Local Brain Temperature Reduction Through Intranasal Cooling With the RhinoChill Device
Preliminary Safety Data in Brain-Injured Patients

Alex Abou-Chebl, MD; Gene Sung, MD; Denise Barbut, MD, MRCP; Michel Torbey, MD

**Background and Purpose**—Hypothermia is neuroprotectant but currently available cooling methods are laborious, invasive, and require whole-body cooling. There is a need for less invasive cooling of the brain. This study was conducted to assess the safety and efficacy of temperature reduction of the RhinoChill transnasal cooling device.

**Methods**—We conducted a prospective single-arm safety and feasibility study of intubated patients for whom temperature reduction was indicated. After rhinoscopy, the device was activated for 1 hour. Brain, tympanic, and core temperatures along with vital signs and laboratory studies were recorded. All general and device-related adverse events were collected for the entire hypothermia treatment.

**Results**—A total of 15 patients (mean age, 50.3 ± 17.1 years) were enrolled. Brain injury was caused by intracerebral hemorrhage, trauma, and ischemic stroke in equal numbers. Hypothermia was induced for fever control in 9 patients and for neuroprotection/intracranial pressure control in 6. Core temperature, brain temperature, and tympanic temperature were reduced an average of 1.1 ± 0.6°C (range, 0.3 to 2.1°C), 1.4 ± 0.4°C (range, 0.8 to 5.1°C), and 2.2 ± 2°C (range, 0.5 to 6.5°C), respectively. Only 2 patients did not achieve the goal of ≥1°C decrease in temperature. Brain temperature, tympanic temperature, and core temperature reductions were similar between the afebrile and febrile patients. There were no unanticipated adverse events and only 1 anticipated adverse event: hypertension in 1 subject that led to discontinuation of cooling after 30 minutes. There were no nasal complications.

**Conclusions**—Intranasal cooling with the RhinoChill device appears safe and effectively lowers brain and core temperatures. Further study is warranted to assess the efficacy of hypothermia through intranasal cooling for brain-injured patients. *(Stroke. 2011;42:00-00.)*

**Key Words:** acute brain injury ■ administration ■ intranasal ■ hypothermia ■ induced ■ neuroprotectants

**Therapeutic hypothermia** is well established as an effective neuroprotectant after both focal and global cerebral ischemia.¹⁻³ There are 2 traditional methods for cooling. The first, external cooling, is effective at temperature reduction but can be laborious and is associated with delayed brain cooling because the periphery and core have to cool first.⁴ The second is internal cooling such as by the use of cold intravascular infusions or indwelling catheters. This method can be very effective and more rapid than surface cooling but is invasive and still requires the cooling of the core before brain temperature reduction can be achieved.⁵ To hasten cooling of the brain, and to avoid the complications associated with total body cooling (eg, ventricular fibrillation, pneumonia, sepsis), numerous investigators have attempted to develop means to cool the brain preferentially at the same time as maintaining the rest of the body normothermic.⁶ Many of these methods require both arterial and venous catheters in the systemic and cerebral circulations and are impractical to use in an emergency or intensive care unit setting.⁷⁻⁸

The RhinoChill Device (RCD; BeneChill Inc, San Diego, CA), which has 2 nasal catheters that spray a proprietary perfluorocarbon–oxygen mixture into the nasopharynx for cooling, has potential advantages over the previously described methods. It takes advantage of the nasal pathways (ie, the conchal folds and turbinates) that provide a highly vascular and large, diffuse surface area that is in close proximity to the cerebral circulation. Cooling in the nasopharynx offers the ability to cool the brain through both direct conductive mechanisms and indirect hematogenous mechanisms preferentially cooling the brain before the body, potentially resulting in faster neuroprotection.⁹⁻¹⁰ Also, RCD catheter placement is minimally invasive, does not require specialized skills to insert or to use, and the entire apparatus...
is small, portable, and battery-operated requiring only medical-grade oxygen.

This study was conducted to test RCD safety and effectiveness of temperature reduction.

Methods

We conducted a prospective, multisite, single-arm study in neurological intensive care units with standardized procedures for implementing therapeutic hypothermia. The 1° end point was to demonstrate a brain (Tb) or tympanic (Tt) temperature reduction ≥1°C after 1 hour induction with the RCD. Secondary end points included time to reduce brain temperature (as measured by either Tb or Tt) 2°C from baseline and extent of core temperature (Tc) reduction during induction. The study received Institutional Review Board approval at all participating sites. Patients in the neurological intensive care unit for whom cooling was ordered (for any reason) and who had experienced an ischemic stroke, intracerebral hemorrhage, head trauma, postoperative aneurysm surgery, or cardiac arrest were enrolled. Patients had to be ≥18 years old and had to be intubated and sedated. Patients were excluded if they had any of the following: skull base fracture, findings on rhinoscopy that precluded placement of the nasal catheters, required >50% FiO2 to maintain SaO2 >98%, hemodynamic instability (ie, systolic blood pressure <90 mm Hg or heart rate <50 beats/min during 3 serial measurements during the 30-minute period before initiating hypothermia), active sepsis not treated with appropriate antibiotics, active coagulopathy or international normalized ratio >3.0×control or partial thromboplastin time >50 seconds, history of contraindications to hypothermia (eg, cryoglobulinemia, sickle cell disease, serum cold agglutinins, or vasospastic disorders [eg, Raynaud, thromboangiitis obliterans]), already hypothermic (Tb, Tt <35°C), pregnant, or participating in another clinical study.

Coolant-Device Description

The RCD works by spraying the evaporative perfluorocarbon coolant (PFH) onto the upper surface of the nasal cavity, where it evaporates absorbing heat from the tissue in the head through both direct conductive mechanisms and indirect hematogenous mechanisms. PFH is an inert liquid that is immiscible in water and is not absorbed in any significant quantity into the body. PFH has a surface tension that is lower than water and will quickly spread uniformly throughout the space in which it is sprayed. Oxygen is delivered with the liquid PFH to maximize evaporation. Local temperatures within the nasal cavity are expected to cool to approximately 2°C. PFH/oxygen escape the nasal cavity through the nostrils or the mouth. In the event that all the PFH is not evaporated, it is possible that it will either trickle out of the nostrils or down the pharynx into the mouth or stomach. The minute quantities of PFH that may be absorbed into the blood or inhaled into the lungs are quickly expired through the lungs.

The RCD consists of 3 components: tubing, control unit, and coolant bottle. The tubing attaches to the control unit to which a hospital oxygen source is connected. PFH is driven out of the bottle by the pressurized oxygen, through a filter and into the nasal catheters, which are similar in size to epistaxis catheters and enable venting through the nostrils. The catheters have rounded atrumatic tips, conform to the nasal anatomy, and have spray ports along their dorsal surface. The system has an overpressure relief valve to prevent excessive catheter and intranasal pressures.

Cooling Protocol

Recumbant subjects were prepared by placement of an orogastric tube with continuous suction to prevent excess condensed PFH liquid from being aspirated or ingested. Their mouths were blocked open to provide venting of the vapor. The individual intranasal catheters were then gently advanced through each nostril so that the distal end was well within the nasal cavity. The inflation pressure of the endotracheal tube cuff was adjusted to 25±1 mm Hg and 100% FiO2 was administered (it was returned to standard settings 30 minutes after the final use of the RCD). The RCD was turned on and oxygen flow was gradually increased to the maximum flow rate (80 L/min) but was decreased if the coolant was noted to be spraying out of the nose. The device was used for 1 hour to initiate temperature reduction after which standard cooling methods were administered according to local procedures. No other cooling method could be used during the 1-hour initiation period. At the end of the 1 hour, the RCD was deactivated, but the intranasal catheters were kept in place at the same time as transitioning to the standard hypothermia protocol; they could be used as needed to prevent rewarming during this transition period. Intermittent cooling with the RCD was targeted at maintaining the Tb or Tt within 0.5°C of the lowest temperature attained during the initial 1-hour cooling period until the Tb equilibrated with Tb or Tt, Tc was measured directly through Camino Micro Intraparenchymal probes (Camino Laboratories, San Diego, CA), when available per institutional protocols. Tb was recorded through bladder, rectal, esophageal, or pulmonary artery probes (per local protocols). Once Tb equilibrated with Tb or Tt, the intranasal catheters were removed.

The institutional-specific cooling procedures, target temperature and hypothermia duration, reversal algorithm, sedation and anti-shivering regimens, and all concomitant medications were recorded through the 24 hours after the termination of hypothermia.

Safety Monitoring

Vital signs, Tb, Tt, Tc, SaO2, intracranial pressure, and pBtO2 (if available) were recorded at baseline and at 15-minute intervals during RCD use. During the transition, these data were recorded every 30 minutes. At the end of the 1-hour RCD induction, rhinoscopy, neurological examination, chest x-ray, complete blood count, basic metabolic panel, prothrombin time, partial thromboplastin time, and cardiac enzymes were obtained.

During standard therapeutic hypothermia treatment, in addition to usual institutional procedures for patient monitoring, the following were performed for the first 24 hours after the initiation of cooling: continuous electrocardiogram, oximetry, blood pressure, Tb, Tt, Tc, and, if applicable, intracranial pressure and/or pBtO2 monitoring. At 24 hours, rhinoscopy and neurological examination were repeated.

Patients were followed for 24 hours after completion of hypothermia treatment at which point vital signs, Tb, Tt, Tc, neurological examination, rhinoscopy, and chest x-ray were obtained. If the subject was able, a Brief Smell Identification Test was performed within 1 hour after regaining consciousness.

All adverse events (AEs) were recorded and categorized as mild, moderate, or severe. The relationship of AEs to the device were categorized as “not,” “probably not,” “probably,” or “definitely” related to the device. Final rhinoscopy and Brief Smell Identification Test (if able) were performed at discharge or 2 weeks after the RCD was used, whichever occurred first.

Statistical Analysis

In summary, descriptive statistics were calculated for all study end points, safety, demographic, and baseline variables. Means, SDs, and ranges were used to describe continuous measurements. Counts and percentages were used to describe categorical parameters. The relationship between body mass index and cooling was analyzed using analysis of covariance models with backward parameter selection, first with P<0.10 as the selection criterion and change in Tc as the outcome variable.

Results

Fifteen patients (mean age, 50.3±17 years; range, 21 to 88 years) were enrolled. All had severe neurological injury with baseline National Institutes of Health Stroke Scale of 26.7±6.7 (range, 15 to 38; median, 28). The indications for hypothermia were fever control in 9 patients and neuroprotection/intracranial pressure control in 6. Table 1 summarizes the patient characteristics and cooling results. All but 1 patient were cooled for at least 60 minutes with the RCD. Tb
(N=15), \(T_b\) (N=11), and \(T_t\) (N=10) were reduced during induction an average of 1.1±0.6°C (range, 0.3 to 2.1°C), 1.4±0.4°C (range, 0.8 to 5.1°C), and 2.2±2°C (range, 0.5 to 6.5°C), respectively (Figure 1). The \(T_t\) readings from 2 patients (Patients 1–1 and 3–5) did not correlate with \(T_c\) and were likely anomalous and if these are excluded then the mean change in \(T_t\) was 1.4±0.6°C.

All patients had temperature reduction ≥0.2°C within 15 minutes: a mean drop of \(T_b\) 0.53±0.24°C, \(T_c\) 0.43±0.35°C, and \(T_t\) 0.65±0.39°C (if 2 outliers were excluded). The median temperature drop at 15 minutes was 0.50°C, 0.30°C, and 0.55°C (outliers removed), respectively. There was no significant lag time between drop in \(T_t\) and \(T_b\) and \(T_c\) with 13 of 15 patients having ≥0.2°C drop of \(T_t\) within 15 minutes and all had a \(T_t\) drop by 30 minutes. One third (5 of 15) had a temperature drop of ≥0.6°C within 30 minutes. There was no difference in the extent of \(T_c\) (1.1±0.6°C versus 1.1±0.5°C, \(P=0.93\)) or \(T_b\) (1.58±0.5°C versus 1.24±0.37°C, \(P=0.08\)) drop between the afebrile and febrile patients, respectively.

Body mass index was seen to be a significant predictor (\(P=0.042\)) of cooling rate (Figure 2): the reduction in \(T_c\) was 0.037°C less per unit of body mass index. Analogous models for \(T_b\) and \(T_t\) showed no significant association with body mass index (\(P=0.86\) and \(P=0.61\), respectively).

The RCD was well tolerated with no major changes to laboratory values other than a mean drop of 0.45 mEq/L of potassium (range, 0.2 to 0.7 mEq/L) in 4 patients, 3 of whom had baseline values ≥3.6 mEq/L. There were no significant nasal complications with only transient minor erythema or nasal discharge seen (Table 2). There was only 1 device-related AE, transient hypertension that resolved with device removal in the only patient not cooled for 60 minutes. Neither baseline nor follow-up smell testing could be performed on any of the patients because 6 were dead at 2-week follow-up and the remainder were comatose or in another minimally conscious state.

**Discussion**

Hypothermia is a highly effective neuroprotectant and hyperthermia is associated with worsening of neurological outcomes.\(^1\) Hypothermia has been studied in the setting of focal cerebral ischemia both experimentally and clinically but has been found clinically efficacious only in the setting of global cerebral ischemia.\(^2,5,11\) Two prospective trials (one randomized) of out-of-hospital cardiac arrest have shown clear clinical efficacy in survival and improved neurological outcomes in patients cooled within hours of the arrest.\(^2,5\) Rapid and early brain cooling is essential for maximizing the neuroprotective effect of hypothermia and applying the hy-

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**Table 1. Patient Characteristics and Individual RhinoChill Cooling Results**

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Sex</th>
<th>Indication</th>
<th>Febrile</th>
<th>(\Delta T_b, ^\circ\text{C})</th>
<th>(\Delta T_c, ^\circ\text{C})</th>
<th>(\Delta T_t, ^\circ\text{C})</th>
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<tr>
<td>1-1</td>
<td>M</td>
<td>ICH</td>
<td>No</td>
<td>2.2</td>
<td>2.1†</td>
<td>6.5</td>
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<tr>
<td>1-2</td>
<td>F</td>
<td>ICH</td>
<td>Yes</td>
<td>1.7</td>
<td>1.8‡</td>
<td>1.9</td>
</tr>
<tr>
<td>1-3</td>
<td>F</td>
<td>ICH/trauma</td>
<td>No</td>
<td>1.7</td>
<td>1.2</td>
<td>NA</td>
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<tr>
<td>2-1</td>
<td>M</td>
<td>IS</td>
<td>No</td>
<td>1.4</td>
<td>1.3</td>
<td>NA</td>
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<tr>
<td>2-2</td>
<td>M</td>
<td>Trauma</td>
<td>Yes</td>
<td>1.5</td>
<td>0.7</td>
<td>NA</td>
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<tr>
<td>2-3</td>
<td>M</td>
<td>ICH/neurogenic fever</td>
<td>Yes</td>
<td>1.2</td>
<td>0.7</td>
<td>NA</td>
</tr>
<tr>
<td>2-4</td>
<td>M</td>
<td>ICH/trauma</td>
<td>Yes</td>
<td>1</td>
<td>1.2‡</td>
<td>NA</td>
</tr>
<tr>
<td>3-1</td>
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<td>ICH</td>
<td>Yes</td>
<td>1.6</td>
<td>1.3</td>
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<td>3-2</td>
<td>F</td>
<td>ICH/trauma</td>
<td>Yes</td>
<td>0.9</td>
<td>0.5†</td>
<td>1.3</td>
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<tr>
<td>3-3</td>
<td>M</td>
<td>IS/trauma/neurogenic fever</td>
<td>Yes</td>
<td>1</td>
<td>0.8</td>
<td>0.6</td>
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<tr>
<td>3-4</td>
<td>F</td>
<td>IS</td>
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<td>NA</td>
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<td>0.5</td>
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<tr>
<td>3-5</td>
<td>F</td>
<td>IS</td>
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<td>NA</td>
<td>1.9§</td>
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<td>3-6</td>
<td>F</td>
<td>IS</td>
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<td>NA</td>
<td>0.4</td>
<td>0.8</td>
</tr>
<tr>
<td>3-7</td>
<td>F</td>
<td>ICH</td>
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<td>1.5</td>
<td>1</td>
<td>1.5</td>
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<tr>
<td>3-8</td>
<td>F</td>
<td>IS</td>
<td>No</td>
<td>NA</td>
<td>1.5</td>
<td>2</td>
</tr>
</tbody>
</table>

Mean (SD) 1.4 (0.4) 1.1 (0.6) 2.2 (2.0)

\(T_b\) indicates brain temperature; \(T_c\), core temperature; \(T_t\), tympanic temperature; M, male; F, female; ICH, intracerebral hemorrhage; IS, ischemic stroke; NA, not available.

*Rectal temperature except: †bladder, ‡pulmonary artery, §esophageal.

Patient cooled for only 30 min.

**Figure 1.** Mean temperature reductions during the 1-hour RhinoChill induction. ICT indicates intracranial temperature.
pothermia earlier in the field may improve outcomes further.\textsuperscript{12} Surface cooling techniques are inefficient, laborious, and slow with cooling rates of 0.25 to 1.6\degree C/h but can be mobile.\textsuperscript{4,13} Endovascular cooling can be more rapid in achieving hypothermia (approximately 3\degree C/h) but is invasive, requires large (and expensive) proprietary cooling apparatuses, and is not mobile.\textsuperscript{5} Furthermore, both of these approaches require cooling of the core before cooling of the brain can begin. There is therefore a need for a rapid, safe, mobile method of cooling the brain. The RCD may be 1 such method. This technique has been used in sheep and porcine models of hypothermia and was more rapid than surface cooling at achieving brain cooling in the former and more rapid than intravenous cold saline infusion in the latter.\textsuperscript{14,15} In the sheep study, surface cooling was unable to achieve the target $T_b$ within 2 hours but with nasal cooling therapeutic $T_b$ was achieved within 15 minutes.\textsuperscript{15} A limitation of these sheep and porcine experiments is they do not perfectly model human physiology and anatomy due to the presence of a rete and a larger nose-to-brain volume; therefore, human testing is essential in conclusively proving cooling efficacy.

In this current safety study, intranasal cooling with the RCD was both safe and feasible. There were no major complications attributable to the device other than hyperten-

\begin{figure}[h]
\centering
\includegraphics[width=\columnwidth]{figure2.png}
\caption{Core temperature reduction was inversely proportional to body mass index (BMI).}
\end{figure}

\begin{table}[h]
\centering
\caption{Rhinoscopy Findings}
\begin{tabular}{|c|c|c|c|c|}
\hline
Patient No. & Baseline & 1 H After RhinoChill & 24 H After RhinoChill & 24 H Postcooling \\
\hline
1-1 & Normal & Erythema, minimal & Normal & Normal \\
1-2 & Normal & Abrasion & & \\
1-3 & Normal & Mucus discharge & Minimal bloody discharge, left nare & \\
2-1 & Not performed & Normal & Not available & Not available \\
2-2 & Normal & Normal & Normal & Not available \\
2-3 & Minor abrasion, left nare & Normal & Normal & Normal \\
2-4 & Minor abrasion, left concha & No change & Normal & Normal \\
3-1 & Mild erythema, small hemorrhage, left nare & Erythema, minimal & Mild to moderate white discharge bilaterally & Not available \\
3-2 & Erythema bilaterally, clear-white discharge, ulcer on columella & Unchanged with erythema right>left, ulcer on columella & Erythema bilaterally & Mild improvement \\
3-3 & Normal & Erythema, medial left nare & Normal & No change \\
3-4 & Erythema, minimal to moderate bilaterally, minimal blood left nare & No change & No change & Erythema, minimal, right nare \\
3-5 & Mild clear discharge bilaterally & Normal mucosa bilaterally & Normal mucosa bilaterally; slight clear white discharge & Not applicable \\
3-6 & Bloody yellow secretions bilaterally & Erythema, mild, bilaterally & Erythema, mild, bilaterally & Erythema, mild \\
3-7 & Erythema, mild, upper left turbinate and right nare & Erythema, mild, bilaterally with clear drainage & Small white plaque, left nare, mild erythema bilaterally & Erythema, minimal, bilaterally \\
3-8 & Erythema, mild, turbinates bilaterally & Clear drainage bilaterally, slight increased erythema left nare, mild erythema right nare & Erythema, moderate, right turbinate & Erythema, mild, right nare \\
\hline
\end{tabular}
\end{table}
sion in 1 patient and transient local nasal mucosal irritation. It is unknown if prolonged use of the device may cause more extensive or permanent injury, which requires further testing. However, because the local nasal temperature should not be <2°C, use of the device should be akin to winter exposure to similar ambient air temperatures, which is not expected to cause major tissue injury. The single case of hypertension was likely attributable to patient discomfort and inadequate sedation and did not lead to any sequelae. The poor neurological outcomes in this study were not related to the use of the RCD but to the underlying critical injuries in all patients. Similarly, cooling with the RCD was found to be safe in 96 of 200 patients with cardiac arrest randomized to treatment in a safety and feasibility study with 18 (21.7%) device-related AEs. The majority of the AEs were mild: 13 nasal disolorations, 3 epistaxis, and 1 perioral bleed. The only nonminor AE was 1 case of periorbital emphysema. All of the patients who survived and could undergo smell testing had no abnormalities.

Importantly, the RCD was effective at quickly lowering Tₛ and Tₑ; within 15 minutes of activation, there was a universal, measurable reduction of Tₛ which in this study was also associated with Tₑ reduction. Previous studies in sheep, pigs, and both previous human studies using RCD all showed preferential brain over body cooling. The differential between brain and body at the end of the 1-hour protocol was 0.9°C in the postresuscitation cardiac arrest study and 0.6°C in the intra-arrest study. There are several reasons why the patient population in this study did not show significant temperature differential at the end of 1-hour induction. First, mean baseline temperature in this group of neurological patients was 38.1°C (range, 36.1 to 40.5°C) as compared with baselines of 35.8°C in the emergency department study and 35.5°C in the intra-arrest study. Many patients in our study were febrile and were cooled using RCD because they had not responded to antipyretic medications. Furthermore, the patients in this study, unlike those in the cardiac arrest studies, had normal or increased circulation, which would facilitate heat exchange from body to brain, minimizing the temperature differential. Still all patients had temperature reduction despite the high numbers who were also febrile. Like with most cooling techniques, cooling efficiency was affected by body mass; however, brain cooling was not. Although larger studies are needed to confirm this finding, it does suggest that the RCD may be a good option for the rapid, noninvasive initiation of brain cooling, even in obese individuals.

The RCD was easy to use with minimal training; virtually anyone capable of inserting a nasogastric tube can use it because there is no imaging required other than rhinoscopy. A limitation to this would be patients with head and facial trauma who may have nasal, orbital, and skull base fractures in whom device insertion would require radiographic imaging. The RCD is highly portable, only requiring high-flow oxygen for use. The control unit is a low-current device and runs on off-the-shelf consumer batteries. The PFH liquid is nonflammable and can be stored at room temperature without any special precautions and can be stored in multiple hospital areas and in ambulances.

This study also showed that the RCD can be safely used as an adjunct to induce hypothermia with either surface or endovascular cooling to achieve quicker induction of brain cooling. It also offers a great deal of flexibility; for example, it may be used during rewarming to avoid rapid increase in Tₑ and possible rebound cerebral edema and intracranial pressure elevation. Theoretically, it may also be used to selectively maintain brain cooling at the same time as simultaneously warming the core if systemic hypothermia is undesirable (this requires validation).

This study and RCD have limitations. This was a safety and feasibility study in a small number of patients; therefore, all possible complications, especially if rare, could not be identified and further study is required. Second, the effect of intranasal cooling on the sense of smell could not be adequately assessed because most of the patients were comatose before initiation of therapy and none were awake at follow-up, requiring further testing; however, given that there was no significant mucosal or deep tissue injury, it is unlikely that there would have been olfactory nerve injury. This study did not assess the ability and safety of the device to maintain hypothermia or to control rewarming. Limitations of this technique in the neurological population include the requirement for sedation and for airway protection. Although it may not be the ideal device for cooling induction in the awake patient who would not otherwise be sedated in the field, it may be extremely useful in comatose patients with head injury during transport, those with less severe injury but still requiring an airway, and also in patients with large strokes requiring an airway in an emergency department setting. Also, because not all patients had intracranial temperature measurements, the rate of brain cooling reported may not be accurate because there is a difference in the correlation between Tₛ and tympanic, rectal, bladder, and esophageal temperatures, with the latter appearing to correlate best with Tₑ; the exact locations of brain probe placement were not recorded and variability in probe depth could explain some of the variability. Future studies should include uniform temperature recording sites, preferably brain and esophageal temperatures. Lastly, the effects of 100% O₂ have not been studied in this population and the safety of this should be validated; however, it is important to note that 100% O₂ is not required for device use but was designed into the protocol as a precautionary measure.

In conclusion, the RCD can initiate hypothermia and effectively and quickly lower Tₛ and Tₑ. The RCD appears to cool the brain more rapidly than the core, which may be desirable in brain-injured patients, and it may be safely used as an adjunct in hypothermia initiation. The device is simple to use and was not associated with any major complications. More study is needed on the safety and efficacy of the device in maintaining hypothermia and in controlling rewarming because it has potentially broad applications in patients with brain injury, particularly after cardiac arrest.

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References
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