Anti- depressants

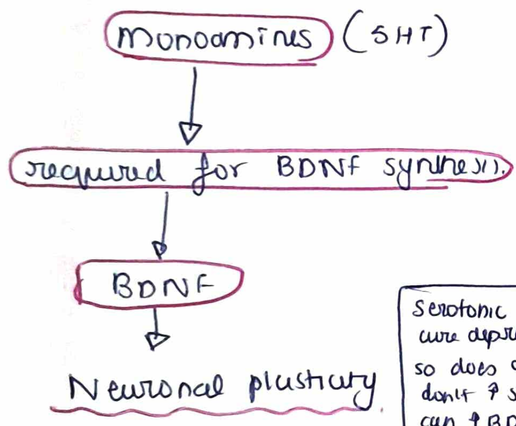
Earlier theory

→ depression occured due to deficiency of monoamines. ie, like 5HT.

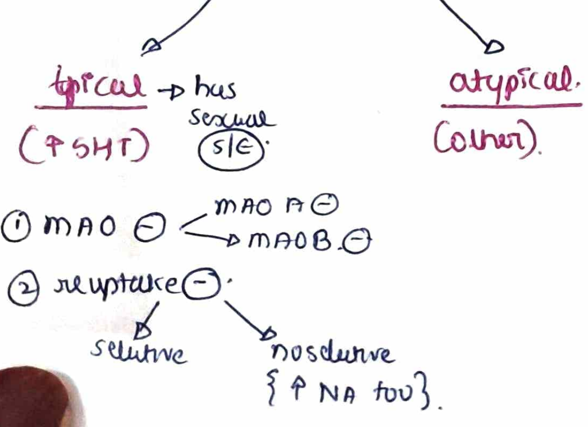
Latest

depression occurs due to deficiency of BDNF - which is required for neuronal plasticity.

actually



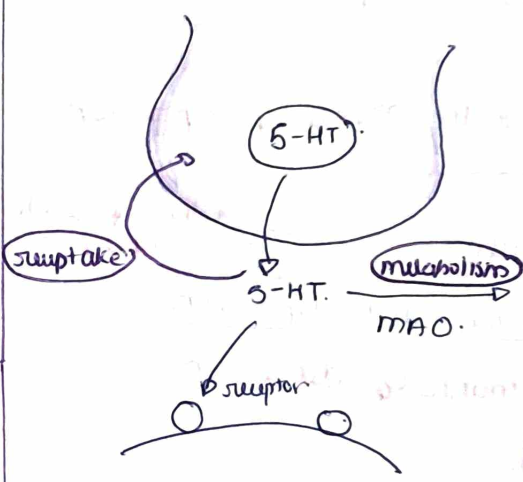
Serotonin can cure depression... so does drugs that don't ↑ 5HT, but can ↑ BDNF.



• drugs that ↑ serotonin has - Sexual side effects. loss of libido, late ejaculation etc.

Typical Antidepressants

they ↑ serotonin.



MAO A is present in Brain & many other areas outside

and MAO A metabolizes all monoamines 5HT, dopamine, adrenaline, Nor ad.

MAO - B - in Brain

It breaks mainly dopamine.

So in depression, we use

Selective MAO-A ⊖ → MOCLOBEMIDE ★ (only reversible MAO ⊖)

→ Reversible Inh. of MA. (RIMA)

reuptake ⊖

- Non-selective ⊖ → ↑5HT, ↑NA → leads to ↑HR, BP, HTN.
- Selective ⊖ → ↑5-HT only
  - Heart dis ✓ can be given
  - severe depression ✗
  - mild - moderate

So Non selective are avoided in Heart disease.

- Non-selective ↑ both (NA) & (5HT), Both are monoamines

So, they are more efficacious

In severe depression they are DOC

SNRI

Serotonin- NA reuptake ⊖.

TCA also does same... so y study seperately ?

TCA does that + H₁#, m# α# etc.

But SNRI only does what it says

i.e, ↑ 5HT & NA & Nothing else.

SNRI

- VENLAFAXINE ☆
- DES venlafaxine.
- MILNACIPRAN. ☆
- DULOXETINE ☆

DOC → ☆ severe depression

Non - Selective reuptake Inhibitors

- TCA
  - Imipramine
  - clomipramine
  - Amitriptyline
  - Nortriptyline.
- SNRI

But TCA are very very toxic

I
- H₁ # - sedation.
- m # → we will Think it's Atropine ! , ppt ACG.
- α # → ↓ BP.
- seizures.

Extreme blocking & seizures.

most lethal s/e → metabolic acidosis.

How to treat ? NaHCO₃ — DOC in TCA poisoning (Ice inde Time)

Tricky Qs (MCQ)

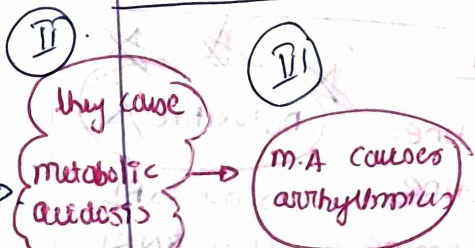
How to treat arrythmias in TCA toxicity ?

Anti arrthmics ? NO!

NaHCO₃ → In TCA arrythmias occurs due to ↑ in M.A so. NaHCO₃ ✓

anti arrthy — No role.

- we don't use TCA now. ✗
- In earlier time TCA were give little amnt at a time to prevent suicide.



they have life threatening & lethal

& we are giving it to ppls with suicide tendency ???

SSRI

Selective Serotonin reuptake Inhibitors

- can be used in ♡ ds.
- mild to moderate depression.

↓ DOC

If they ask you specifically...

1) what is DOC for severe depression

→ SNRI

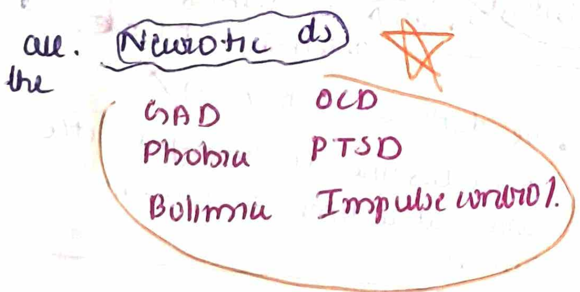
. If they ask u what is DOC in depression. {they mean mild-mod} • If severe, they'll specify.

→ SSRI.

SSRI is also DOC in ☆.

↓

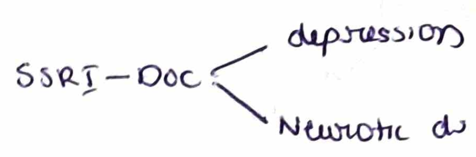
all the Neurotic ds



SSRI.

Fluoxetine.
Paroxetine.
Fluvoxamine.
Sertraline
Citalopram.

Duloxetine X is not SSRI. it is SNRI confusing in MCQ



S/E

so used in premature ejaculation.

MC: Nausea, Vomiting.
Sexual S/E – ↓ libido, delayed ejaculation.
• anxiety at begining of treatment.

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what? It's used in GAD & and it causes anxiety? How?

↑ serotonin causes anxiety at begining...
then receptors get's desensitized.
ie, they are given in chronic anxiety ✓
not in acute X

Discontinuation Syndrome

. If symptoms like tachycardia etc..

So, Tapering is done.

Fluoxetine. – min risk of discontinuation syndrome.

Atypical → ↓ sexual S/E

Bupropion → anti-smoking
Amineptin } ↑ 5HT reuptake. → we don't know how they ↑ BDNF.
Tianeptin }
MCQ which is SSRE enhancer? Amineptin & Tianeptin.
Mirtazapine
Amoxapine → anti-psychotic → D_2 # → can cause EPS → MCQ
Vilazodone } ↑ 5HT, $5HT_{1A}$ (+)
Vortioxetine }
Esketamine — NMDA R # → nasal route → in treatment resistant depression
Brexanolone → used in post partum depression

Bupropione → antismoking.
Buspirone → antianxiety.
→ drug in anxiety.

MIRTAZAPINE

* NSSA.

- mirtazapine → causes break fail by # α_2 in both 5HT & NA receptors
- also it blocks $5HT_2$, $5HT_3$, $5HT_4$ receptors

so, 5HT - only acts on $5HT_1$
But NA acts on α & β receptors.
∴ it is called NSSA

Nor adrenergic & specific serotonergic antidepressant