Human brain cooling during breath-holding

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Target audience

Physiologists and clinicians studying therapeutic brain cooling and metabolic rate suppression.

Purpose

Whether humans can affect cooling of vital organs, especially the brain, under any conditions is a controversial issue in thermal physiology. The aim of this exploratory study was to establish whether brain cooling could be induced by breath-holding (apnea).

Methods

A 43 year old diving instructor (S.M.) and expert breath-hold diver volunteered for this pilot study. Apnea was initiated at functional residual and terminated upon reaching the initial urge to breathe. Six measurement sessions were performed over a period of several months. Experiments were performed using a 1.5 T scanner (Achieva, Philips, The Netherlands). The head was immobilized with foam pads. Measurements were performed using a 2D rf spoiled gradient echo sequence in transversal plane (Fig. 1); FOV 280×280 mm; acquisition matrix (256, 128); TR/TE 40/6 ms; slice thickness 6 mm; bandwidth/pixel 125.4 Hz; 2 acquisitions. Net measurement time was 10 sec. Magnetic field homogeneity in the measured slice was improved by first order shimming before recording the first image. This shimming state was kept constant during the entire measurement session. Since shimming to switch-off was not possible, drift of the shimming system was expected, in addition to the B_0 drift. Magnetic field drift was monitored using the two probes P1, P2 filled with water at room temperature (22°C; Fig. 1). Reference probes were cylindrical (100 mm length, 24 mm diameter). The experiment began with ten baseline image recordings at 30 sec intervals. Apnea started at time t = 0 min (Fig. 2, 3). The next ten acquisitions were every 15 sec, followed by the ten measurements at 30 sec intervals. Mean phase was averaged from the water probes and from volume of interest (VOI) 16.4×16.4×6 mm^3 placed in the parietal lobe (black square, Fig. 1). Drift of the magnetic field was fitted by line (Fig. 2). Only points unaffected by apnea were fitted, i.e., points from the interval $0 \leq t \leq 4$ min were excluded (Fig. 2a). After corrections for linear drift, temperature changes were computed using the brain-water chemical shift coefficient -0.019 ppm/°C [1]. Measured data were smoothed by adjacent-averaging of 5 (Fig. 3a, b) or 3 points (Fig. 3c). Standard deviation of residues of water probe phase fits (Fig. 2b) in °C was used as a measure of accuracy of relative temperature estimations.

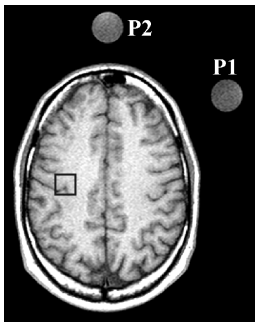

Fig. 1: VOI (black square) in parietal lobe. Water probe P1 and P2.

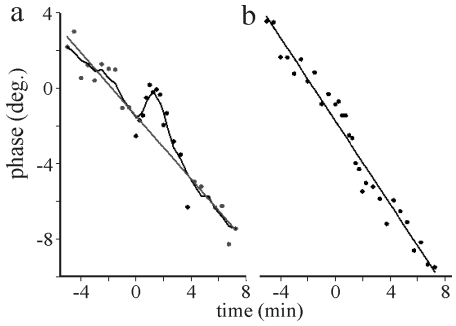

Fig. 2: Mean phase vs. time. Figure 1 shows VOI: (a) black square; (b) water probe P1. Fitted lines show drift of magnetic field.

Results

Figure 2 represents time course of the phase, as measured. In this example, the drift in magnetic field in selected VOI, water probe P1, and P2 were -0.00607, -0.00798, and -0.00774 ppm/min, respectively. Standard deviation of residues of water probes was ±0.3 °C. Relative brain temperature changes during experiment are shown in Fig. 3. Apnea duration varied between 75 and 105 sec. Brain temperature promptly decreased during apnea by ca 1 °C in 1 min and similarly returned to baseline upon termination of apnea. There was no sign of the temperature decrease abating when apnea was terminated. The subject did not report feeling cold during apnea. Brain cooling and re-warming were global, but regional differences were observed (not shown).

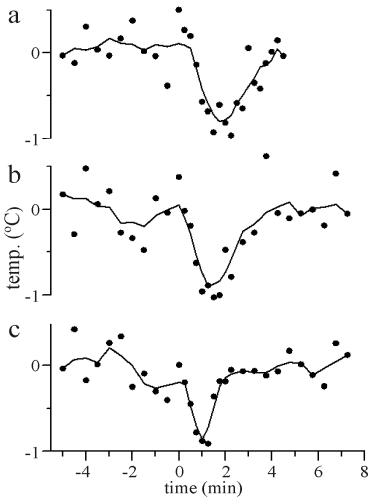

Fig. 3: Relative brain temperature vs. breath-hold duration: (a) 105 sec; (b) 90 sec; (c) 75 sec.

Discussion

To our knowledge, this is the first observation of human brain cooling under natural conditions, albeit by breath-holding. The phenomenon of brain cooling has only previously been observed in diving and hibernating animals with implanted brain temperature sensors. For example, in seals, forced dives lasting ca 10 min decreased brain temperature by ca 3°C [2]. Metabolic rate is recognized to be suppressed by 6-8% per °C. The promptness and rate of brain cooling detected here is probably the fastest ever recorded in a human over this range [3]. As neither hypercapnia (build-up of CO_2) nor hypoxia was present at the beginning of the experiment, the decrease in brain temperature was probably initiated by apnea, but there was evidence of an anticipatory response. Such rapid and extensive cooling can be explained by cooled blood entering the brain. Brain cooling was facilitated by a low lung volume, progressive hypercapnia and, possibly, some degree of hypoxia, i.e., additional components of the so called, dive response. Cooling of the brain at such a rate, e.g., during therapeutic hypothermia, would normally result in a dramatic increase in shivering and, hence, O_2-consumption. The dive response, however, is auto-regulated and neuroprotective, suppressing and matching metabolic rate to the limited O_2-supply. Consequently, there was no shivering.

Conclusion

As in other diving species, this study suggests that brain cooling in humans is a latent, natural state that can be triggered not only at room temperature but also in the absence of a carotid rete. Cooling of the brain develops promptly, globally and reversibly. The response is neuroprotective, suppressing metabolism and matching energy need to availability. The outcome of this exploratory study opens the possibility of devising new therapies that offer more effective neuroprotection in critically ill patients, e.g., stroke, cardiac arrest, brain injury, etc, possibly extending to the first-aid setting. Importantly, the response side-steps many shortcomings associated with therapeutic hypothermia.

References

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