Head and Neck Cooling Decreases Tympanic and Skin Temperature, But Significantly Increases Blood Pressure

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Background and Purpose—Localized head and neck cooling might be suited to induce therapeutic hypothermia in acute brain injury such as stroke. Safety issues of head and neck cooling are undetermined and may include cardiovascular autonomic side effects that were identified in this study.

Methods—Ten healthy men (age 35±13 years) underwent 120 minutes of combined head and neck cooling (Sovika, HVM Medical). Before and after onset of cooling, after 60 and 120 minutes, we determined rectal, tympanic, and forehead skin temperatures, RR intervals, systolic and diastolic blood pressures (BP), laser-Doppler skin blood flow at the index finger and cheek, and spectral powers of mainly sympathetic low-frequency (0.04–0.15 Hz) and parasympathetic high-frequency (0.15–0.5 Hz) RR interval oscillations and sympathetic low-frequency oscillations of BP. We compared values before and during cooling using analysis of variance with post hoc analysis; (significance, P<0.05).

Results—Forehead skin temperature dropped by 5.5±2.2°C with cooling onset and by 12.4±3.2°C after 20 minutes. Tympanic temperature decreased by 4.7±0.7°C within 40 minutes, and rectal temperature by only 0.3±0.3°C after 120 minutes. Systolic and diastolic BP increased immediately on cooling onset and rose by 15.3±20.8 mm Hg and 16.5±13.4 mm Hg (P=0.004) after 120 minutes, whereas skin blood flow fell significantly during cooling. RR intervals and parasympathetic RR interval high-frequency powers increased with cooling onset and were significantly higher after 60 and 120 minutes than they were before cooling.

Conclusions—Head and neck cooling prominently reduced tympanic temperature and thus might also induce intracerebral hypothermia; however, it did not significantly lower body core temperature. Profound skin temperature decrease induced sympathetically mediated peripheral vasoconstriction and prominent BP increases that are not offset by simultaneous parasympathetic heart rate slowing. Prominent peripheral vasoconstriction and BP increase must be considered as possibly harmful during head and neck cooling. (Stroke. 2012;43:2142-2148.)

Key Words: head cooling ■ hypothermia ■ sympathetic activation ■ blood pressure increase ■ peripheral vasoconstriction

Therapeutic hypothermia is neuroprotective after cardiac arrest1 or hypoxic ischemic encephalopathy,2 and after traumatic brain injury,3 and seems to be promising in acute stroke treatment.4 Ongoing clinical phase 3 trials such as EuroHYP-1 and ICTUS 2/3 evaluate therapeutic benefits of hypothermia for acute ischemic stroke.

Even mild hypothermia might be beneficial, but neuroprotective effects most likely depend on early induction of hypothermia. Induction by endovascular or surface-cooling devices is complex, invasive, and often requires sedation and an intensive care setting.5 In contrast, localized head and neck cooling is an easily applicable method that might be suited for immediate hypothermia induction already in the prehospital setting.6 However, hypothermia also influences the autonomic nervous system.7 8 Even local cold stimulation, eg, by immersion of a limb into ice water—known as the cold pressor test—activates sympathetic cardiac and peripheral vasomotor outflow and may increase heart rate (HR) and blood pressure (BP);9 whereas, cold stimulation of the facial area—known as the cold face test—induces cardiovagal outflow with HR-slowing and peripheral sympathetic activation with vasoconstriction and BP increase.9

Kallmünzer et al recently showed that local head and neck cooling may slightly reduce body core temperature.6 Therefore, the method might be suited for noninvasive hypothermia induction.6 However, cardiovascular autonomic effects of

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localized head and neck cooling are still unknown and might be similar to effects of cold pressor or cold face testing with changes in BP and HR that might limit benefits of cooling.

In this study, we therefore evaluated changes of cardiovascular parameters and autonomic modulation during local head cooling.

**Patients and Methods**

Ten healthy men (mean age, 35 ± 13 years) with a mean body weight of 78.4 ± 12.0 kg and a mean body mass index of 24.2 ± 3.2 kg/m² participated in the study. None of the participants had any known disease or was taking medication known to affect the cardiovascular or autonomic system. Before testing, all participants refrained from nicotine, caffeine, or alcohol for at least 18 hours. The Institutional Ethics Committee of the University of Erlangen-Nuremberg had approved the study, and written informed consent had been obtained from all study participants according to the Declaration of Helsinki.

**Measurement of RR Intervals, Blood Pressure, Respiration, Skin Blood Flow and Temperatures**

We continuously recorded electrocardiographic RR intervals (RRI) using a standard 3-lead electrocardiogram, and noninvasively monitored beat-to-beat systolic (BPsys) and diastolic blood pressures (BPdia) by means of radial artery plexus-tonometry at the wrist (Colin Pilot, Colin Medical), with oscillometric BP calibration at the brachial artery. 10

We recorded respiratory frequency using calibrated 2-belt chest-abdomen inductance plethysmography (Respiracitit Calibrator, Ambulatory Monitoring, Inc.) with 1 belt at the level of maximal thoracic and the other at maximal abdominal respiratory excursions. 11

Skin blood flow (SBF) was monitored at the right index finger pulp and the left cheek using laser Doppler flowmetry (Perimed). The laser probe emits a divergent narrow band light at a wavelength of approximately 780 nm with an intensity ≤0.8mW. 10 The volume measured in the skin is a hemisphere with an approximate radius of 1 mm. 10 However, the instrument does not measure perfusion in absolute values (ml/min g⁻¹), given that the measured volume is tissue-dependent and not exactly known. 10–12 Therefore, after calibration of the instrument with a moityl standard according to the manufacturer, flow was measured in arbitrary perfusion units (PU). 10–12

Temperature was measured continuously with 1 skin probe attached to the forehead (Bio Thermosat BTH-5), 1 probe inserted into the rectum (Temprecise, Arizant Healthcare, Inc,) and a third probe inserted into the outer ear canal for tympanic temperature recording (EElan Med GmbH).

**Baseline Recordings at Supine Rest and Recordings During Head Cooling**

Participants were tested between 9 AM and 2 PM. They were lying on a comfortable stretcher in a quiet room with an ambient temperature of 24°C and stable humidity. All participants initially rested for ≥40 minutes to ensure a stable cardiovascular situation while we attached the monitoring devices.

Cold stimulation was accomplished with a head and neck cooling device (Sovika, HVM Medical) containing free-floating cooling gel and consisting of a central portion, covering the skull and forehead, and 3 flaps covering the dorsal and parietofrontal cervical regions. Before use, the cooling device was kept at 4°C.

For RRRs, BPsys, BPdia, SBF, and respiratory frequency, we assessed individual average values from 5-minute signal epochs recorded at baseline, immediately after cooling onset, after 60 minutes cooling, and after 120 minutes cooling.

For RRIs and BPsys, BPdia, SBF, and respiratory frequency, we used individual average values from 5-minute signal epochs recorded at baseline, immediately after cooling onset, after 60 minutes cooling, and after 120 minutes cooling.

Forehead skin, tympanic, and rectal temperatures were measured at baseline, immediately after cooling onset, and after every 10 minutes of cooling. Simultaneously, participants had to rate the perception of frostiness and overall discomfort on a visual analog scale ranging from 0 (no frostiness/discomfort) to 10 (maximum conceivable frostiness/discomfort).

After 120 minutes, the cooling device was removed while measurements continued for another 20 minutes for safety reasons and to assure return of parameters to baseline values.

Criteria to abort cooling were a decrease in BP by more than 20 mm Hg, bradycardia above 1200 ms, tachycardia below 500 ms, arrhythmias, and complaints about significant discomfort or pain indicating the participant’s desire to end the test.

**Data Storage and Offline Analysis**

RRI, BPsys, SBF, and respiratory data were digitized by a custom-made analog-to-digital converter, sampled at a rate of 300Hz, and fed to a Macintosh PowerBook computer (Apple Inc). Artifacts were manually cleaned by linear interpolation and data were stored for offline analysis. 13 A C-language program identified all electrocardiographic QRS complexes in each sequence, located the peak of each R-wave, and calculated consecutive RRRs. From the continuous waveforms of all parameters, beat-to-beat mean values were automatically calculated and interpolated linearly between adjacent values to construct a corresponding continuous time series. 13 From 5-minute recordings taken at baseline, immediately after onset of head cooling, after 60 and after 120 minutes of head cooling, we selected 60-second epochs without artifacts to calculate mean values and standard deviations of RRRs, BPsys, BPdia, SBF, and respiratory frequency.

**Analysis of Spectral Powers of Autonomic Modulation**

RRI and BP values show underlying fluctuations that are largely mediated by the undulating activity of the sympathetic and parasympathetic nervous systems. 14 These underlying fluctuations were characterized by autoregressive analysis using a linear detrending option and model order estimation according to Akaike information criterion. 15 The autoregressive algorithm gives reliable estimates of the frequencies and powers of the relevant oscillations with a relatively small amount of data that still assures stationarity of signals. 13

We identified peaks of oscillations in the so-called low-frequency (LF; 0.04–0.14 Hz) and high-frequency (HF; 0.15–0.50 Hz) ranges of RRI and BPsys, 9,14 LF oscillations of RRI at rest are considered to be mediated by sympathetic outflow and, to an undetermined degree, also by parasympathetic activity. 9 In contrast, LF oscillations of BP are related to sympathetic outflow only. 9,14 HF oscillations of RRI reflect parasympathetic activity, 9,14 whereas HF oscillations of BP are primarily a mechanical consequence of respiration-induced fluctuations in venous return and cardiac output. 9,14 The magnitude of LF and HF oscillations was determined as the integral under the power spectral density curves of RRI (ms²/Hz) and BP (mm Hg²/Hz) for the 2 frequency bands, and was expressed as LF and HF powers of RRI (ms²) and BP (mm Hg²). 9,14 In addition, we calculated the RRI-LF/HF ratios as an index of sympathovagal balance. 9

**Statistical Analysis**

We tested data for normal distribution using the Shapiro-Wilk test. Differences in RRIs, BPsys, BPdia, SBF, and respiratory frequency values at baseline, on cooling onset, and after 60 and 120 minutes of head cooling were assessed by an ANOVA for repeated measurements (general linear model). Differences in skin, rectal, and tympanic temperature values, in values of frostiness and overall discomfort at baseline, on cooling onset, and after every 10 minutes of cooling were also assessed by ANOVA. The suitability of the ANOVA model was determined by Mauchly’s sphericity test. In case of violation of the sphericity assumption, the Greenhouse Geisser correction was employed.

In case of significant ANOVA results, we performed post hoc single comparisons. For comparison of values at rest and during head cooling, we used t tests for paired samples in case of normal distribution, and the Wilcoxon test in case of non-normal distribution of data. A commercially available statistical program (SPSS, SPSS Inc) was used for data analysis. Significance was set at P<0.05.
Results
Data are presented as Mean±SD. Perception of frostiness (Figure 1) significantly increased from baseline visual analog scale scores of 0.1±0.3, to highest visual analog scale scores immediately after onset of cooling (3.4±1.6; P=0.005). After 20 minutes cooling, perception of frostiness constantly improved until the end of cooling (2.1±1.7 after 60 minutes; 1.6±1.8 after 120 minutes). Three participants rated their maximum frostiness at a score of 5, and 2 participants even rated their maximum frostiness at 6.

Perception of discomfort (Figure 1) also increased significantly during cooling, from baseline visual analog scale scores of 0.3±0.7 to scores of 1.9±1.4 immediately after cooling onset, similar scores after 60 minutes cooling (1.9±1.9), and highest scores after 120 minutes cooling (2.2±1.8; P=0.018). Two participants rated their maximum discomfort at a score of 4, 1 participant rated his maximum discomfort at 5, and 1 participant rated his maximum discomfort at 6.

Head cooling significantly decreased forehead skin, tympanic, and rectal temperatures (Figure 1). Forehead skin temperature decreased from 37.3±0.3°C at baseline to 31.8±2.2°C immediately after cooling onset, and to lowest values of 24.9±3.2°C (P<0.001) after 20 minutes cooling; then, forehead skin temperature slowly increased again until the cooling device was removed (28.5±3.5°C after 60 minutes, 34.1±2.4°C after 120 minutes).

Tympanic temperature decreased from 36.6±0.7°C at baseline to 34.9±1.2°C immediately after cooling onset, to lowest values of 31.8±1.2°C after 40 minutes cooling (P<0.001), and then slowly increased again until the end of cooling (32.1±1.1°C after 60 minutes, 33.3±1.0°C after 120 minutes). Rectal temperature slowly decreased from 36.7±0.4°C at baseline to 36.5±0.3°C after 60 minutes, and to 36.4±0.3°C after 120 minutes (P=0.021) of head cooling.

HR slowed significantly during head cooling, measured as RRI increase from 983.4±192.0 ms at baseline, to 1006.5±178.0 ms on cooling onset (P>0.05), and to highest RRIs (1101.5±231.2 ms; P=0.005) after 60 minutes cooling. Then, RRIs slightly decreased again to 1099.3±215.5 ms after 120 minutes, values that were still higher than at baseline (P=0.011; Figure 2; Table).

BPsys increased steadily during head cooling, from 116.8±7.0 mm Hg at baseline to 123.0±9.0 mm Hg after cooling onset, to 125.9±9.1 mm Hg after 60 minutes and 132.1±16.5 mm Hg after 120 minutes. However, differences compared with baseline values were not significant (P>0.05; Figure 2; Table).

BPdia increased steadily and significantly from 64.8±5.0 mm Hg at baseline to 69.3±7.0 mm Hg after cooling onset, to 72.9±6.0 mm Hg after 60 minutes, and to maximum values of 81.3±13.7 mm Hg after 120 minutes of cooling (P=0.004; Figure 2; Table).

SBF decreased significantly during cold stimulation, with SBF at the right index finger decreasing from 189.7±84.9 PU at baseline to 94.8±45.2 PU immediately after cooling onset, and to a minimum of 33.8±20.8 PU after 60 minutes cooling (P=0.001). Then, index finger SBF slightly increased again to 35.3±30.2 PU after 120 minutes (Table).

SBF at the left cheek decreased from 77.5±46.0 PU at baseline to 55.8±31.9 PU after cooling onset, to 37.3±24.2 PU after 60 minutes, and reached a minimum value of 34.2±22.5 PU after 120 minutes cooling (P=0.013; Table).

Respiratory frequency remained unchanged during head cooling (P>0.05). Parasympathetically mediated RRI-HF powers increased immediately after cooling onset from 464.9±289.1 ms² at baseline to 1446.3±1019.6 ms² (P=0.014). RRI-HF powers after 60 minutes (1271.6±1071.9 ms²) and after 120 minutes cooling (1230.2±1644.9 ms²) were still significantly higher than at baseline, but no longer as high as after cooling onset (Figure 2; Table).

RRI-LF powers, RRI-LF/HF ratios, and spectral powers of BPsys and BPdia did not change significantly during cold stimulation (P>0.05; Table).
Discussion

In all our participants, head and neck cooling significantly decreased forehead skin, tympanic, and to some extent, even rectal temperatures.

Although mean scores of frostiness and discomfort did not exceed 3.4 and 2.2 on the 0 to 10 scales, 5 of the 10 participants indicated their maximum frostiness at 5 and 6, and 40% scored their maximum discomfort between 4 and 6. These rather high values most likely contributed to the sympathetic activation and BP increase during head and neck cooling given that stimuli perceived as noxious, cold, or painful cause A-δ- and C-nerve-fiber activation9,16–18 and elicit cold pressor responses with surges in sympathetic outflow.9

Despite frostiness, head and neck cooling had a rather slow and limited effect on rectal temperature, with a decrease from 36.7±0.4°C at baseline to only 36.5±0.3°C after 60 minutes and 36.4±0.3°C after 120 minutes.

In individual participants, rectal temperature decreased by more than the average 0.3°C after 120 minutes cooling and even dropped by 0.6°C, showing that there is some effect of local head and neck cooling on body core temperature. Yet, the effect still seems rather small.

Using the same head and neck cooling device in 10 healthy volunteers, Kallmünzer et al observed a maximum decrease in rectal temperature by 0.65°C after 60±54 minutes cooling.6 The more prominent effect on body core temperature might be caused by the fact that the authors included 4 women in their study. Although their and our participants had similar body weight, body mass index, and ages, body mass index very likely differed between women and men of the group reported by Kallmünzer et al; this might have accounted for a more prominent effect of head and neck cooling in individual, slender participants.

Although head and neck cooling has some effect on body core temperature, this effect might vary with the body stature and composition; therefore, this still needs to be validated in a large cohort comprising participants with a wide range in age, weight, and body mass index before effects of local head and neck cooling on body-core temperature can be determined to be clinically efficient.
In contrast to rectal temperature, tympanic temperature rapidly decreased from 36.6 ± 0.7°C at baseline to 34.9 ± 1.2°C immediately after cooling onset, and dropped further to 31.8 ± 1.2°C within 40 minutes of cooling.

The therapeutic goal of head cooling is a significant decrease in intracranial temperature. Unfortunately, we were limited to monitoring tympanic temperature instead of the actual intracerebral temperature, as we had to avoid invasive procedures in our volunteers. Therefore, we could not assess temperatures more closely related to actual brain temperatures. Measurement of intracranial temperature via external ventricular drains may be an option in intensive care unit patients to assess closer correlations with intracerebral temperatures; however, we first had to evaluate cardiovascular effects of head and neck cooling in young, healthy volunteers before considering the method in intensive care unit patients. As most of our volunteers also rejected the placement of an internal jugular vein catheter, we also could not measure the temperature of blood returning from the brain, which could be a better parameter of brain-cooling effects than would the decrease in tympanic temperature.

Thus, we cannot rule out that the decrease in tympanic temperature was largely because of local effects on superficial tissue cooling and cooling of blood in the external carotid artery. However, we assume that the decrease in tympanic temperature of more than 4.5°C, ie, from 36.6 ± 0.7°C at baseline to 31.8 ± 1.2°C after 40 minutes, also induced thermal conduction through the skull and cerebrospinal fluid into brain tissue, and thus it had some intracerebral cooling effect. Thoresen et al applied selective head cooling to newborn piglets and were able to lower brain temperatures more effectively than rectal temperatures. Haaland et al induced hypothermia also in newborn piglets and showed closer correlations of cooling-induced intracerebral temperature decreases with tympanic than with rectal temperature decreases. However, the findings in newborn piglets do not necessarily imply similarly close correlations between tympanic and cerebral temperatures in human adults; this is because thermal convection differs significantly between the small animals and our adult humans because of different surface-area-to-mass ratios.

Thus, we cannot only assume that head and neck cooling not only effectively reduces tympanic temperature, but might also lower intracranial temperature. Yet, only more invasive measurements will verify the actual extent of head-and-neck-cooling-induced intracerebral hypothermia.

Forehead skin temperature decreased more quickly and more prominently than did the tympanic temperature. The cooling of skin temperature by, on average, as much as 5.5°C immediately on cooling onset, and by approximately 9°C within 20 minutes, not only caused prominent frostiness and discomfort in some of our participants, but also induced significant changes in cardiovascular parameters that might limit benefits of head-cooling-induced hypothermia.

The onset of head cooling resulted in a rapid reduction of superficial SBF, ie, peripheral vasoconstriction, and in an increase in BP, particularly BPdia; whereas HR slowed, ie, RRI increased, because of the activation of parasympathetically mediated HF powers of RRI modulation immediately on and during head cooling.

For all parameters, cooling-induced changes became more prominent during the first 60 minutes and even longer for cheek SBF and for systolic and diastolic BP until the end of cold stimulation, when discomfort also was highest. Cardiovascular changes are similar to those observed during the cold face test that increases BP because of augmented peripheral sympathetic vasomotor tone and lowers HR via enhanced parasympathetic outflow. In contrast to our young and healthy study participants, the sympathetic responses with vasoconstriction and BP increases.
are very likely even more prominent in most stroke patients who are usually older than are our volunteers. In older persons, and particularly in patients after stroke, autonomic modulation is shifted toward more prominent sympathetic and less parasympathetic activity. Thus, we assume that head and neck cooling in older persons, and particularly in stroke patients, might result in at least similar, but most likely more pronounced, peripheral vasoconstriction and BP elevation in our healthy participants. In contrast, limited cardiac modulation of stroke patients most likely yields less cardiac activation with head cooling, and thus less buffering of HR than was observed in our young participants. Consequently, head and neck cooling might further more disadvantageous cardiovascular effects in older persons and after stroke than in our participants.

The cooling-induced cardiac activation with slowing of HR may have beneficial effects in patients with cerebral lesions caused by stroke, traumatic brain injury, or epilepsy, i.e., in patients with the abovementioned shifts of autonomic balance toward predominant sympathetic outflow. This increase in cardiac outflow with subsequent HR slowing may be mediated by direct, cold-induced stimulation of the trigeminal brain stem reflex and of parasympathetic efferent pathways, as described with cold face stimulation; or, it might be secondary to the cold-evoked increase in sympathetic activity and BP elevation, i.e., to baroreflex activation.

On cooling onset, BPsys increased immediately and prominently, by an average of 6.2 ± 10.5 mm Hg, and continued to rise to values that were more than 15 mm Hg higher after 120 minutes cooling than at baseline. Because of high interindividual differences, the increase was not statistically significant. Yet, the high SD indicates an even more pronounced sympathetic vasoconstriction and BP elevation in individual participants; this implies a risk of harmful cardiovascular and cerebrovascular side effects of head cooling. Very likely, the concomitantly parasympathetic activation and HR slowing prevented the rise of BPsys to even higher values.

Similar to BPsys, BPdia also increased immediately, and significantly, by an average of 4.5 ± 5.9 mm Hg on cooling onset, and continued to rise to values that were 8.1 ± 7.2 mm Hg and 16.5 ± 13.4 mm Hg higher after 60 and 120 minutes cooling, respectively, than at baseline.

The prominent rise in diastolic pressure reflects an increase in peripheral resistance caused by peripheral vasoconstriction. Evidently, the rapid decrease in forehead skin temperature by more than 12°C after only 20 minutes cooling and the high level of frostiness trigger a strong sympathetic response, with drop in peripheral SBF and critical increase in BPs.

Particularly in acute stroke patients, potential benefits of head-and-neck-cooling-induced cerebral hypothermia, which still need to be proven by more direct measures of intracranial temperature, may be offset by such prominent increases in systolic and diastolic BPs.

There is evidence that acute hypertension increases the risk and severity of hemorrhagic transformation in acute stroke. In rabbits with experimental ischemic stroke, Bowes et al showed that acute hypertension is associated with an increased risk and severity of hemorrhagic transformation. In a follow-up study, Fagan et al showed that acute hypertension is not only a marker related to increased risk of hemorrhagic transformation, but is causative for hemorrhagic transformations. In 793 acute ischemic stroke patients of the ECASS-II trial, Yong and Kaste demonstrated that increased variation in BP profiles, including the within-patient BPsys maximum, is associated with an increased hazard ratio of parenchymal hemorrhages. The relevance of BPdia elevation, which was particularly prominent in our participants with head and neck cooling, is underlined by the findings of Ko et al. The authors monitored BP variability in 792 acute ischemic stroke patients during the first 72 hours after hospital admission and showed that increased variability, particularly of BPdia, is associated with the development of hemorrhagic transformation.

As mentioned above, older persons, and especially acute stroke patients, show even more pronounced sympathetic responses to activating stimuli. Therefore, head and neck cooling might not be indicated in stroke patients unless there is stringent pharmacological BP control or buffering of sympathetic stimulation by pain-relieving and sedating medication. Yet, sedating medication negatively reduces the ability to judge the patient’s clinical status and to monitor any disease deterioration.

Moreover, the significant vasoconstriction with a decrease in cheek SBF by 21.5% ± 39.0% and in finger-pulp SBF by 43.3% ± 29.8% on cooling onset may compromise or delay the attempt to lower body core temperature. Vasoconstriction of superficial skin vessels and the subsequent decrease in SBF is a powerful defense against cooling and delays the decrease in core temperature by slowing temperature exchange between body core and skin surface by reducing cutaneous heat loss. Consequently, superficial skin cooling with activation of sympathetic outflow and vasoconstriction might result in a delayed induction of central hypothermia unless there is sedation or analgesia.

Moreover, prominent peripheral vasoconstriction, as observed in our volunteers, lowers the ratio between blood flow velocity and arterial BP, i.e., the vascular conductance; this occurs not only in superficial cutaneous, but also in deeper peripheral, as well as visceral arteries, and thus contributes importantly to the BP increases seen in our study. Thus, pronounced peripheral vasoconstriction may be potentially harmful in fragile patients such as acute stroke patients.

Only patients with low baseline BP might possibly benefit from blood pooling toward the central circulation and brain caused by cold-induced increases in peripheral resistance and BP.

Summary
The prominent decrease in tympanic temperature by 4.7 ± 0.7°C within 40 minutes suggests that head and neck cooling might also lower cerebral temperature. However, more direct measurements of intracerebral temperature are needed to confirm this assumption.

In contrast, head cooling had no relevant effects on body core temperature as shown by the only marginal, 0.3 ± 0.3°C decrease in rectal temperature despite 120 minutes cooling.
However, the pronounced decrease in forehead skin temperature by more than 12°C within 20 minutes and the significant increase in frostiness triggered prominent sympathetic responses with peripheral vasoconstriction and steadily increasing systolic and diastolic BPs by more than 15 mm Hg within 120 minutes head cooling. The concomitant cardiovagal activation and HR slowing could not adequately buffer the sympathetic activation. Thus, BP increased by more than 2148 Stroke August 2012

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