

ATROPINE-COMA THERAPY (ACT)

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Introduction and Definition

Atropine-Coma Therapy (ACT) stands as a notable, albeit controversial and ultimately abandoned, methodology within the history of psychiatric treatment. Defined precisely, ACT was a historical method employed primarily during the mid-twentieth century aimed at treating severe cases of agitation, intractable psychoses, and certain symptom clusters associated with **schizophrenia** by intentionally inducing a profound state of coma. This therapeutic approach relied upon the administration of massive, controlled doses of **atropine sulfate**, a potent anticholinergic agent, which temporarily disrupted central nervous system function to the extent required to render the patient unconscious for a prescribed period. The underlying, though scientifically tenuous, rationale was that such a drastic physiological interruption might reset pathological neural circuits or alleviate acute, distressing psychological states that were refractory to conventional sedation or early pharmacological interventions of the time.

The core procedure of ACT involved pushing the patient past the standard therapeutic threshold for atropine--a drug typically used in much smaller doses to manage bradycardia or reduce secretions--into a state of extreme anticholinergic toxicity. This severe systemic challenge resulted in a deep, pharmacologically induced stupor or coma, often accompanied by intense peripheral side effects such as extreme tachycardia, severe xerostomia (dry mouth), urinary retention, and dangerous hyperthermia due to the suppression of thermoregulatory sweating mechanisms. Because of the inherent toxicity and the narrow margin between the coma-inducing dose and a lethal dose, ACT demanded meticulous medical supervision, often requiring specialized hospital units equipped to handle the acute risks associated with severe poisoning, including the potential for respiratory failure or cardiac collapse.

It is crucial to categorize ACT not merely as deep sedation, but as a form of **pharmacological shock therapy**, aligning conceptually with other drastic treatments popular in the era, such as Insulin Shock Therapy (IST) and the early use of Electroconvulsive Therapy (ECT). While ECT utilized electrical energy and IST utilized hypoglycemia, ACT leveraged profound anticholinergic blockade to achieve its desired "shock" effect. The formal, precise definition of ACT centers on the specific use of atropine sulfate to achieve a coma state lasting several hours, often repeated over a course of weeks, distinguishing it from general sedation techniques or continuous narcosis aimed merely at prolonged sleep without the requisite depth of coma induced by atropine toxicity.

Historical Context and Development

The origins of Atropine-Coma Therapy are rooted in the broader mid-20th century psychiatric environment, characterized by limited understanding of neurotransmitter function and a pervasive frustration regarding the lack of effective, reliable treatments for severe, chronic mental illnesses, particularly schizophrenia. The decade leading up to the 1950s was marked by the ascendancy of

"shock" therapies, driven by the belief that a massive physiological insult could somehow interrupt or reverse the progression of severe psychopathology. ACT specifically gained traction in certain European psychiatric circles, particularly in the Soviet Union and other Eastern Bloc nations, where the technique was refined and promoted, though it remained controversial and less widespread than ECT or IST in Western Europe and the United States.

The proponents of ACT often cited anecdotal evidence suggesting that patients emerging from the induced coma demonstrated a temporary, and sometimes sustained, reduction in psychotic symptoms, particularly agitation, catatonia, and severe anxiety. This perceived efficacy fueled its adoption, despite the overwhelming physiological burden placed upon the patient. The therapeutic approach was sometimes linked to the idea of "pathophysiological mechanisms" where psychiatric illness was viewed as a sustained, damaging functional disequilibrium that required a powerful, disruptive intervention to force the system back into equilibrium. The intense anticholinergic state achieved by massive atropine dosing served as this powerful disruptor.

The development trajectory of ACT paralleled that of other dangerous interventions. Initial enthusiasm was often based on small, uncontrolled series of cases, and the lack of standardization across institutions meant that treatment protocols varied widely, contributing significantly to inconsistency in outcomes and mortality rates. While some clinicians viewed ACT as a last resort for otherwise hopeless cases, its increasing documentation of severe morbidity and outright mortality meant that it never achieved the relative acceptance or widespread integration into mainstream psychiatric practice that ECT eventually secured following its own refinements and safety improvements.

The context of its use is also critical: ACT was utilized during a period when the primary pharmacological tools available were limited to barbiturates, bromides, and early sedatives. The eventual introduction of the first generation of effective antipsychotic medications, beginning with **chlorpromazine** in the early 1950s, dramatically shifted the therapeutic landscape. These new medications offered a far safer, more specific, and manageable way to control psychotic symptoms, rendering the extreme risks associated with ACT increasingly unjustifiable and leading to its rapid obsolescence in most global psychiatric centers.

Pharmacological Mechanism of Atropine

Atropine sulfate is pharmacologically classified as a competitive antagonist of muscarinic acetylcholine receptors (mAChRs), meaning it blocks the action of the neurotransmitter acetylcholine at these receptor sites throughout the central and peripheral nervous systems. In standard medical use, small doses of atropine are utilized for their peripheral effects, such as increasing heart rate (by blocking vagal stimulation) or drying up secretions (by blocking glandular activation). However, Atropine-Coma Therapy required doses exponentially higher than standard

therapeutic levels to penetrate the blood-brain barrier effectively and induce the desired central nervous system depression leading to coma.

The mechanism of inducing coma through atropine involves the generation of a severe **central anticholinergic syndrome**. Acetylcholine plays a critical role in memory, learning, and arousal; massive blockade of central mAChRs profoundly disrupts these functions, leading rapidly to confusion, delirium, hallucinations, and ultimately, severe CNS depression, stupor, and coma. The psychological symptoms targeted by ACT--agitation and psychosis--were theorized to be related to or relieved by this complete systemic shutdown, although the exact neural pathways involved in this "therapeutic" effect were poorly understood and remain speculative in retrospect.

The peripheral effects of ACT were intense and dangerous. The typical anticholinergic triad of symptoms--often summarized as "hot as a hare, blind as a bat, dry as a bone, red as a beet, and mad as a hatter"--was intentionally maximized. The blockade of sweating combined with increased metabolic activity often led to severe, life-threatening **hyperthermia** (fever), requiring continuous external cooling measures. Furthermore, the extreme tachycardia resulting from vagal blockade placed immense strain on the cardiovascular system, posing a major risk of arrhythmia, myocardial ischemia, and subsequent cardiac arrest, especially in patients with underlying cardiac vulnerabilities.

Achieving the required depth of coma without causing fatal respiratory or circulatory collapse demanded highly precise dosing and constant titration, often involving the administration of hundreds of milligrams of atropine over a short period, doses that would be considered fatal in non-monitored settings. The therapy essentially operated at the very edge of pharmacological lethality, underscoring why intensive medical monitoring, including continuous cardiac and respiratory support preparation, was non-negotiable for anyone undergoing the ACT procedure.

The Procedure and Administration Protocol

The established protocol for Atropine-Coma Therapy was complex, resource-intensive, and highly standardized across institutions that practiced it, largely due to the extreme danger involved. The initial step involved extensive preparatory work, including thorough physical examinations to rule out underlying conditions (particularly cardiovascular or renal impairment) that would render the patient incapable of surviving the stress of severe anticholinergic poisoning. Patients were admitted to specialized, highly staffed units, sometimes referred to as 'shock wards,' where continuous observation by physicians and nurses trained in toxicology management was mandatory throughout the entire course of treatment.

The actual administration of the **atropine sulfate** was a carefully titrated process. ACT was typically administered intravenously or intramuscularly, beginning with small initial doses which were then rapidly escalated over successive hours or days until the coma threshold was reached.

This titration phase was critical; the goal was to induce the desired depth of unconsciousness--a state where the patient was unresponsive to painful stimuli--while carefully monitoring vital signs, particularly heart rate, body temperature, and respiration, to avoid lethal overdose. Achieving the therapeutic coma often required doses ranging from 100 mg to well over 200 mg of atropine sulfate per session, depending on individual patient metabolism and tolerance.

Once the coma was induced, it was typically maintained for a duration ranging from two to six hours. During this period, the medical team's primary focus shifted to managing the severe side effects. This included aggressive cooling techniques (ice packs, cooling blankets) to counteract hyperthermia, maintaining airway patency, and potentially administering fluids. Following the prescribed duration, supportive care was utilized to allow the drug concentration to decrease gradually until the patient slowly emerged from the comatose state, which was usually marked by a period of extreme confusion, delirium, and residual anticholinergic effects that could persist for hours.

A full course of ACT was rarely a single session; rather, it often involved a series of induced comas, typically administered every few days, totaling anywhere from six to twenty sessions over a course of several weeks. This repetitive exposure was believed necessary to achieve a sustained therapeutic effect, akin to the protocols used for Insulin Shock Therapy. The cumulative nature of the treatment amplified the risks, placing a prolonged physiological burden on the patient's organs and increasing the likelihood of complications such as aspiration pneumonia, persistent cardiac issues, or irreversible central nervous system damage stemming from prolonged hyperthermia or circulatory strain.

Clinical Application and Target Patient Populations

Atropine-Coma Therapy was never intended as a general psychiatric treatment; rather, it was reserved for specific, highly resistant clinical scenarios. The primary target populations were individuals suffering from acute, severe psychotic episodes, particularly those characterized by extreme agitation, destructive behavior, or severe catatonic stupor that did not respond adequately to less invasive treatments available at the time. The rationale for its use in these cases was one of desperation: clinicians felt compelled to try any intervention that offered even a remote chance of alleviating profound suffering and dangerous behavioral instability.

Within the scope of **schizophrenia**, ACT was sometimes applied to patients experiencing persistent excitement or chronic destructive behavior. Early proponents suggested that the forced neurological reset provided by the deep coma might break the cycle of pathological thinking and behavior that characterized these severe states. It was hoped that the patient, upon awakening, might exhibit a temporary remission or be rendered more amenable to psychotherapy or rehabilitation efforts that had previously been thwarted by the intensity of their illness.

However, the therapeutic scope of ACT was inherently limited. It was not utilized for mood disorders like depression (where ECT was already established) or for anxiety disorders. Its application was almost exclusively focused on the severe end of the psychotic spectrum, reflecting the desperate search for biological interventions in the absence of effective targeted medications. Critically, the therapy was largely palliative for acute symptoms; there was little evidence suggesting that ACT offered a curative effect or prevented future psychotic episodes, meaning relapses were common once the treatment course concluded, further calling into question the extreme risks undertaken.

Risks, Side Effects, and Ethical Considerations

The inherent risks associated with Atropine-Coma Therapy were staggering, making it one of the most perilous psychiatric interventions ever routinely practiced. The most immediate and life-threatening risk was **cardiovascular collapse** due to extreme tachycardia and arrhythmias induced by the massive anticholinergic load. Respiratory failure was also a significant concern, sometimes secondary to central nervous system depression, or due to complications like aspiration during the unconscious state. Furthermore, the combination of high fever (hyperthermia) and circulatory strain posed a substantial risk of permanent brain damage or death.

Beyond mortality, patients frequently experienced severe morbidity. Side effects during the coma included prolonged periods of delirium and confusion upon emergence, severe dehydration, persistent urinary retention requiring catheterization, and potential damage to vital organs due to prolonged stress. The long-term physiological impact of repeated near-fatal poisonings was substantial, although detailed, longitudinal studies documenting chronic effects were lacking, largely because the therapy was abandoned shortly after effective antipsychotics emerged.

The ethical considerations surrounding ACT are profound. Given the severity of the illness in the target population and the era in which it was practiced, questions regarding informed consent are highly relevant. It is questionable whether patients suffering from acute, severe psychosis could provide truly voluntary and informed consent for a procedure that carried such a high risk of death or permanent injury. Moreover, the therapy's effectiveness was poorly substantiated by modern scientific standards, leading to significant moral and ethical debates regarding the justification of utilizing such a dangerous intervention when the therapeutic gains were often temporary and difficult to quantify reliably.

The high death rate associated with ACT--reports vary, but rates were significantly higher than those associated with contemporary ECT--contributed heavily to its eventual condemnation. The sheer physiological assault the therapy inflicted, combined with its limited long-term success, positioned it as a highly unethical and unsafe intervention by contemporary medical standards, serving today as a stark reminder of the lengths to which medicine went in the pre-pharmacological

era to treat severe mental illness.

Perceived Efficacy and Professional Critique

The professional assessment of Atropine-Coma Therapy has historically been bifurcated. Proponents, primarily those institutions that specialized in its administration, often reported high rates of short-term success, noting that extremely agitated or psychotic patients became calmer, more cooperative, and showed a reduction in acute symptomatology immediately following the course of treatment. This perception of efficacy was usually based on clinical observation and highly subjective outcome measures, focusing heavily on behavioral improvements rather than objective, psychometric changes.

However, professional critique, particularly from centers utilizing more data-driven methodologies, was robust. Critics consistently pointed out the lack of rigorous, controlled clinical trials necessary to establish ACT's superiority over simple deep sedation or placebo, especially when weighed against the extreme risk profile. The temporary nature of the improvements was a major weakness; if ACT merely interrupted the acute phase but did not prevent relapse, the justification for risking the patient's life repeatedly became untenable. Furthermore, the improvements observed were often difficult to separate from the intense nursing care, environmental control, and high level of attention afforded to patients undergoing such a critical procedure.

Many established medical bodies and leading figures in pharmacology viewed ACT skeptically, characterizing it as a form of physiological abuse or medical extremism rather than a legitimate therapeutic tool. The high mortality and morbidity rates often overshadowed any purported therapeutic benefit in the eyes of the broader medical community, leading to professional discouragement against its adoption outside of specialized, often isolated, psychiatric hospitals. Ultimately, ACT failed the critical test of scientific validation, relying too heavily on anecdotal success stories and falling short when scrutinized against the emerging standards of evidence-based medicine.

Decline and Replacement by Modern Treatments

The demise of Atropine-Coma Therapy was swift and definitive, largely dictated by two major advancements: the widespread adoption of safer, more effective pharmacological agents and a growing global emphasis on patient safety and ethical treatment standards. The single most impactful factor was the introduction of **antipsychotic medications**, beginning with phenothiazines like chlorpromazine in the early 1950s. These drugs offered a targeted, manageable, and significantly safer means of controlling psychotic symptoms and severe agitation without requiring the induction of a life-threatening coma.

As specific pharmacological antagonists became available, the rationale for using a generalized,

toxic physiological assault like ACT evaporated. Why risk cardiac arrest and brain damage via massive atropine dosing when oral medication could achieve comparable or superior symptomatic control with minimal risk? The shift marked a pivotal transition in psychiatry from reliance on broad "shock" interventions aimed at resetting the entire system to targeted neurochemical interventions.

Furthermore, as medical ethics evolved and the field demanded higher standards of patient safety and evidence-based practice, therapies with documented high mortality rates, such as ACT and Insulin Shock Therapy, became indefensible. Institutional practices were scrutinized, and dangerous procedures lacking strong empirical support were systematically phased out, often through hospital policy changes and shifts in psychiatric education.

Today, Atropine-Coma Therapy is strictly recognized as a historical footnote--a dangerous, resource-intensive, and largely ineffective intervention that represents the desperate state of psychiatric care prior to the advent of modern psychopharmacology. Its abandonment serves as a powerful illustration of medical progress, highlighting the critical importance of safety, specificity, and scientific validation in the development and deployment of psychiatric treatments. Modern interventions for severe agitation and psychosis rely on carefully managed doses of antipsychotics and benzodiazepines, vastly safer methodologies that have entirely supplanted the brutal risks inherent in ACT.