Whilst a physiological function for the adult mammalian carotid body (CB) remains disputed, there is now burgeoning evidence supporting a pathophysiological role for this systemic chemoreceptor. However, the development of clinical interventions for carotid body dysfunction in patients with sleep disordered breathing, congestive heart failure and insulin resistance has been restricted by a lack of fundamental knowledge of the mechanism(s) accounting for CB activation by hypoxia. Of the proposed O$_2$ sensors, the type I cell mitochondria appear particularly sensitive to relevant arterial O$_2$ tensions. In particular, the exceptionally low O$_2$ affinity of complex IV causes mitochondrial electron flux to be more susceptible to small falls in O$_2$ compared with other cell types. Whether or not the mitochondria have a functional role in establishing the unique O$_2$ sensitivity of the whole CB organ is the focus of our current investigations together with an examination of potential pharmacological approaches to influencing chemosensitivity.