

# Sudden Death Caused by Neurological Events



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The role of the central nervous system in the modulation and precipitation of sudden cardiac death has been a matter of controversy for decades. The links between the central nervous system and autonomic nervous system are anatomically and physiologically relevant to understand the high prevalence of cardiovascular events during the acute and post-acute period of cerebral events such as stroke of all types, seizures and epilepsy, head injury, other neurological conditions, and neurosurgical procedures. Much experimental evidence indicates that the final common pathway for most cardiac arrhythmias induced by central neural stimulation is related to the limbic system, specifically the amygdala and insular regions. Stimulation of insular area induces marked sympathetic activation that is persistent even after vagal stimulation or vagal section. Acute stroke, both experimentally and clinically, has been documented to induce focal myocytolysis in a series of patients with acute stroke that died suddenly due to ventricular fibrillation. There is evidence to support lateralization, which is a differential effect from each cerebral hemisphere, in neurocardiac control. Heart rate increases with left hemispheric inactivation and decreases with right hemispheric inactivation. Experimentally, the right cerebral hemisphere has been shown to modulate sympathetic activity. Clinically, this correlates with increased arrhythmias and arrhythmic death following acute right-hemispheric stroke. Sudden unexpected death in epilepsy patients (SUDEP) is a common and devastating cause of death among patients with epilepsy. Animal experiments have implicated postictal central apnea, prone position, and neurogenic pulmonary edema as substrates for life-threatening ventricular arrhythmias. Future studies should focus on identification of at-risk patients, confirmation of a vulnerable period of cerebrogenic sudden death in those with different neurological conditions, and clarification of the neurochemical mediators.

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