Physiology Team 439

MED439
KING SAUD UNIVERSITY

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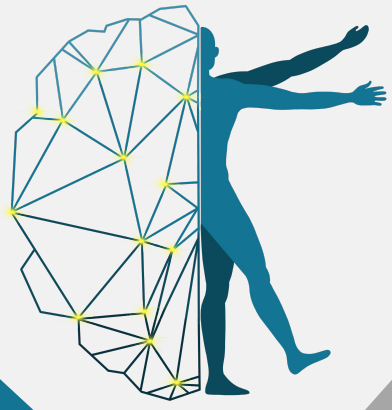

# Aging and Brain

# Objectives:

- Define Aging and its consequences.
- Describe the theories of aging and terms Used.
- Name some Brief Geriatric Assessment Instruments.
- Body Changes in Aging.
- Describe brain changes associated with healthy aging related to structure, chemical, neuropsychological and genetic.
- Memory Changes in Aging.
- Describe Important clinical conditions eg; geriatric syndrome, Alzheimer, carotid hypersensitivity.

**Color index:**

- Important.
- Girls slide only.
- Boys slide only.
- Dr's note.
- Extra information.

**Editing File**

# Aging

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**Definition**

aging is the progressive, universal decline first in functional reserve and then in function that occurs in organisms over time functional reserve:part of the function that isn't needed in adulthood, decreases daily(المخزون) function: what's actually needed

| Elderly (Young old) | Aged (Middle old) | Very Old (Oldest old) |
|---|---|---|
| Age 65-74* | Age 75-84* | Ages 85+* |

- Aging is not a disease; however, the risk of developing disease is increased, often dramatically, as a function of age.
- "Ageing is a development issue. Healthy older persons are a resource for their families, their communities and the economy."( goal is to keep elderly healthy)

## AGEING TERMS

**UNIVERSAL AGEING**
age changes that all people share)
eg. graying of hair, wrinkles

**PROBABILISTIC AGEING**
age changes that may happen to some (eg type two diabetes).
probability, not everyone will get it

**SOCIAL AGEING**
society's expectations of how people should act as they grow older
eg. unacceptable marriage/acts due to age

**CHRONOLOGICAL AGEING**
referring to how old a person is

**BIOLOGICAL AGEING**
an organism's physical state as it ages

# SUCCESSFUL AGEING*

**Aging can be better by having:**

- Active engagement with life
- Low probability of disease or disability
- High Cognitive & Physical function capacity

## Some Theories of Aging

These are all hypothetical mechanisms, as there isn't only one accepted mechanism for aging

| Hypothesis | How It May Work |
|---|---|
| Genetic | Aging is a genetic program activated in post-reproductive life when an individual's evolutionary mission is accomplished(regression after adulthood) |
| Oxidative stress It's the most acceptable mechanism | Accumulation of oxidative damage (by free radicals) to DNA, proteins, and lipids interferes with normal function and produces a decrease in stress responses |
| Mitochondrial dysfunction | A common deletion in mitochondrial DNA with age compromises function and alters cell metabolic processes and adaptability to environmental change |
| Hormonal changes | the decline and loss of circadian rhythm (hormones secreted during certain times eg.during the day/night) in secretion of some hormones produces a functional hormone deficiency state |
| Telomere shortening | Aging is related to a decline in the ability of cells to replicate in chromosomes |
| Defective host defenses | The failure of the immune system to respond to infectious agents and the overactivity of natural immunity create vulnerability to Infections |
| Accumulation of senescent cells | Renewing tissues become dysfunctional through loss of ability to renew → accumulation of senescent (aging) cells and body's inability to get rid of them |

# OXYGEN -free radicals (FR) & reactive oxygen species(ROS)

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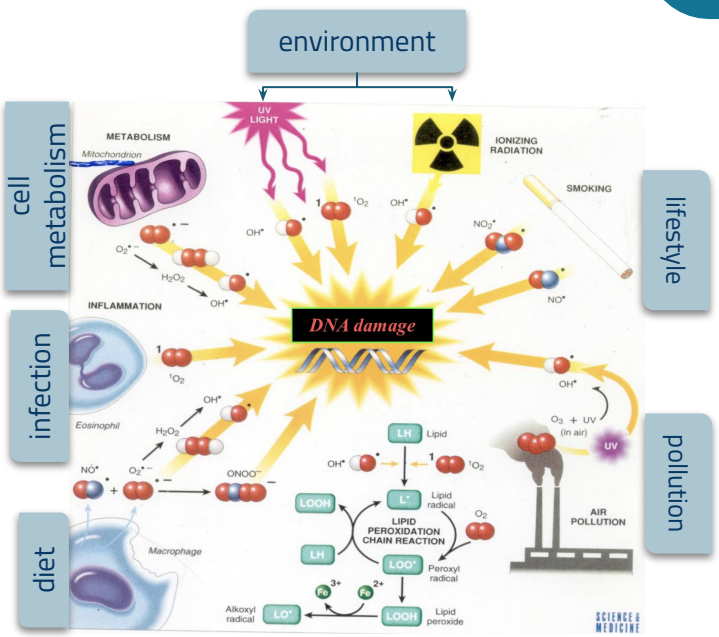
- Oxidative stress theory is the most famous theory of aging.
- OXYGEN - free radicals (O2-,H2O2,HO-) and reactive oxygen species (ROS) are produced in Mitochondria (NO+O2→ONOO-)
- So from where do we get these FRs and ROSs? Mitochondria

- oxidative stress comes from a group of oxygen derivatives. and **normally** oxygen is used in mitochondria to oxidise nutrients and produce energy, as a byproduct of these oxidative processes some oxygen free radical release.
- other sources for oxygen radicals to be released"not normal": exposure of ionizing radiation, smoking/inhale smoke, exposure to atmospheric pollution, multiple previous infections(autoimmunity by macrophages and neutrophils during their action release FR) , diets with a lot of preservatives or genetically modified foods.




## Mitochondria produce ROS

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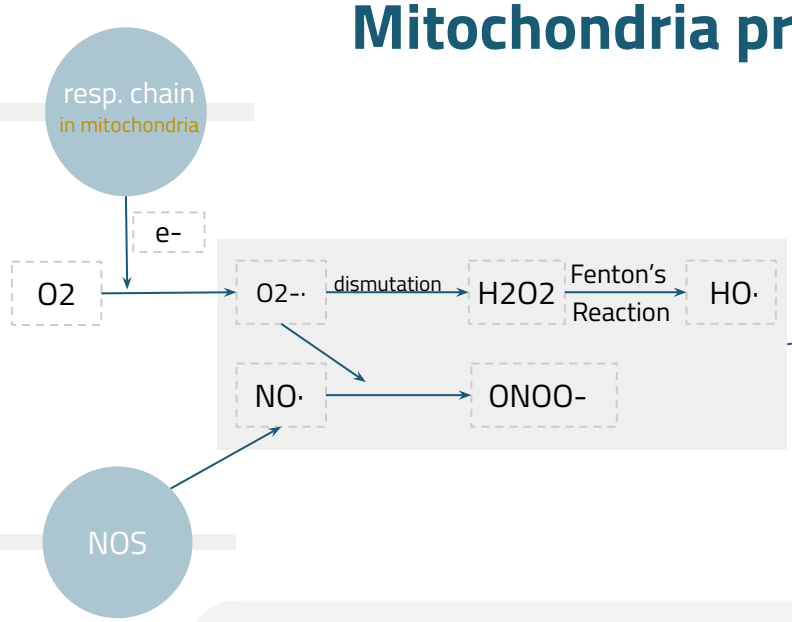


Molecules damaged

proportional to amount of free radicals

- protein thiol oxidation- MPT
- Lipid oxidation
- DNA oxidation
- Respiratory impairment
- Oxidation of extra-mitochondrial components

→ impairment of cell constituents → impaired function

The respiratory chain (resp. chain) produces superoxide radicals (O2-·), which generate hydrogen peroxide (H2O2) and hydroxyl radicals (HO·). Mitochondrial nitric oxide synthase (NOS) produces nitric oxide (NO·), which combines with O2-· to generate peroxynitrite (ONOO-). All these ROS may cause mitochondrial and cellular damage if present in excess. MPT, Mitochondrial permeability transition.

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# Leading Causes of Death Age 65+ "Medical Diagnoses"

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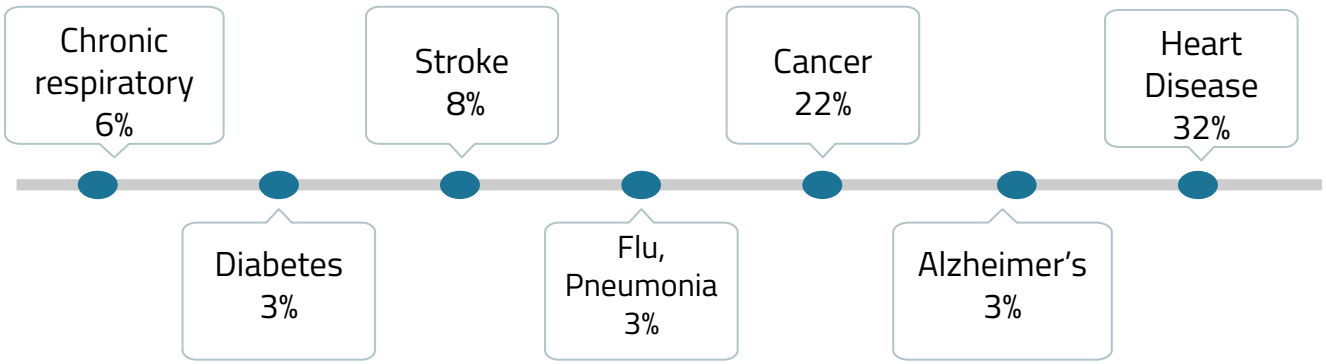


## Age Related Changes

1. Decreased height due to collapse of vertebrae, lean body mass and body water
2. Increased body fat Less body fluid
3. Consequence Changes in pharmacokinetics
4. Lower metabolic rate & Longer reaction times (between stimulus and response)
5. A functional decline in audition olfaction, and vision
6. Declines in certain memory functions Short term memory especially
7. Declines in kidney, pulmonary, and immune functions, declines in exercise performance, and multiple endocrine changes
8. Declines in sexual activity and in women menopause & in male impotence

(Craik and Salthouse, 1992; Hayflick, 1994, pp. 137-186; Spence, 1995

# AGING NERVOUS SYSTEM

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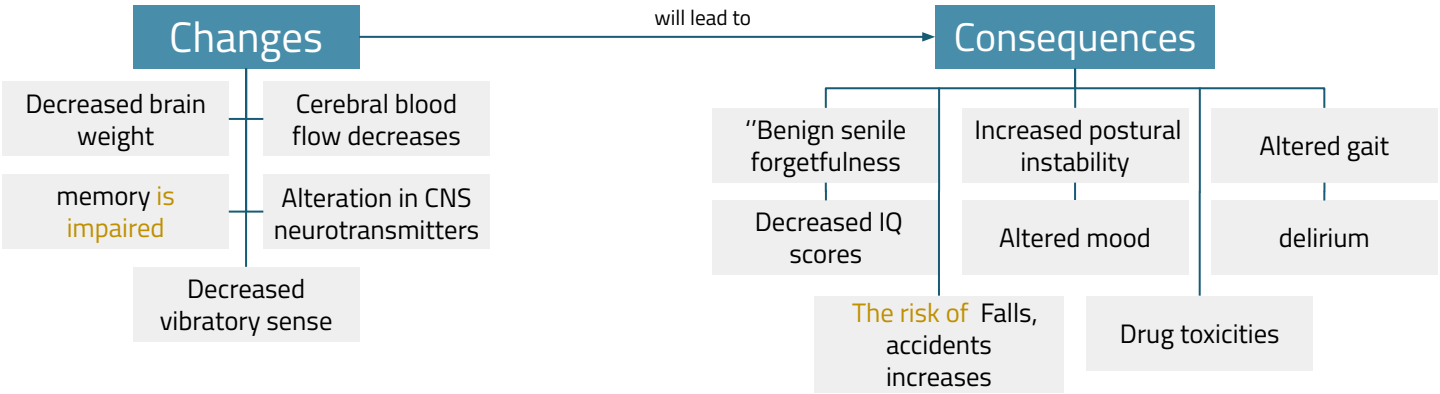
## "Use it or lose it!"

Individuals who remain mentally active perform better on cognitive tasks than those who engage in mental activity less often.

- Accelerated rate of brain shrinkage after age 50.
- Loss of 100,000 neurons in the cortex per day.
- Irreversible process of brain dysfunction.

brain weight is less with age ( less white matter→ functions are less "like memory") and less cerebral blood flow and less vibratory sense




## Aging & Brain Regions important

| Structure | Regional function |
|---|---|
| Basal ganglia | Becomes bright in appearance due to iron accumulation (movements affected) |
| Subarachnoid space | **Increase** in size due to brain shrinkage |
| Hippocampus | **Reduction in size** due to cell loss in the structure.<br>Part of limbic system Involved in learning & long term memory |
| Ventricles | **Increase** in size due brain shrinkage. |
| White Matter | **Reduction** in size due to neuronal atrophy in the deep brain.<br>Involved in information transmission. |

The cerebellum is the youngest brain region least affected by aging*

# Nervous System changes

| | |
|---|---|
| 1 | Neuronal loss is normal in the aging brain but the ability to learn remains generally unchanged |
| 2 | There is loss of dendritic arborization so less communication between neurons |
| 3 | Recall memory is affected more than cognitive function in normal aging |
| 4 | Lowered seizure threshold* |
| 5 | Reduced Sympathetic nervous system activity risk of bradycardia |
| 6 | Reduced Neurotransmitter levels (Dopamine, Serotonin, Glutamate) |
| 7 | changes in sleep patterns |
| 8 | increased risk of stroke |
| 9 | Intellectual functioning defined as "Stored" memory increases with age |
| 10 | Problem solving skills increase with age* |
| 11 | Cerebral atrophy shows up on CTs and MRI scans* |
| 12 | Abnormalities in EEG tracings* |
| 13 | Loss of lipids, and lipid turnover rate, and a decrease in catabolism and synthesis* |
| 14 | Loss of RNA (messenger and transcription) but not DNA* |
| 15 | Aging leads to increased cerebral amyloid* |
| 16 | Average amount of brain protein is reduced with a marked loss in multiple enzymes (carbonic anhydrase and the dehydrogenases) but with a relative increase in abnormal proteins such as amyloid in tangles and plaques* |

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# Aging Changes in Nervous System

- Aging is a major risk factor for most common neurodegenerative diseases, including mild cognitive impairment, dementias including Alzheimer's disease, cerebrovascular disease, Parkinson's disease and Lou Gehrig's disease.
- While much research has focused on diseases of aging, there are few informative studies on the molecular biology of the aging brain in the absence of neurodegenerative disease or the neuropsychological profile of healthy older adults.

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# Aging Changes in Nervous System

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**This Lecture will focus on reviewing the brain changes associated with healthy aging**

- **Structural changes**
- **Chemical changes**
- **Neuropsychological**
- **Genetic changes**

# 1-Structural changes.

## 1 Loss of neural circuits and brain plasticity:

- Some areas would be more vulnerable to aging eg: hippocampus and neocortical circuits.
- Age-related cognitive decline is not due to neuronal death but to synaptic alterations.
- This cognitive deficit is due to functional and biochemical factors such as changes in enzymatic activity, chemical messengers (calcium), or gene expression in cortical circuits

## 2 Thinning of the cortex:

- There is a **decrease** in grey matter volume between adulthood and old age, whereas white matter volume was found to **increase** from age 19-40, and decline after this age.

## 3 Age-related neuronal morphology:

- Dendritic arbors and dendritic spines* of cortical pyramidal neurons decrease in size and/or number in specific regions and layers of human and non-human primate cortex as a result of age.
- A 46% decrease in spine number and spine density has been reported in humans older than 50 compared with younger individuals.

*from 438 team:Dendritic arborization also called dendritic branching, describes the tree-like branching out of dendrites to make new synaptic connections, the branches themselves are called spines.

## 4 Neurofibrillary tangles:

- One of the important differences between normal aging and pathological aging is the location of neurofibrillary tangles. In normal, non-demented aging, the number of tangles in each affected cell body is relatively low. However, unlike tangles, plaques have not been found to be a consistent feature of normal aging

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# 2-Chemical changes.

## 1 Dopamine:

- Significant age-related decline in dopamine synthesis —> notably in the striatum
  the Striatum is a part of the basal ganglia and one of the components of the reward system and extrastriatal regions.
- Significant age-related decreases in all dopamine receptors D1 , D2 , and D3.
- The loss of dopamine with age is thought to be responsible for many neurological symptoms that increase in frequency with age, such as decreased arm swing and increased rigidity.
- Changes in dopamine levels may also cause age-related changes in cognitive flexibility.
- Substantia Nigra of mid brain secrets Dopamine

## 2 Serotonin:

- Decreasing levels of different serotonin receptors and the serotonin transporter, 5-HTT, have also been shown to occur with age. it will decline with age in the caudate nucleus, putamen, and frontal cerebral cortex.

## 3 Glutamate:

- Glutamate is another neurotransmitter that tends to decrease with age.
- lower glutamate concentration in the motor cortex compared to younger subjects especially in the parietal gray matter, basal ganglia, and to a lesser degree, the frontal white matter.
- glutamate may be useful as a marker of brain diseases that are affected by aging.

# 3-Neuropsychological changes.

## 1 Changes in orientation:

- Deficits in orientation are one of the most common symptoms of brain disease, hence tests of orientation are included in almost all medical and neuropsychological evaluations. Results of studies are somewhat inconclusive. So although current research suggests that normal aging is not usually associated with significant declines in orientation, mild difficulties may be a part of normal aging and not necessarily a sign of pathology. Orientation is the awareness of one's environment, with reference to place, time and people

## 2 Changes in attention:

- Many older adults notice a decline in their attentional abilities, results suggest that sustained attention increases in early adulthood and then remains relatively stable, at least through the seventh decade of life. It is worth noting that there are factors other than true attentional abilities that might relate to difficulty paying attention. For example, sensory deficits like hearing or vision may make attention it more difficult.

## 3 Changes in memory:

- Memory functions, more specifically those associated with the medial temporal lobe are especially vulnerable to age-related decline. ( decrease working & explicit memory but not implicit). (more details next slide)
- Changes in language.

## 4 Changes in performance:

- on verbal tasks, vary in predictable patterns with age. For example, behavioral changes associated with age include compromised performance on tasks related to word retrieval, comprehension of sentences with high syntactic and/or working memory demands, and production of such sentences

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# 4-Genetic changes.

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- Research focused on discovering the genetic component in developing Alzheimer's disease has also contributed greatly to the understanding the genetics behind normal or "non-pathological" aging. The human brain shows a decline in function and a change in gene expression This modulation in gene expression may be due to oxidative DNA damage at promoter regions in the genome.

دكتور العيال: مو مهم تعرف أنواع الجينات أهم شيء تعرف أنه مع زيادة العمر بيصير فيه جينات بيقل expression حقها وفيه جينات العكس وبيكون بينهم توازن أي خلل بالتوازن بيسبب early ageing

**Genes modulation**

**Genes that are down regulated over the age of 40 include:**

- GABA-A receptor gene
- Genes involved in long-term potentiation e.g. calmodulin
- Calcium signaling genes
- Synaptic plasticity genes
- Synaptic vesicle release and recycling genes

**Genes that are upregulated include:**

- Genes associated with stress response and DNA repair
- Antioxidant defense

# Cognitive changes in aging: memory & mental processing

Which type of explicit memory is is affected more? Episodic

**Short term memory (20s to 30s)**
IMMEDIATE RECALL DECLINE
WORKING MEMORY DECLINE

Example of working memory: Doing a task that involves multitasking such as Typing during a lecture, where you will be 1)typing 2)listening 3)lockin at the screen

**Long term memory**

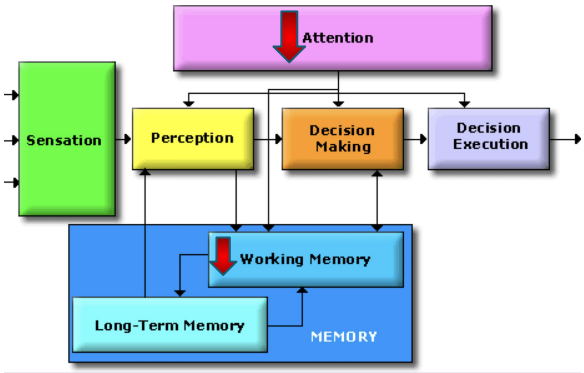
It is divided into:

1.Declarative or Explicit memory:stored in hippocampus and you retrieve it after consciously thinking about it

- Semantic memory:Words and language is retained & late to decline
- Episodic memory events: start to decline from middle age.

2.Skill memory or Implicit memory:Involves cerebellum, motor cortex, sensory cortex, visual areas and does not involve awareness. Procedural memory is retained.

- There is decline in mental processing via reduction of attentional ability and decline in ability in forming working memory (mainly includes short term memory) There is decline in explicit memory but implicit memory is retained (eg; driving a car, tying a shoe).



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# Hearing

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**Decreased hearing ability in old people could be due to:**

**Presbycusis:**
Part of normal aging (35% people over 60 years of age have bilateral, symmetric & progressive impairment for high pitched sounds sensorineural hearing loss)

**Cerumen impaction:**
is one of the most common reversible cause of conductive hearing loss in elderly

## -Sensorineural Hearing Loss:

Damage to the hair cells of the organ of Corti may be caused by intense noise, viral infections, ototoxic drugs (e.g., salicylates, quinine and its synthetic analogues, aminoglycoside antibiotics, loop diuretics such as furosemide and ethacrynic acid, and cancer chemotherapeutic agents such as cisplatin), fractures of the temporal bone, meningitis, cochlear otosclerosis (see above), Ménière's disease, and aging

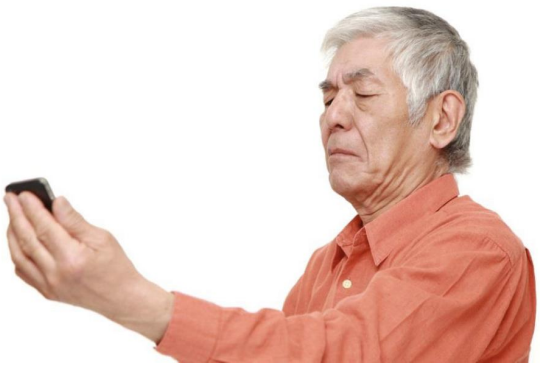
**Old people develop some hearing signs such as:**

- Paradoxical hypersensitivity to loud sounds
- Exacerbated in the presence of competing background noise


**In conclusion: Slow, persistent decline in hearing with age. The hearing loss begins in the sixth decade and is typically symmetrical and bilateral, beginning in the high-frequency range.**

# Vision

- Loss of ability to see items that are close up begins in the 40's (Presbyopia)
- Size of pupil grows smaller with age: focusing becomes less accurate
- Lens of eye yellows making it more difficult to see red and green colors
- Night vision not as acute
- Opacification of lens (Cataract) Cloudy lens
- Pupil less sensitive to light
- Arcus Senilis grey pigmentation of the eyes
- Sensitivity to glare increases



# Taste sensation disorders

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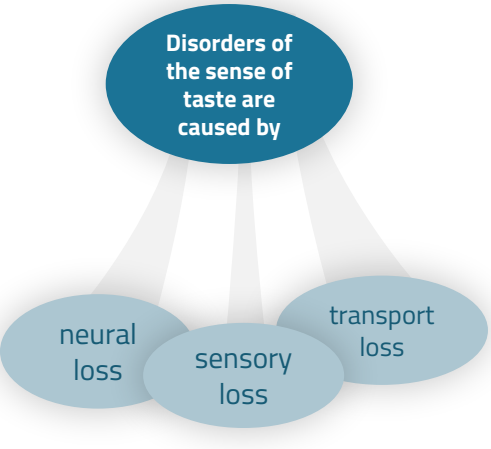
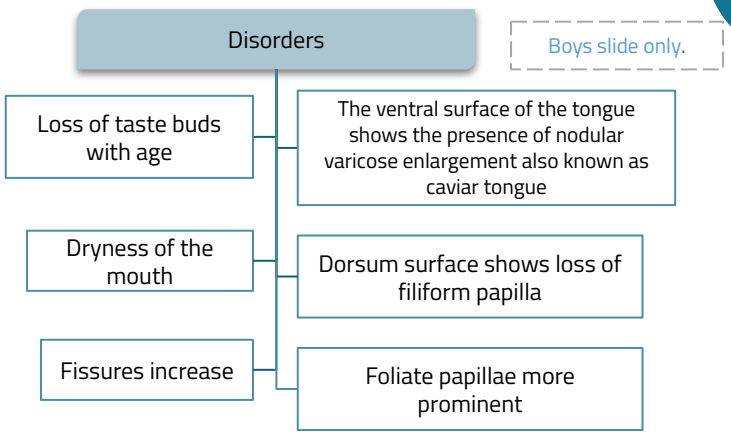
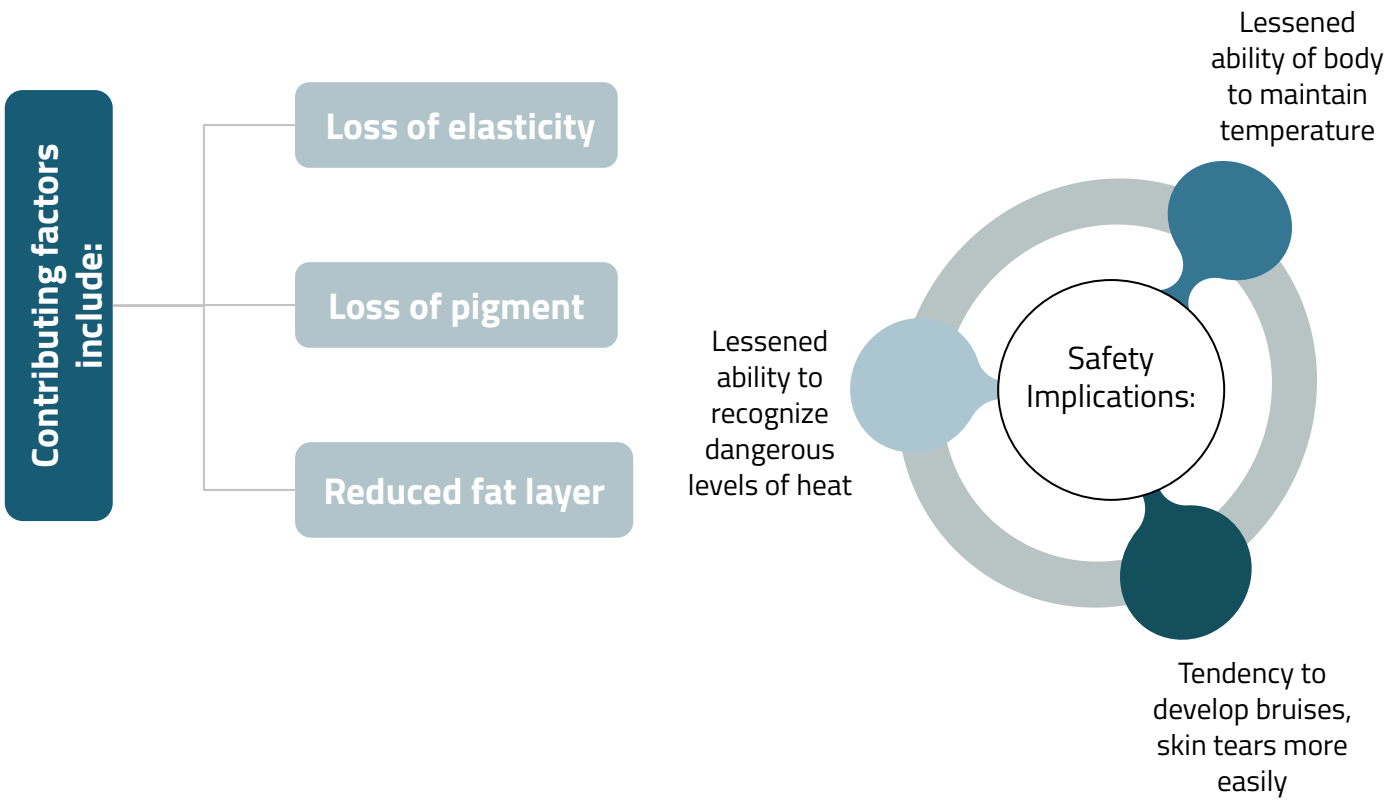



**Sensory gustatory losses**

caused by inflammatory and degenerative diseases in the oral cavity, a vast number of drugs, such as antithyroid and antineoplastic agents, radiation therapy to the oral cavity and pharynx, viral infections, endocrine disorders, neoplasms,and aging

# Pain and Sense of Touch



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# SLEEP PATTERNS

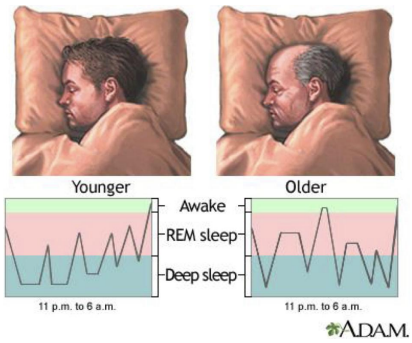


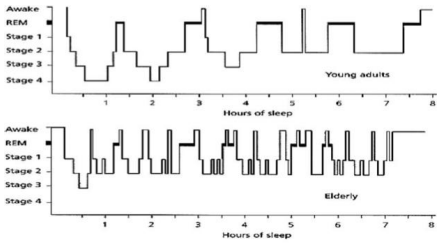
It tends to become more fragmented, with more awakenings during the night. They will get awake Three or four times each night.

The transition between sleep and waking up is often abrupt, which makes older people feel like they are a lighter sleeper than when they were younger.

Total sleep time stays the same or is slightly decreased (6.5 to 7 hours per night).

The proportion of slow wave sleep decreases relative to total sleep time, but the proportion of sleep that is REM sleep decrease or is unchanged




⬆ sleep latency
⬆ awakenings
⬇ REM sleep
⬇ Slow wave sleep

⬇ Total Sleep Time
⬆ Arousals

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# Sexual Dysfunction

Erectile dysfunction (ED) is not considered a normal part of the aging process. Nonetheless, it is associated with certain physiologic and psychological changes related to age.

In the Massachusetts Male Aging Study (MMAS), a community-based survey of men between the ages of 40 and 70, 52% of responders reported some degree of ED. Complete ED occurred in 10% of respondents, moderate ED occurred in 25%, and minimal ED in 17%

# Geriatric Syndromes

**It includes:**

- Dementia and Delirium
- Falls
- Urinary Incontinence
- Pressure Ulcers
  التقرحات اللي تحصل لهم بسبب ملازمة السرير لأنه يكون في ضغط مستمر على أجسامهم
- Functional Decline

# Dementia and Delirium

**Dementia**

Dementia is a syndrome of progressive decline in which multiple intellectual abilities deteriorate (worsen), causing both cognitive and functional impairment.

**Delirium**

Delirium is an acute state of confusion. فجأة ما يعرف الشخص اللي قدامه أو هو فين
Delirium may be the only manifestation of a life threatening illness (Infection or Drugs) in the older adult. Impairment cerebral blood flow: Transient ischemia

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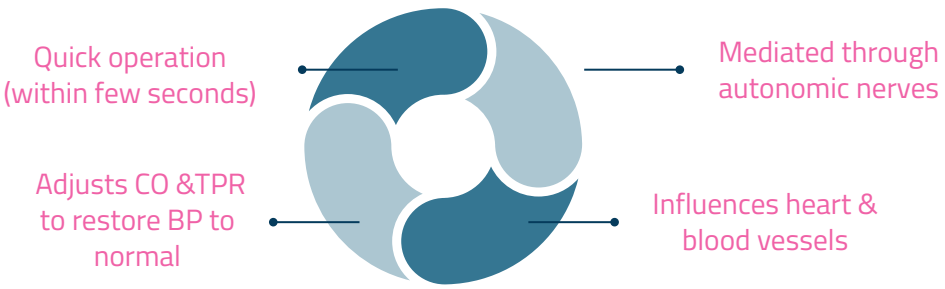
# Decline in Autonomic Functions

- Aging is associated with decreased Heart rate variability which is associated with increased mortality
- Changing position from supine to standing may trigger dizziness more frequently with ageing reflect the diminished cardiovascular sympathetic modulations and significant decline in overall autonomic functions
- Variation in heart rate response to deep breathing and valsalva manoeuvre is decreased because of impaired vagal control of heart rate with increasing age.

# Baroreceptor Reflex



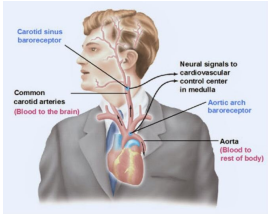

# Carotid sinus hypersensitivity

Carotid sinus syncope occurs when there is an exaggerated vagal response to carotid sinus stimulation

Provoked by wearing a tight collar, looking upwards or turning the head

Carotid sinus syndrome occurs in the elderly and mainly results in bradycardia

-Most common etiologies of atrioventricular block
-do not massage both carotids simultaneously



# Brief Geriatric Assessment Instruments

Old people have a special clinic in hospitals known as Geriatric clinic.

| Domain | Instrument | Comments |
|---|---|---|
| Cognition | | |
| Dementia | MMSE (mini-mental state examination) | Widely studied and accepted |
| | Time and change (T&C) test | Sensitive and quick |
| Delirium | CAM (confusion assessment method) | Sensitive and easy to apply |
| Affective disorders | GDS 5-question form (Geriatric depression scale) | Rapid screen |
| Visual impairment | Snellen chart | Universally used |
| Hearing impairment | Whispered voice | No special equipment needed |
| | Pure tone audiometry | Can be performed by trained office staff |
| Dental health | DENTAL | |
| Nutritional status | Weight loss of >4.5 kg (>10 lb) in 6 months or weight <45 kg (<100 lb) | |
| Gait and balance | "Timed Get Up and Go" test | Requires no special equipment |

# Alzheimer's Disease

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## Definition

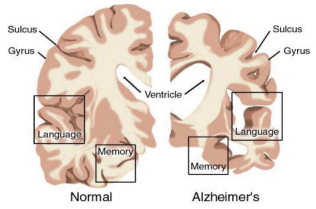
Alzheimer's disease defined as premature aging of the brain, usually beginning in mid-adult life and progressing rapidly to extreme loss of mental powers similar to that seen in very, very old age.

## Features:

| | |
|---|---|
| 1 | An amnesic type of memory impairment |
| 2 | Deterioration of language |
| 3 | Visuospatial deficits مشاكل في تحديد الاتجاهات |

## Signs:

| | |
|---|---|
| 1 | Motor and sensory abnormalities |
| 2 | Gait disturbances |
| 3 | Seizures. They are uncommon until the late phases of the disease. |

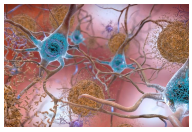



## Amyloid Plaques

Found outside the neuron

Plaques have not been found to be a consistent feature of normal aging. It is hallmark of Alzheimer's disease.

There is accumulation of amyloid plaques between nerve cells (neurons) in the brain.



Amyloid is a general term for protein fragments that the body produces normally. Beta amyloid is a protein fragment snipped from an amyloid precursor protein (APP).

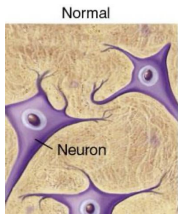
In a healthy brain, these protein fragments are broken down and eliminated. In Alzheimer's disease, the fragments accumulate to form hard, insoluble plaques.

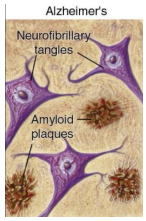
Amyloid plaques are seen in very old age 80 but if we find it in a person who is 40 this is due to Alzheimer's disease

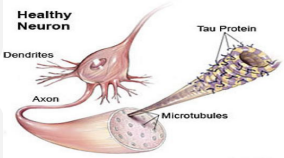
## Neurofibrillary Tangles

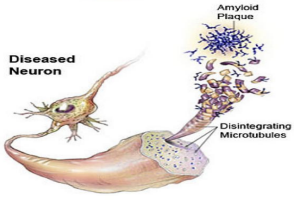
Found inside the neuron

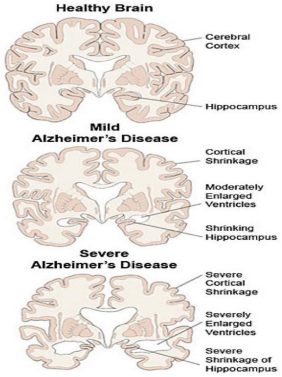
- These are insoluble twisted fibers found inside the brain's cells.
- Consist primarily of a protein called tau, which forms part of a structure called a microtubule. The microtubule helps transport nutrients and other important substances from one part of the nerve cell to another.
- In normal, non-demented aging, the number of tangles in each affected cell body is relatively low
- In Alzheimer's disease, however, the tau protein is abnormal and the microtubule structures collapse.



# MCQ & SAQ:



**Q1: oxygen free radicals (FR) and reactive oxygen species (ROS) are produced in:**
A. lungs
B.Mitochondria
C. chromosome
D. telomere

**Q2: leading cause of death age 65+?**
A.heart disease
B. smoking
C. stroke
D. cancer

**Q3: how many neurons does the cortex lose per day?**
A.100,000 neurons
B. 200,000 neurons
C. 2,000 neurons
D. 1,000 neurons

**Q4: Which one of these is a Neuropsychological change?**
A. Thinning of the cortex
B. Decrease Glutamate
C. Changes in memory
D. Amyloid Plaques

**Q5: ..... is a syndrome of progressive decline in which multiple intellectual abilities deteriorate.**

A. Dementia
B. Alzheimer's Disease
C. Delirium
D. Declarative memory

**Q6: Which one is an Autonomic Function that will decrease with age?**

A. Taste sensation
B. Urinary Incontinence
C. Arcus Senilis
D. Heart rate variability

answer key:
1: B
2: A
3: A
4: C
5: A
6: D

**1- What damage can the ROS cause if found excessively in MPT?**

**2- What happens to the Hippocampus with aging?**

**3-Enumerate 4 things that will happen to the vision with ageing.**

**4- Enumerate 3 Brief Geriatric Instruments with their uses.**

**A1: 1- protein thiol oxidation- MPT 2- Lipid oxidation 3- DNA oxidation 4- Respiratory impairment 5- Oxidation of extra-mitochondrial components**

**A2: Reduction in size due to cell loss in the structure. (Par of limbic system Involved in learning & long term memory)**

**A3: Slide 12**

**A4: 1-MMSE used for Dementia 2-CAM used for Delirium 3-GDS 5 used for Affective disorders**

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- Homoud Algadheb.
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