Hypothermia Does Not Alter Somatosensory Evoked Potential Amplitude and Global Cerebral Oxygen Extraction during Marked Sodium Nitroprusside–induced Arterial Hypotension

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Background: To prevent neurologic damage, monitoring cerebral function by somatosensory evoked potentials is used in selected settings. Excision of intraocular melanoma provides a unique opportunity to assess independently during anesthesia the effects on median nerve somatosensory evoked potentials (MN-SSEPs) and cerebral oxygen extraction of sodium nitroprusside–evoked arterial hypotension with and without hypothermia.

Methods: Median nerve somatosensory evoked potentials, arterial pressure, jugular venous bulb oxygen saturation (Sjo_2) and lactate concentration, and arterial–jugular bulb oxygen content difference were assessed during propofol–remifentanil anesthesia under sodium nitroprusside–evoked arterial hypotension (mean arterial pressure, 40 mmHg) with and without surface hypothermia (32°C) in 11 otherwise healthy patients undergoing resection of choroidal melanoma.

Results: Hypothermia alone did not affect peak-to-peak amplitude of N20/P25 but prolonged cortical latency of N20 (22.6 ± 2.2 *vs.* 25.9 ± 2.5 ms, $P < 0.05$), cervical latency of N13 (14.3 ± 1.2 *vs.* 15.7 ± 1.6 ms, $P < 0.05$), and central conduction time (8.3 ± 1.4 *vs.* 10.2 ± 1.6 ms, $P < 0.05$). Evoked arterial hypotension did not depress MN-SSEP N20/P25 amplitude either with or without hypothermia (−0.31 *vs.* −0.28 μV, $P > 0.05$) or alter latency (0.08 *vs.* 0.1 ms, $P > 0.05$). Furthermore, hypotension with or without hypothermia did not change Sjo_2, arterial–jugular bulb oxygen content difference, or lactate concentration.

Conclusions: Thus, hypothermia to 32°C does not alter MN-SSEP amplitude and global cerebral oxygen extraction during marked sodium nitroprusside–induced arterial hypotension with a mean arterial pressure of 40 mmHg but prolongs MN-SSEP latencies during propofol–remifentanil anesthesia in individuals without cerebrovascular disease.

CEREBRAL ischemia is one of the most devastating complications of perioperative arterial hypotension. Somatosensory evoked potentials (SEPs), used to assess neural function during anesthesia for cerebrovascular surgery[1,2] or hypothermia,[3–7] may be particularly important to prevent neuronal damage by directing the anesthesiologist's attention to possible ischemia. Unfortunately, knowledge of the relationship of median nerve somatosensory evoked potentials (MN-SSEPs) amplitude or latency to hypotension and hypothermia is scarce, with most data derived from experiments in animals in which graded hypotension was induced by blood withdrawal,[8,9] by ganglion-blocking agents,[10,11] or during cardiac bypass.[12] In fact, the relation between hypotension, hypothermia, or their combination and MN-SSEP amplitude in humans has not been systematically studied. However, if MN-SSEPs are to be used as an intraoperative monitoring tool or to define limits of arterial hypotension, temperature-dependent and arterial pressure–dependent effects on MN-SSEPs and cerebral oxygen extraction must be separated. This is particularly true when hypotension is deliberately induced by sodium nitroprusside (SNP) since both hypothermia and SNP may or may not[13–20] interfere with cerebrovascular autoregulation.

Accordingly, we report our experience with evoked arterial hypotension and hypothermia during propofol–remifentanil anesthesia for excision of intraocular malignant melanoma. Technically, this surgery involves lamellar dissection of sclera, regional *en bloc* removal of sclera and choroid, and resuturing the remaining sclera to cover the resected area.[21] This transscleral resection is performed in a few centers worldwide for large uveal tumors that cannot be eradicated by radiotherapy alone, to preserve visual function and avoid enucleation. Since surgery can easily result in intraocular hemorrhage leading to loss of vision or of the eye, controlled arterial hypotension with a mean arterial pressure around 40 mmHg during the critical parts of surgery is required.[22] Furthermore, since hypotension in this range is near if not below the lower inflection point of the cerebral autoregulatory pressure–flow relation, surface hypothermia has been suggested to lessen the risk of cerebral ischemia and improve surgical conditions.[23]

Accordingly, this setting provides a unique opportunity to assess independently in the same individuals the effects on MN-SSEPs and cerebral oxygen extraction of SNP-evoked arterial hypotension with and without hypothermia.

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Materials and Methods

Patients

Nineteen otherwise healthy patients (American Society of Anesthesiologists physical status class I) were

scheduled for possible resection of intraocular melanoma under hypotension and hypothermia following informed written consent. To minimize risks evoked by unrecognized cerebrovascular or coronary artery disease, all patients had undergone extensive preoperative assessment, including history, physical examination, 24-h arterial pressure measurements, echocardiography, stress electrocardiography, cerebrovascular duplex sonography, pulmonary function tests, and standard laboratory tests. In 8 patients, the tumor eventually proved to be nonresectable intraoperatively, leaving for final analysis 11 patients undergoing evoked hypotension both before and during induced hypothermia (5 men, 6 women; age, 54 ± 13.7 yr [mean $\pm$ SD]; weight, 74 ± 13.5 kg; height, 173 ± 8.6 cm).

General Procedures

After premedication with 1 mg oral flunitrazepam, both on the evening before surgery and in the morning 1 h before induction of anesthesia, preoperative MN-SSEPs were recorded. A five-lead electrocardiography was applied to monitor heart rate, rhythm, and ST segments (leads II and V_5), a pulse oximeter was attached, and a peripheral venous cannula was placed. For continuous measurement of arterial pressure and arterial blood sampling, a radial artery catheter was inserted under local anesthesia. General anesthesia was induced by 200 $\mu g \cdot kg^{-1} \cdot min^{-1}$ propofol (Klimofol®; IVAmed, Mannheim, Germany) and 0.5 $\mu g \cdot kg^{-1} \cdot min^{-1}$ remifentanil (Ultiva®; Glaxo Welcome, Hamburg, Germany) followed by 0.1 mg/kg vecuronium (Norcuron®; Organon Teknika, Oberschleißheim, Germany). Following tracheal intubation, patients were mechanically ventilated with 30% oxygen in air. Anesthesia was maintained with propofol (120 $\mu g \cdot kg^{-1} \cdot min^{-1}$), remifentanil (0.3 $\mu g \cdot kg^{-1} \cdot min^{-1}$), and vecuronium (2 mg/h), and no bolus injections were administered at any time. A gastric tube, esophageal and rectal temperature probes, and a Foley catheter were also placed. Normocapnia was established using mainstream capnography (Siemens, Erlangen, Germany) and repeatedly confirmed by arterial blood gas analysis (α-stat). For central venous pressure measurements, drug infusion and blood sampling, a triple-lumen catheter was inserted *via* the right internal jugular vein, and its position close to the right atrium was confirmed by intravascular electrocardiography. For cerebrovenous blood sampling, a catheter (20 gauge; Braun, Melsungen, Germany) was advanced into the jugular bulb *via* a 6-French introducer following retrograde cannulation of the left internal jugular vein, as described.[24] Jugular bulb blood was sampled at a rate of 0.5 ml/min to avoid extracranial contamination.[25] In three patients, jugular bulb blood could not be analyzed because samples turned out to be coagulated.

Following anesthetic induction, 500 ml hydroxyethyl starch, 10%, and 500 ml normal saline were infused to compensate for fluid losses, to optimize cardiac preload, and to minimize possible reflex tachycardia during induced arterial hypotension.

Measurements

Cardiovascular Variables. Heart rate was determined from the electrocardiogram (Sirecust 1281; Siemens), lead II. Arterial and central venous pressures were continuously measured by electromanometry relative to barometric pressure with transducers referenced to the mid axillary line (Sirecust 1281) and recorded on a multichannel strip chart recorder (Siredoc 220; Siemens) at slow speed.

Body Temperature. Esophageal and rectal temperatures were continuously measured (Sirecust 1281) with thermistor probes.

Blood Gas Tensions, Lactate concentration, and Oxygen Saturation. Blood oxygen and carbon dioxide partial pressures, pH, oxygen saturations, hemoglobin concentration, and lactate concentrations were measured using electrodes at 37°C (α-stat) or spectrophotometry (ABL-725; Radiometer, Copenhagen, Denmark) in arterial, central venous, and jugular bulb blood samples withdrawn simultaneously into chilled tubes and placed in crushed ice until analysis.

Neurophysiological Measurements. For median nerve stimulation and MN-SSEP acquisition, a commercially available system was used (Viking IV; Nicolet Biomedical Instruments, Madison, WI). Right and left median nerves were identified at the wrists with a stimulator probe and marked. Pairs of platinum needle stimulating electrodes were inserted subcutaneously over the median nerve at both wrists with the cathode placed 3 cm proximal to the anode. The median nerve was stimulated with 25 mA using a constant square wave impulse with a duration of 0.1 ms.

Median nerve somatosensory evoked potentials were recorded using platinum needle electrodes inserted subcutaneously and with an electrode impedance of less than 1 kΩ. The recording electrode positions over the left and right primary somatosensory cortex were chosen according to the international 10–20 classification system. The cortical MN-SSEP N20/P25 component was recorded from the C3' and C4' electrode positions over the left and right primary somatosensory cortex. The cervical MN-SSEP N13 component was recorded from an electrode over the spinous process C2. A midfrontal electrode (Fz) served as the reference for all channels. The central electrode (Cz) was used as ground.

Cortical responses to 200 stimuli delivered at a frequency of 4.7 Hz were averaged by computer and recorded simultaneously with cervical MN-SSEPs. The low-frequency response of the recording apparatus was 30 Hz, and the high-frequency response was 1,000 Hz. During measurements, averaged wave forms were displayed on an oscilloscope, and the data were stored on

floppy discs until analysis. Peak-to-peak amplitudes of N20/P25 and latencies of N13 and N20 waves were determined by cursor measurements, and central conduction time was calculated as the difference between cortical N20 latency and cervical N13 latency. Continuous recordings of MN-SSEPs then commenced and lasted throughout surgery.

Clinical Interventions and Data Sampling

Two episodes of evoked arterial hypotension were evaluated, *i.e.*, a hypotensive period of approximately 15 min before induced hypothermia (with body temperature allowed to decrease without attempts of active warming), and a second, more prolonged hypotensive period following induction of hypothermia, as required for completion of surgery. The initial hypotensive period allowed the surgeon to safely dissect the tissues in the vicinity of the tumor, making a final determination of its resectability, and also served as a final clinical check that the patient would likely tolerate more prolonged arterial hypotension as indicated by absent ST-segment changes and no major decrease in MN-SSEP amplitude. In eight patients, following the first hypotensive episode, the surgeon decided that the tumor could not safely be resected, and anesthesia was terminated without data acquisition.

In case the surgeon determined that the tumor appeared resectable (n = 11), further surgery was halted at normotension until hypothermia to an esophageal temperature of approximately 32°C had been induced using ice packs and a cool (8°C) circulating-water mattress. Once target hypothermia was reached, arterial hypotension was induced again, and surgery proceeded.

For evoked hypotension, SNP was infused together with sodium thiosulfate, with doses being increased as needed to evoke a decrease of mean arterial pressure to a final plateau of 40 mmHg, with mean arterial pressure decreased gradually at a rate of approximately 5 mmHg every 2 min.

Mean arterial blood pressure, heart rate, and esophageal temperature were recorded with each evoked potential recording. Arterial and jugular venous bulb blood gas tensions, pH, hemoglobin concentration, lactate concentrations, and oxygen saturations were determined before and after 15 min of arterial hypotension both before or during marked hypothermia, *i.e.*, at the nadir decrease of arterial pressure.

Statistical Analysis

Data are reported as mean ± SD. Differences in mean values of MN-SSEP variables between interventions (before/during hypothermia: yes/no) over mean arterial pressure were assessed by two-way repeated-measures analysis of variance followed by the Newman-Keuls *post hoc* test, if indicated. Other variables were compared during normotension and plateau hypotension (approx-

imately 40 mmHg) both before and during hypothermia. To assess temperature-dependent effects on MN-SSEP latencies, regression analysis was used. The following *a priori* null hypotheses were tested: There is no difference in means of variables (latencies and amplitudes) when compared between (1) normotension and hypotension and (2) before and during hypothermia. An α-error P of less than 0.05 was considered statistically significant.

Results

High-quality MN-SSEPs were recorded in all patients, and a representative course of anesthesia and surgery is depicted in figure 1.

Sodium nitroprusside–evoked arterial hypotension with a mean nadir pressure of 40 mmHg did not significantly alter either MN-SSEP N20/P25 amplitude or latency (figs. 2A and B and table 1) at an esophageal temperature of approximately 36°C. Furthermore, there was no evidence of increased global cerebral oxygen extraction or lactate production, as shown by unchanged jugular venous bulb oxygen saturation (Sjo_2), arterial–jugular bulb oxygen content difference, and lactate concentrations (table 1). Arterial oxygen partial pressure and pH_a did not change (data not shown), nor did arterial carbon dioxide partial pressure or hemoglobin concentration (table 1).

Hypothermia *per se* did not alter MN-SSEP N20/P25 amplitude but increased latencies. During arterial normotension, the decrease in esophageal temperature to 31.6 ± 0.25°C was associated with a slight but highly significant increase in latency of all MN-SSEP components (fig. 3). Cortical N20 latency increased by 14.6% (22.6 ± 2.2 *vs.* 25.9 ± 2.5 ms, $P < 0.05$), and cervical N13 latency increased by 9.8% (14.3 ± 1.2 *vs.* 15.7 ± 1.6 ms, $P < 0.05$). Central conduction time, *i.e.*, the interval between N13 and N20 also increased (8.3 ± 1.4 *vs.* 10.2 ± 1.6 ms, $P < 0.05$) by 22.9% during hypothermia. Latencies as a function of body temperature are presented in figure 3, demonstrating a linear relation ($P < 0.05$) between latency of MN-SSEP components and esophageal temperature. Thus, for a 1°C decline in temperature (at unchanged pressure), N20 latency increased by 1 ms, N13 latency increased by 0.4 ms, and central conduction time increased by 0.1 ms. Hypothermia alone was associated with a slight but nonsignificant increase in Sjo_2 and decrease in arterial–jugular bulb oxygen content difference (table 1).

In contrast, arterial hypotension to a mean arterial pressure as low as 40 mmHg during hypothermia *per se* did not change latencies (fig. 2B and table 1).

Effects on MN-SSEP amplitude of SNP-evoked arterial hypotension during hypothermia are shown in figure 2A. Like before induced hypothermia, N20/P25 amplitude
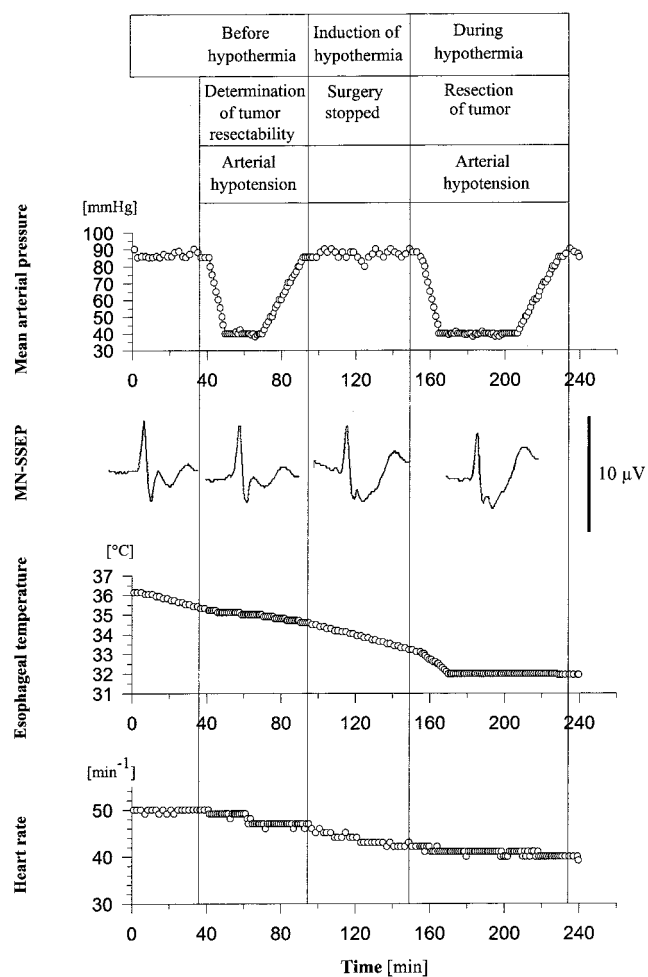
Fig. 1. Time course of mean arterial pressure, MN-SSEP amplitude, esophageal temperature, and heart rate during anesthesia and surgery in a patient undergoing resection of a choroidal melanoma delineating the two episodes of evoked arterial hypotension in their relation to body temperature. A similar time course was observed in other patients except for variation in the duration of the final stage of tumor resection. Values of variables during hypotension before and during hypothermia were compared 15 min after hypotension had been achieved. Hypotension with a mean arterial pressure of 40 mmHg lasted approximately 20 min before hypothermia and averaged approximately 60 min during hypothermia of 32°C. Heart rate continuously decreased as esophageal temperature decreased from 36.1°C to 31.9°C.

did not change during combined hypothermia and hypotension (table 1). Thus, arterial hypotension did not evoke different effects on MN-SSEP amplitude before and after hypothermia at plateau hypotension. Furthermore, the course of pressure-related MN-SSEP amplitudes was not significantly different before and during hypothermia (fig. 2A).

Arterial hypotension during hypothermia did not significantly change Sjo_2, arterial–jugular bulb oxygen content difference, or lactate concentration difference (table 1).

The SNP dose required to decrease mean arterial pressure to a plateau of 40 mmHg averaged $0.7 \pm 0.5\ \mu g \cdot$

$kg^{-1} \cdot min^{-1}$ before and $1.0 \pm 0.7\ \mu g \cdot kg^{-1} \cdot min^{-1}$ during ($P > 0.05$) induced hypothermia.

Duration of arterial hypotension during hypothermia averaged 60 ± 17 min, as required for resection of choroidal melanoma, and all patients underwent successful surgery. None of the patients showed neurologic deficits or other anesthesia-related complications.

Discussion

In this confirmatory study, we assessed and compared in humans the effects on MN-SSEP amplitude and latency of marked arterial hypotension evoked by SNP with or without marked hypothermia along with variables of global cerebral oxygen extraction. SNP-evoked hypotension to a mean arterial pressure of as low as 40 mmHg did not significantly alter MN-SSEP amplitude or latency or cerebral oxygen extraction before or during hypothermia of 32°C. Furthermore, hypothermia *per se* did not change MN-SSEP amplitude but significantly prolonged MN-SSEP latency. Thus, in individuals without evidence of cerebrovascular disease, hypothermia does not alter effects on MN-SSEP amplitude and global cerebral oxygen extraction of prolonged SNP-evoked arterial hypotension with a mean arterial pressure of 40 mmHg.

These results were observed during propofol–remifentanil anesthesia and with arterial partial pressure of carbon dioxide (Pco_2) maintained unchanged (α-stat). Remifentanil does not accumulate even during long procedures and, in dosages of $0.125–0.4\ \mu g \cdot kg^{-1} \cdot min^{-1}$, does not change MN-SSEP amplitude or latency.[26,27] Propofol has also been recommended during MN-SSEP monitoring since MN-SSEPs are affected to a lesser degree compared to inhaled anesthetics.[28] It might be speculated that the anesthetic effect of propofol and remifentanil may have increased during hypothermia by decreased clearance because of decreased hepatic blood flow and/or metabolism. However, this effect, if any, on MN-SSEP variables is likely small and would only introduce a conservative error with regard to MN-SSEP depression. In fact, MN-SSEP amplitude remained unchanged as body temperature decreased to 32°C, excluding major effects of increased anesthetic depth.

The SNP dose required to decrease mean arterial pressure to 40 mmHg was not different before and during hypothermia, excluding differential effects of SNP dose. Progressive decreases in mean arterial pressure were evoked by increasing SNP dosage, resulting in a pressure decrease of approximately 2.5 mmHg/min. This rate is considered consistent with unimpaired autoregulation within the known autoregulatory pressure range in rats subjected to graded hypotension by bleeding.[29] Thus, neither the anesthetics used nor the rate of arterial pressure changes are likely to have introduced a bias on results observed.
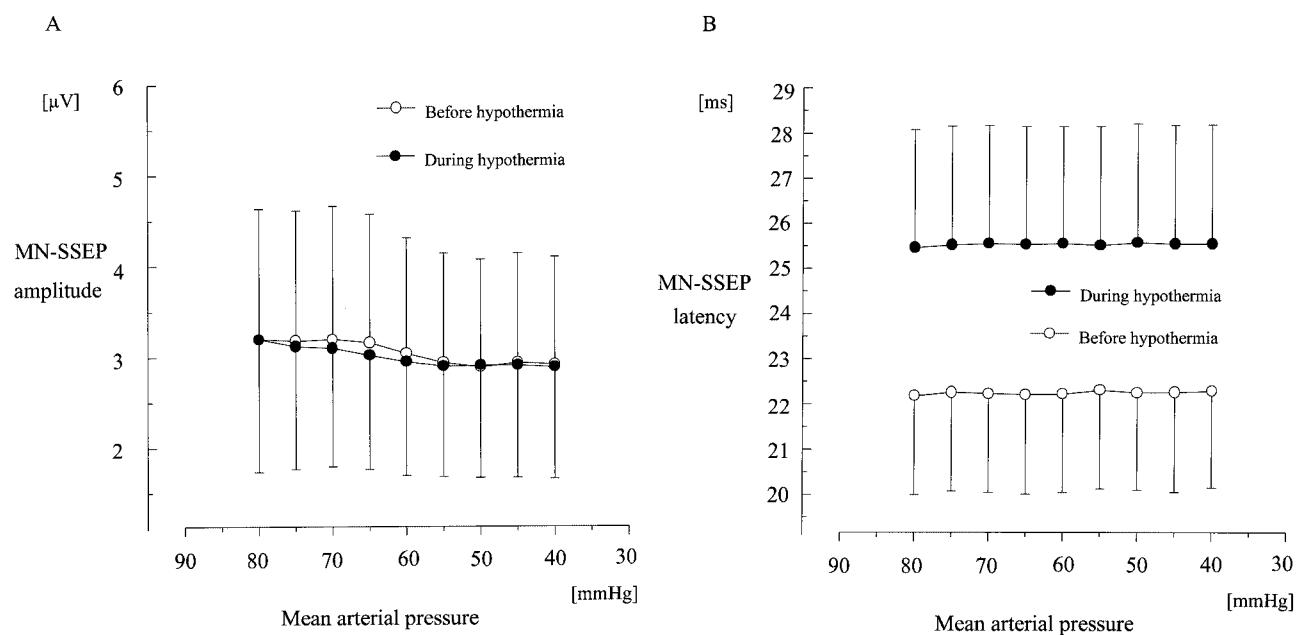
Fig. 2. MN-SSEP peak to peak amplitude of N20/P25 (*A*) and MN-SSEP latency of N20 (*B*) in relation to mean arterial pressure before (open circles) and during hypothermia (full circles). Means ± SDs from 11 patients undergoing resection of intraocular melanoma during progressive arterial hypotension with or without hypothermia of approximately 32°C.

Monitoring cortical function by MN-SSEPs during evoked hypotension or hypothermia is considered important for prevention of neural damage.[1,2,10] Unfortunately, for obvious reasons, safe limits of SNP-evoked arterial hypotension in humans and effects of marked hypotension on MN-SSEPs have not been defined. Controlled arterial hypotension during intraocular tumor resection, providing a bloodless surgical field, facilitating dissection, and possibly decreasing surgical time,[22] is believed to be essential in preventing choroidal hemorrhage and loss of the eye but obviously carries a risk of cerebral ischemia. Accordingly, this setting can provide insights since interpretation is not corroborated by the simultaneous presence of hypothermia, hypotension, or hemodilution, such as during cardiopulmonary bypass, allowing effects of hypotension to be clearly separated from those of hypothermia. Furthermore, it allows assessment of whether hypothermia interacts with SNP-evoked arterial hypotension.

In the intact brain, autoregulation maintains cerebral blood flow constant over a wide pressure range, with a lower limit in humans believed to be 60 or 50 mmHg.[30-32] During normothermia and SNP-induced hypotension with a mean arterial blood pressure of 50–55 mmHg or even less, cerebral blood flow appears to be little affected in the awake or anesthetized state.[33-39] This has been attributed to an intact cerebral autoregulation[36,40] but may or may not[17] also be explained by a direct vasodilating effect of

Table 1. Data from Patients Undergoing Resection of Intraocular Melanoma during Sodium Nitroprusside–evoked Arterial Hypotension before and during Induced Hypothermia

	Before Hypothermia		During Hypothermia	
	Plateau Arterial Hypotension		Plateau Arterial Hypotension	
	Before	During	Before	During
Esophageal temperature, °C	35.9 ± 0.2	35.4 ± 0.2	32.6 ± 0.7	31.8 ± 0.3
Mean arterial pressure, mmHg	81 ± 9.5	40 ± 5.2	86 ± 8.9	38 ± 3.0
N20/P25 amplitude, μV	3.20 ± 1.4	2.92 ± 1.2	3.20 ± 1.5	2.89 ± 1.2
N20 latency, ms	22.18 ± 2.2	22.28 ± 2.6	25.46 ± 2.1*	25.54 ± 2.7*
SjO_2, %	53.2 ± 11.4	54.2 ± 5.8	60.6 ± 2.6	57.6 ± 5.5
$Cao_2–CjO_2$, ml O_2/dl	7.4 ± 1.7	7.1 ± 1.0	6.4 ± 0.4	6.5 ± 0.2
$Lactate_a–Lactate_j$, mm	−0.03 ± 0.1	−0.01 ± 0.1	0 ± 0.1	0.01 ± 0.1
$Lactate_j$, mm	0.83 ± 0.2	0.84 ± 0.1	0.75 ± 0.1	0.78 ± 0.1
$Paco_2$, mmHg	36.4 ± 3.0	37.8 ± 1.0	37.8 ± 1.0	37.7 ± 1.0
$Hemoglobin_a$, g/dl	11.7 ± 1.1	11.6 ± 1.4	11.9 ± 1.1	11.7 ± 1.2

Data represent values of variables (means ± SD) before and after 15 min of marked arterial hypotension evoked by sodium nitroprusside before and during induced hypothermia. Subscript a indicates values measured in arterial blood; subscript j indicates values measured in jugular bulb blood.

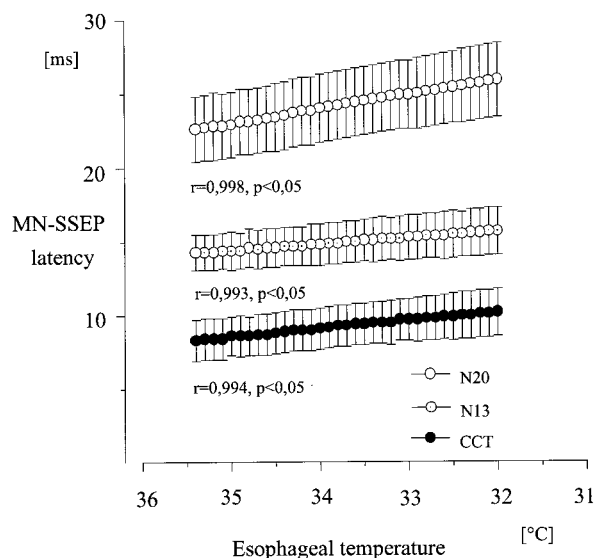* $P < 0.05$ *vs.* values before hypothermia

Fig. 3. MN-SSEP latencies of cortical N20 and cervical N13 at arterial normotension in relation to esophageal temperature. Central conduction time (CCT), *i.e.*, the difference between N13 and N20, is also presented. Means ± SDs from 11 patients undergoing resection of intraocular melanoma during hypothermia of approximately 32°C.

SNP. However, others have reported a disturbed autoregulation with SNP[13-15,41-43] and longer hypotension.[13] In our study, SNP-evoked hypotension with a mean pressure to 40 mmHg, *i.e.*, 10–20 mmHg below the presumed limit of autoregulation, failed to decrease MN-SSEP amplitude and was not associated with a change in global cerebral oxygen extraction. Absence of critical cortical ischemia despite marked SNP-evoked hypotension is supported by unchanged Sjo_2 and unchanged global cerebral oxygen content difference.

Hypothermia alone during propofol–remifentanil anesthesia did not significantly affect MN-SSEP amplitude but linearly prolonged cortical latency, cervical latency, and central conduction time, with prolongation of the N20 component by 1 ms/°C. This is consistent with decreased axonal conduction and delayed synaptic transmission[44] and close to values of 1–1.6 ms/°C reported by others.[45-47] In our study, however, unlike studies using cardiopulmonary bypass, arterial pressure and body temperature were assessed independently, and arterial oxygen content did not change.

The effect of body temperature on MN-SSEP amplitude is not well defined. Larger temperature decreases to 25.8°C[7] or 27.8°C[46] using cardiopulmonary bypass in humans decreased MN-SSEP amplitude by 11–19%. In our study, N20/P25 peak-to-peak amplitude remained unchanged, indicating in humans during remifentanil–propofol anesthesia that surface hypothermia with a mean core temperature of 31.8°C alone does not alter MN-SSEP amplitude but only delays MN-SSEP latency.

Few studies, all performed in cats or rats, have addressed the impact of hypothermia on cerebrovascular autoregulation without using cardiopulmonary bypass,[18-20] and whether hypothermia interacts with effects of SNP evoked hypotension is unknown. Surface hypothermia (30.5–32°C) blunts the vasodilatory response of pial arterioles to hemorrhagic hypotension, suggesting impaired cerebral autoregulation in anesthetized cats and rats.[18-20] Finally, hypothermia may decrease vascular nitric oxide release, resulting in less vasodilatation during hypotension.[48] Although these data suggest that cerebral blood flow autoregulation or SNP response may be impaired by hypothermia, our data indicate in humans that even hypothermia of 32°C alters neither the MN-SSEP response nor global cerebral oxygen extraction during marked SNP-evoked arterial hypotension.

In conclusion, during remifentanil–propofol anesthesia in individuals without evidence of cerebrovascular disease, marked SNP-evoked arterial hypotension with a mean arterial pressure of 40 mmHg does not depress either MN-SSEP amplitude nor prolong MN-SSEP latency and is associated with unchanged global cerebral oxygen extraction and lactate production. Furthermore, surface hypothermia with a core temperature of 32°C alone does not depress MN-SSEP amplitude but prolongs MN-SSEP latencies. Finally, there is no evidence that hypothermia alters the effects of SNP-evoked marked arterial hypotension.

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