The effect of sustained hypoxia on arterial stiffness

H. Vedam, D.J. Barnes, J. Hedner, C. Phillips, R.R. Grunstein, Woolcock Institute of Medical Research, Camperdown, NSW, Australia

In humans, the net effect of acute systemic hypoxia is limb vasodilatation despite increases in muscle sympathetic vasoconstrictor nerve activity. However, the response of large arteries to hypoxia has not previously been studied. Large conduit arterial stiffness has become an important concept, and is based on number of factors including smooth muscle tone, vessel wall collagen/elastin structure, the endothelium, heart rate (HR) and mean arterial pressure (MAP).

AIM: To assess the effect of sustained isocapnic hypoxia on arterial stiffness in healthy men.

METHODS: Eight subjects aged 31±4 years (mean ± SD) were exposed to 20 minutes of isocapnic hypoxia (saturation 81.6 ± 0.9%) and five subjects aged 28 ± 6 years were exposed to sham hypoxia. Arterial stiffness was measured using pulse wave analysis and calculation of the augmentation index (AIx). Mean arterial pressure (MAP), systolic blood pressure (BP), diastolic BP, heart rate (HR) and ventilatory responses were also measured.

RESULTS: After adjusting for the changes in MAP and HR, hypoxia had a significant effect on the corrected AIx ($p = 0.027$). After an initial period of nonsignificant increase in arterial stiffness, there was a significant reduction in the corrected AIx over the hypoxic period. Resaturation resulted in a further fall in the AIx, before it returned to normal during the next 6 mins of recovery.

CONCLUSIONS: Hypoxia induced a gradual reduction in arterial stiffness in the setting of an increased sympathetic output state. One potential mechanism is increasing endothelium derived nitric oxide mediated vasodilation.

Supported by NHMRC Grant Number 301936 and University of Sydney Postgraduate Award (UPA)