**Mechanism of Action**

Target of Benzodiazepine are on GABA receptor, because GABA is major inhibitory neurotransmitter in CNS. GABA is consisting of five-alpha, beta, gamma subunits that span the postsynaptic membrane. The influx of chloride ions cause hyperpolarization of the neuron and decrease neurotransmitter by inhibiting the formation of action potentials.

- **Empty receptor is inactive and coupled chloride channel is closed**
- **Binding of GABA open the chloride channel cause hyperpolarization**
- **Entries of chloride hyper polarize cells, make them difficult to depolarize and reduce neural excitability**
- **Binding of GABA is enhanced by benzodiazepine; result in more entry of negative chloride ions.**

### Sedative - Hypnotics

These drugs make exited patient calm and cool with and without causing sleep. They are differ only in concentration.

- **SEDATIVE**
  - Sedative are those drugs which make patient calm and relax without causing sleep, although patient may feel dizziness and may loss alertness or responsiveness.
  - Benzodiazepine
  - Newer Non BZD Hypnotics
    - Zopiclone
    - Zolpidem
    - Zaleplon

- **HYPNOTICS**
  - Hypnotics are the drugs which make person calm but also induce sleep. This is extension of sedative dose of any drugs. Sedative in large dose act as hypnotics.

### Different stages of Sleep pattern

- **Stage – 0**
  - Awake
  - Constitute 1 – 2%

- **Stage – 01**
  - Dozing
  - Constitute 3 – 6%

- **Stage – 02**
  - Unequivocal Sleep
  - Constitute 40 – 50%

- **Stage – 03**
  - Deep sleep transition
  - Constitute 5 – 8%

- **Stage – 04**
  - Cerebral Sleep
  - Constitute 10 – 20%

  - REM sleep
  - Paradoxical Sleep
  - Constitute 20 – 30%

### Pharmacological Action

- **Reduction of Anxiety**
- **Sedative-Hypnotics**
- **Anterograde Amnesia**
- **Muscle Relaxant**

### Benzodiazepine Mechanism Schematic Diagram

- Benzodiazepine binding to GABA receptor
- Chloride channel opening
- Hyperpolarization of neuron

### Classification - KD Tripathi

- **Sedation – Sleep – Anaesthesia**
- **Barbiturate**
- **Benzodiazepine**
- **Newer Non BZD Hypnotics**
  - Zopiclone
  - Zolpidem
  - Zaleplon

### Long Acting Phenytoine

- **Short Acting**
  - Butobarbitone
  - Phenobarbitone

- **Ultra Short Acting**
  - Thiopentone
  - Methohexitone

- **Stage – 01**
  - Awake
  - Constitute 1 – 2%

- **Stage – 02**
  - Dozing
  - Constitute 3 – 6%

- **Stage – 03**
  - Unequivocal Sleep
  - Constitute 40 – 50%

- **Stage – 04**
  - Cerebral Sleep
  - Constitute 10 – 20%

  - REM sleep
  - Paradoxical Sleep
  - Constitute 20 – 30%

- **Hypnotic**
  - Diazepam
  - Lorazepam
  - Clonazepam
  - Clobazam
  - Oxazepam
  - Alprazolam
  - Chlordiazepoxide

- **Antianxiety**
  - Diazepam
  - Lorazepam
  - Temazepam
  - Oxazepam
  - Flurazepam
  - Nitrazepam
  - Alprazolam

- **Anticonvulsant**
  - Diazepam
  - Lorazepam
  - Temazepam
  - Oxazepam
  - Flurazepam
  - Nitrazepam
  - Alprazolam
  - Chlordiazepoxide

- **Cell Membrane**
  - GABA Site
  - Chloride Channel

- **BZD Site**
  - Benzodiazepine Binding site

- **α, β, γ subunits**

- **Brain – GABA Site**

- **Brain – Chloride Channel Wide open**

- **Empty receptor is inactive and coupled chloride channel is closed**

- **Binding of GABA open the chloride channel cause hyperpolarization**

- **Entries of chloride hyper polarize cells, make them difficult to depolarize and reduce neural excitability**

- **Binding of GABA is enhanced by benzodiazepine; result in more entry of negative chloride ions.**

- **By Targeting GABA receptor**