Lidocaine (Lignocaine Local anaesthetic) Pharmacology

Lidocaine (also known as lignocaine) is a commonly used **amide-type local anesthetic** and a **Class 1B antiarrhythmic drug** . It acts primarily by **blocking voltage-gated sodium channels** , thereby preventing the initiation and conduction of nerve impulses and abnormal cardiac electrical activity.

Chemical Structure and Classification

Lidocaine belongs to the **amide group** of local anesthetics, which is characterized by the presence of an **amide bond (–CONH–)** . This bond features:

- A **nitrogen-hydrogen (–NH–)** group connected to a **carbonyl group (C=O)** .
- The aromatic ring in lidocaine is substituted with **two methyl groups in the ortho positions** , enhancing its lipophilicity.
- The amide nitrogen is linked to a **diethylamino side chain** , contributing to its anesthetic and antiarrhythmic properties

High-Yield

- **Ester anesthetics** (e.g., *procaine* , *cocaine*) have **one "i"** in their names.
- **Amide anesthetics** (e.g., *lidocaine* , *bupivacaine*) have **two or more "i"s** .

Indications

Lidocaine is widely used for both **local anesthesia** and **management of ventricular arrhythmias** . Key indications include:

- **Acute ventricular tachycardia** , especially post-myocardial infarction.
- **Ventricular arrhythmias related to digoxin toxicity.**
- **Open-heart surgery** as an antiarrhythmic agent.
- **Local anesthesia** for:
    - Infiltration and nerve block
    - Epidural and spinal anesthesia
    - Ocular procedures (eyelid or globe surgeries)
    - Minor surgical or dental procedures
- **Topical anesthesia** (gel, spray, or aerosol) for mucosal surfaces (e.g., larynx, bronchi, urethra)

Mechanism of Action

Lidocaine acts by:

- **Blocking open and inactivated voltage-gated sodium channels** , preventing sodium influx into neurons or cardiac cells.

- This **inhibits depolarization**, thereby halting nerve signal propagation or ectopic pacemaker activity.
- Lidocaine has **rapid association and dissociation kinetics**, making it more effective in **ischemic or depolarized cardiac tissue**.
- In nerves: it **prevents pain signal transmission**.
- In the heart: it **suppresses abnormal ventricular activity** without significantly affecting atrial conduction.

Pharmacokinetics and Pharmacodynamics

- **Onset**: Rapid
- **Duration**: Intermediate (1–2 hours)
- **Route**: Poor oral bioavailability ? **IV or parenteral administration** is preferred
- **Metabolism**: Hepatic (via CYP450 enzymes, primarily **oxidative N-dealkylation**)
- **Clearance**: Hepatic blood flow-dependent
- **Half-life**: ~2 hours in healthy individuals
- **Therapeutic range**: **1.5–5.0 mcg/mL**
- **Toxic levels**: >5 mcg/mL

Reduced clearance is seen in **heart failure** or **liver disease**, increasing the risk of toxicity.

Dosing and Administration

- For arrhythmias: **IV bolus** followed by continuous infusion.
- For minor surgery: **1% plain lidocaine**
- For dental procedures: **2% lidocaine with epinephrine**

Dose adjustments are necessary in:

- **Congestive heart failure**
- **Liver impairment**

Side Effects

Central Nervous System (CNS):

- Drowsiness
- Nausea, vomiting
- Paresthesia
- Twitching
- Seizures (focal ? generalized)

Cardiovascular System:

- Bradycardia
- Hypotension
- Negative inotropy
- Cardiac arrest or asystole (at toxic doses)

Lidocaine is considered **less cardiotoxic** compared to other antiarrhythmic drugs.

Toxicity

Toxicity occurs when plasma levels exceed **5 mcg/mL** .

Mnemonic: **SAMS**

- **S** : Slurred speech
- **A** : Altered CNS
- **M** : Muscle twitching
- **S** : Seizures

Drug Interactions

- **Negative inotropic agents** (e.g., beta-blockers, calcium channel blockers) may **reduce hepatic blood flow** , **increasing lidocaine plasma levels** and predisposing to toxicity.
- **Enzyme inducers/inhibitors** may also affect metabolism.