

Drug	Function
Glyceral Trinitrate Spray	<ul style="list-style-type: none"> <li>- Vasodilates, redistributes and venodilates</li> <li>- High first pass metabolism</li> <li>- Prodrug that becomes NO</li> <li>- Glutathione S-transferase acts on GTN to become NO</li> <li>- NO activated <b>GUANYLYL CYCLASE</b> to increase cGMP levels</li> <li>- cGMP dephosphorylates LC of myosin</li> </ul>
Aspirin	<ul style="list-style-type: none"> <li>- prevents thrombosis in coronary vessels by blocking platelet function</li> <li>- inhibits <b>CYCLOOXYGENASE</b> (covalent irreversible binding)</li> <li>- cyclooxygenase makes thromboxane and prostaglandins</li> <li>- is an NSAID (non-steroidal anti-inflammatory drug)</li> <li>- when used as NSAID → 350mg/4 hrs</li> </ul> <p><b>Low dose aspirin (50mg daily)</b></p> <ul style="list-style-type: none"> <li>- supresses platelet function (without anti-inflammatory effects)</li> <li>- platelets are in blood, so when aspirin is absorbed into splanchnic venous circulation at high concentration, it already <b>begins inactivating platelets</b></li> <li>- ALREADY PERFORMING FUNCTION so only low dose required</li> </ul> <p><b>Aspirin for Analgesia</b></p> <ul style="list-style-type: none"> <li>- Tissues that are targeted by aspirin to reduce pain make new COX frequently</li> <li>- Regular, large doses are therefore required</li> </ul> <p><b>REABSORPTION OF ASPIRIN</b></p> <ul style="list-style-type: none"> <li>- Aspirin is an ACID → so it is uncharged when the environment is acidic</li> <li>- When urine is acidified → aspirin uncharged → lipid soluble → reabsorbed</li> <li>- In <b>aspirin overdose</b>: forced ALKALINE DIURESIS <ul style="list-style-type: none"> <li>■ Give bicarbonate solution and lots of WATER</li> <li>■ Urine becomes basic → aspirin is charged → unable to be reabsorbed</li> </ul> </li> </ul>
Propofol	<ul style="list-style-type: none"> <li>- anaesthesia (intravenous)</li> <li>- acts on CNS and rapidly equilibrates with the brain (compartment theory)</li> <li>- QUICK drug action</li> </ul>
D-Tubocurarine	<ul style="list-style-type: none"> <li>- Causes <b>paralysis</b> and <b>respiratory arrest</b></li> <li>- <b>ANTAGONIST</b> of nicotinic receptor</li> <li>- Has quaternary ammonium ion (same as ACh) that prevents it from being absorbed in GIT because of the charge</li> <li>- Positive charged ions have difficulty getting through lipid environment</li> <li>- Curare like drugs used for anaesthetic (surgery)</li> <li>- Competitively binds <b>ALPHA SUBUNIT OF NICOTINIC RECEPTOR</b></li> </ul>
Beta Blockers	<ul style="list-style-type: none"> <li>- <b>Beta-1 adrenoceptor</b> antagonists</li> <li>- Block overactive sympathetic NS</li> </ul>
Atropine	<ul style="list-style-type: none"> <li>- Increases HR (treats bradycardia)</li> <li>- Inhibits parasympathetic tone of vagus</li> <li>- <b>ANTAGONIST</b> of MUSCARINIC receptor</li> <li>- Was used as anaesthetic, prevents glandular secretions (parasympathetic)</li> <li>- Dilates pupil</li> <li>- Anticholinergic</li> </ul>
Propranolol	<ul style="list-style-type: none"> <li>- Competitive <b>ANTAGONIST</b> at <b>beta-1 adrenoceptor</b> (also beta-2)</li> <li>- Decreases heart rate (treats tachycardia)</li> <li>- BETA BLOCKER</li> <li>- Modification of the catecholamine structure of noradrenaline</li> </ul>
Morphine	<ul style="list-style-type: none"> <li>- From opium poppy (analgesia)</li> <li>- High first pass metabolism (intravenous or intramuscular admin)</li> <li>- <b>Competitive AGONIST</b> of OPIOID RECEPTORS (GPCR)</li> </ul>