



Alzheimer

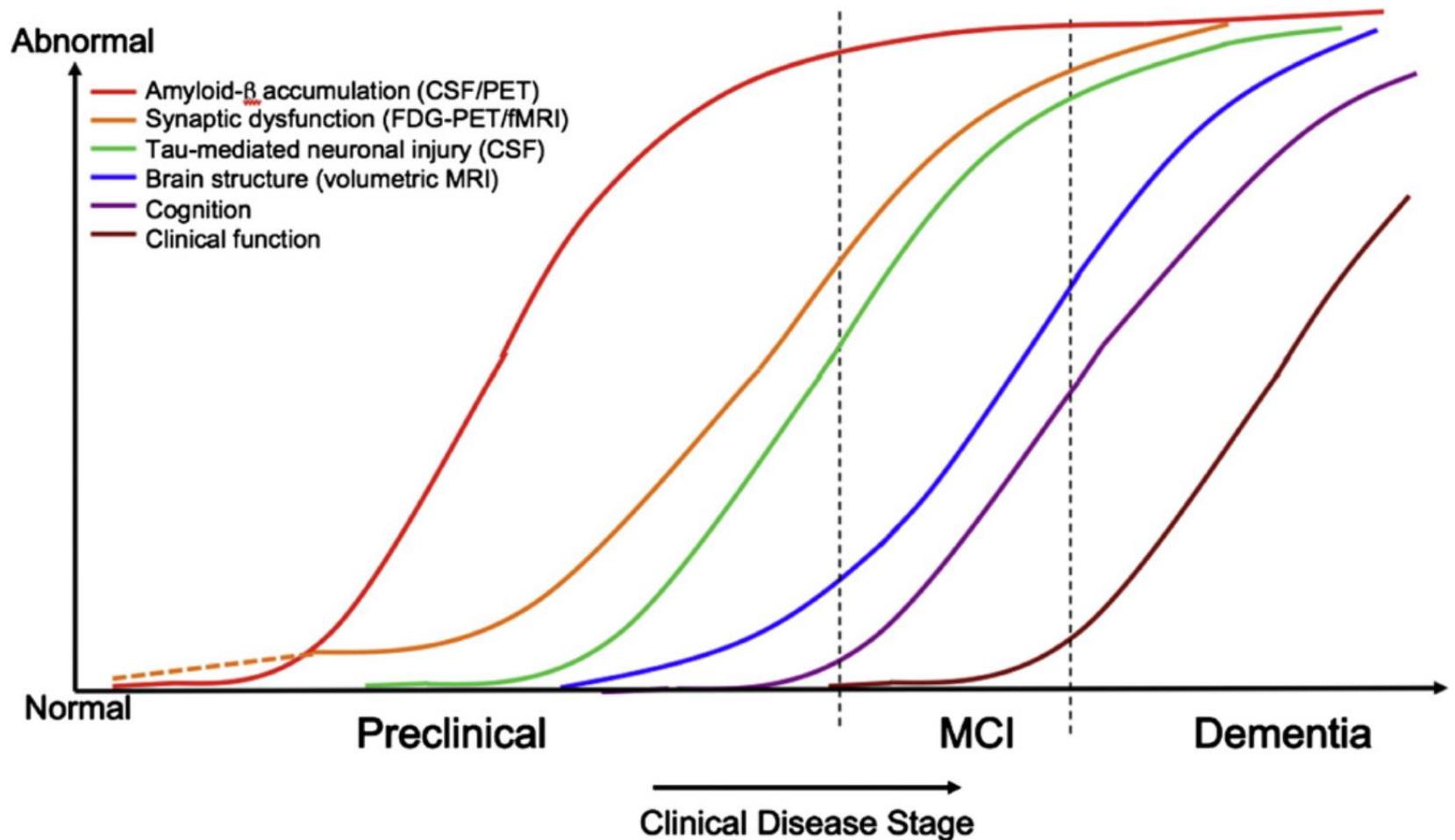




tangle

plaque

Model of the Dynamic Biomarkers of Alzheimer's Disease



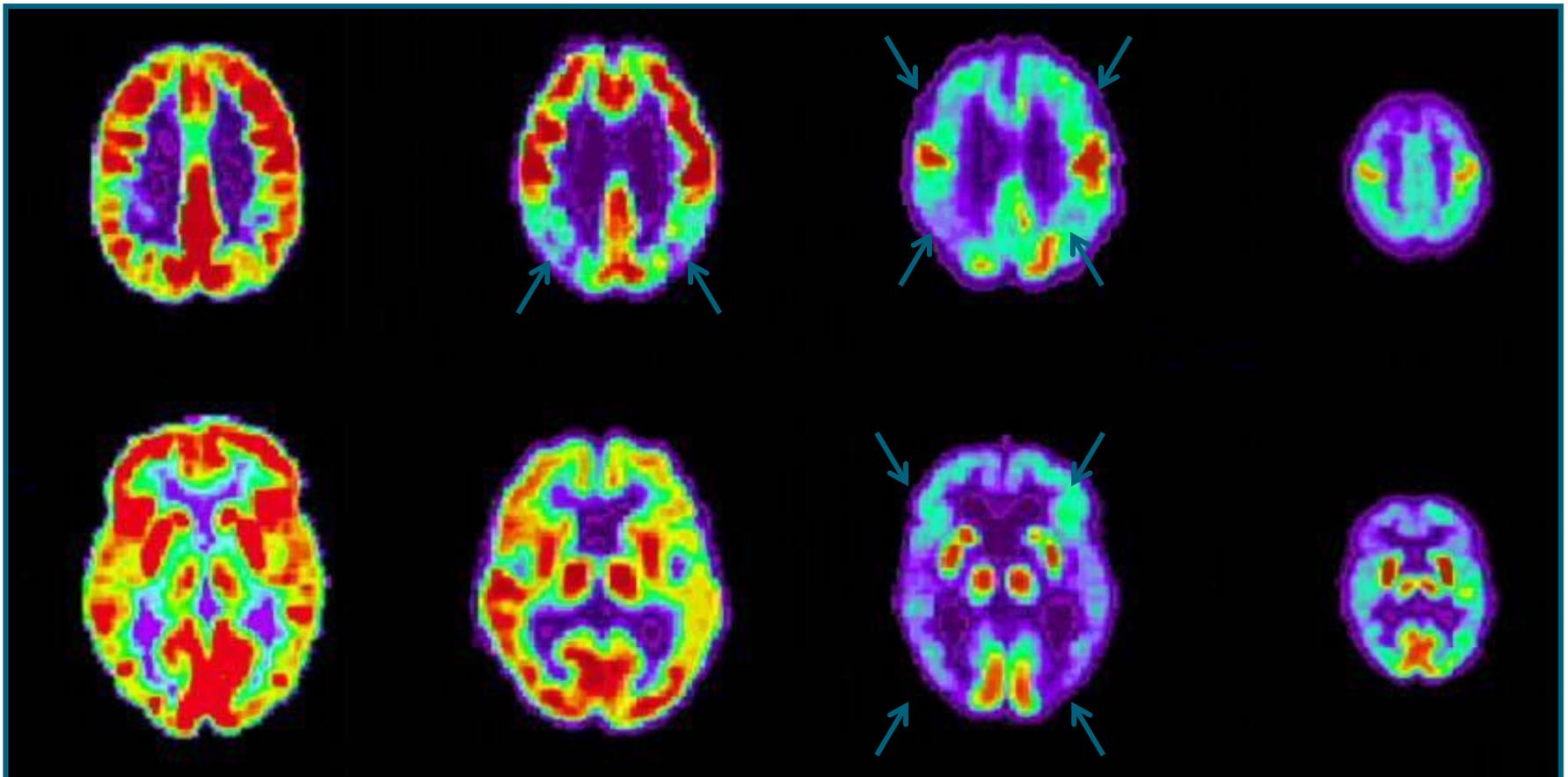
Positron Emission Tomography (PET)

Normal

**Early
Alzheimer's**

**Late
Alzheimer's**

Child



Alzheimer's disease: treatment

- Incurable, only stabilizing/slowing of the process
- Death after +/- 8 years after first symptoms
- Mild to moderate stages: cholinesterase inhibitors
- Prevents decline of acetylcholinesterase (ACh)
- Supports communication between neurons by keeping ACh levels higher
- Postponing symptoms 6-12 months in +/- 50% of patients

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Vascular dementia



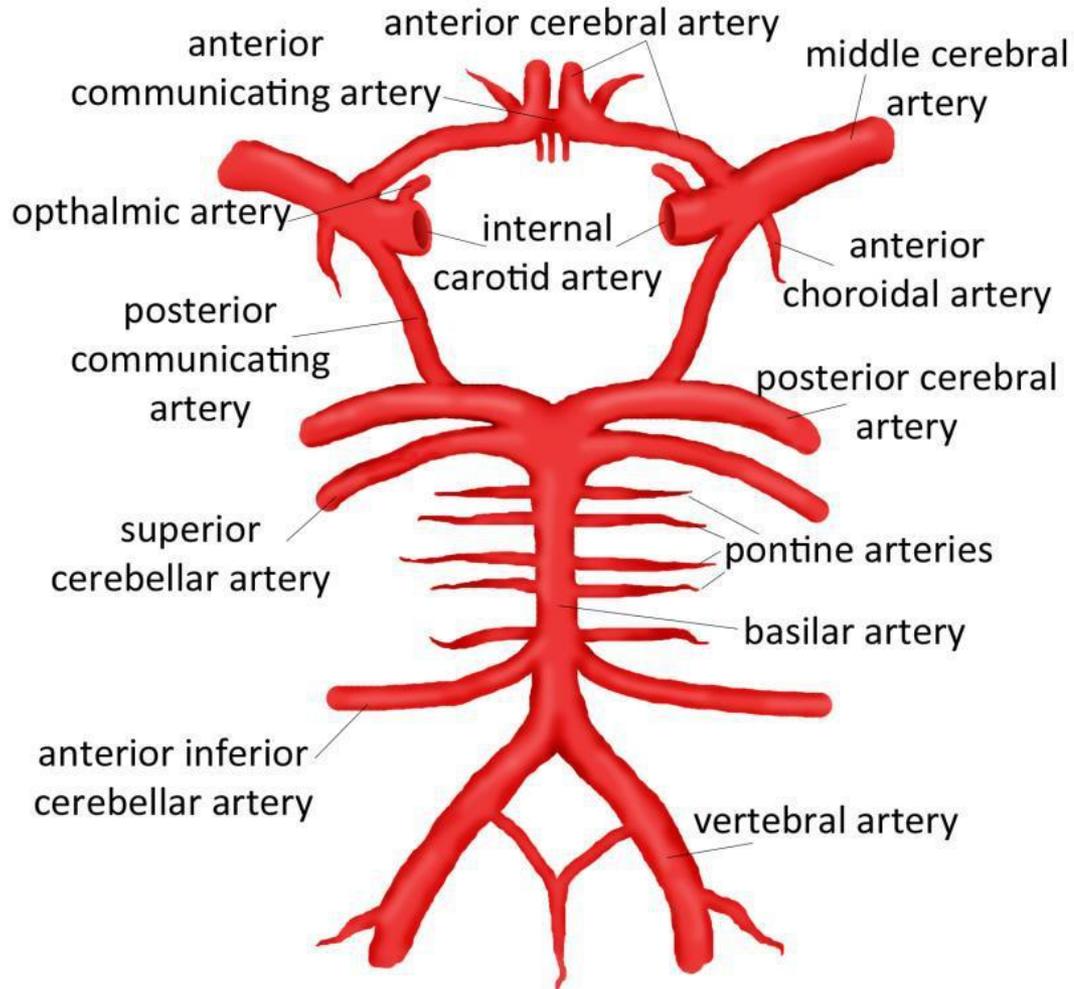
Multi-infarct dementia

Post-stroke dementia

Anatomy of the blood vessels

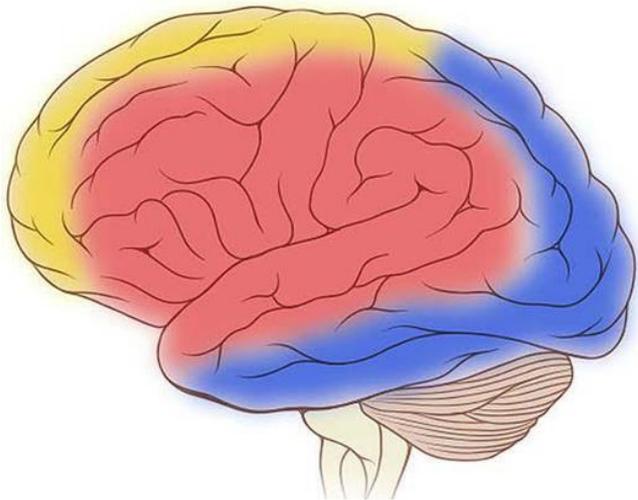


Anatomy of the blood vessels

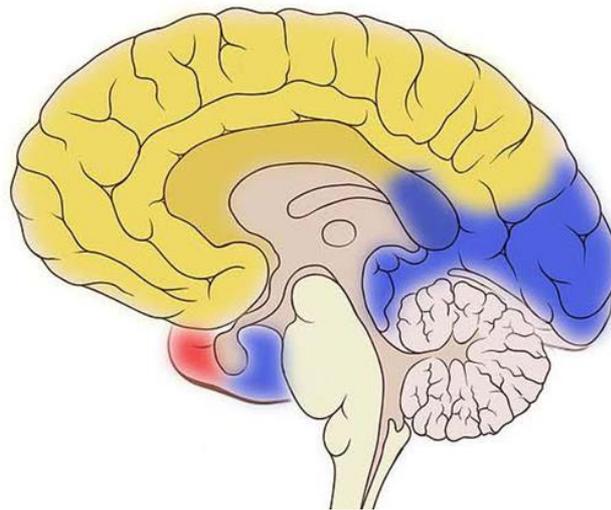


Anatomy of the blood vessels

Lateral Brain



Medial Brain



-  Anterior Cerebral Artery
-  Middle Cerebral Artery
-  Posterior Cerebral Artery

Vascular dementia

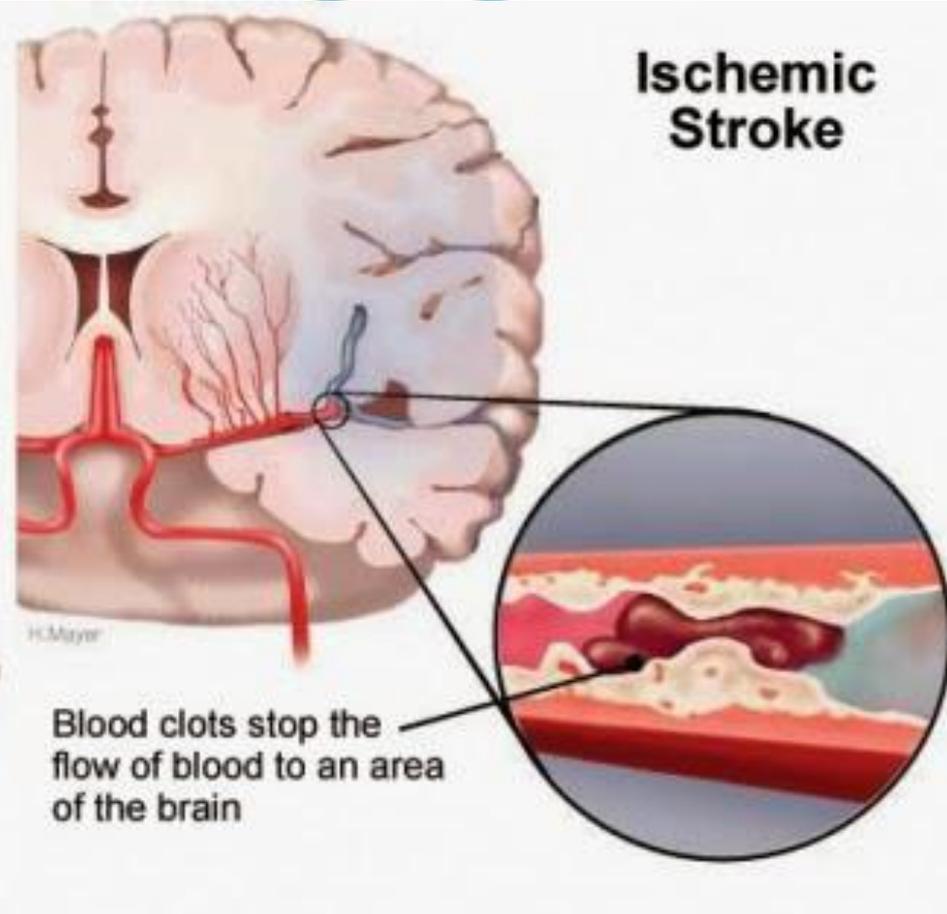
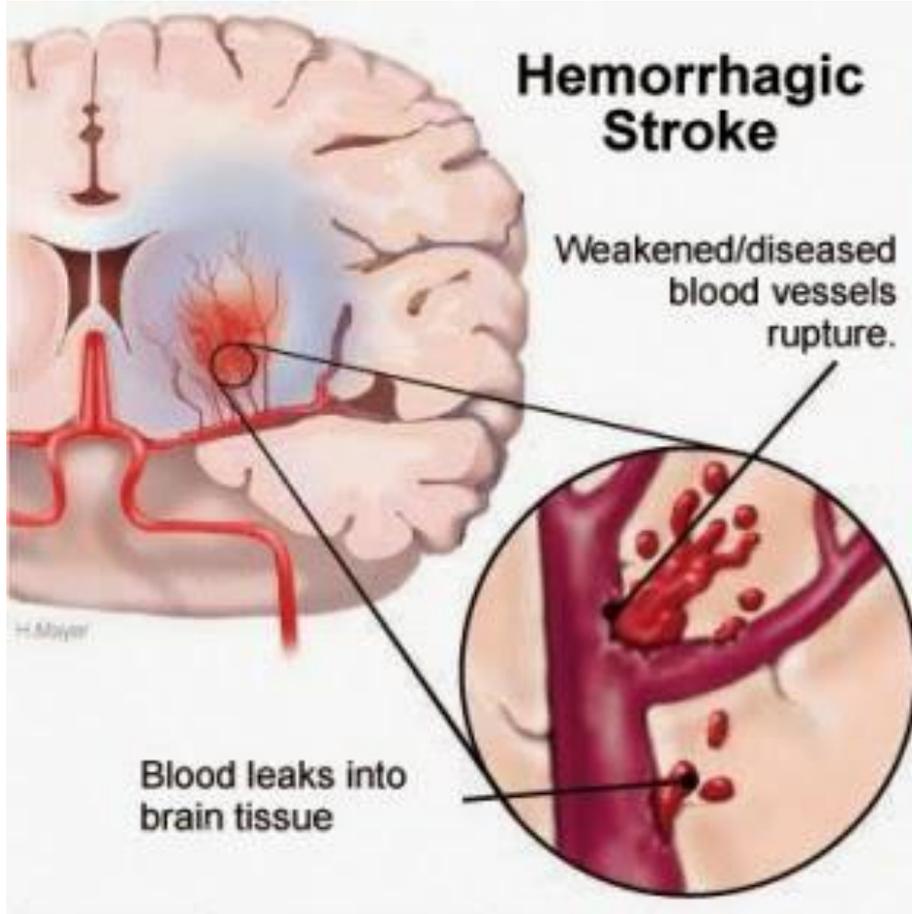
- Course of the disease
- Sudden onset
- Stepwise deterioration
- After each deterioration, partial recovery is possible
- Thinking process becomes slower and less flexible
- Persons are conscious of the decline

- Symptoms

Vascular dementia

Pathology

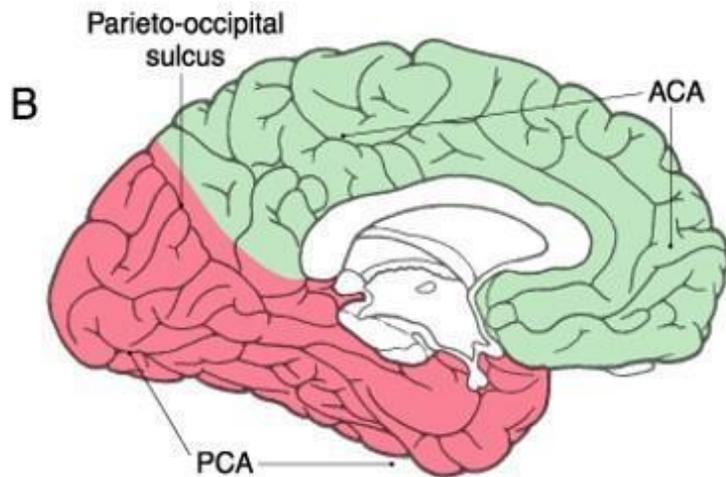
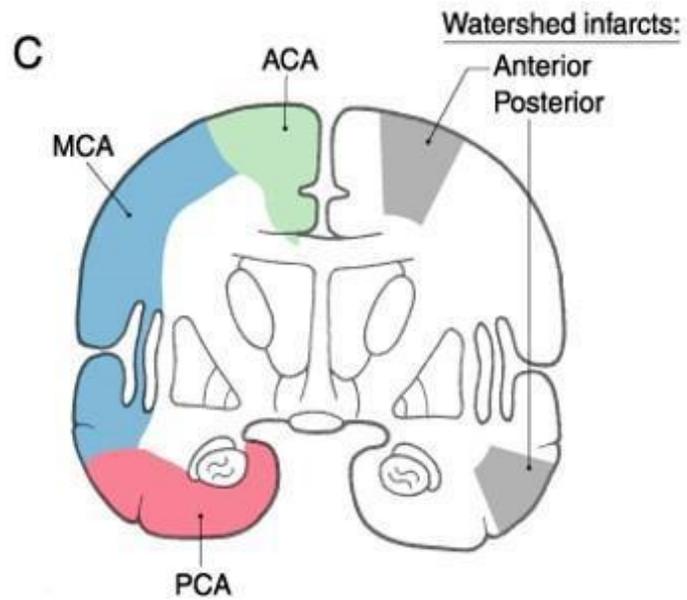
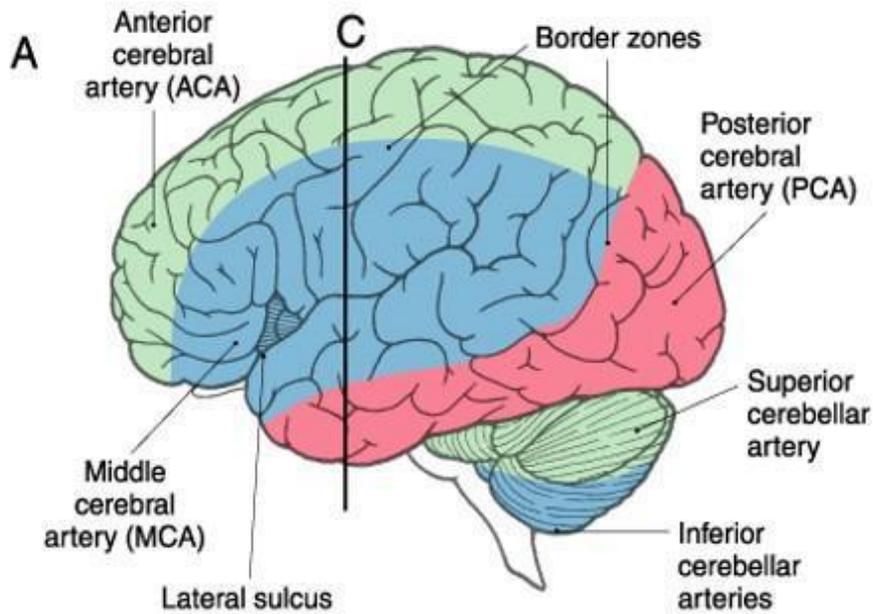
- Blood vessel => blood clot => infarct
- Tear of a blood vessel => bleeding
- Constriction of a blood vessel



CVA and TIA

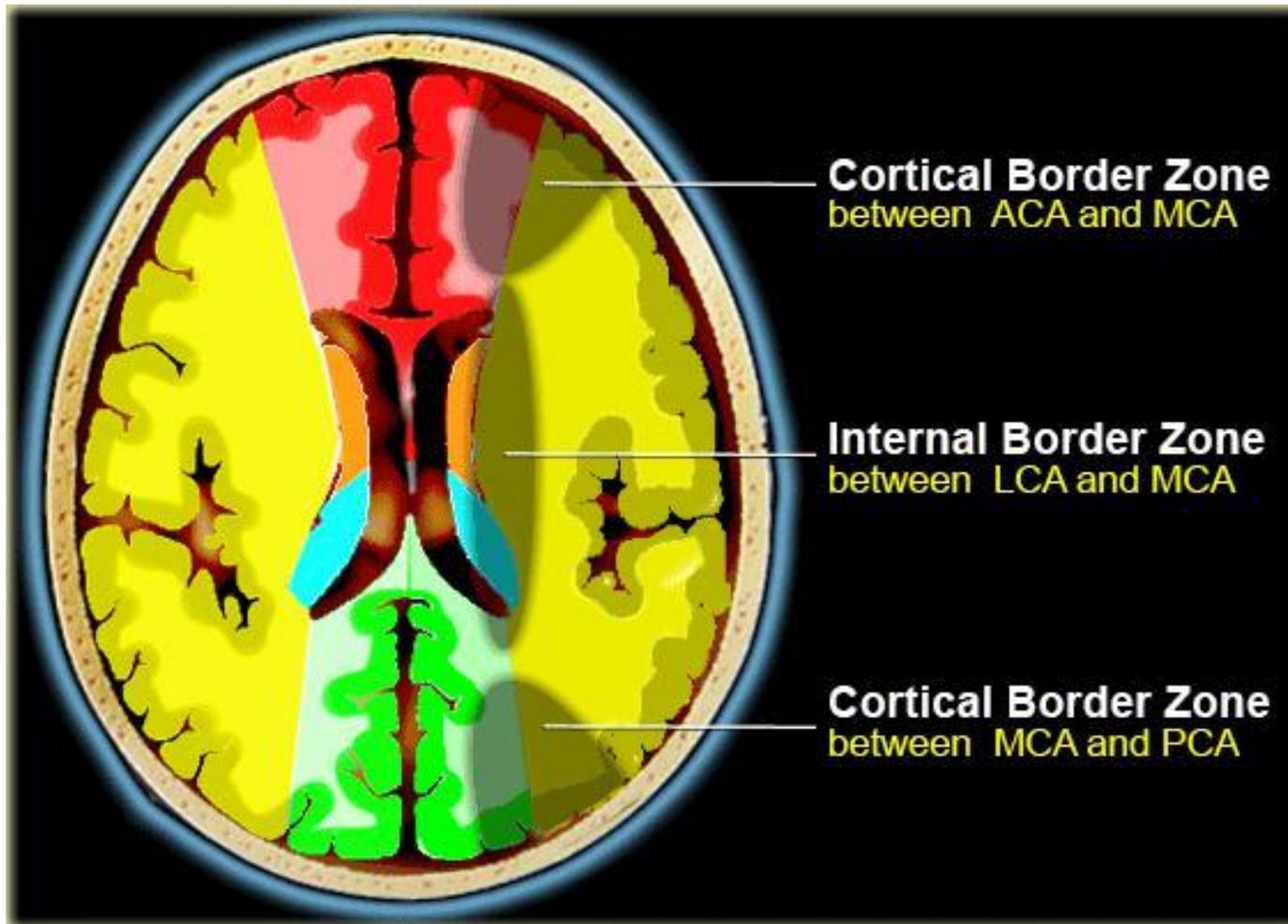
Cerebrovascular accident

- ▶ Ischemic or hemorrhagic
- ▶ Symptoms are dependant on localisation
 - Possible: paralysis, speech disorders, sensory disorders, blindness
 - Special type: watershed infarct



Watershed infarct

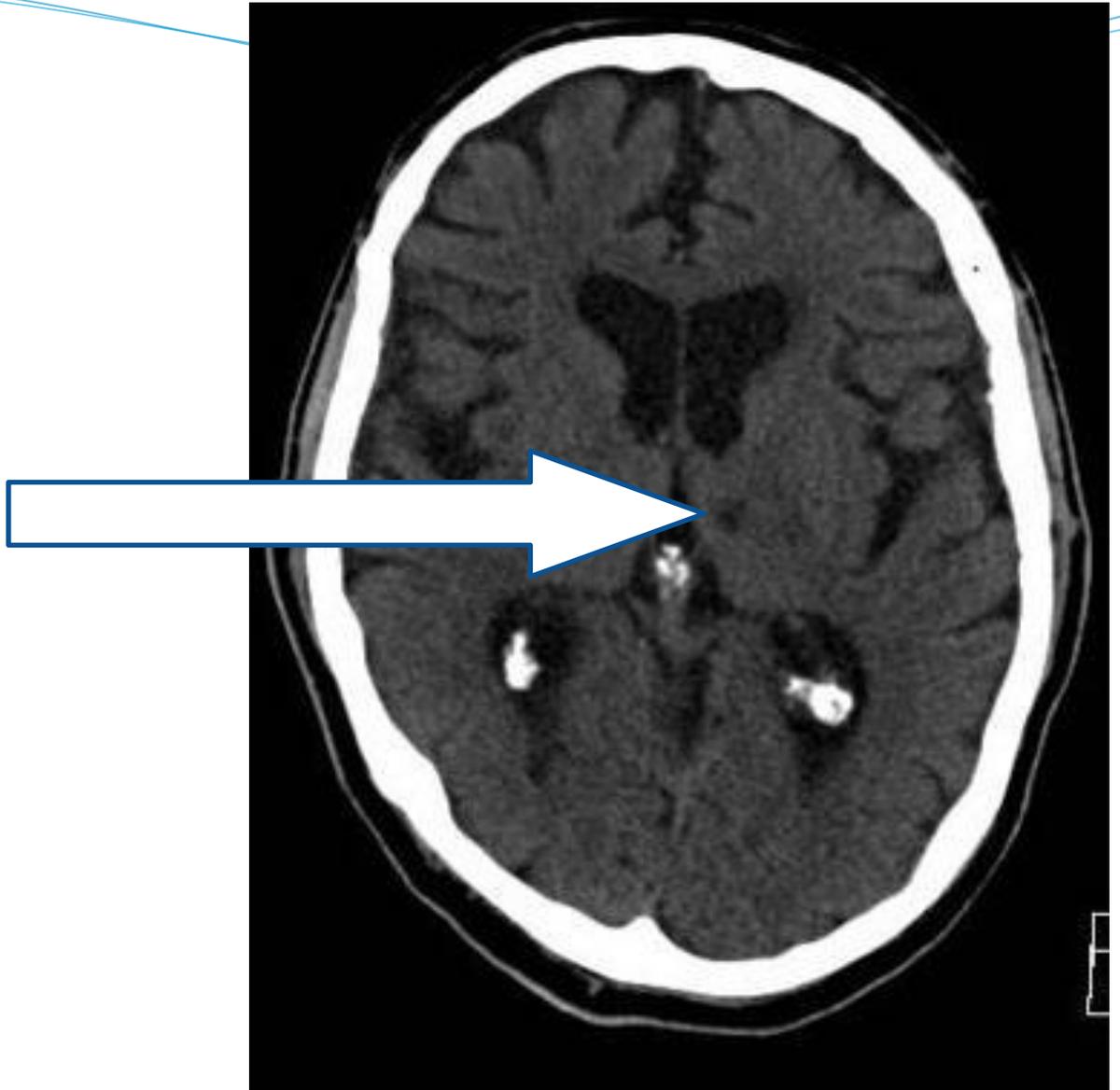
Watershed infarct



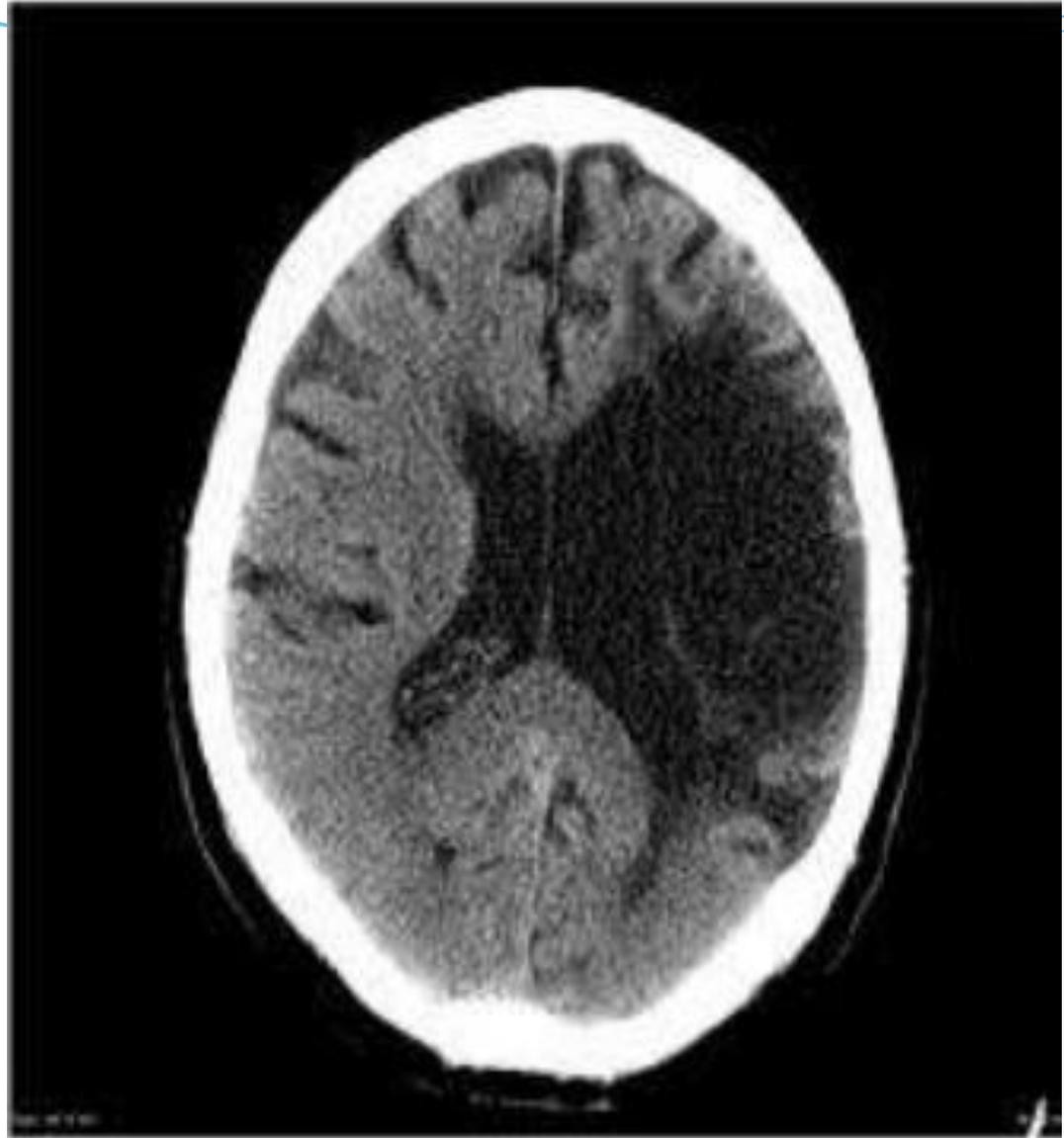
Watershe d infarct



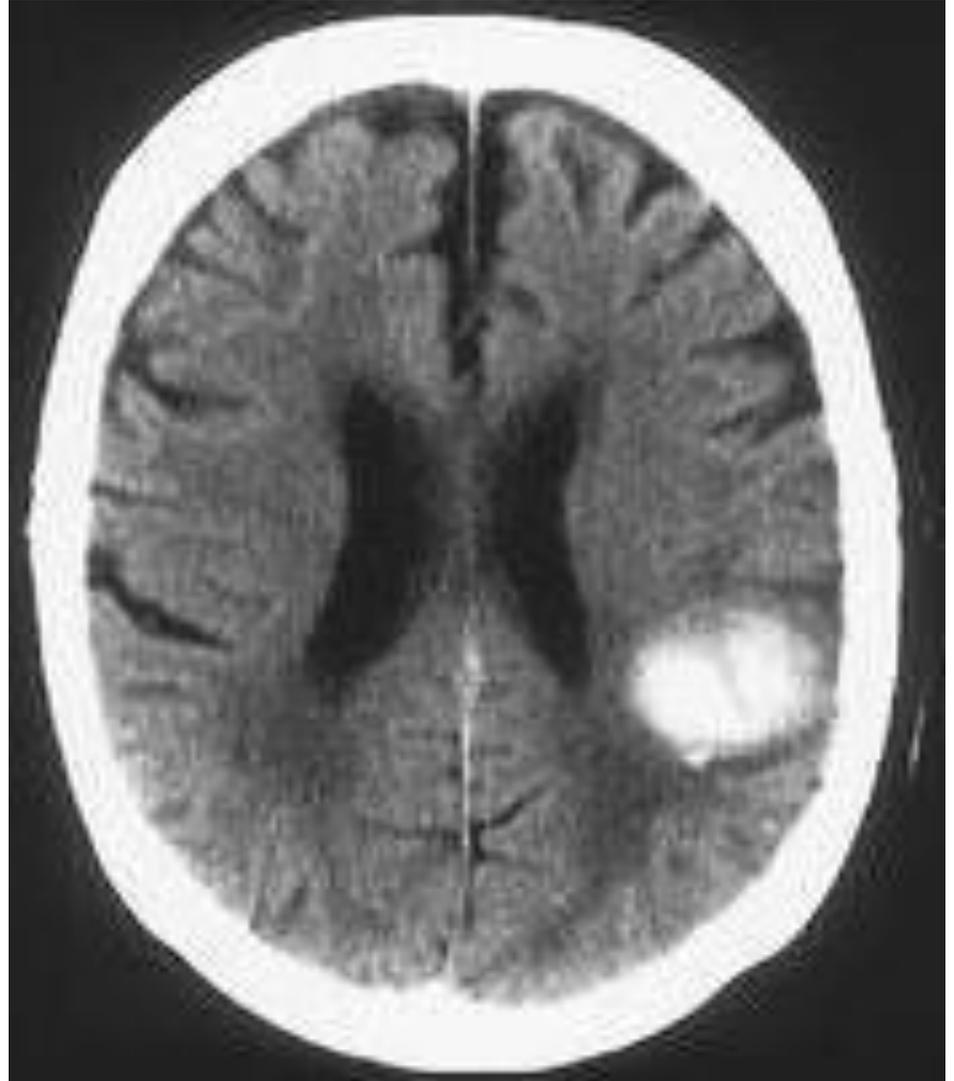
Lacunar infarct



Old infarct



Hemorrhagic infarct



Pop
Quiz!



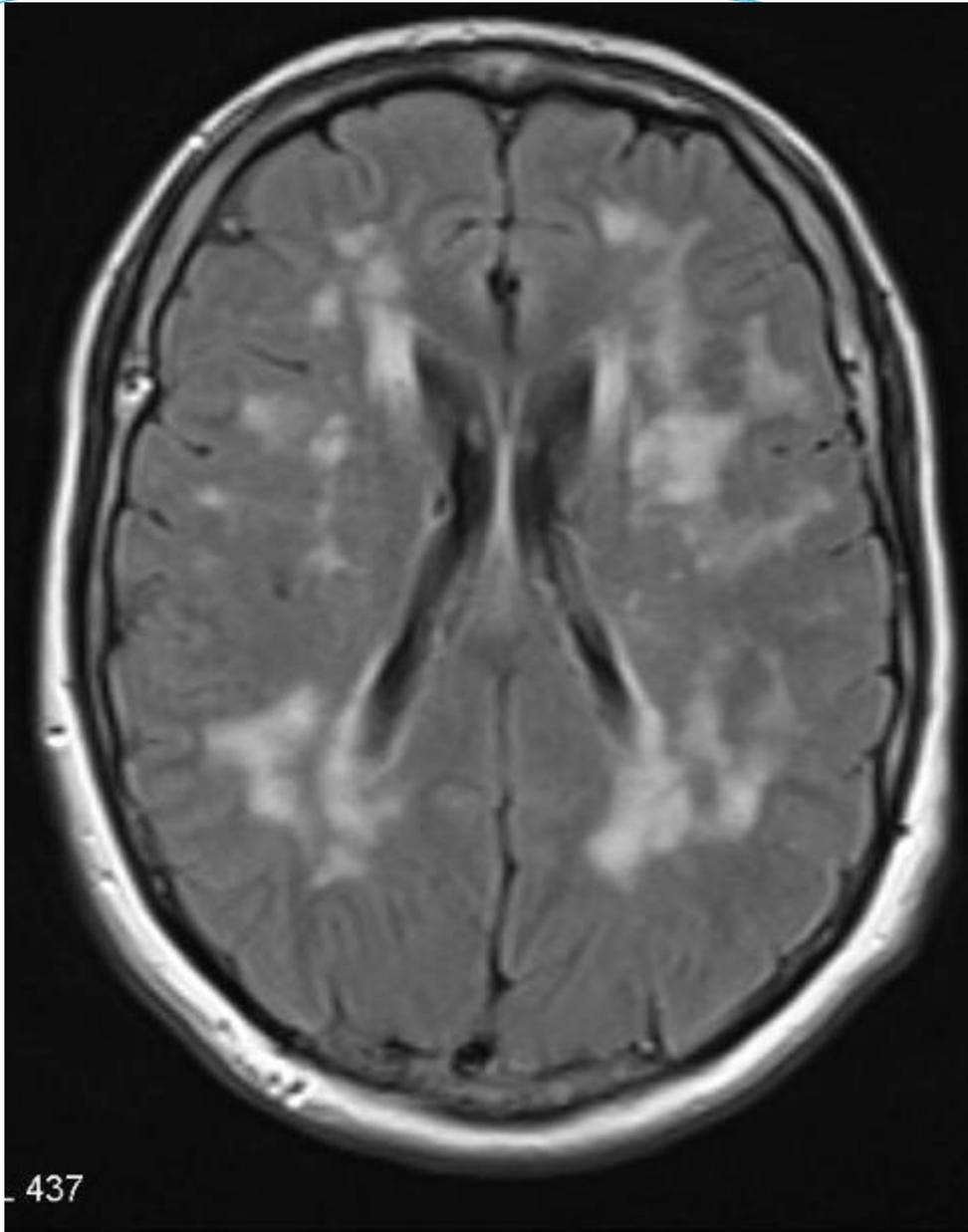
CADASIL

What is not correct?

1. It is hereditary
2. It is autosomal recessive
3. Patients often have migraine with aura
4. Notch gene affects white matter

CADASIL

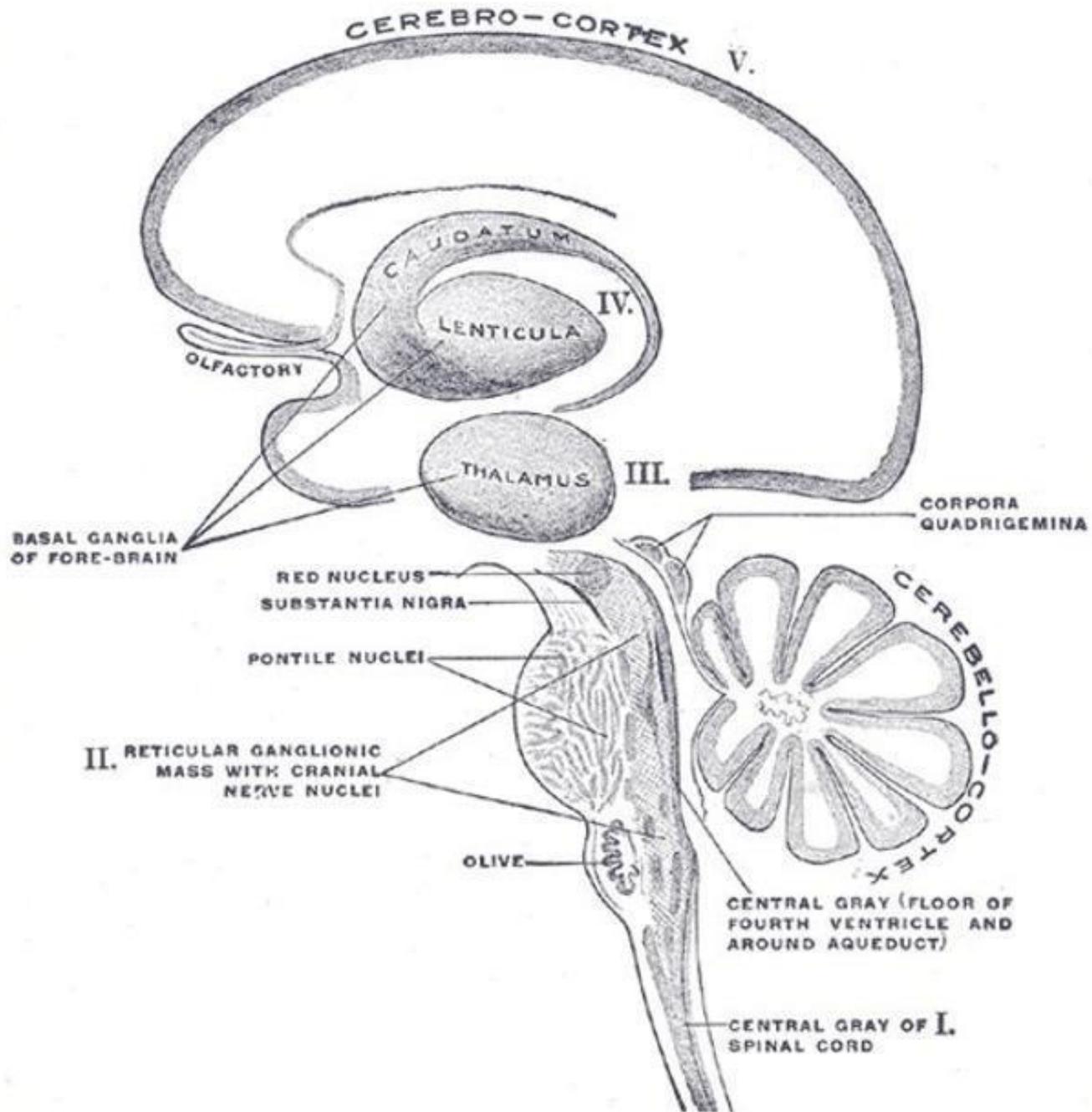
- Cerebral Autosomal Dominant Arteriopathy with Subcortical Infarcts and Leuko-encefalopathy
- Affects white matter through depositions of granular material in the blood vessels which causes a fibrous thickening.
- Symptoms: subcortical stroke, migraine with aura (1/3) psychiatric disturbances, at the late stage of development: dementia.



Non-specific white
matter laesions

Which structure that serves as a relay station is often affected in vascular dementia and leads to altered consciousness?

1. The basal ganglium
2. The corpus callosum
3. The thalamus
4. The globus pallidus



Thalamus

Contributes to functions of

- consciousness
- sleep
- memory
- sensory and motor functions

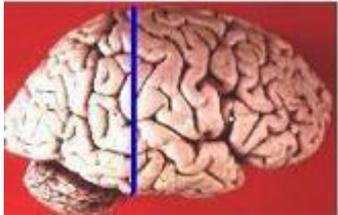
Destruction of this brain area may alter consciousness and impair memory, attention and motivation.

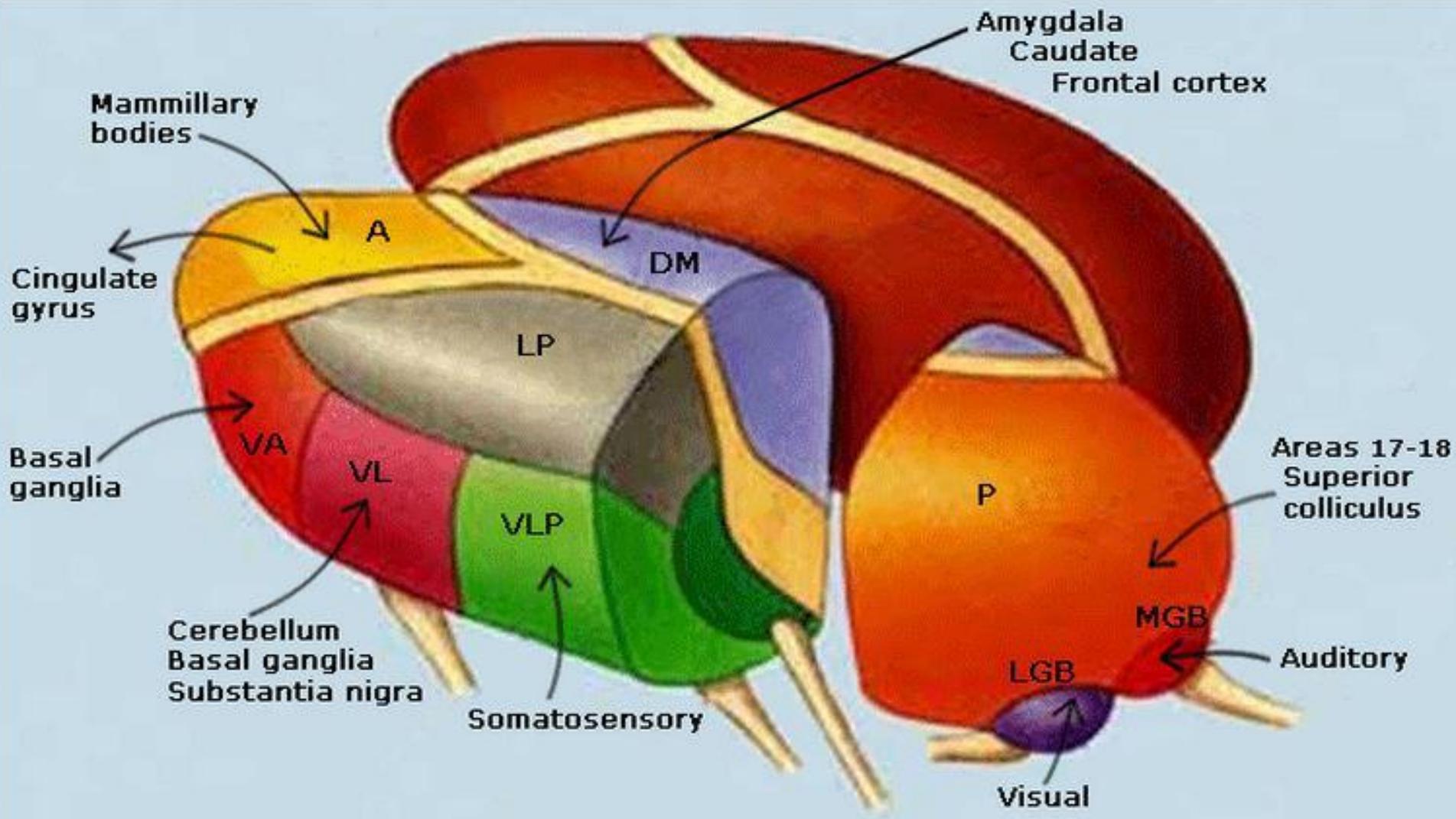
How many thalami are there?

1.1

2.2

3.4 (consciousness, sleep, memory, sensory and motor functions)





Vascular dementia

Treatment

Treating the predisposing risk factors...

Content

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Lewy body dementia

Cortical Lewy body dementia

Diffuse Lewy body dementia (DLBD)

Frederich Heinrich Lewy
1885-1950



Lewy body dementia

Course of the disease

- Present in 10-15% of dementia cases?
- Cfr. techniques
- Autopsy studies: 15-36% of dementia patients
- Male > female (1.5:1), man have worse prognosis
- Progression can be rapid (1-5 years) or more like

Lewy body dementias

Lewy body dementias include two clinical diagnoses, dementia with Lewy bodies (DLB) and Parkinson's disease dementia (PDD), which share essentially the same array of symptoms

Similarities with Parkinson dementia

- Memory impairment starts more than 1 year after physical complaints: more likely Parkinson's disease
- Memory impairment starts within 1 year after physical complaints: more likely DLBD

Lewy body dementia

Symptoms

- Fluctuations in cognition, strong variation in attention and alertness
- DLBD: 80-90%
- AD: 20%
- Repeated visual hallucinations, detailed (animals, children)
- Spontaneous motor symptoms (parkinsonism)

Lewy body dementia

Symptoms

- Strong reaction when using neuroleptics
- Other hallucinations (other than visual), sometimes delusions
- REM-sleep disorder

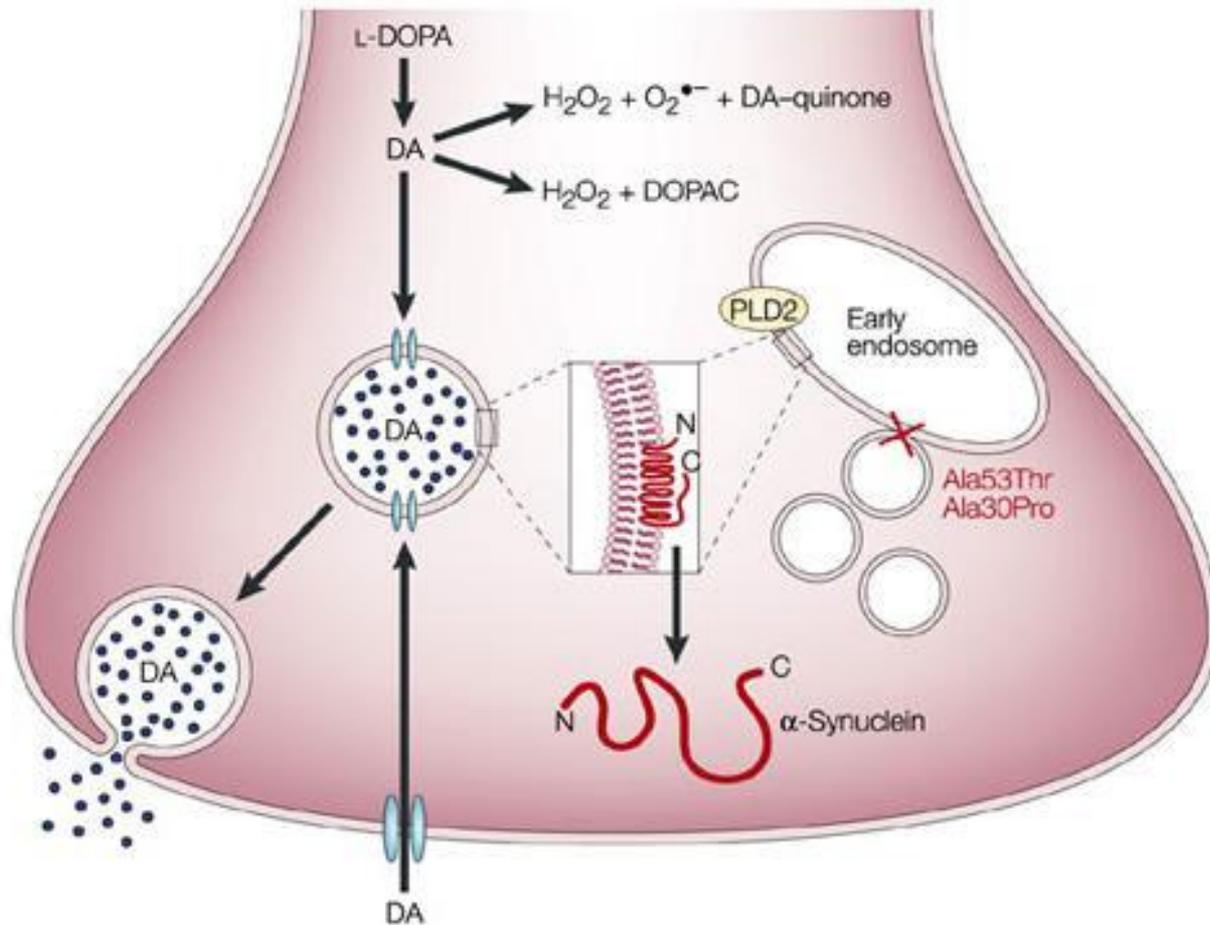
Lewy body dementia:

pathology

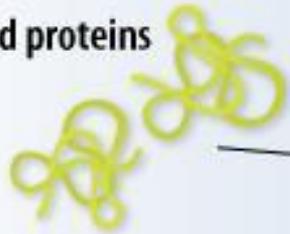
- Lewy body:

- Alfa synucleïne protein inclusion in cel
- Comparable to tau protein
- Lewy bodies in cortex and brain stem
- Subcortical nuclei, limbic cortex, neocortex (temporal > frontal = parietal)
- DD Parkinson: substantia nigra

Lewy body: alfa synucleine



Misfolded proteins



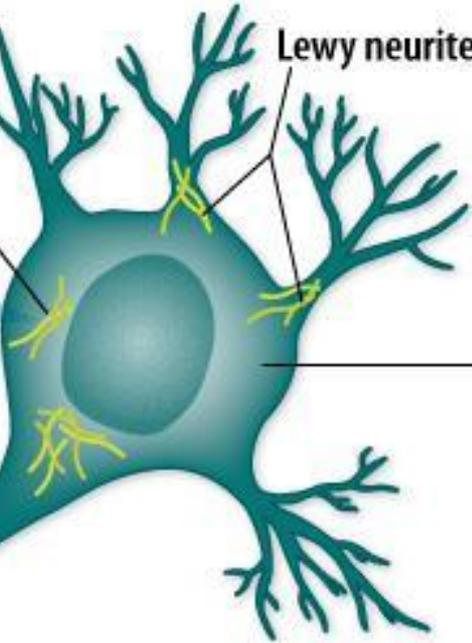
Oligomers



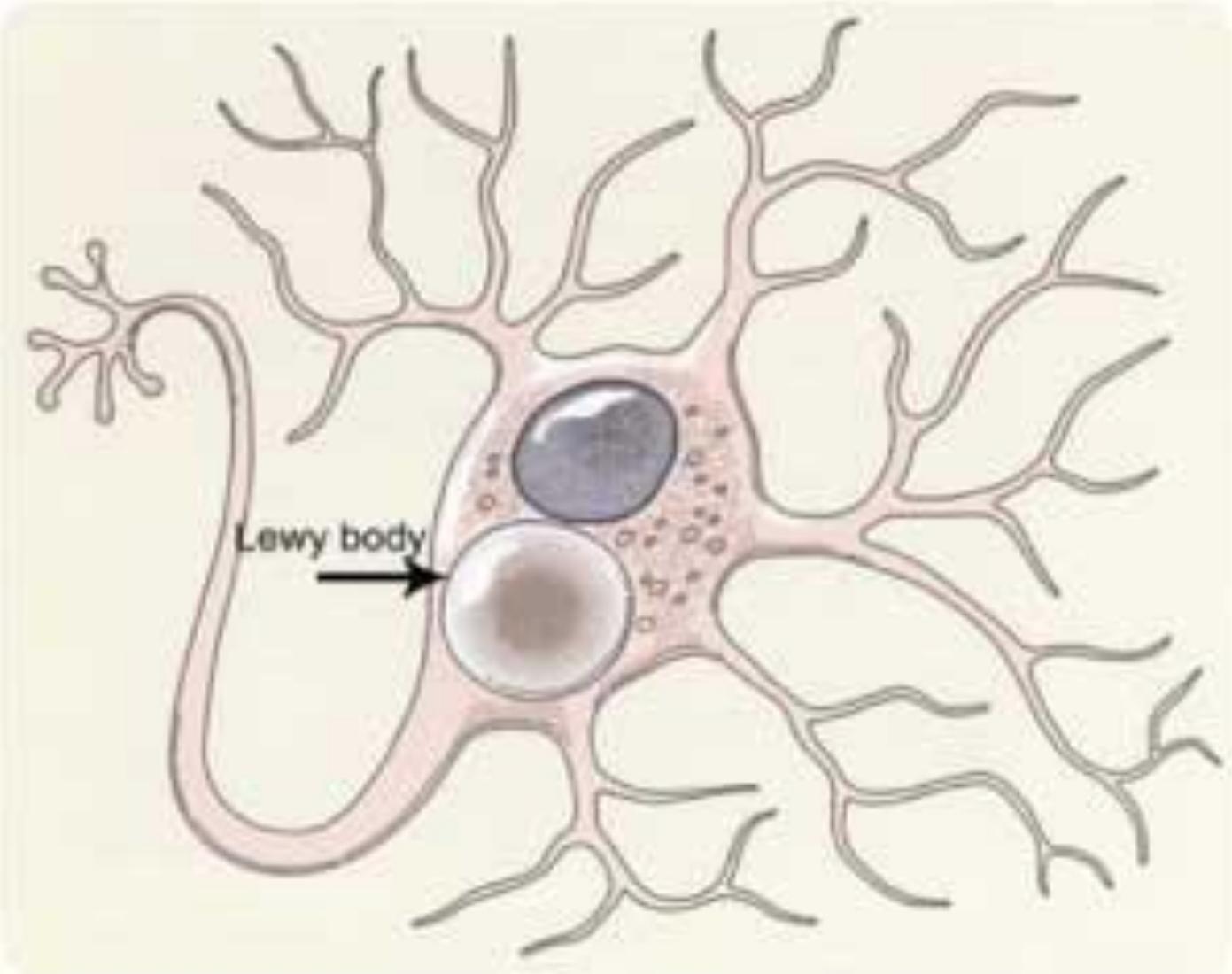
Fibrils
(β pleated sheet)



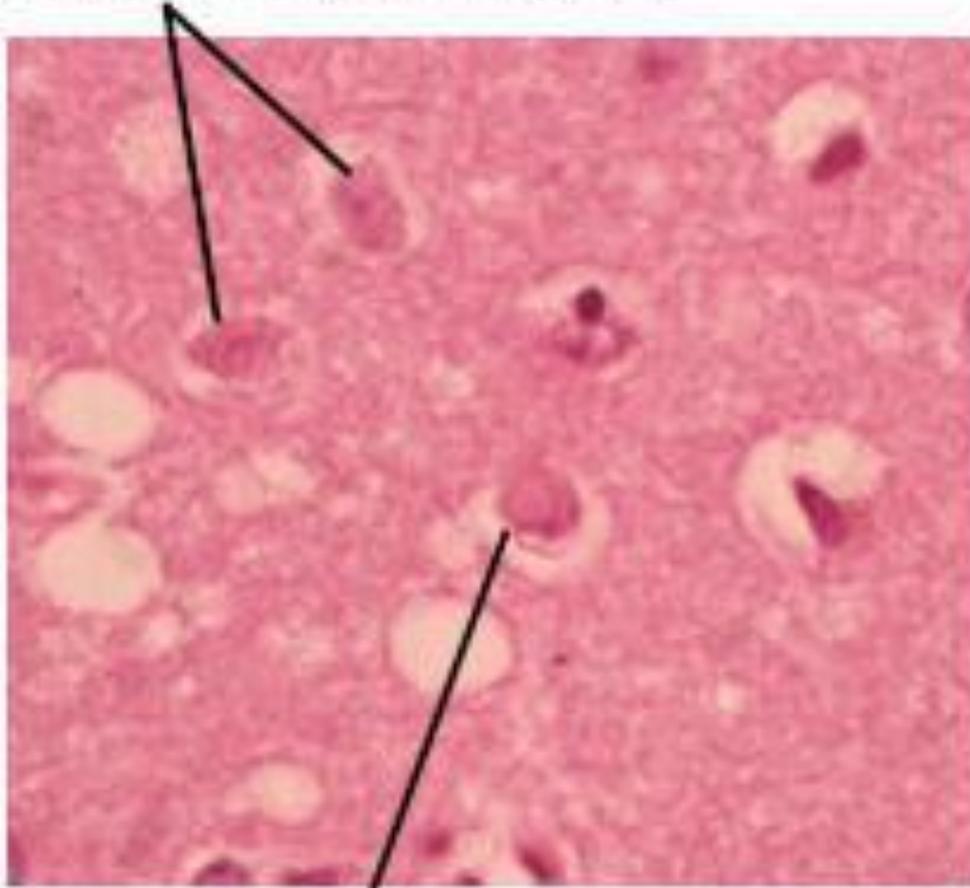
Lewy bodies



Lewy neurites

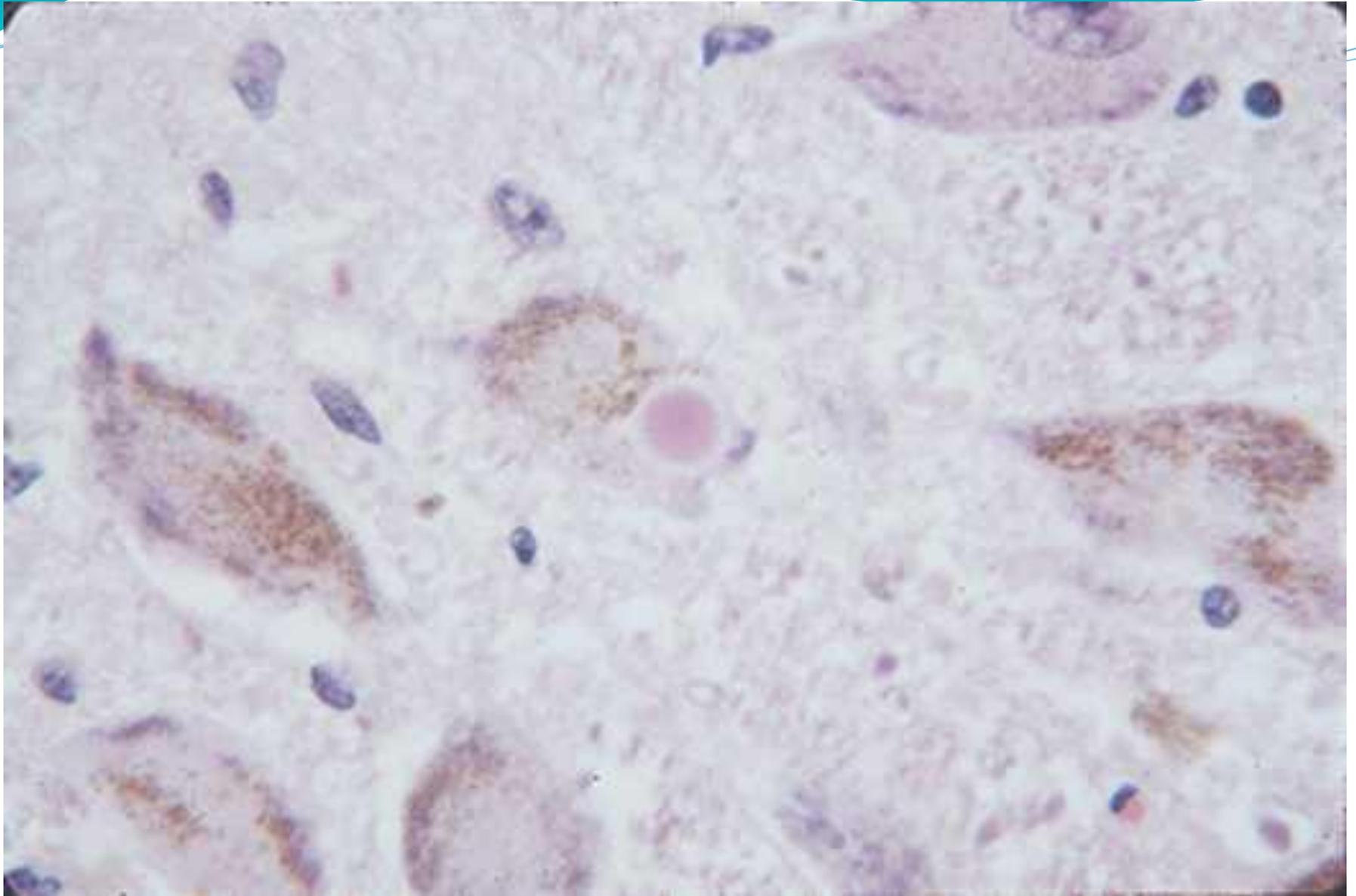


Nerve cells in cerebral cortex



**Cortical Lewy body
(Haematoxylin and eosin stain)**





DLBD Criteria

The diagnostic criteria for *probable DLB* require:

- The presence of dementia
- At least two of three core features:
 - fluctuating attention and concentration,
 - recurrent well-formed visual hallucinations, and
 - spontaneous parkinsonian motor signs.

Suggestive clinical features include:

Features of dementia associated with Parkinson's disease

Features are divided in 4 groups

Group I

The core feature requires a prior diagnosis of Parkinson's disease, and dementia causing a decline in function severe enough to impair the patient in daily activities and in at least one cognitive domain.

Features of dementia associated with Parkinson's disease

Group II - The clinical features include both the cognitive and behavioral domains described below:

Cognitive domains:

- Attention
- Executive function
- Visuo-spatial ability

Features of dementia associated with Parkinson's disease

Group III - The third category includes two features that will not rule out a diagnosis of PDD, but may make the diagnosis more uncertain:

- Existence of an abnormality such as vascular disease which causes cognitive impairment although not determined to cause dementia

Features of dementia associated with Parkinson's disease

Group IV - The last domain contains two features which suggest that other existing conditions impair the patient's cognitive functioning to such an extent that reliable diagnosis of PDD becomes impossible.

- Cognitive or behavioral symptoms which occur only in the context of existing conditions, such as systemic diseases, drug intoxication, or major depression

Lewy body dementia

Treatment

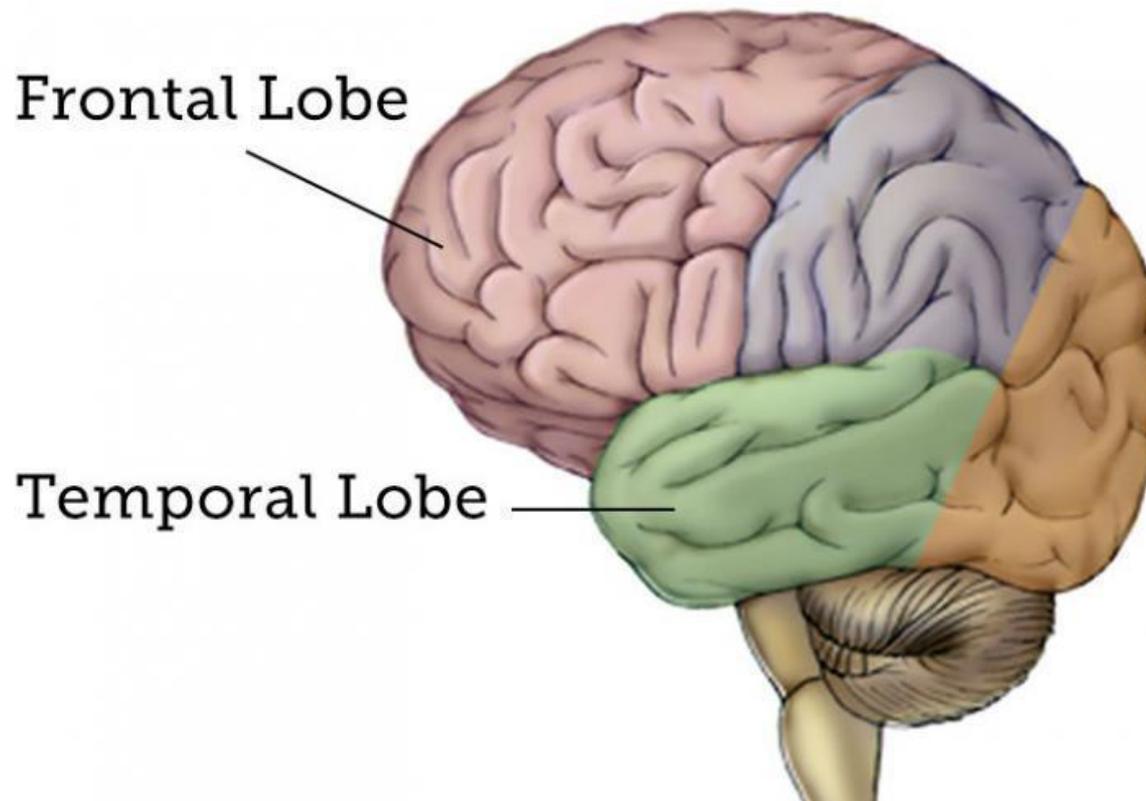
None...

Only symptomatic

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Frontotemporal dementia



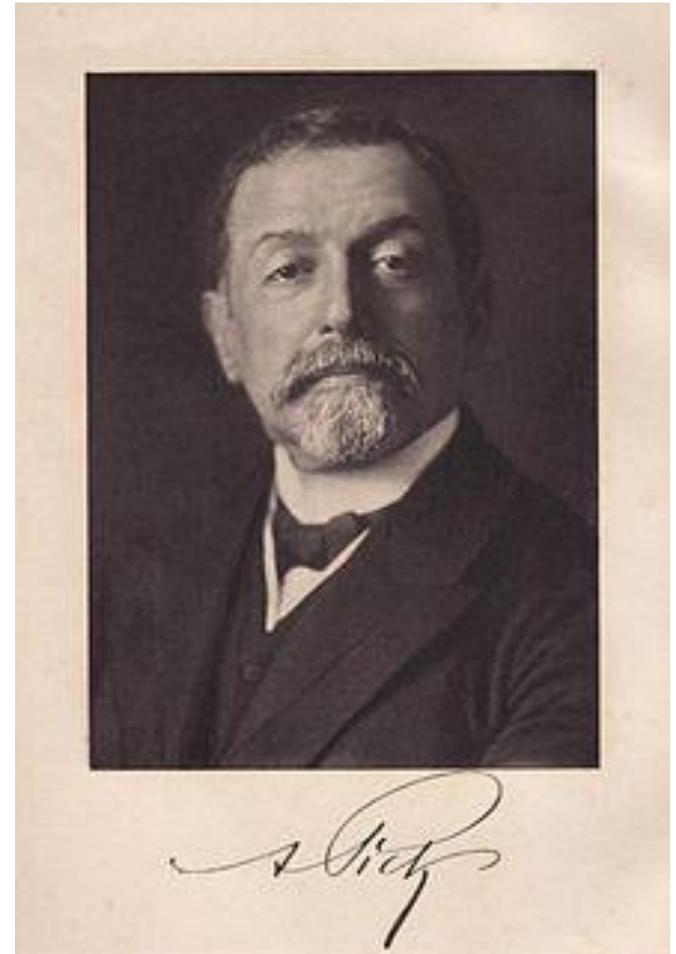
Frontotemporale demencie

Used to be Pick's disease

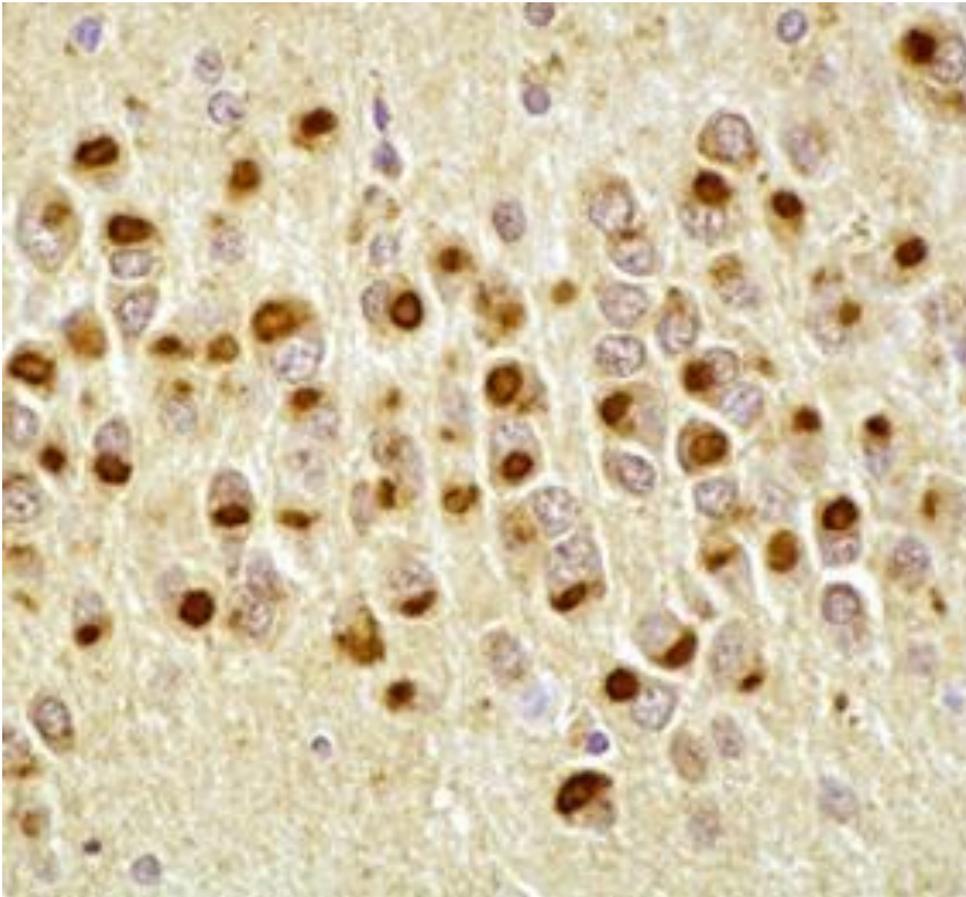
This is more and more abandoned, since there are newer insights into the different types

Now Pick's disease is only used when Pick bodies are found

Arnold Pick
1851-1924



Pick bodies (tau)



Frontotemporal dementia

Different variants (FTD)

- Behavior variant frontotemporal dementia (bvFTD)
- Primary progressive aphasia (PPA)
- Semantic variant
- Nonfluent/agrammatical variant

Frontotemporale dementie

Behavior variant frontotemporal dementia (bvFTD)

Important changes in

- Personality, interpersonal relations
- Behaviour

Mood-, emotional- and neurological changes

bvFTD

Behavioral symptoms

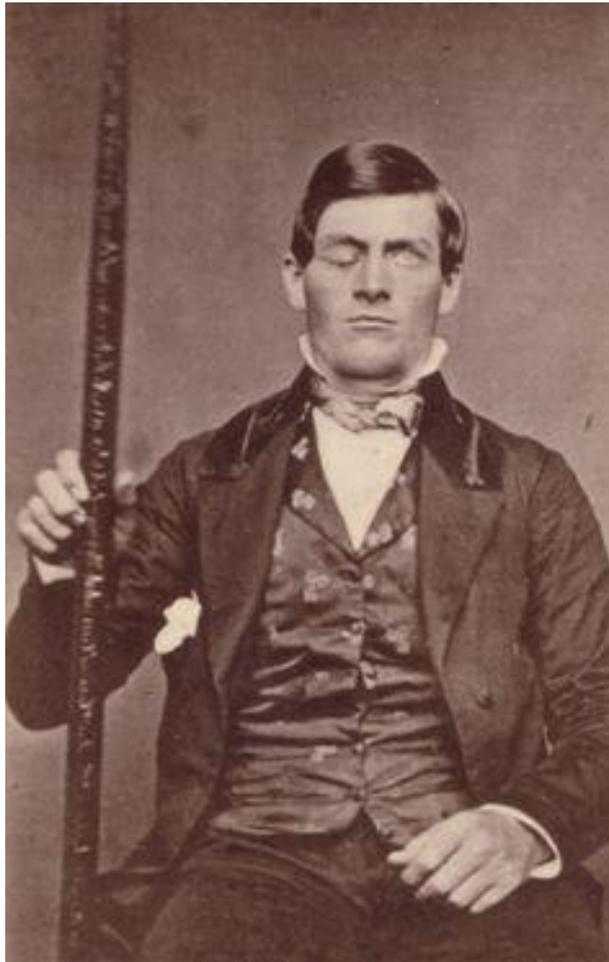
- Hyperoral: overeating/drinking, compulsive behaviour regarding diet (specific nutrients, inedible things)
- Stereotypical/repetitive: clapping of hands, same walk over and over ...
- Personal hygiene: fails in an early stage

bvFTD

Emotional symptoms

- Apathy: taking an initiative is diminished, lack of motivation
- Lack of insights into disease: present in early stage
- Emotional numbing: present in early stage, loss of empathy and sympathy, growing indifference

Accidental frontal behaviour



Phineas Gage
1823-1860



Frontotemporal dementia

Primary progressive aphasia (PPA)

- Affects language, writing and understanding abilities
- Mostly before age of 65, but possible later onset
- Three types
 - Semantic variant: loss of possibility of understanding or formulating words in a spoken sentence

Frontotemporal dementia

Semantic variant

- Progressive deterioration in
- Understanding words (mainly nouns) and meaning of words
- Recognition of objects
- Speech is still fluent, but becomes more difficult to understand

Frontotemporal dementia

Nonfluent/agrammatical variant

- Predominantly difficulties with language production
- Initially hesitant speech
- Later stage: just less speaking
- End stage: mutistic, and development of swallowing disorders
- Understanding language, writing and reading is preserved longer

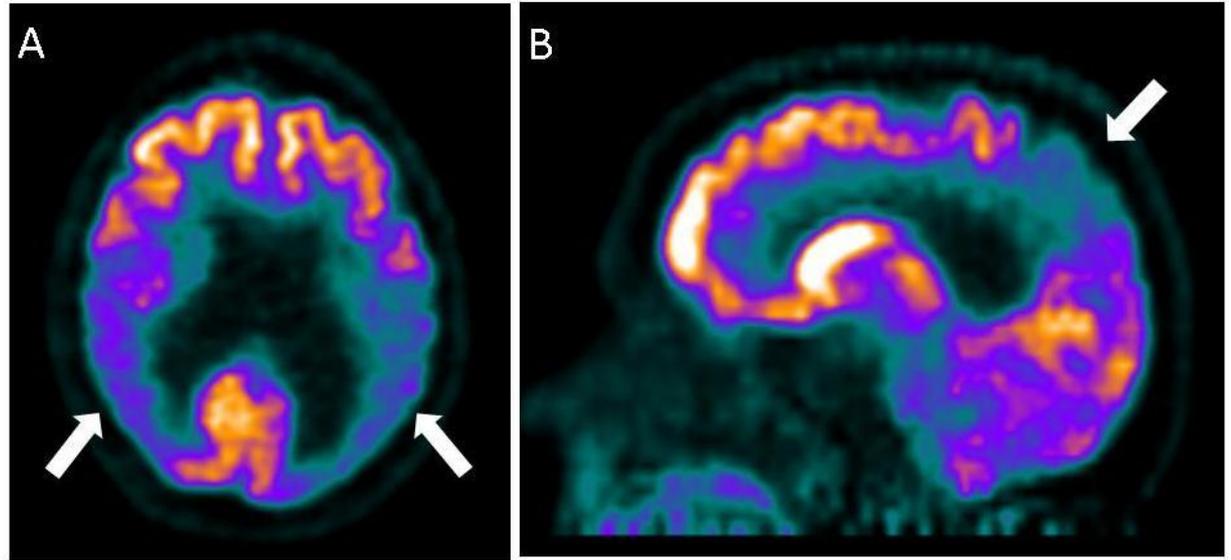
Frontotemporal dementia

Logopenic variant

- Predominantly decline in possibility of retrieving words
- Presentation: slow speech, frequent pauses (=word retrieval difficulties)
- Fundamental loss of phonological short term memory / working memory (auditive attention span)

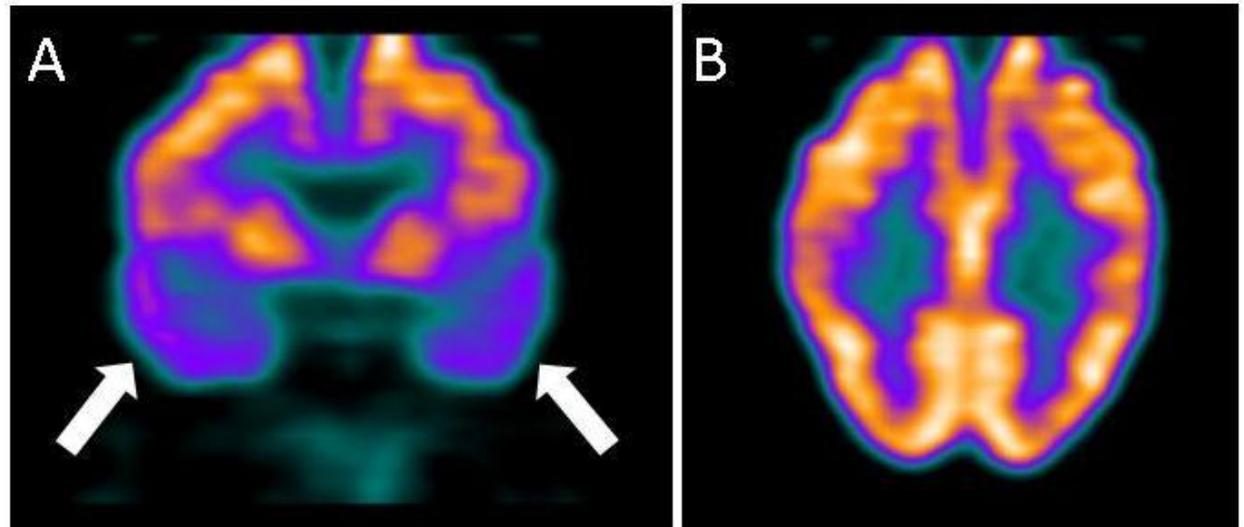
FDG PET

- AD



- FTD

- PPA

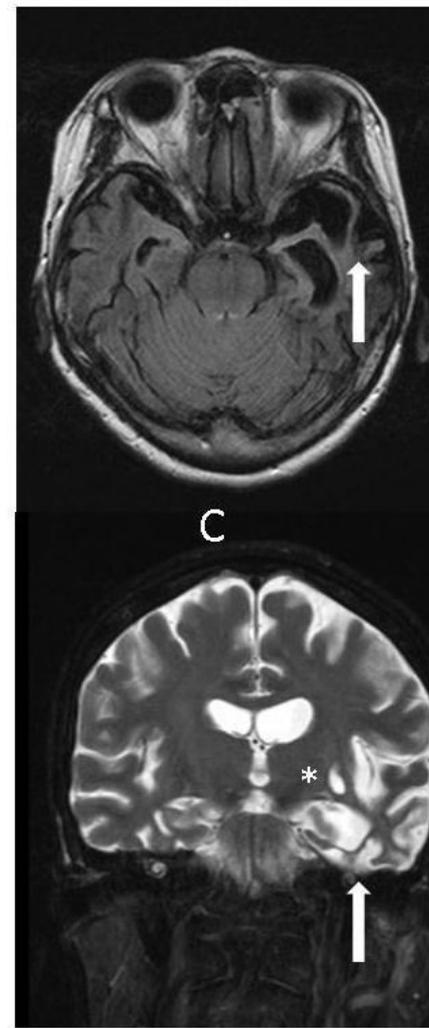
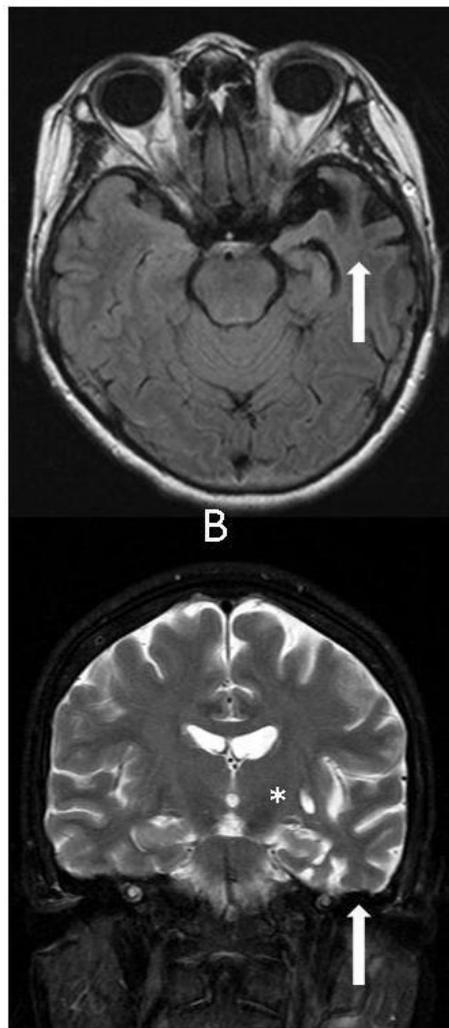
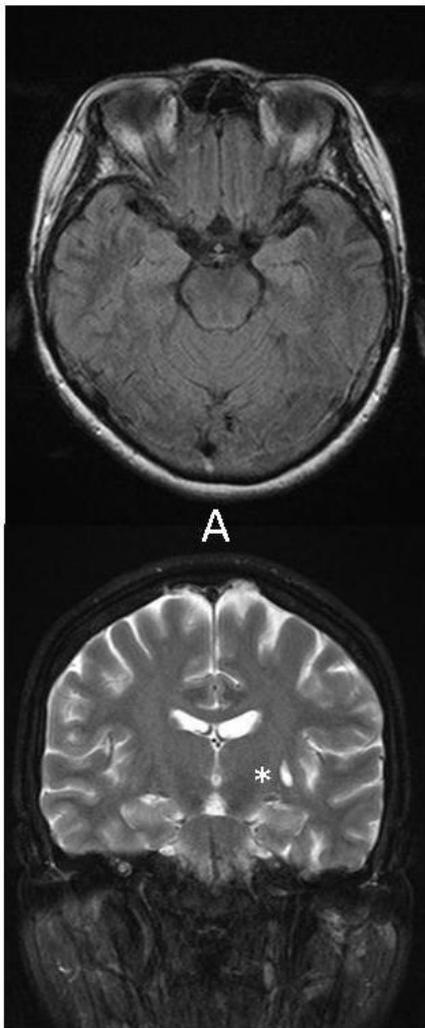


T=0 years

T=3 years

T=8 years

PPA
RRP
IOH
MGA
ARS
REI
YSA
S
I
V
E



Frontotemporal dementia

Treatment

None...

Unless symptomatic when behaviour disorders start

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Other types of dementia

- Alcohol – Korsakov
- Huntington dementia
- Creutzfeldt-Jakob
- Dementia pugilistica

Other types of dementia

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Alcohol – Korsakov

Sergei Korsakov

1854-1900

Lack of vitamin B₁

Syndrome of Wernicke

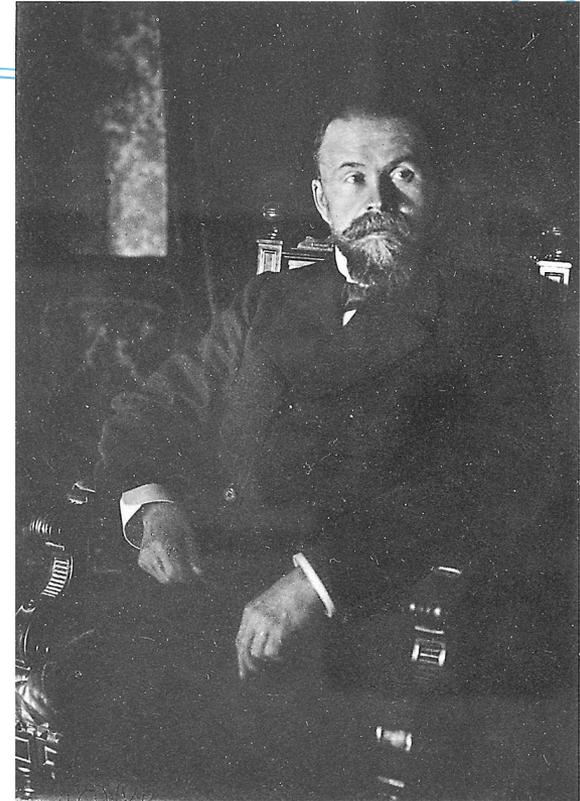
60% of these evolve into



Alcohol – Korsakov

Syndrome of Wernicke

- Palsy of the eye muscles
 - Ataxia
 - Delirium
-
- Could lead to coma and death



Carl Wernicke
1848-1905

Alcohol – Korsakov

- Males > females, age between 45 and 65 years
- Criteria (DSM IV)
- Anterograde amnesia (no new memories after event)
- Variabel presentation of retrograde amnesia (memories from before the event)
- And one of the following: aphasia, apraxia, agnosia or a deficit in executive function
- Also: not during a delirium or withdrawal

Alcohol – Korsakov

- Symptoms:
- Memory loss
- Memory deficit are filled up by made-up stories = confabulations, not lies
- Intellectual possibilities are mostly spared
- Desorientation in time and space
- Gait and balance disorders
- Changes in character: lack of initiative, paranoid, depressie en agressief gedrag

How to write Korsakov/w/ff?

Originally Russian: Корсаков

So actually you can choose.

Most often:

In Dutch: Korsakov

In English: Korsakoff

In German: Korsakow

Korsakov

T2/FLAIR: symmetrically increased signal intensity in the

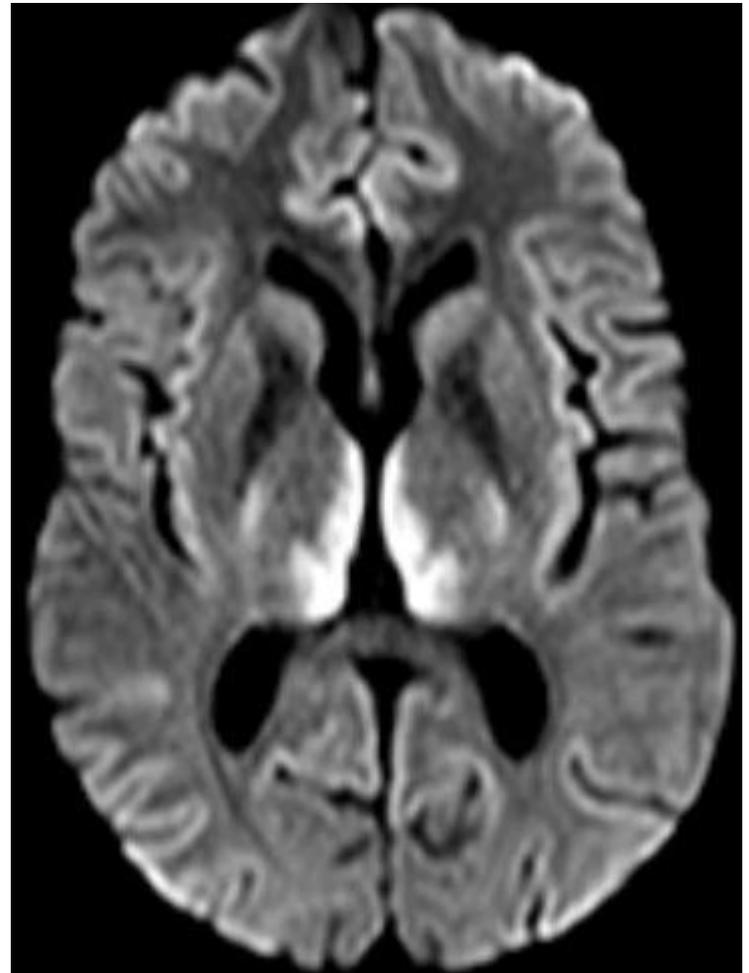
Mamillary bodies

Dorsomedial thalami

Tectal plate

Peri aqueductal area

Around the third ventricle



Other types of dementia

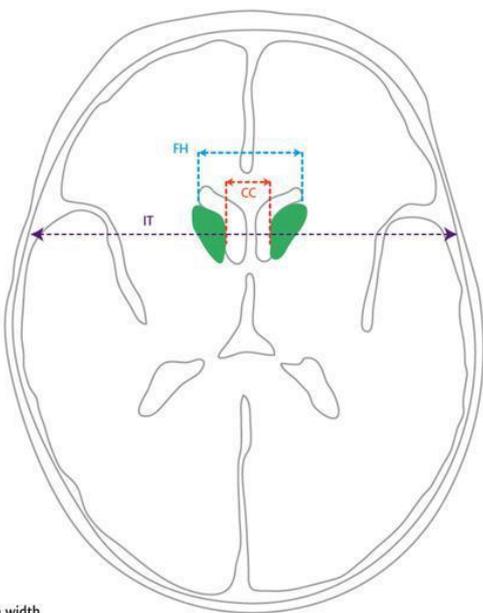
- Alcohol – Korsakov
- Huntington dementia
- Creutzfeldt-Jakob
- Dementia pugilistica

Huntington

- Progressive cell death because of Huntington protein (Huntingtin)
- The protein has a functional role in cytoskeletal anchoring or transport of mitochondria
- Gives way to a loss of GABAergic neurons of the basal ganglia, especially atrophy of the caudate nucleus and putamen.

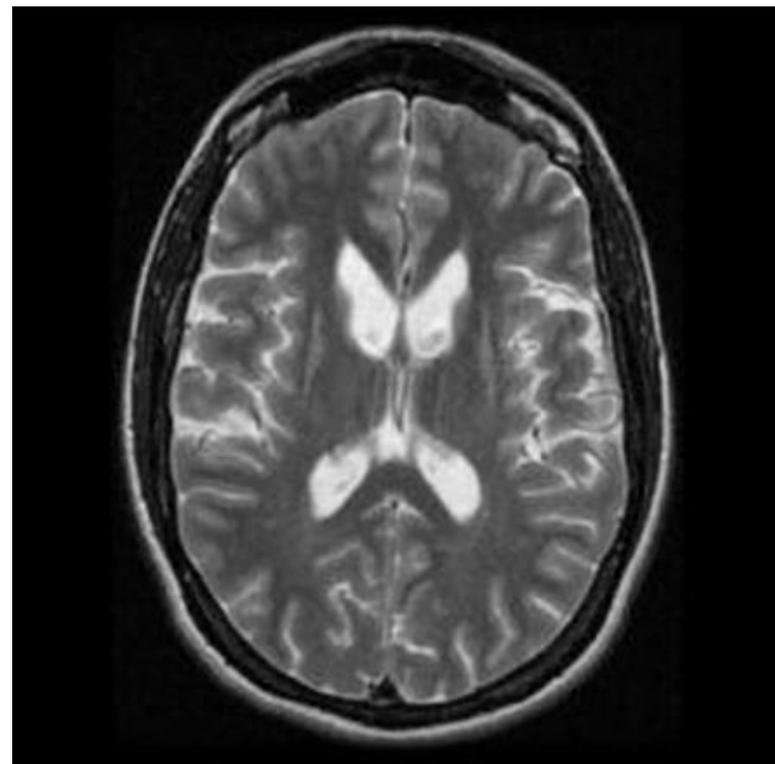
Huntington

classically characterised by atrophy of the caudate nucleus with concomitant enlargement of the frontal horns of the lateral ventricles.



FH = frontal horn width
CC = intercaudate distance
IT = inner table width

F. Gaillard 2009
Radiopaedia.org CC BY-NC-SA 4.0



Other types of dementia

- Alcohol – Korsakov
- Huntington dementia
- Creutzfeldt-Jakob
- Dementia pugilistica

Creutzfeldt-Jakob

- Neurodegenerative prion-disease (wrongly folded proteins that affect other protein's structure)

- 4 forms

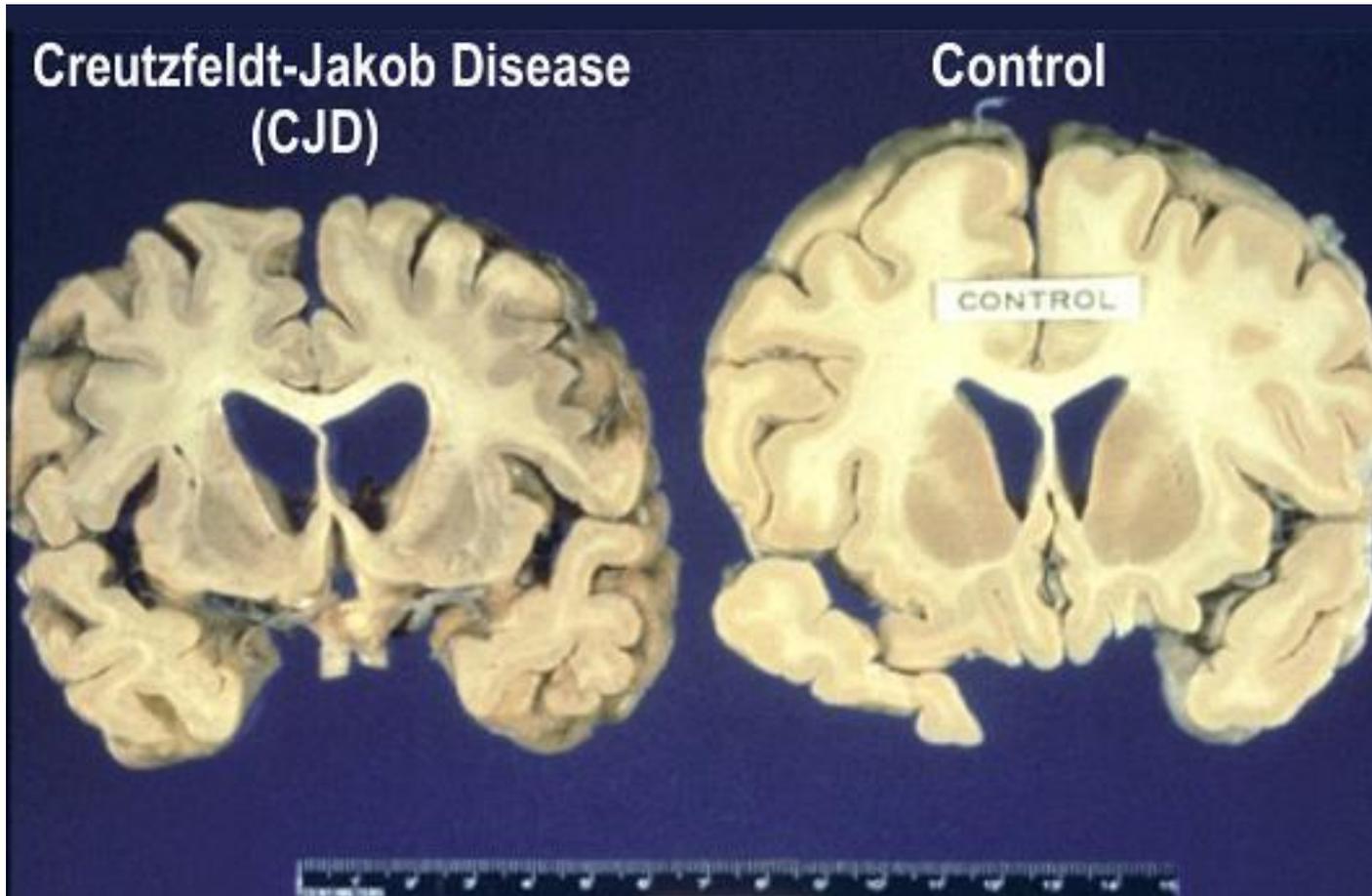
- Variant (vCJD): consumption of contaminated food (prions): BSE/kannibalism

- Sporadic (sCJD): +/- 85% of the cases

- Familial (fCJD): +/- 15% of the cases

- Iatrogenic: contamination of tissue of affected persons

Creutzfeldt-Jakob



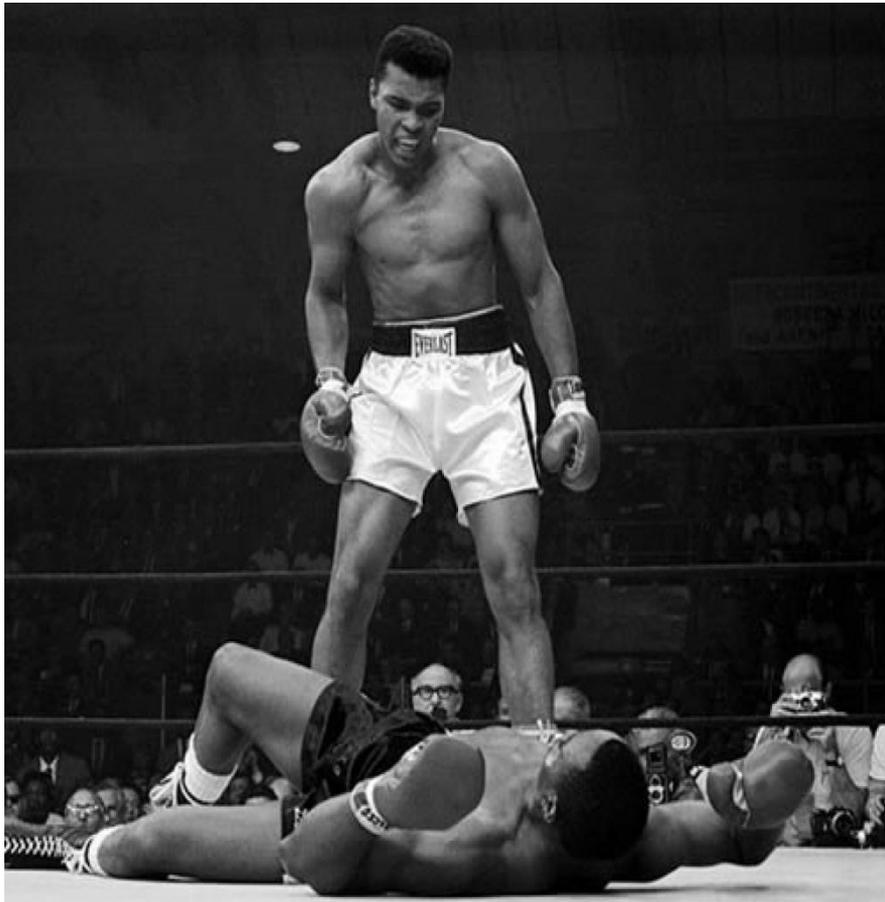
Other types of dementia

- Alcohol – Korsakov
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- Dementia pugilistica

Dementia pugilistica

- Chronic traumatic encephalopathy (CTE)
- AKA chronic boxer's encephalopathy, traumatic boxer's encephalopathy, boxer's dementia, chronic traumatic brain injury associated with boxing (CTBI-B), and punch-drunk syndrome
- Amateur/professional boxers, wrestlers, other (football, ...)

Dementia pugilistica



Parkinson? Or variant DemPug?

Dementia pugilistica

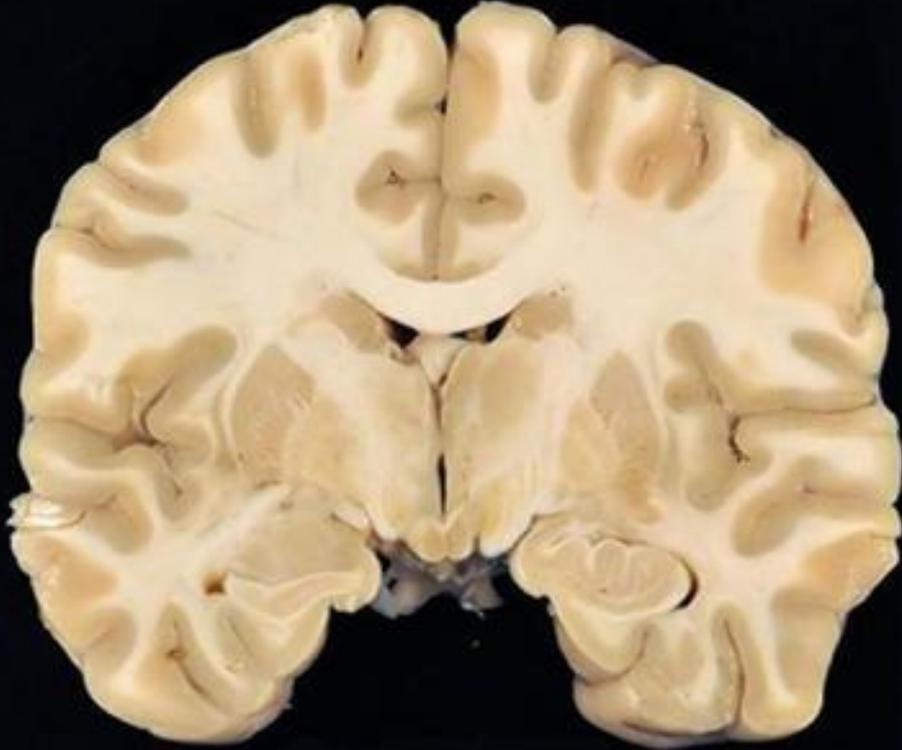
Symptoms

aggressiveness, explosiveness, impulsivity, depression, memory loss

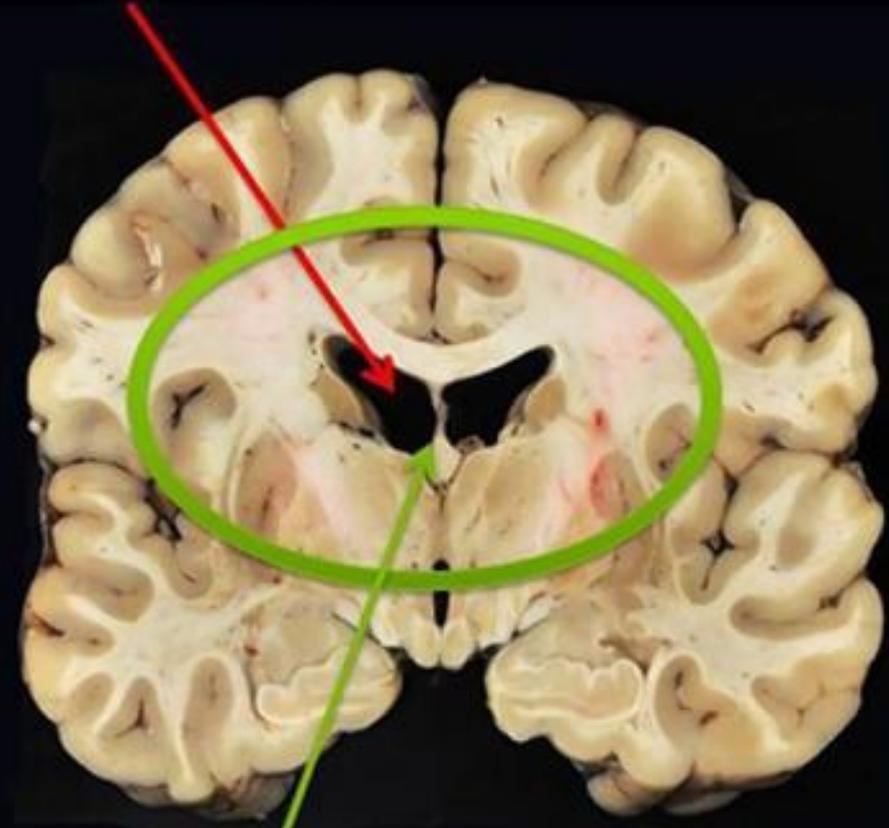
Radiology

generalised cortical atrophy, hippocampal atrophy, vermian atrophy, cavum septum pellucidum, features of diffuse axonal injury (e.g. microhaemorrhages)

Ventricular enlargement



Normal 27 year old



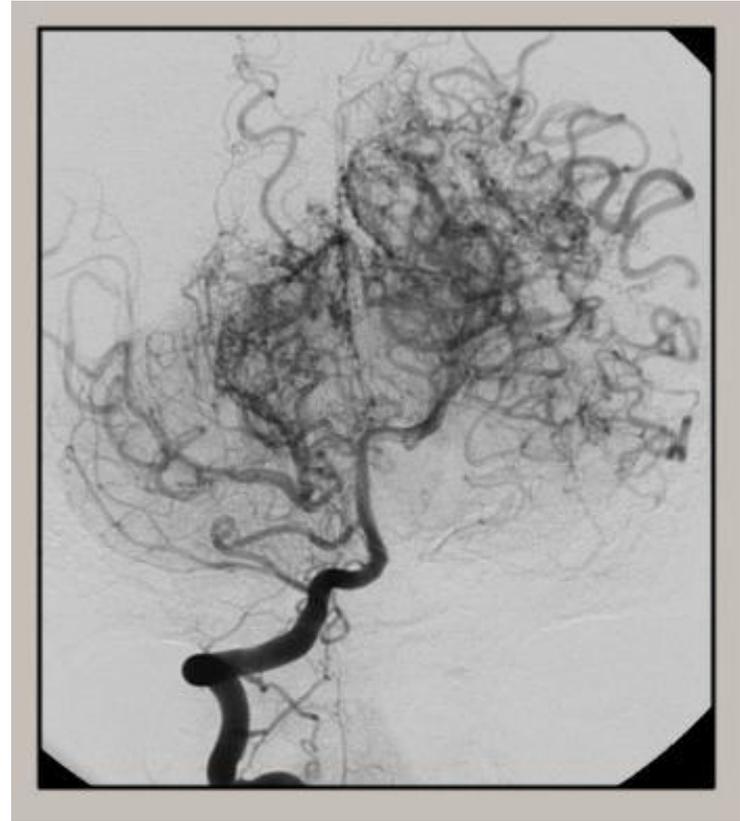
Aaron Hernandez

Atrophy of the fornix

Other types of dementia

- Alcohol – Korsakov
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- Dementia pugilistica

Moyamoya disease



Puff of smoke sign (Japanese)

Moyamoya disease

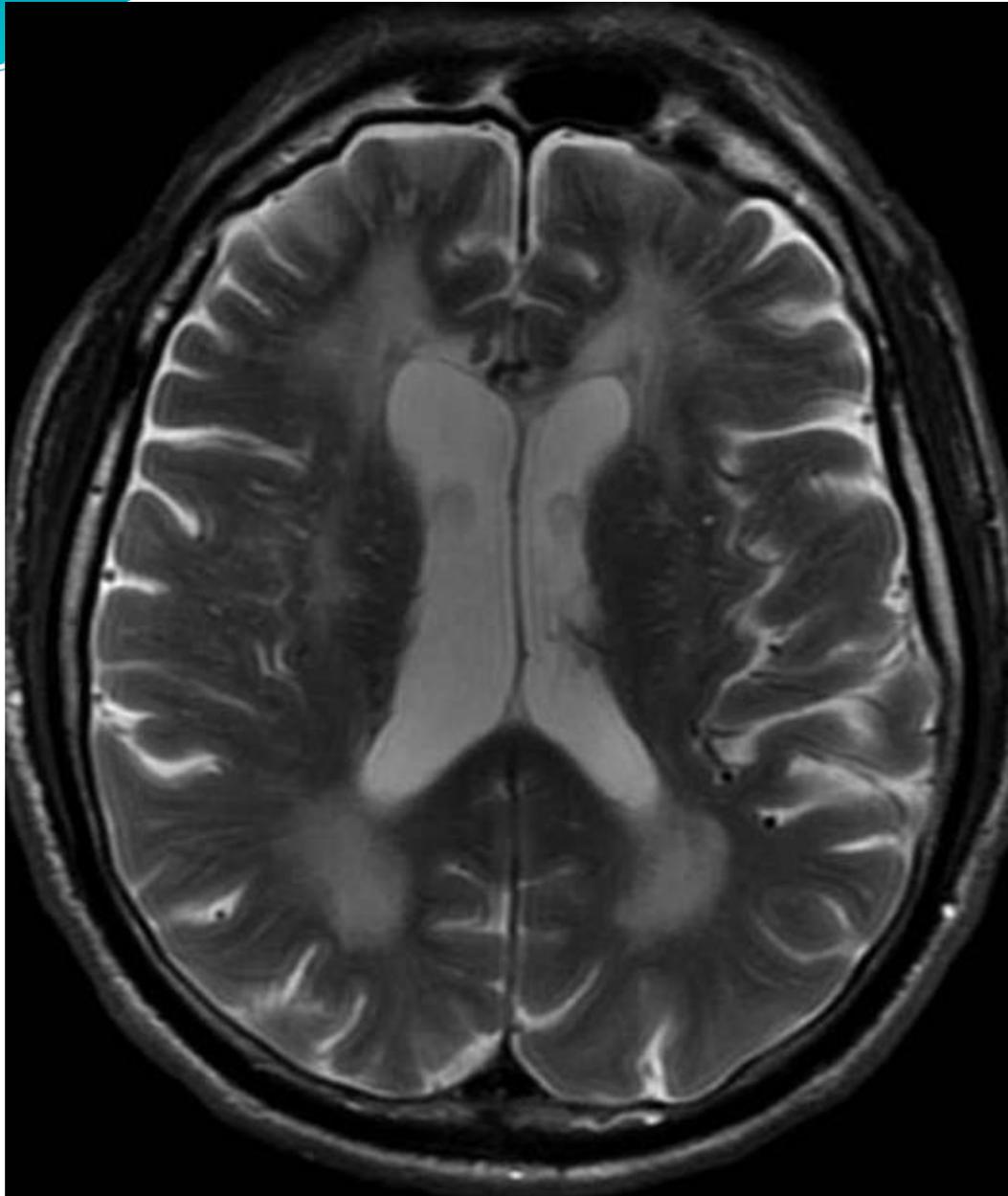
- Occlusions of cerebral arteries => tiny abnormal intracranial collateral vessel networks
- Prevalence:
 - Worldwide: 1/1.000.000
 - Western countries: 1/2.000.000
 - More in people of Asian descent

Other types of dementia

- Alcohol – Korsakov
- Huntington dementia
- Creutzfeldt-Jakob
- Dementia pugilistica

HIV dementia – AIDS dementia complex

- Affected neurons in aids-patients
- Rare since antiretroviral treatment
- < 1% of HIV-positives
- Symptoms: inattention and reduced concentration, apathy and dulling of personality, psychomotor slowing, marked motor slowing and ataxia



CT:

symmetric periventricular
and deep white matter
hypoattenuation

MRI:

symmetric periventricular
and deep white matter T2
hyperintensity

relative sparing of the
subcortical white matter and
posterior fossa structures

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Take home messages

- There are different forms of dementia
- Try to find the symptoms and correlate them to a pathology
- It is imperative to know your brain anatomy and function



WHO

WHAT

WHERE

WHEN

WHY

HOW

QUESTIONS

ANSWERS