Effects of 24 hour sleep deprivation on cerebral blood flow measured by ASL

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Purpose Sleep of sufficient duration, continuity and depth is paramount to maintain neurobehavioral functioning. Despite the fact that lack of sleep can have a severe impact on neurobehavioral functioning and increase morbidity, sleep deprivation is common in everyday life due to extended work hours, shift work and jet lags[1]. Since the detrimental effects of sleep loss can vary greatly among individuals, there is a need for non-invasive measurements of the effects of sleep deprivation. The current study investigates the effect of sleep deprivation on cerebral blood flow (CBF) as measured by arterial spin labeling (ASL).

Methods 39 healthy male volunteers, age 18-26 yrs, were scanned on a first morning after sufficient sleep and on a second morning after either normal sleep (n=20, age 22.7 ± 2.2 yrs) or 24 hours supervised sleep deprivation (n=19, age 21.6 ± 2.2 yrs). All subjects were refrained from perfusion confounders such as physical exercise, caffeine or nicotine. The MRI scan protocol (3T Philips Achieva, 8 channel HC) included a 2D gradient-echo single-shot EPI PCASL scan (SENSE 2.5; TE/TR 11/4400 ms; FOV 240x240 mm; matrix 64x64; 22 slices; 6 mm slice thickness; no gap; 2 background suppression pulses; PLD 1800-2680 ms; labeling duration 1800 ms; NSA 60; total scan duration 8:14 min, a separate M0-scan and a 1 mm isotropic 3D T1-scan[2]. Perfusion-weighted maps were motion corrected, quantified using a single compartment model and spatially normalized using both diffeomorphic anatomical and perfusion-weighted registration[2].

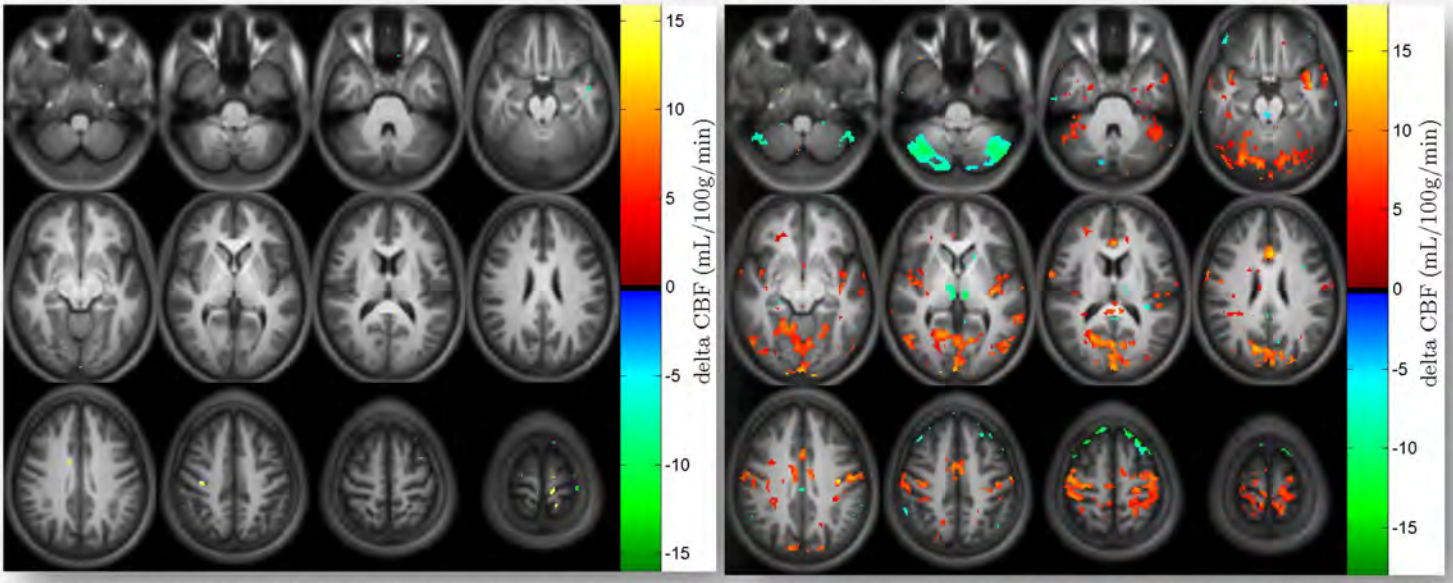

Figure. Cerebral blood flow (CBF) difference maps [morning 2 minus morning 1], showing differences (p<0.001 uncorr., colors ranging from -15 to +15 mL/100g/min difference) for normal sleep (n=20, left Figure) and for supervised sleep deprivation (n=19, right Figure).

Results Whereas the 24 hour test-retest showed nearly no CBF changes for the normal sleepers (left Figure), there were wide-spread perfusion increases for the sleep deprived (right Figure), most notably in the primary visual, lateral occipital, lateral motor cortex and white matter (Table). In addition, there were perfusion decreases in the cerebellum for the sleep deprived but not for the normal sleepers. There was an increase in urine osmolality after normal sleep but not after sleep deprivation, and there was a decrease in hematocrit after sleep deprivation but not after normal sleep (Table).

Discussion This is the first study to show significant changes in CBF after sleep deprivation that are not observed after normal sleep. The fact that the largest differences were observed in the occipital cortex and white matter may suggest that there are changes in arterial transit time after sleep deprivation. Based on hematocrit changes, we estimate to have overestimated the CBF increase after sleep deprivation by 1 mL/100g/min[3]. Physiological changes such as hydration status may play a role in the CBF changes after sleep deprivation.

Regions of interest (mL/100g/min)	Normal sleep (n=20)	Sleep deprivation (n=19)
Frontal cortex	0.7	0.9
Temporal cortex	-0.3	2.8 †
Parietal cortex	0.6	2.7 *
Occipital cortex	0.2	5.0 †
White matter	0.3	1.5 †
Cerebellum	0.5	-0.9
Parameters		
Urine osmolality (mOsm/kg)	3.0 †	1.4
Hematocrit (% L/L)	-0.5	-1.4 *

Table summarizes differences between the morning scan sessions [morning 2 minus morning 1]. * p<0.05 † p<0.005

References

[1]*Poudel, Sleep 2012* [2]*Alsop, MRM 2014* [3]*Varela, NMR Biomed 2011*