# Impact of Anti-seizure Medications on the Brain's Salience Network in Generalized Seizures

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## Introduction

Epilepsy is a neurological disorder marked by recurrent, unprovoked seizures. These seizures can manifest in various forms, depending on the region of the brain affected. Generalized Tonic-clonic Seizures (GTCS), previously known as grand mal seizures, are one of the most severe types, affecting the entire brain and resulting in a loss of consciousness, muscle rigidity, and violent jerking movements. GTCS can have serious consequences, including physical injury, cognitive impairments, and even death. For individuals who experience frequent GTCS, Antiseizure Drugs (ASDs) become essential in controlling the occurrence and intensity of seizures, as well as improving quality of life. Antiseizure drugs work by modulating neuronal activity in the brain. They generally function by enhancing inhibitory neurotransmission, for example through the GABAergic system, or by inhibiting excitatory pathways such as those involving glutamate. These medications play a crucial role in preventing seizures, but their impact on the brain's underlying neural circuits, particularly those responsible for emotional processing, cognitive control, and attention, has gained significant interest. One such circuit that may be influenced by antiseizure medications is the salience network [1].

#### Description

Antiseizure drugs function by targeting various mechanisms that stabilize neuronal activity in the brain, with the ultimate goal of preventing seizures. These medications work by influencing both excitatory and inhibitory neurotransmission. Some drugs enhance the action of Gamma-Amino Butyric Acid (GABA), a neurotransmitter that inhibits brain activity and helps to suppress seizures. Other drugs target glutamate, which is the brain's primary excitatory neurotransmitter, in order to reduce excessive neuronal firing. Medications such as valproate, phenobarbital, and benzodiazepines are commonly used for their GABAergic effects, while drugs like lamotrigine and topiramate act on glutamate pathways. Additionally, certain ASDs, including carbamazepine and phenytoin, modify sodium and calcium channels, contributing to stabilization of neuronal activity. The salience network plays a vital role in detecting, prioritizing, and responding to significant stimuli in the environment. This network helps to direct attention to important events and assists in regulating emotional responses to those events. The key components of the salience network include the anterior insula (AI), which is involved in interoception, emotional awareness, and autonomic regulation; the Anterior Cingulate Cortex (ACC), which plays a critical role in cognitive control, decision-making, and emotional processing; and the amygdala, which is central to processing emotions, particularly fear and anxiety. Disruptions to

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the functioning of this network can result in impairments in emotion regulation, attention, and overall cognitive performance [2].

In the context of epilepsy, the salience network can be significantly impacted by seizure activity. For individuals with generalized tonic-clonic seizures, the rapid and widespread neuronal firing associated with these seizures may disrupt normal network connectivity. This disruption can lead to difficulties in regulating emotions, paying attention, or making decisions. Seizure-induced dysfunction of the salience network may contribute to cognitive and emotional symptoms that persist even when seizures are under control. Thus, understanding the interactions between antiseizure drugs and the salience network is crucial for optimizing treatment strategies for epilepsy patients. Antiseizure drugs, while primarily designed to control seizures, can have unintended effects on the brain's neural networks, including the salience network. For example, GABAergic drugs that enhance inhibitory transmission may reduce the excitability of regions involved in the salience network, potentially improving emotional regulation and cognitive processing in some patients. Conversely, certain drugs that modulate excitatory neurotransmission or ion channels may have a different impact, either restoring or further disrupting network activity. The effects of these medications on the salience network are not fully understood, but emerging evidence suggests that some antiseizure drugs may influence the functional connectivity between the AI, ACC, and amygdala. This modulation could have implications for mood, cognition, and autonomic control in patients with epilepsy [3].

For example, studies have shown altered connectivity in the salience network in patients with epilepsy, particularly in the insula and anterior cingulate cortex. This finding suggests that the disruption of salience network connectivity may be a key feature of the disease. Furthermore, Electroencephalography (EEG) studies have observed changes in neuronal firing patterns during and after seizures, which can be influenced by the use of antiseizure drugs. These changes may impact the salience network's ability to regulate attention and emotion [4].

Clinical practice, patients on antiseizure medications may report changes in cognitive function, emotional well-being, and overall quality of life. These side effects are often linked to alterations in brain network activity, including the salience network. For example, some patients may experience cognitive difficulties such as memory impairment or attention deficits, which may be exacerbated by certain antiseizure drugs. Emotional symptoms, including anxiety or irritability, have also been reported by patients undergoing longterm treatment with drugs like levetiracetam, carbamazepine, or valproate. These findings underscore the importance of understanding the impact of antiseizure drugs on the salience network, as such effects can significantly influence patient outcomes [5].

## Conclusion

In conclusion, antiseizure drugs have a profound impact on the functioning of the salience network in patients with generalized tonic-clonic seizures. While these medications effectively control seizure activity by stabilizing neuronal excitability, their influence on brain networks, particularly those related to emotion regulation, attention, and decision-making, is also significant. The salience network, which plays a central role in processing important stimuli and regulating responses to them, can be disrupted by both epileptic seizures and the medications used to treat them. Understanding how antiseizure drugs modulate this network is crucial for optimizing treatment plans, as disruptions to salience network activity may lead to cognitive and emotional difficulties that affect the patient's quality of life. Neuroimaging studies and clinical observations suggest that antiseizure drugs can modulate the connectivity of the salience network in different ways, depending on the drug's mechanism of action. While some drugs may restore normal network functioning, others can induce side effects that interfere with emotional regulation, attention, and cognitive function. Therefore, personalized treatment approaches that consider not only the efficacy of seizure control but also the potential impact on the salience network are essential for improving outcomes in patients with epilepsy.

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### **Conflict of Interest**

There are no conflicts of interest by author.

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