Enhanced carotid body chemosensory activity and the cardiovascular alterations induced by intermittent hypoxia

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The carotid body (CB) plays a main role in the maintenance of the oxygen homeostasis. The hypoxic stimulation of the CB increases the chemosensory discharge, which in turn elicits reflex sympathetic, cardiovascular and ventilatory adjustments. An exacerbate carotid chemosensory activity has been associated with human sympathetic-mediated diseases such as hypertension, insulin resistance, heart failure and obstructive sleep apnea (OSA). Indeed, the CB chemosensory discharge becomes tonically hypereactive in experimental models of OSA and heart failure. Chronic intermittent hypoxia (CIH), a main feature of OSA, enhances CB chemosensory baseline discharges in normoxia and in response to hypoxia, inducing sympathetic overactivity and hypertension. Oxidative stress, increased levels of ET-1, Angiotensin II and pro-inflammatory cytokines, along with a reduced production of NO in the CB, have been associated with the enhanced carotid chemosensory activity. In this review, we will discuss new evidence supporting a main role for the CB chemoreceptor in the autonomic and cardiorespiratory alterations induced by intermittent hypoxia, as well as the molecular mechanisms involved in the CB chemosensory potentiation.

Autonomic dysfunction

Carotid body

Hypertension

Intermittent hypoxia

Oxidative stress

angiotensin II

bosentan

cyclo(dextro tryptophyl dextro aspartylprolyl dextro valylleucyl)

cytokine

endothelin 1

hypoxia inducible factor 1alpha

immunoglobulin enhancer binding protein

interleukin 1beta

interleukin 6

nitric oxide

noradrenalin

reactive nitrogen species

reactive oxygen metabolite

reduced nicotinamide adenine dinucleotide phosphate oxidase 2

tempol

transcription factor NFAT3

tumor necrosis factor alpha

autonomic dysfunction

cardiovascular disease

carotid body chemoreceptor

chronic intermittent hypoxia

clinical feature

disease association

disease classification

disease course

disease severity

endothelial dysfunction

enzyme activation

human

hyperactivity

hypertension

immunoreactivity

inflammation

molecular mechanics

nonhuman

oxidative stress

pathophysiology

protein expression

protein function

protein synthesis

renin angiotensin aldosterone system

Review

risk factor

sleep disordered breathing

upregulation