AFTERPOTENTIAL

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AFTERPOTENTIAL

Primary Disciplinary Field(s): Neurophysiology, Biopsychology, Pharmacology

1. Core Definition

The **Afterpotential** is a transient, post-spike change in the membrane potential of an excitable cell--typically a neuron or muscle fiber--that immediately follows the rapid repolarization phase of an action potential. It represents a lingering electrical state, or "aftercurrent," that deviates from the cell's standard resting membrane potential (RMP) before returning to a steady baseline. This phenomenon is a direct consequence of the slow kinetics of voltage-gated ion channels, particularly potassium and calcium channels, which remain open or active for a brief period after the primary action potential spike has subsided.

As referenced in initial definitions, afterpotential is sometimes referred to as **aftercurrent**, emphasizing that it is the sustained flow of ionic charge that governs this lingering electrical state. While the bulk of the electrical energy associated with the action potential is expended during the massive, rapid influx of sodium ions and subsequent efflux of potassium ions, the afterpotential captures the residual component of excitability. This residual energy profoundly influences the cell's immediate recovery state, determining how soon, or if, it can be stimulated to fire another action potential.

Crucially, afterpotentials can be either inhibitory or excitatory. An inhibitory afterpotential, known as a hyperpolarizing afterpotential (AHP), drives the membrane potential below the RMP, making the cell temporarily less excitable. Conversely, an excitatory afterpotential, known as a depolarizing afterpotential (DAP), drives the membrane potential closer to the threshold, enhancing the likelihood of repetitive firing. The precise balance and timing of these two types of afterpotentials are fundamental regulators of neural coding and firing patterns within the central nervous system.

2. Physiological Mechanism and Phases

The generation of the afterpotential is inextricably linked to the sequence of ionic events that terminate the action potential. Repolarization of the membrane is primarily driven by the opening of voltage-gated **Potassium (K+) channels**, allowing K+ ions to flow out of the cell, neutralizing the positive charge that entered during depolarization. However, these K+ channels typically close more slowly than the voltage-gated Sodium (Na+) channels that initiated the spike. This difference in kinetic speed is the fundamental physiological basis for the subsequent membrane fluctuation.

The delay in K+ channel closure means that, for a brief window, K+ ions continue to exit the cell even after the membrane potential has crossed the resting potential threshold (approximately -70 mV). This continued efflux of positive charge drives the internal potential to a value more negative

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than the RMP, resulting in the most common form of afterpotential: the **Hyperpolarizing Afterpotential**. This temporary state of hyperpolarization serves as an essential brake on neural activity, enforcing the relative refractory period by requiring a stronger stimulus to overcome the deeper negative potential.

Furthermore, in many neuronal types, particularly those associated with burst firing, afterpotentials are heavily modulated by intracellular **Calcium (Ca2+) ions**. The influx of Ca2+ during the action potential or subsequent repetitive activity triggers the opening of calcium-activated potassium channels (KCa channels). These KCa channels contribute significantly to the slower components of the hyperpolarizing afterpotential, linking the recent history of electrical activity (Ca2+ concentration) to the future excitability profile of the cell. This coupling mechanism allows neurons to adapt their firing rate based on prior activity levels.

3. Types of Afterpotentials and Components

Afterpotentials are complex phenomena, often comprising multiple distinct currents that manifest sequentially, each having a unique molecular basis and duration. The classification generally distinguishes between membrane shifts that suppress activity and those that promote it, alongside classifications based on duration.

Hyperpolarizing Afterpotential (AHP): This is the inhibitory component where the membrane potential temporarily falls below the RMP. AHPs are universally crucial for limiting the cell's firing frequency. They are subdivided based on their kinetics and underlying ion channels:

Fast AHP (fAHP): Occurs within milliseconds immediately following the spike. Primarily mediated by voltage-gated K+ channels (like Kv3 channels) that are rapidly activated and deactivated.
Medium AHP (mAHP): Lasts for tens to hundreds of milliseconds. Often mediated by specific KCa channels, which link transient increases in intracellular calcium to membrane hyperpolarization. The mAHP is key for regulating spike frequency adaptation (SFA).
Slow AHP (sAHP): Can persist for several seconds. These are typically mediated by muscarinic-sensitive KCa channels (such as SK channels), and their sustained effect profoundly dampens excitability, regulating overall network activity and plasticity.

Depolarizing Afterpotential (DAP): This is the excitatory component where the membrane potential temporarily remains slightly positive relative to the RMP, without reaching the action potential threshold. DAPs can be generated by persistent inward currents, such as slow-inactivating Na+ currents or transient Ca2+ currents.

The DAP is essential for promoting **repetitive or burst firing**. If the DAP is strong enough, it can bring the membrane potential back to the threshold shortly after the initial spike, leading to a train of action potentials. This mechanism is crucial in pacemaker neurons that generate rhythmic

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activity without external input, providing an intrinsic means of oscillation.

The interplay between DAPs and AHPs dictates the output mode of the neuron. In many neurons, the DAP occurs early, followed by the inhibitory AHP, creating a precise window for excitability. This complex sequence allows the nervous system to generate highly specific temporal codes rather than simple, sporadic discharges.

4. Role in Neural Plasticity and Coding

The duration and magnitude of afterpotentials serve as critical determinants of how a neuron processes incoming information and translates it into a firing code. The afterpotential acts as an internal memory mechanism, reflecting the recent history of neural activity and thereby influencing future response patterns. This concept is central to understanding neural network dynamics.

For example, during sustained high-frequency input, the accumulation of intracellular calcium leads to an increasingly larger and longer-lasting slow AHP. This mechanism causes **spike frequency adaptation (SFA)**, where a neuron initially fires rapidly but then slows its rate even if the excitatory input remains constant. The slow AHP thus prevents neuronal exhaustion and shapes the temporal characteristics of information transmission, moving the coding strategy from simple frequency representation to temporal dynamics.

Furthermore, afterpotentials play a significant role in synaptic plasticity, the biological basis for learning and memory. Changes in the properties of the AHP, particularly the slow components, have been shown to modulate the induction of long-term potentiation (LTP) and long-term depression (LTD). By controlling the excitability state of the postsynaptic neuron, afterpotentials influence whether synaptic input arriving shortly after an action potential will be effective in modifying synaptic strength, demonstrating their impact at the intersection of electrophysiology and cognition.

5. Clinical Significance and Pharmacology

Because afterpotentials are governed by highly specific ion channels, they represent major targets for therapeutic intervention in various neurological and cardiac disorders. Modulating the magnitude or duration of AHPs and DAPs can restore normal rhythmicity or reduce pathologically enhanced excitability.

In the field of epilepsy, hyperexcitability and abnormal burst firing are common hallmarks. Pharmacological agents that enhance the AHP (by increasing K+ conductance) can dampen this hyperexcitability, reducing the likelihood of seizures. Conversely, disorders characterized by low excitability or impaired neural rhythmicity might benefit from drugs that suppress the AHP or enhance the DAP.

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The role of afterpotentials is particularly critical in cardiac electrophysiology. In myocardial cells, abnormal depolarizing afterpotentials--specifically **Early Afterdepolarizations (EADs)** and **Delayed Afterdepolarizations (DADs)**--are major causes of life-threatening cardiac arrhythmias. EADs occur during the prolonged plateau phase of the cardiac action potential and are often related to residual inward currents or reactivation of L-type calcium channels. DADs occur after full repolarization and are typically mediated by abnormal calcium release from internal stores. Targeting the ion channels responsible for these pathological afterpotentials is a cornerstone of anti-arrhythmic drug design.

Further Reading

Action potential (Wikipedia)
Hyperpolarization (biology) (Wikipedia)
Afterpotential (ScienceDirect)
Refractory period (physiology) (Wikipedia)

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