

ATROPINE

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Primary Disciplinary Field(s): Pharmacology, Toxicology, Ophthalmology, Anesthesiology

1. Core Definition and Classification

Atropine is a highly significant pharmaceutical agent classified chemically as a tropane alkaloid and pharmacologically as a potent antimuscarinic agent. It functions as a competitive, reversible antagonist of muscarinic acetylcholine receptors (mAChRs). This mechanism places it squarely within the class of **anticholinergic drugs**. Derived naturally from plants belonging to the Solanaceae family, notably Deadly Nightshade (*Atropa belladonna*) and Jimsonweed (*Datura stramonium*), it is one of the principal belladonna alkaloids. While historically extracted directly from these sources, modern pharmaceutical applications often utilize synthetically produced atropine to ensure purity and consistency, although the chemical structure remains identical to the naturally occurring levorotatory form (hyoscyamine) and the racemic mixture (atropine).

The primary therapeutic utility of atropine stems from its ability to inhibit the parasympathetic nervous system (PNS). By blocking the action of acetylcholine at peripheral muscarinic receptor sites, atropine disrupts the signal transmission responsible for "rest and digest" functions, leading to systemic effects such as increased heart rate, decreased glandular secretions, and relaxation of smooth muscles. Its widespread physiological impact makes it a versatile tool in clinical medicine, ranging from routine diagnostic procedures to critical emergency interventions, including its use to counteract certain types of poisoning and to manage cardiac dysfunction.

2. Chemical Structure and Source

Chemically, atropine is designated as a racemic mixture of (R)- and (S)-hyoscyamine. The biologically active component found in natural plant sources is primarily the levorotatory isomer, L-hyoscyamine. The core molecular structure is based on the tropane skeleton, a bicyclic organic compound common to several important alkaloids found in nightshade plants. The presence of this tropane ring system is critical for its ability to bind effectively to and antagonize muscarinic receptors throughout the body. The natural derivation from *Atropa belladonna*, often referred to as the **belladonna alkaloid**, highlights its historical link to traditional medicine and toxicology, given the plant's inherent toxicity.

The biosynthesis of atropine involves the esterification of tropine and tropic acid. While plant extraction remains a potential source, synthetic production methods allow for the large-scale manufacturing and stringent quality control necessary for modern pharmacological standards. The close chemical relationship between atropine and other tropane alkaloids, such as **scopolamine** (hyoscine), underscores a shared general pharmacological profile, though scopolamine exhibits

more pronounced effects on the central nervous system (CNS) compared to atropine's predominantly peripheral action. Understanding this structural relationship is key to differentiating their nuanced clinical uses and comparative side effect profiles in various patient populations.

3. Mechanism of Anticholinergic Action

Atropine acts primarily as a non-selective, competitive antagonist at all five known subtypes of muscarinic receptors (M1 to M5). Its competitive binding means it occupies the receptor site normally reserved for the excitatory neurotransmitter **acetylcholine** (ACh), thereby preventing ACh from initiating a parasympathetic response. This antagonism is reversible; high concentrations of ACh can overcome the block, although clinically, atropine exerts a profound and rapid inhibitory effect on parasympathetic activity, leading to immediate physiological changes. The degree of blockade is proportional to the concentration of atropine and the receptor density in the target tissue.

The clinical consequence of blocking these muscarinic receptors is a systemic cessation of various cholinergic functions. For example, in the heart, M2 receptor blockade leads to increased sinus node firing rate and enhanced conduction velocity through the atrioventricular node, resulting in **tachycardia** (increased heart rate). In glandular tissue, M3 blockade halts secretion, causing characteristic symptoms such as dry mouth (xerostomia) and reduced sweating (anhidrosis). In the eye, blockade of M3 receptors in the iris sphincter muscle causes relaxation and subsequent pupil dilation (mydriasis), while blockade in the ciliary muscle inhibits the focusing reflex (cycloplegia).

4. Detailed Pharmacological Effects

The pharmacological profile of atropine is highly diverse, impacting multiple organ systems through the effective disruption of parasympathetic control. Cardiovascular effects are critical in acute settings, where atropine is used to rapidly counteract severe **bradycardia** (abnormally slow heart rate), particularly that resulting from excessive vagal stimulation or certain drug toxicities. By accelerating the heart rate, atropine helps stabilize cardiac output and restore perfusion pressure, which is vital in hypotensive or shocked patients. However, caution must be exercised, as excessive dosing can lead to dangerously high heart rates.

Atropine significantly affects the gastrointestinal and respiratory systems. It causes relaxation of smooth muscles throughout the GI tract, leading to a marked reduction in motility and the cessation of intestinal spasms. This antispasmodic property was historically leveraged in the treatment of various gastrointestinal disorders. In the respiratory tract, atropine induces **bronchodilation** by relaxing the bronchial smooth muscles and, crucially, reduces the volume of bronchial and salivary secretions. This reduction in bodily secretions is invaluable when atropine is employed as an **adjunct to anesthesia**, as it minimizes the risk of laryngeal spasm or aspiration pneumonia during

endotracheal intubation and surgical procedures.

Furthermore, the widespread reduction of exocrine secretions affects nearly all glands, including sweat, salivary, and lacrimal glands. While this secretory inhibition is beneficial pre-operatively, the resulting dryness of the mouth, skin, and eyes are the most frequently reported side effects. The constellation of severe anticholinergic symptoms resulting from toxicity is often summarized by the mnemonic: "hot as a hare" (hyperthermia due to lack of sweating), "blind as a bat" (mydriasis and cycloplegia), "dry as a bone" (anhidrosis and xerostomia), "red as a beet" (cutaneous vasodilation), and "mad as a hatter" (CNS effects like delirium and psychosis).

5. Primary Clinical Applications

Atropine holds specific critical roles across several medical disciplines. Its most routine and widely recognized application is in **ophthalmology**. Topical administration of atropine eye drops is utilized to achieve potent and prolonged mydriasis (pupil dilation) and cycloplegia (paralysis of the ciliary muscle). This dual effect ensures the ophthalmologist has an unobstructed, still view of the posterior segment of the eye, aiding in comprehensive retinal examinations and accurate refraction measurements. Due to its extended duration of action, atropine is often reserved for therapeutic interventions, such as treating painful inflammatory conditions like iritis and uveitis, or for specific pediatric refractions where reliable cycloplegia is essential.

In **cardiology and critical care medicine**, atropine is designated as the standard initial pharmacologic treatment for clinically significant symptomatic bradycardia, defined as a dangerously slow heart rate often accompanied by symptoms such as hypotension, dizziness, or chest pain. It works rapidly via intravenous administration to reverse the pathological effects of excessive vagal tone. Moreover, atropine is an indispensable antidote in the management of **organophosphate poisoning** (resulting from exposure to certain pesticides or chemical nerve agents). These toxins inhibit acetylcholinesterase, leading to massive, potentially fatal accumulation of acetylcholine; atropine acts by competitively blocking the overstimulated muscarinic receptors, thus reversing the life-threatening cholinergic crisis, particularly the pulmonary and cardiovascular manifestations.

Its role as an **adjunct to anesthesia** remains vital, though evolving. While newer, shorter-acting agents have replaced it for routine drying of secretions, atropine is still essential in the operating room for specific scenarios. These include managing reflex bradycardia that can occur during surgical manipulation of vagally innervated structures (such as the abdominal viscera or ocular muscles) or when used to rapidly reverse the effects of certain non-depolarizing neuromuscular blocking agents, allowing the patient to regain spontaneous muscular control post-surgery.

6. Pharmacokinetics and Dosage

Atropine is characterized by rapid and effective absorption following all common routes of administration, including oral, intramuscular, and intravenous injections. When administered intravenously, its therapeutic effects on the heart are typically observed within one to two minutes, a characteristic that makes it uniquely suited for rapid emergency intervention in cardiac arrests or severe bradyarrhythmias. Following absorption, atropine is widely distributed throughout the body tissues, and, importantly, it possesses sufficient lipophilicity to cross the blood-brain barrier, resulting in the potential for central nervous system effects, especially at higher concentrations.

Metabolism of atropine occurs predominantly in the liver through enzymatic hydrolysis, yielding inactive metabolites, and conjugation reactions. A significant portion of the administered dose is also excreted unchanged via the kidneys. The elimination half-life generally ranges from 2 to 4 hours in healthy adults, but careful consideration must be given to dose adjustments in vulnerable populations. The half-life can be notably prolonged in neonates, the elderly (who are also more susceptible to CNS side effects), and patients suffering from significant hepatic or renal impairment, necessitating a reduction in dosage frequency or total amount to prevent toxic accumulation. Dosage is highly variable based on the clinical indication; for instance, the required dosing for ophthalmology is miniscule compared to the extremely high, repeated doses necessary to counteract severe organophosphate poisoning.

7. Adverse Effects and Toxicity

The adverse effect profile of atropine is extensive and predictable, as it is a direct consequence of its therapeutic mechanism--a systemic suppression of parasympathetic tone. Common, mild side effects experienced at therapeutic doses include profound xerostomia (dry mouth), reduced tear production leading to dry eyes, temporary blurred vision resulting from cycloplegia, difficulty with urination (urinary retention), and constipation due to decreased gastrointestinal smooth muscle motility. These peripheral effects are generally well-tolerated but require monitoring.

In sensitive individuals, particularly pediatric and geriatric patients, or when administered at elevated dosages, atropine can induce significant central nervous system effects. These can range from mild agitation and restlessness to severe manifestations such as disorientation, confusion, hallucinations, and frank psychosis (delirium), often accompanied by amnesia. The potential for CNS toxicity underscores the need for careful dose calculation and surveillance, especially in prolonged therapeutic regimens.

Acute **atropine poisoning** or overdose represents a serious medical emergency, presenting as a severe, life-threatening manifestation of the anticholinergic syndrome. Symptoms include dangerously fast heart rates (severe tachycardia), malignant hyperthermia (due to failure of sweating), dangerously elevated blood pressure, severe urinary retention, and profound,

sometimes violent, psychiatric disturbances. The definitive treatment involves robust supportive care, including rapid cooling to manage hyperthermia and the use of the specific pharmacological antidote, **physostigmine**, which is a reversible acetylcholinesterase inhibitor that effectively raises synaptic acetylcholine levels to competitively overcome the atropine blockade at the receptor sites.

8. Historical Context and Legacy

The therapeutic and toxic properties of belladonna-derived compounds have been recognized for centuries. The plant *Atropa belladonna* itself derives its name ("beautiful lady") from its use during the Renaissance, particularly in Italy, where women would apply extracts to their eyes to induce dilation of the pupils (mydriasis), which was considered aesthetically desirable and associated with beauty. This ancient practice provides clear historical evidence of the potent ocular effects of atropine, long before its chemical isolation.

In the 19th century, atropine was successfully isolated in its pure alkaloid form, marking a key milestone in the development of modern pharmacology. This isolation permitted precise dosing and rigorous scientific study, fundamentally advancing the understanding of the autonomic nervous system. Today, while pharmaceutical research has yielded more receptor-selective anticholinergics for chronic diseases, atropine remains essential in acute care settings. Its rapid efficacy in managing cardiac emergencies and its unparalleled role as an antidote for cholinergic poisoning cement its legacy as one of the most fundamental and indispensable drugs in clinical medicine.

Further Reading

[Atropine \(Wikipedia\)](#)

[Atropine - StatPearls](#)

[Atropine Drug Information](#)