G119 Autonomic Nervous System and Sudden Death

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After attending this presentation, attendees will improve their understanding about "unexplained" sudden deaths with negative autopsy findings caused by the activation of the utonomic nervous system, as well as cardiac deaths following injuries to other organs and related to the effects of the autonomic nervous system.

This presentation will impact the forensic science community by providing detailed knowledge about the most common mechanisms of death following activation of the autonomic nervous system.

Although the cardiovascular system is often considered a unique physiological entity, the evidence of a link between sudden cardiac death and autonomic nervous system is compelling. It is in fact well recognized that physical and emotional stress (such as anxiety or anger) may lead to sudden death as a result of arrhythmias, myocardial ischaemia, and infarction. Experimental evidence from human and animal models proves that stimulation of certain brain areas (mainly the insular cortex, the infralimbic cortex, and the amygdala) or sympathetic efferent fibers to the heart can produce fatal abnormaities of the heart rhythm. Psychiatric diseases are also known to cause sudden cardiac death. There is integration between the limbic system and the cortical autonomic "control sites." Circuitry between these areas might result in abnormal electrical stimulation to the SA node resulting in sudden death.

This catastrophic event is known to be responsible for more than 300,000 sudden cardiac deaths every year in the United States. The majority of victims are thought to have suffered ventricular tachycardia or ventricular fibrillation. Several clinical and experimental studies suggest that heart rate variability and baroreflex sensitivity are the most reliable predicting factors for possible future cardiac events.

Central autonomic dysfunctions causing cardiovascular complications such as ECG changes, cardiac arrhythmias, ischaemic damage to the myocardial muscle, and disturbances of blood pressure regulation might often occur as a result of brain injuries or acute cerebrovascular disease. These often life-threatening complications are thought to be due to increased sympathetic tone with subsequent elevation of circulating catecholamines. Several experimental and human studies have suggested that the most arrhythmogenic areas, if injured, are the prefrontal areas or the insula. These brain structures might also give rise to further cardiac complications involving heart rate variability and possible heart failure. Although head trauma-induced heart rate variability has been recently studied with modern techniques, cardiac complications following brain disease are now being redefined.

The ECG changes following brain damage or ischaemia can often mimic myocardial ischaemia. The most commonly described abnormalities are regarded to be ST segment depression, flat or inverted T waves, prolonged QT intervals, and U waves. These phenomena have been proved to be due to autonomic nervous system reaction to the brain disease, and are not related to any kind of heart or coronary artery disease. In comparison with cardiac related ECG changes, these seem to appear later, with a peak about two days later from the cerebrovascular event (or trauma) and revertion within two weeks. It is interesting that, although in the majority of cases the ECG changes do not reflect real myocardial damage, sometimes macroscopic and microscopic changes to the myocardium have been observed at autopsy without coronary artery disease. These changes were observed to be myocardial necrosis with histiocytic infiltration, subendocardial hemorrhage, and myofibrillar degeneration.

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 ** Presenting Author*