ARE AGING PROCESSES DEPENDENT ON CAROTID BODY ACTIVITY ?

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The aging process is characterized by a decline in several physiological functions resulting in a reduced capability to maintain homeostasis. This lowered homeostatic capacity seems to involve the carotid body (CB), whose role is to modulate ventilation and tissue oxygen supply thus playing a prime role in all aging processes. Chronic intermittent or episodic hypoxia, as occurs during a number of disease states, can have devastating effects, and prolonged exposure to this hypoxia can result in cell injury or cell death. Aging causes marked changes in CB morphology. With increased age man CB shows a reduction in the number and volume of mitochondria, fewer synaptic junctions between glomi, along with a reduction in CB content of neurotransmitters, leading to a sort of ‘physiological denervation’. The increase in HIF-1 - VEGF - ET and NOS-1 expression during chronic hypoxia is less evident in CBs of old rats as compared to the young ones. Aging could be interpreted as a cumulative result of oxidative damage to cells, which derives from aerobic metabolism. Moreover, metabolism rate is tightly correlated with life span, thus a loss in mitochondrial function is one of the prime factors affecting CB aging processes. The age-related reduction in synaptic junctions might be a self-protective mechanism through which cells buffer themselves against accumulation of reactive oxygen species during aging. The correlation between oxygen supply and life-span of CB cells remains open until the question of how and why cells sense oxygen is solved.