The acute effects of alcohol intoxication on the volume of internal carotid artery and brain volume dynamics

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Background: Animal studies of acute ethanol administration on vascular motor responsivity showed both vasodilatation and vasoconstriction of cerebral blood vessels, with higher doses of ethanol associated with vasoconstriction [1]. Application of ethanol doses equivalent to 1-2g/kg of body mass on *in-vitro* preparations of isolated human peripheral blood vessels was associated with vasodilatation [2]. Also *in-vivo* Doppler studies of the middle cerebral artery in humans after alcohol intake of 1g/kg of body weight suggested vasodilatation [3]. Change in volume of arteries and arterioles after ethanol consumption may result in acute changes of brain tissue volume [4].

Methods: Seven healthy male, non-smoking, light-drinking individuals (LD, 33±6 years; 9±9 alcoholic drinks/month over 1-year prior to study) underwent two MRI sessions (baseline and after acute alcohol consumption). TOF 3D MRA (TR/TE = 39/7ms; flip angle = 25; voxel size: 0.39 x 0.39 x 0.79 mm^3; orientation of 3D volume: oblique axial, perpendicular to basilar artery) was used to assess the lumen of precavernous segment of internal carotid artery (ICA-PC) and 3D MPRAGE (TR/TE/TI=10/4/300ms) and double spin echo ($TR/TE_1/TE_2$=5000/20/80ms) to measure brain tissue volume. After baseline examination, participants consumed approx. 0.9g/kg body weight of ethanol in fruit juice (equivalent to 4-5 standard alcoholic drinks) over 15-20 minutes. Intoxication MRI was acquired near peak breath alcohol levels (BrAC). Three of the participants underwent another MRI session without alcohol ingestion about 3 weeks later to assess the measurement variance of our method. Identification of ICA-PC was performed in two steps: (1) the MRA was thresholded at 35% of the maximum intensity of the entire 3D angiogram and manually edited to mask and isolate the left and right ICA-PC, as well as remove extraneous signals. (2) The segments were automatically identified on each slice by thresholding the angiogram within expanded arterial masks at 50% of maximum intensity (within the expanded mask). This second analysis was to assure that the maximum intensities defining the thresholds originated from the arterial segments of interest and to account for differences between maximum signal intensities between left and right ICA-PC among different slices. Changes in blood flow velocity after ethanol intoxication and possible artifacts caused by change in flow velocity were assessed numerically [5]. Brain tissue volume changes between baseline and intoxication condition were assessed with boundary shift integral (BSI) [6][7].

Results: Near peak BrAC (0.10±0.02%), ICA-PC lumen volume was greater than at baseline by 22±13% and 29±17% for left and right ICA-PC respectively, and significantly greater than the error determined in the reliability study (both $p<0.01$). No significant differences in total brain tissue volume were observed between baseline and intoxication.

The reliability studies on 3 LD (without alcohol ingestion) showed the mean volumetric difference of ICA-PC segment was -3±2% for the left- and -5±7% for the right ICA-PC. The increase of maximum intensities within the left and right ICA-PC from baseline to intoxication was approx. 10%. Assuming laminar flow, this corresponds to an increase of maximum blood flow velocities of about 1%, which causes only 12-15% increase in the arterial volume due to flow artifact, as estimated by numerical simulations [5]. Thus, the observed increases of left and right ICA-PC lumen volumes appears to be over and above what can be explained by measurement variance and flow-related changes and is therefore interpreted to represent a direct response to acute alcohol intoxication. The error associated with the BSI method was –0.6±2.2cc, approx. 0.4% of intracranial volume [7].

Discussion: Vasodilatation of the precavernous segment of the internal carotid arteries during acute alcohol intoxication is consistent with previous human studies indicating vasodilation of select intracranial arteries. The non-significant changes in overall brain tissue volume during intoxication suggest two mechanisms: (1) non-homogeneous dilatation: i.e., some intracranial arteries dilate, while others constrict, so that the total brain tissue volume is constant, (2) osmotic forces: acute ethanol intoxication significantly increases plasma osmolality, so that interstitial fluid flows into the plasma to equalize osmotic imbalance. This latter mechanism is supported by decreased apparent diffusion coefficients as a consequence of acute alcohol intoxication [8].

References:

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