Neurobiology of Sudden Death

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Fava et al. [1] suggest that anxiety or panic is an affective prodrome of cerebral insufficiency. This hypothesis is supported by a report that anxiety may increase the risk of fatal coronary heart disease through hyperventilation during an acute attack, which could in turn induce coronary spasm, or an acute attack of anxiety triggering an episode of fatal ventricular arrhythmias [2]. The neurobiology is suggested by studies linking brain stem cardiovascular control, cardiovascular reactivity in challenging tasks, the induction of breathing, panic and vasospasm to dopa-mine lateralized to the right hemisphere [3, 4]. This hypothesis is supported by an abnormal parahip-pocampal region and its afferent and efferent connections in patients vulnerable to lactate-induced panic [5]. It is also supported by optimal response organization at intermediate dopamine tone in a medial-frontal-striatal activation system [4] and by a critical role for the medial frontal cortex in emotional experience and expression and in complete sympathetic activation of cardiovascular responses to both severely and mildly stressful stimuli [6]. These findings suggest that subclinical obstruction of lung airways in patients with panic disorder might be due to the dys-regulation of airways smooth-muscle tone by abnormalities of dopa-mine [7,8] subserving cognitive and emotional functioning, reflecting properties of neuronal activity and firing [9-12]. They also prompt attention to the time of onset of symp-

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toms and their longitudinal development in the stages of illness [1] possibly altered by slower, deeper breathing contributing to 6.5-fold reduction in mortality [13].

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