Neurobiology of Anger-Hostility and Coronary Risk

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Ricci Bitti et al. [1] find that proneness to coronary heart disease is more significantly correlated with Aggressive Responding ($p < 0.0001$) than with Anger Arousal ($p < 0.002$). The neurobiologic features are suggested by reports linking disruption of brain stem cardiovascular control, cardiovascular reactivity in challenging tasks, and subclinical impairment of lung airways to dopamine abnormalities lateralized to the right hemisphere. This hypothesis is supported by optimal response organization at intermediate dopamine tone in a medial-frontal-striatal activation system, and by deactivation of the right hemisphere, a state marker of depression, promoting dominance of the left hemisphere associated with cardiac arrhythmia, vasoconstriction [2] and violence [3]. It is also supported by a neurochemical model underlying differences in reaction times between introverts and extroverts [2, 4, 5].

The fact that delay-dependent speeding of reaction time, indicating motor readiness, is abolished by depletion of dopamine [2, 6], prompts a multidisciplinary approach involving neuropharmacology and cardiovascular physiology [7] in investigations of particular components of anger-hostility [1] by monitoring behavioral correlates of asymmetric brain functions in emotionally charged dialog, reflecting properties of neuronal activity and firing. This method is supported by the correlation of the frequency and duration of speech hesitation pauses to 6-fold coronary heart disease and mood, respectively, by the blood pressure reduction effect of longer, less recurrent pauses [2] predictive of response to neuropharmacologic intervention [8], and by studies linking pause rate and variability of pause duration to the left and right hemisphere, respectively [2, 9]. It is also supported by reports suggesting temporal coding in the central nervous system manifested by the association of interspike intervals with specific behavioral events [10].

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