3.1 The Autonomic Nervous System and Sudden Death.
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Learning Objectives

At the end of this presentation participants will be able to:

1. Explain their understanding of “unexplained” sudden deaths caused by the activation of the Autonomic Nervous System.
2. Recite the pathophysiology of cardiac deaths following injuries to other organs related to the effects of the Autonomic Nervous System.
3. Explain the most common mechanisms of death through a logic process that involves anatomy, physiology and physiopathology.

Abstract

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 pain, restraint, anxiety or anger) may lead to sudden death as a result of arrhythmias, myocardial ischemia 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 abnormalities 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 sinoatrial (SA) node to result 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, ischemic 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 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, 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 ischemia can often mimic myocardial ischemia. 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 2 days later from the cerebrovascular event (or trauma) and reversion within 2 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 haemorrhage, and myofibrillar degeneration.