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THE EFFECT OF CONTINUOUS POSITIVE AIRWAY PRESSURE ON TOTAL CEREBRAL BLOOD FLOW IN 23 HEALTHY AWAKE VOLUNTEERS

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ABSTRACT

Continuous Positive Airway Pressure (CPAP) is used as the gold standard treatment for sleep disordered breathing, acting as a pneumatic splint to prevent collapse of the pharyngeal airway. However, the influence that CPAP has on Cerebral Blood Flow (CBF) dynamics is not well understood. This preliminary study investigates the influence of CPAP on total CBF in 23 healthy awake subjects by measuring flow velocity and lumen diameter of the left and right proximal Internal Carotid Arteries (ICA), Vertebral Arteries (VA), and Middle Cerebral Arteries (MCA) using Duplex Color Doppler Ultrasound (US) with and without CPAP at a level of 15 cm H_2O. Transcutaneous Carbon Dioxide (PtcCO2) level, heart rate, Blood Pressure (BP), and oxygen saturation (SaO2) were monitored before and after each test. The preliminary measurements indicate that CPAP results in a decrease of CBF by 17% (p-value < 0.05). The theoretically predicted decrease in CBF from PtcCO2 variation was 6%. The study should be further explored in patients with sleep apnea and various types of cerebrovascular and craniospinal disorders.

INTRODUCTION

Continuous positive airway pressure (CPAP) is the most effective and widely used therapy for Obstructive Sleep Apnea Syndrome (OSAS) a chronic medical condition associated with intermittent hypoxemia during sleep that occurs in 2 to 4% of people. OSAS is known to increase the risk of stroke and stroke risk factors, such as hypertension. CPAP therapy alleviates breathing conditions for patients with OSAS preventing the collapse of the pharyngeal airway during sleep [1].

Data about the effects of CPAP upon cerebral perfusion are conflicting [1-4]. A number of Transcranial Doppler Ultrasound (TCD) studies have found that MCA flow velocity was not influenced by CPAP usage. However, TCD measurements do not take into account changes in artery diameter and it is unclear if changes in MCA flow accurately reflect total change in CBF. In addition, blood Carbon Dioxide (PCO2) level is an agent important for cerebral circulatory autoregulation. In the previous CPAP studies [1-4], end tidal CO2 level was monitored, which can be significantly different than the CO2 level in the blood.

METHODS

US measurements were obtained in 23 healthy, young, non-smoking volunteers (12 male; mean age 25; mean BMI 21.8 kg/m^2), with and without CPAP at 15 cm H_2O applied through a full face mask, in a randomized order following a structured protocol. The US measurements were performed during the afternoon at atmospheric pressure a minimum of two hours after the last meal and drink with caffeine in a standard room with controlled temperature. Measurements were obtained with the subject in the supine position with a head tilt of 30 degrees. Peak systolic, end diastolic flow velocity and arterial diameter in M-mode were obtained for three time points at the left and right proximal ICA and VA, while only flow velocity was studied in both MCA. $PtcCO_2$ was monitored using a single sensor applied to the chest. SaO_2 and pulse rate were monitored with a finger pulseoximeter during the entire measurement period. BP was recorded before and after each test. US measurements under CPAP began after $PtcCO_2$ level returned to baseline (±2mmHg) or 15 minutes following the fitting of the mask.

All velocity and diameter measurements based on the US images were conducted by a single blind operator. Mean flow velocity, V, in each vessel was calculated by measuring the peak systolic, $V_{sys.}$, and end diastolic, $V_{dia.}$, velocity for three cardiac cycles (Eq. 1).

$$V = \frac{V_{sys.}+2V_{dia.}}{3} \quad (1)$$

Similarly, mean diameter, $\overline{D}$, of each vessel for three cardiac cycles was calculated based on M-mode US measurements, where $D_{sys.}$ and $D_{dia.}$ are the peak systolic and end diastolic vessel diameters (Eq. 2).

$$\bar{D} = \frac{Dsys.+2D_{dia.}}{3} \quad (2)$$

Flow rate Q (ml/min) in each vessel was calculated by Eq. (3) and total experimental CBF, CBF_{exp}, was determined by the sum of the flow rates in the left and right ICAs and VAs assuming a parabolic velocity profile (Eq. 4).

$$Q = \frac{V}{2} * \frac{\pi \bar{D}^2}{4} \quad (3)$$

$$CBF_{exp.} = Q_{ICAs} + Q_{VAs} \quad (4)$$

Pulsatility index (PI) was calculated as the ratio of the difference between the peak systolic and end diastolic velocity and mean velocity (Eq. 5).

$$PI = (V_{sys.} - V_{dia.})/V \quad (5)$$

The relation between PCO_2 (mm Hg) and CBF (ml/min/100 g brain tissue) has been quantified in the Rhesus monkey by Reivich [5] (Eq. 6).

$$CBF_{monkey} = 20.9 + \frac{92.8}{1+10570e^{-5.251\log(PtcCO2)}} \quad (6)$$

It has also been found that every 100 g of human brain tissue receives about 52.2 ml/min of blood while the monkey brain receives about 48.8 [5]. Additionally, the human brain is ~ 14 times the weight of a Rhesus monkey. Based on these assumptions, a theoretical CBF vs. PCO_2 curve can be calculated (Figure 1). Using this curve, the theoretical change in CBF, $CBF_{theor.}$, was determined given the experimentally measured change in $PtcCO_2$.

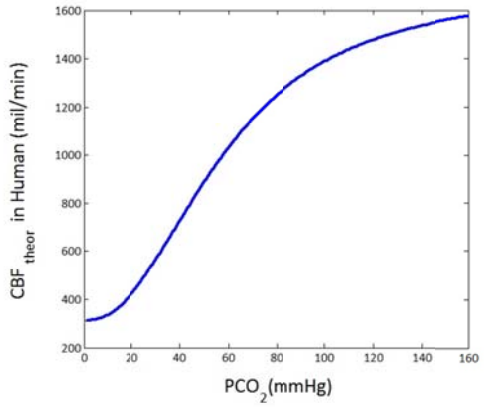

Figure.1. Theoretical relation of human arterial $PtCO_2$ and CBF based on Reivich study [5].

RESULTS

Table 1 summarizes the results with and without CPAP for the 23 subjects in terms of mean values for: PtcCO2, BP, $CBF_{exp.}$, flow velocity, and PI. The $CBF_{exp.}$ decreased by 17% with CPAP (p-value<0.05). The mean theoretical decrease in CBF was 6%.

Flow velocity decreased by 9, 3 and 11% at the ICA, VA, and MCA, respectively with CPAP.

Table 1. Comparison of mean results for the 23 healthy subjects with and without CPAP (STD is standard deviation).

	NO CPAP	STD	With CPAP	STD
PtcCO2 [mm Hg]	36.4	5.3	33.7	6.2
BP [mm Hg]	87.3	6.6	90	7.1
CBF_{exp} [ml/min]	507.6	168.4	419.4	119.4
V_{ICA}[m/s]	0.46	0.08	0.42	0.06
V_{VA}[m/s]	0.30	0.06	0.29	0.07
V_{MCA}[m/s]	0.63	0.13	0.59	0.13
PI_{ICA}	0.99	0.19	0.99	0.25
PI_{VA}	1.24	0.23	1.23	0.26
PI_{MCA}	0.79	0.12	0.84	0.11

DISCUSSION

These preliminary findings suggest that an increase in mean airway pressure of 15 cm H_2O could significantly decrease total CBF in healthy awake volunteers, and that the decrease was greater than that predicted by $PtcCO_2$ changes alone. However, the US measurements require further examination to determine operator dependent measurement variability. It is yet unclear how these results would translate to sleeping people, subjects with OSAS and various types of cerebrovascular and craniospinal disorders such as stroke and hydrocephalus.

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