Pulse Wave Velocity Is Increased With Experimental Sleep Restriction in Healthy Humans

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Objectives: Increased carotid-femoral pulse wave velocity is indicative of vascular stiffening of the central arterial tree. Aortic stiffness is a key risk factor for the development of hypertension and cardiovascular disease. Following acute (24-hour) sleep deprivation, healthy adults exhibit an increase in carotid-femoral pulse wave velocity; however, acute sleep deprivation poorly represents sleep patterns observed in everyday life. With this information in mind, we hypothesized a prolonged (9 day) exposure to restricted sleep (4 hours of sleep per night) would result in increases in carotid-femoral pulse wave velocity in healthy humans.

Methods: Seven (3M, 5F) young (23±1 yrs), healthy adults underwent a 4-day period of acclimation followed by 9 days of experimental sleep restriction (4 hours of sleep per night – from 12:30 AM to 4:30 AM). High-fidelity radial arterial pressure waveforms and carotid-femoral pulse wave velocity were assessed using applanation tonometry (SphygmoCor, AtCor Medical). Subjects were studied on Day 2 (Acclimation) and Day 13 (Restriction).

Results: Sleep restriction resulted in an increase in carotid-femoral pulse wave velocity (5.6±0.2 to 5.9±0.2 m/s, $p=0.05$) and a decrease in round trip time (179±8 to 150±11 ms, $p<0.01$) when compared to the acclimation period. A reduction in the Buckberg subendocardial viability ratio (SEVR, indicative of myocardial oxygen supply/demand, $p=0.02$) and an increase in the Pressure-Time Integral Systole (PTI, an index of cardiac load, $p=0.01$) were also observed following sleep restriction.

Conclusions: prolonged (9-day) exposure to experimental sleep restriction in young healthy humans results in unfavorable changes in central macrovascular function, including an increase in central arterial stiffness and cardiac load. These results may have important implications for the increase in cardiovascular disease risk in individuals experiencing limited sleep.