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Neurological Institute Journal

Inside:

- **Keeping It Cool: Temperature Regulation in the Neuro Intensive Care Unit**
- **Managing Skull Base Chondrosarcoma**
- **Transforming the Way We View the Brain and Its Blood Supply**
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FROM THE EDITOR



Dear Colleague,

I am pleased to bring you the Winter 2009 issue of the Neurological Institute Journal.

Through continuing collaboration with scientists at Case Western Reserve University School of Medicine, physicians at the University Hospitals Neurological Institute test and refine the latest advances in treatment

for patients with disabling neurological disorders. The NI Journal highlights these advances and demonstrates our interdisciplinary strengths. As an added benefit for our readers, CME credit is readily available in each issue for the busy practitioner interested in receiving *AMA PRA Category 1 Credits*TM.

Our winter issue features articles written by physicians, nurses, fellows, residents and researchers. In this issue, Alan Hoffer, MD, and colleagues reflect on the uses of therapeutic hypothermia for patients with neurological injuries, particularly as it applies to patients suffering from cerebral vasospasm following subarachnoid hemorrhage. As physicians and scientists gain a better understanding of how to avoid its complications, temperature regulation in the neuro-intensive care unit re-emerges as a promising therapy.

Jeffery Sunshine, MD, and colleagues present an overview of the recent advances made by magnetic resonance research staff at UH. This team of technicians and clinicians describe several advanced neuroradiological imaging techniques and their application for the care of neurological patients.

Alia Hdeib, MD, and colleagues explore the management of skull base chondrosarcomas, rare lesions that can be difficult to treat. The authors follow a neurosurgical patient at UH and examine the goals of treatment for these recurrent tumors.

Finally, Lori Mertz, CNP, shows us the advantages of intermediate care. The Neurological Intermediate Care Unit at UH offers an alternative to the intensive care unit for patients with less critical needs but still requiring significant care.

We thank these authors for contributing their time to this issue of the NI Journal and hope our readers find the articles insightful and smart. Your comments and inquiries are welcome.



Nicholas C. Bambakidis, MD
Editor-in-Chief
216-844-8758
Nicholas.Bambakidis@UHhospitals.org



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On the cover: The effects of vasospasm on surrounding cerebral blood vessels following aneurysmal subarachnoid hemorrhage, one of the neurologic conditions that may benefit from therapeutic hypothermia. Read more about this case in the article by Hoffer and colleagues on page 2. (Illustration by Ravin Art & Design.)



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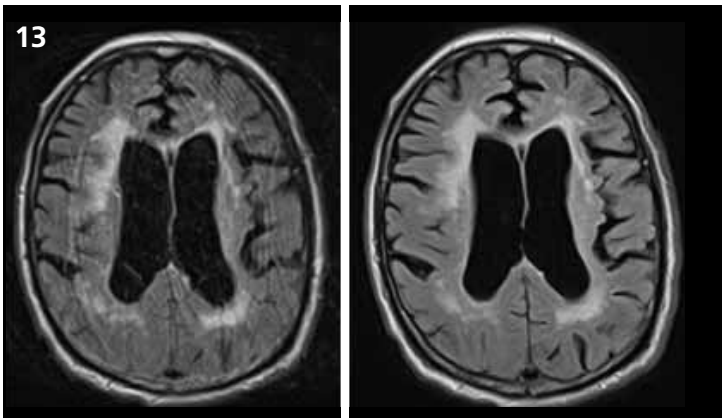
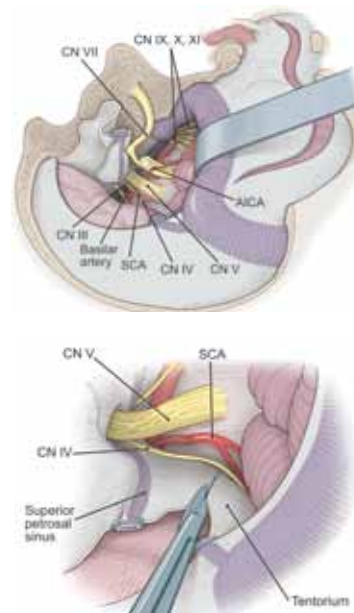
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University Hospitals Neurological Institute is Northeast Ohio's first designated institute for the comprehensive care of patients with diseases affecting the nervous system. The institute comprises 15 Centers of Excellence, which bring together some of the country's foremost experts in neurology, neurosurgery, neuroradiology, neuro-oncology, neuro-ophthalmology, neurotology, neuropathology, neuropsychology, neuropsychiatry and related specialties.

The Neurological Institute offers an interdisciplinary approach to highly individualized therapies and offers leading-edge care, including stereotactic radiosurgery, endovascular stroke and aneurysm treatments, neurostimulation and artificial disc replacement.

Keeping It Cool: Temperature Regulation in the Neuro Intensive Care Unit

By
Alan Hoffer, MD
Christopher Horn, MD
Michael DeGeorgia, MD

A Novel Therapy?

In recent years, a great deal of attention has returned to temperature regulation in patients with neurological diseases, reviving a topic that has been dormant for almost 40 years. In the 1950s and '60s, the first clinical studies on therapeutic hypothermia for neurological injuries were performed. Early attempts at therapeutic hypothermia were thwarted by imprecise cooling techniques and lack of understanding of the physiologic consequences of cooling. Cooling often took days, and the methods used resulted in imprecise and inconsistent therapy. Meanwhile, in an era before modern intensive care units, the many physiologic sequelae of hypothermia could not be monitored or corrected. Despite the potential benefits of this therapy, it was not possible to demonstrate clear improvements after neurological insults.¹⁻³

A New Understanding

Much has changed since then. With an improved understanding of temperature-dependant cellular and molecular processes, better and more precise methods of cooling, and intensive care unit (ICU) measures that can prevent and treat the complications of temperature therapy, therapeutic hypothermia is again emerging as a treatment option for anoxic, ischemic, and traumatic brain injuries.

The molecular revolution in the second half of the 20th century and the first decade of the 21st century has given us a new understanding of the mechanisms of injury on a cellular level. This is particularly true for the central nervous system. It has long been known that virtually all the chemical reactions in the body are temperature-sensitive. It was not until recently, however, that the processes of damage and repair of neurons were elucidated. The early theories on the mechanism by which hypothermia attenuated neurological injury focused primarily on the decrease in metabolic rate of the cooled tissues with the central concept being that improving the supply-demand ratio of the tissues was sufficient to diminish the injury. Indeed, the cellular metabolic rate drops 6-10% per degree Celsius during cooling.^{4,5} It has become apparent, however, that other processes that actively result in neuronal death are also affected by temperature. A number of pathways contribute to the ongoing injury of the brain after an insult (Figure 1). In many instances, these pathways overlap, regardless of the etiology.⁶ Activation of inflammatory and pro-apoptotic cascades can exacerbate tissue damage far beyond that of the primary injury. A neurological insult results in cytokine release, opening of the blood brain barrier, and an influx of white blood cells that further propagate the stimulus for inflammation.⁷⁻⁹ Meanwhile, within neurons, mitochondrial dysfunction, energy depletion, failure of ion homeostasis, uncontrolled release of excitatory amino acids, and activation of pro-apoptotic factors, such as caspases, push the cell toward its demise.¹⁰⁻¹² It is clear that temperature accelerates these processes and has a significant effect on patients' clinical status and outcome. Multiple studies have shown that fever at presentation and during hospitalization correlates well as an independent risk factor with poor neurological outcome after anoxic, ischemic, and traumatic brain injuries.

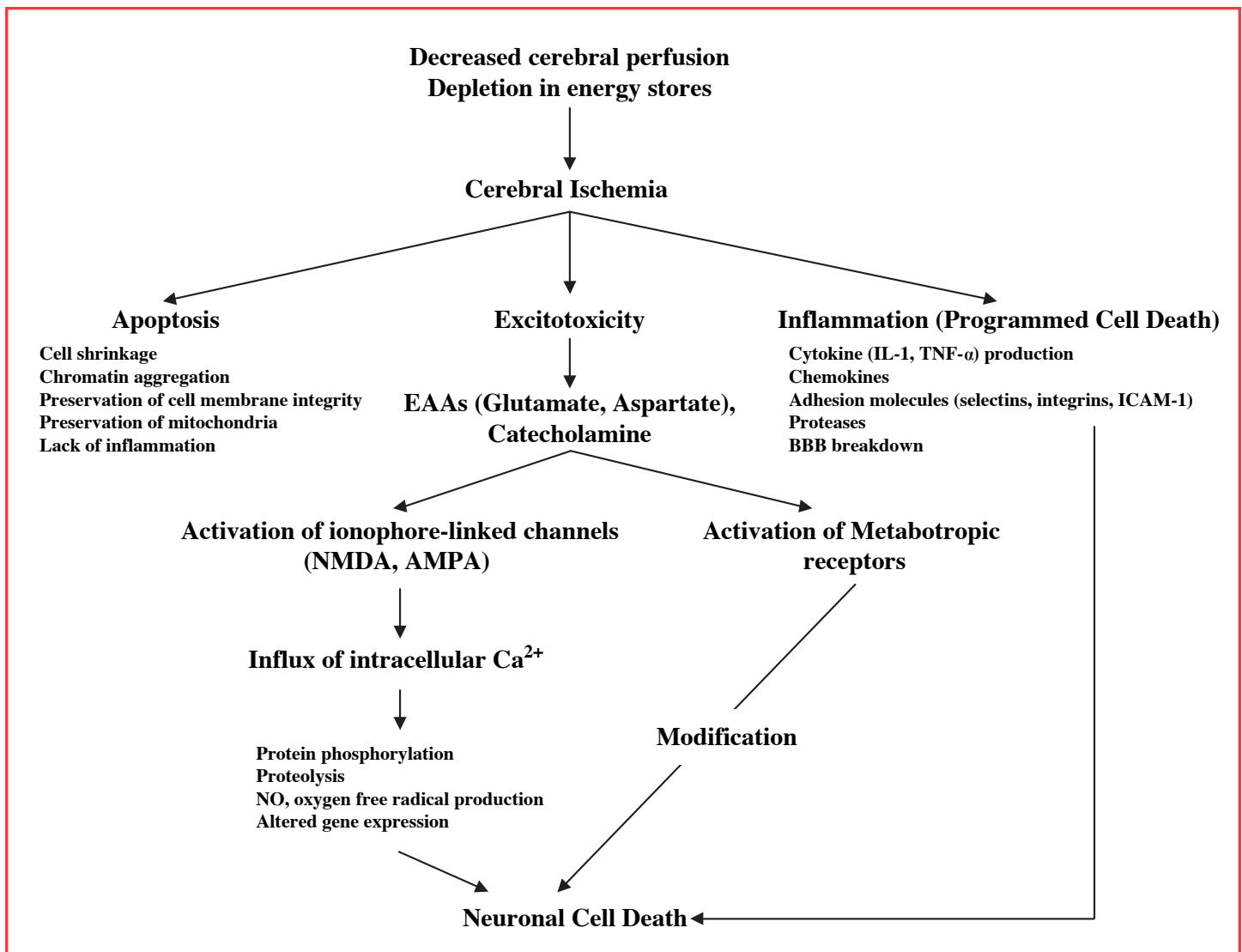


Figure 1. Molecular cascade after stroke.⁵¹ Image courtesy of Elsevier.

Subarachnoid Hemorrhage

Fever after subarachnoid hemorrhage (SAH) occurs in up to 80% in all patients within the first 10 days. Risk factors for developing fever are poor clinical grade and intraventricular blood.^{13,14} It has been shown in several recent studies that hyperthermia independently predicts morbidity and mortality and is implicated in the development of cerebral vasospasm.^{15,16} Fever is unequivocally related to poor outcome, but the use of hypothermia has not been translated into improved outcomes, though the application of hypothermia has not been rigorously studied in the setting of SAH. Todd and colleagues investigated the use of intra-operative hypothermia in 1,001 patients with a preoperative World Federation of Neurological Surgeons score of I, II, or III ("good-grade patients"). The trial showed no significant difference in outcome between intra-operative hypothermia and normothermic groups.¹⁷ In the setting of postoperative hypothermia, more clinical work has been centered on maintenance of normothermia or mild hypothermia. Gasser and colleagues evaluated the feasibility of long-term hypothermia in the setting of poor-grade SAH and brain edema in 21 patients. These patients were treated with hypothermia and barbiturate coma. Overall, there was no difference in three-month outcome between the two groups.¹⁸ Oddo and colleagues investigated the use of normothermia in refractory hyperthermia SAH patients with microdialysis. They concluded that patients with induced normothermia

had significantly less intracerebral metabolic crisis as compared to when they were hyperthermic, irrespective of the patients' intracranial pressure.¹⁹ Badjatia and colleagues presented a case control study that matched SAH patients by age, severity, and blood amount showing that normothermia in the first two weeks after ictus was associated with improved outcomes at 12 months. This study also highlights that maintaining normothermia comes with a higher risk of pneumonia and longer ICU stay.²⁰

Traumatic Brain Injury

Poor outcomes of patients with traumatic brain injury (TBI) have been strongly correlated with hyperthermia, Glasgow coma scale (GCS) score at presentation, age, pupillary response and size, hypoxia, and high intracranial pressure (ICP).^{21,22} Hyperthermia in the first week after initial injury has been associated with increased ICP, prolonged ICU stay, and neurological impairment as well

as diffuse axonal damage, cerebral edema, hypotension, and leukocytosis.^{22,23} Although hyperthermia is a harbinger for poor outcomes, the medical evidence to support the use of hypothermia in TBI is mixed. Henderson et al and McIntyre et al produced meta-analyses with contradictory conclusions.^{24,25} Both of these meta-analyses reviewed roughly the same studies but Henderson et al included studies using patients with normal ICPs. With the removal of these two studies, the Henderson et al meta-analysis shows a positive effect of hypothermia on improved neurological outcome.²⁶ Also of note is the National Acute Brain Injury Study: Hypothermia (NABISH) that investigated the use of hypothermia in 392 patients randomized to normothermia or hypothermia (33°C).²⁷ This study provided the bulk of patients in both of the meta-analyses. This becomes problematic as Polderman and colleagues note that cerebral perfusion pressure and mean arterial pressure cutoffs are lower than what is recommended in current guidelines and significant heterogeneity in terms of treatment protocols between the multiple centers. In the NABISH trial, no significant difference was noted between the normothermic and hypothermic groups, though there was an increased rate of complications and length of stay in the hypothermic group. Patients with a GCS of 5-7 and hypothermia on admission without being actively rewarmed showed a trend toward significant benefit. Similarly, a recent small comparative cohort study by Puccio and colleagues showed that induced normothermia (36-36.5°C) can reduce ICP and maintain a lower ICP, which may signify an overall improvement and less secondary injury.²⁸

Ischemic Stroke

As with all neurological injury, hyperthermia has a deleterious role in the ischemic stroke patient. In a large meta-analysis of 1,443 patients from various studies, hyperthermia was shown to be consistently associated with worse outcomes, especially when fever occurred late in the first week from ictus.^{29,30} Fever has also been counterproductive in the setting of thrombolytics. Millán and colleagues studied 254 patients treated with tissue plasminogen activator within three hours from stroke onset. The results showed that body temperature at 24 hours correlated to stroke severity and volume of hypodensity on CT scan at 24 hours and was higher in patients who did not re-canalize and who had symptomatic hemorrhagic transformation and poor outcome.³¹

Animal models of transient middle cerebral artery occlusion consistently demonstrate reduction in stroke volumes ranging from 27% to 92% with prompt hypothermia. But as the time to hypothermia increases, the possibility of decreasing the size of infarct becomes much less.³² It appears that hypothermia mirrors the use of thrombolytics

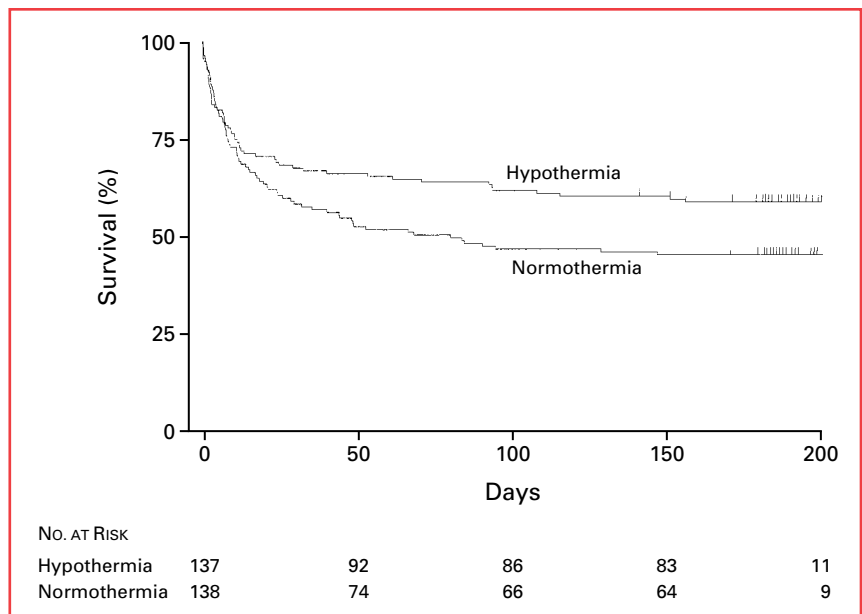


Figure 2. Survival curve of comatose patients randomized to therapeutic hypothermia or no temperature regulation after cardiac arrest with return of spontaneous circulation.⁵² © 2002 Massachusetts Medical Society. All rights reserved.

in terms of a three-hour window and is most useful in the transient occlusion models.³³ There have been several clinical studies investigating the usefulness of hypothermia in the ischemic stroke patient in various settings. Hypothermia has been shown to temper edema formation associated with ischemic stroke as well as decrease ICP elevations seen in malignant MCA syndrome.^{34,35} Hypothermia as an adjunct to thrombolysis was investigated by Krieger et al and DeGeorgia et al in two feasibility studies.^{36,37} DeGeorgia et al reported a significant reduction of infarct size by MRI in the patients that underwent moderate hypothermia. Both trials showed feasibility of surface and intravascular cooling in combination with thrombolysis but were too small to comment on efficacy.

Cardiac Arrest

The clearest indication for hypothermia has been found in the setting of cardiac arrest. Similar to stroke literature, hypothermia has been shown to improve neurological and histological endpoints in various animal models of global ischemia and cardiac arrest. Unlike with stroke, there have been randomized clinical trials that have shown a clear benefit in neurological outcome after therapeutic hypothermia. Bernard and colleagues randomized 77 patients to the normothermic or hypothermic treatments after return of spontaneous circulation.³⁸ Hypothermia to 33°C was induced within two hours of return of spontaneous circulation. Patients remained in hypothermia for 12 hours and were then gradually rewarmed. Twenty-one out of the 43 patients treated with hypothermia (49%) survived and had a good outcome compared to nine of the 34 treated with normothermia (26%). The odds ratio for a good outcome with hypothermia as compared with normothermia was 5.25. The hypothermia after cardiac arrest (HACA) investigators published a multicenter randomized trial that studied the effect of hypothermia on patients that suffered an out-of-hospital ventricular fibrillation arrest.³⁹ Therapeutic hypothermia was defined as 32-34°C with a duration of 24 hours and a slow rewarming period. 137 patients were randomized to normothermia and 136 to the hypothermia group. Fifty-five percent of the hypothermia group had a good neurological outcome compared to 39% of the normothermic group. There was also a significant reduction in three-month mortality (Figure 2). The

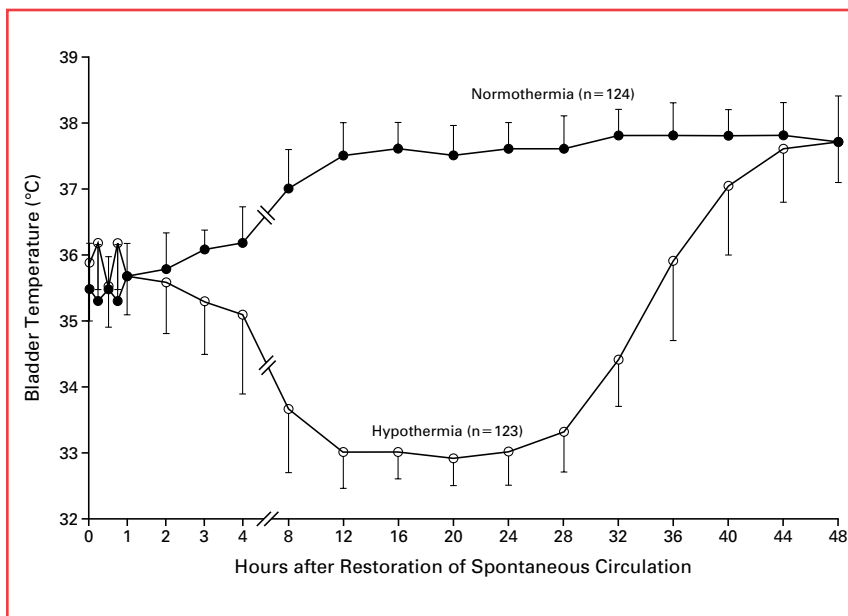


Figure 3. Time course of therapeutic cooling demonstrating the initiation of hypothermia, maintenance phase and slow rewarming.⁵²

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significant effect of hypothermia was further demonstrated by Holzer and colleagues who combined the two feasibility trials as well as a smaller third trial, elucidating that more patients in the hypothermia group were discharged with favorable neurological recovery, and survival at six months with favorable functional neurological recovery was more likely in the hypothermia group.⁴⁰ The number-needed-to-treat to allow one additional patient to leave the hospital with favorable neurological recovery was six. Because of the significant difference in neurological outcomes, therapeutic hypothermia after cardiac arrest was given Class IIa level of evidence in the 2005 American Heart Association and Emergency Cardiovascular Care CPR guidelines. Sagalyn and colleagues reviewed the literature since the HACA and Bernard studies in 2002 for post-resuscitation care of cardiac arrest patients using hypothermia.⁴¹ They included 13 published reports outside of the randomized control trials, confirming that the real world application of hypothermia affords similar benefit in neurological outcome and survival of the post cardiac arrest patient.

Technical Aspects of Temperature Regulation

The process of therapeutic hypothermia can be divided into three stages: initiation, maintenance, and rewarming (Figure 3). In the past, cooling was achieved by surrounding the patient with ice packs. This technique was successful but could take hours to days to reach the target temperature. Renewed interest in temperature management has stimulated new technological advances in cooling. Thanks to computer-controlled large-area surface cooling devices and endovascular catheters, patients can be precisely cooled in minutes to a few hours. Because the interval from the inciting event to reaching therapeutic hypothermia correlates with improved outcome, rapid cooling is crucial for neuroprotection. In light of this, pre-hospitalization cooling with iced saline is currently being evaluated to further reduce the interval until hypothermia is achieved.⁴² Additionally, rapid cooling can avoid some of the complications associated with the induction phase, such as hypovolemia, electrolyte disorders, and hyperglycemia.⁴³ The body, however, does not acquiesce to induced-hypothermia without exhibiting normal compensatory responses. In particular, shivering must be controlled in order to reach and maintain the target temperature. The mainstay of shiver

control is good sedation and analgesia. First-line anti-shiver agents include fentanyl, meperidine, dexmedetomidine, propofol, and magnesium.⁴² Paralytics may be necessary during the induction phase, but prolonged paralysis is rarely required. Warm-air counter-warming can also lower the shiver threshold.⁴⁴

Avoiding complications in the maintenance phase requires an understanding of the physiologic consequences of hypothermia. As the metabolic rate drops, so does carbon dioxide (CO₂) production. Attempts to maintain a "normal" P_{CO₂} level can result in a relative hypercapnia that has the potential to cause cerebral vasodilation and increased intracranial pressure. Conversely, an excessively low P_{CO₂} will cause vasoconstriction and decrease cerebral blood flow that may exacerbate neurological injury.⁴² Target P_{CO₂} values at 32°C should be 32-36 mmHg. This correlates with a level of 42-46 mmHg at 37°C.⁴⁵ Meanwhile, hypothermia-induced increases in fat metabolism result in increased levels of glycerol, lactate, ketones, and free fatty acids. The production of these molecules causes a metabolic acidosis with a pH that rarely drops below 7.25 and does not require treatment as the intracellular pH actually increases slightly.⁴⁶

An intracellular shift of electrolytes coupled with renal tubular dysfunction can severely deplete serum potassium, magnesium, and phosphate levels.⁴⁷ The loss of extracellular electrolyte homeostasis increases the risk for cardiac arrhythmia. The most common EKG changes in the setting of hypothermia are prolonged PR intervals, increased QT interval, and widening of the QRS complex. Meanwhile, hypothermic myocardium is less responsive to anti-arrhythmic drugs and more difficult to defibrillate.⁴² This is particularly true with severe hypothermia (< 32°C). In severe hypothermia, the risk of arrhythmia increases, usually starting with atrial fibrillation and progressing to ventricular tachycardia and even ventricular fibrillation as the temperature decreases. Because the treatment of cardiac arrhythmia is more difficult, prophylaxis is particularly important.

Normally, mild hypothermia results in bradycardia and an increase in cardiac contractility. Though these have opposite effects, the net result is a decrease in cardiac output in sedated and euvolemic patients. Despite the drop in cardiac output, there is little concern for tissue ischemia as the decrease in metabolic rate usually exceeds the lowered energy supply.⁴⁸ The heart rate can be increased with external pacing or the administration of chronotropic drugs; however, this is usually unnecessary. Care should be taken to avoid hypotension in the hypovolemic patient. A "cold diuresis" can occur through a combination of a catecholamine-induced increase in venous return, activation of atrial natriuretic peptide,

NEUROCRITICAL CARE CENTER

Michael A. DeGeorgia, MD

Director

The Neurocritical Care Center provides continuous state-of-the-art monitoring and treatment for critically ill patients with neurological or neurosurgical disease. UH introduced the world to neurological intensive care medicine nearly three decades ago. Since then, the center has earned international respect for training physicians worldwide. The Reinberger Neuroscience Intensive Care Unit is staffed 24/7 by a dedicated team of neuro-intensive care specialists as well as specially trained critical care nurses, pharmacists, respiratory therapists, physical therapists, nutritionists, case managers, social workers, and bioethicists. Our team is skilled in managing the full range of neurology and neurosurgery patients. We offer highly sophisticated treatment options for patients who are critically ill, need postoperative care, or require intensive neurological monitoring. Studies show that involvement of a dedicated neurological intensive care team correlates with significantly reduced length-of-stay and controlled cost and most importantly with reduced in-hospital mortality and better patient outcomes.

(Keeping It Cool: Temperature Regulation in the Neuro Intensive Care Unit continued)

and decreased levels of antidiuretic hormone and renal antidiuretic hormone receptor levels.⁴⁹ These processes may culminate in hypotension and an increase in blood viscosity. The risk of developing hypotension is worsened with the administration of diuretics, including mannitol.

Rewarming following hypothermia requires vigilant supervision. As the body returns to normothermia, it can experience rebound effects from the cooling period. This is particularly true for potassium homeostasis.⁴² Life-threatening hyperkalemia can occur. Glucose levels must also be carefully monitored as hypothermia-induced insulin resistance is reversed and hypoglycemia may emerge. Finally, the neuroprotective effects of hypothermia may be erased by rapid rewarming.⁵⁰ The risk for all of these complications can be minimized by rewarming the patient in a slow, controlled fashion. This permits normal compensatory mechanisms to adapt to and ameliorate the sequelae of increasing temperature. As such, rewarming should not occur at a rate faster than a quarter of a degree per hour.

Conclusion

A great deal has changed since the first trials of hypothermia. New technologies in the laboratory and the ICU have permitted better understanding and implementation of therapeutic temperature control at UH and elsewhere. With an increased number of studies being performed, the indications for cooling are on the verge of expanding to new disease processes. One reason for this may be the broad effects of hypothermia. Unlike many of the modern strategies to minimize neuronal injury that focus on a particular molecule, hypothermia affects multiple secondary injury pathways that lead to neuronal loss. As we learn more about how to use this treatment and avoid the complications that have been associated with it, the less likely it will be that therapeutic hypothermia will go into hibernation again.

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Authors

Alan Hoffer, MD

Assistant Professor of Neurosurgery
UH Neurological Institute
Department of Neurosurgery
University Hospitals Case Medical Center
216-844-5744
Alan.Hoffer@UHospitals.org



Christopher Horn, MD

Fellow
UH Neurological Institute
Department of Neurology
University Hospitals Case Medical Center
216-844-3192
Christopher.Horn@UHospitals.org



Michael DeGeorgia, MD

Director, Neurocritical Care Center
UH Neurological Institute
Department of Neurology
University Hospitals Case Medical Center
216-844-1552
Michael.DeGeorgia@UHospitals.org



Managing Skull Base Chondrosarcoma

By
Alia Hdeib, MD
Cliff A. Megerian, MD
Nicholas C. Bambakidis, MD

Skull base chondrosarcomas are rare lesions of the skull base, which are often difficult lesions to cure. They typically occur laterally within the petrous bone of the skull base and can extend intradurally with compression of the brainstem and lower neurovascular structures.¹⁻³ Because they are relatively rare lesions, individual practitioners may only have an opportunity to treat small numbers of patients over the course of their careers. Despite the suffix “-sarcoma,” these lesions behave in an indolent fashion and respond well to aggressive surgical treatment and adjuvant therapy quite unlike chordomas with which they are often confused.⁴ This article highlights some of the goals of treatment, in the context of a case example, and reviews the pertinent literature available regarding these lesions.

Case Illustration

A 64-year-old man, in otherwise good health, presented to the Department of Neurological Surgery at UH with a history of acute severe headache. The patient noted that when his headache developed, he experienced transient double vision when looking to the right, which quickly went away. His past medical history was significant only for mild hypertension, and he was taking a low dose of diuretic medication to control it. He continues to work daily as an attorney. His neurological function was normal upon physical examination. The exam included a detailed evaluation of his cranial nerve function as well as an evaluation of his hearing. Imaging demonstrated the presence of a heterogeneously enhancing lesion arising from the petrous apex on the right side extending into the posterior and middle cranial fossae (Figure 1). Evidence of brainstem compression was apparent on axial imaging, and there was evidence of hemorrhage within the lesion. Significant bony erosion of the petrous apex was also observed on computed tomography imaging (Figure 2). Imaging was consistent with the diagnosis of a chondrosarcoma of the skull base.

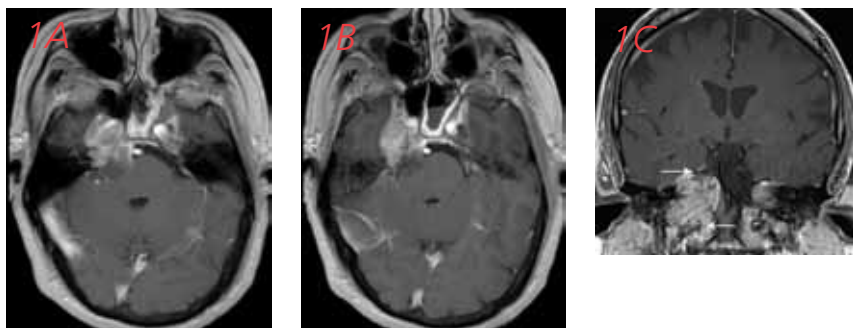


Figure 1: Preoperative T1 magnetic resonance images (MRI) with contrast show a heterogeneously enhancing lesion arising from the right petrous bone extending into the posterior (A) and middle (B) cranial fossa. Coronal views (C) demonstrate the extent of the tumor cranio-caudally (arrows).



Figure 2: Computed-tomography (CT) shows lytic destruction of the petrous apex (arrow), a characteristic of chondrosarcomas.

After further discussion regarding treatment options, the patient was taken to the operating room for a combined transpetrosal approach to the posterior and middle cranial vaults. Following a craniectomy, which crossed the transverse sinus, a partial petrosectomy was performed, sparing the labyrinth in an effort to preserve hearing (Figure 3).^{5,6} A presigmoid approach was then used to debulk the tumor within the posterior fossa. Pathological analysis was consistent with a chondrosarcoma with acute hemorrhage within it (Figure 4). Following ligation of the superior petrosal sinus and section of the tentorium, the middle fossa was exposed; the tumor within Meckel's cave and the petrous apex was also removed (see Surgical Video at UHhospitals.org/skullbasevideo). The patient did well postoperatively without any neurological deficits and was discharged after a stay of approximately seven days. Imaging demonstrated excellent decompression of the neural elements with a small amount of residual tumor present within the petrous bone (Figure 5). He has since been scheduled for adjunctive radiosurgical treatment.

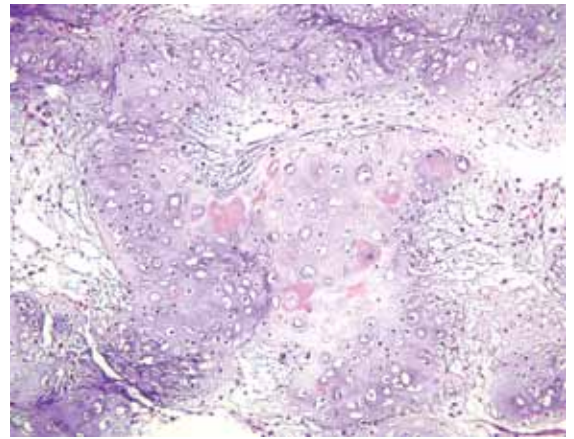
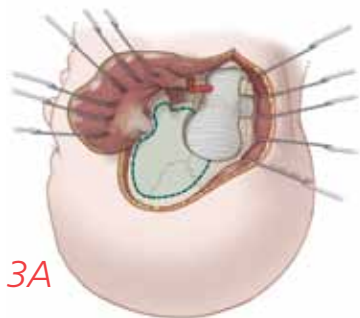
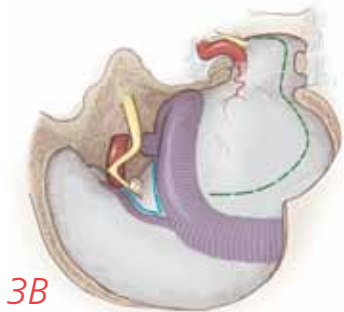


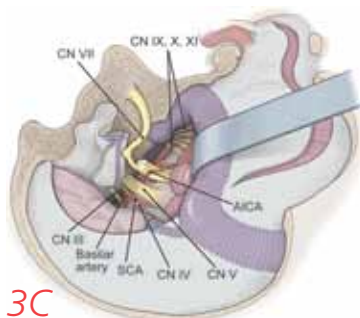
Figure 4: Histopathology showing large cells in a cartilaginous matrix, consistent with chondrosarcoma. Acute hemorrhage is also present in select areas.



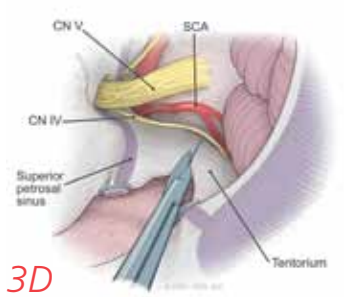
3A



3B



3C



3D

Figure 3: Illustrations depicting the surgical approach. With the patient in the lateral position and the head turned, a subtemporal craniotomy is combined with a posterior fossa craniotomy, which can be extended to the foramen magnum if necessary (A). Exposure of the posterior fossa may be done either presigmoid or retrosigmoid (B). In the present case, only a presigmoid exposure was necessary (C). The superior petrosal sinus can be ligated and the tentorium incised to allow simultaneous access to the middle cranial fossa (D). Such a maneuver must avoid damage to the trochlear nerve (CN IV), which travels along the tentorial border. SCA = superior cerebellar artery, AICA = anterior inferior cerebellar artery; CN III = oculomotor nerve, CN IV = trochlear nerve, CN V = trigeminal nerve, CN VII = facial nerve, CN IX = glossopharyngeal nerve, CN X = vagus nerve, CN XI = spinal accessory nerve, CN XII = hypoglossal nerve, PICA = posterior inferior cerebellar artery, and SCA = superior cerebellar artery. Images courtesy of Barrow Neurological Institute and Journal of Neurosurgery.

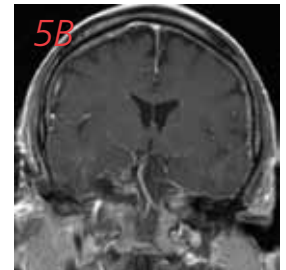
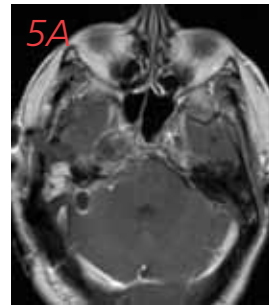


Figure 5: Postoperative contrasted magnetic resonance images in axial (A) and coronal (B) planes showing decompression of the brainstem and near-total removal of the tumor from within the petrous bone.

Discussion

Chondrosarcomas are rare skull base tumors previously thought to be similar to chordomas, but they follow a different clinical course. They are a less aggressive tumor, with better prognosis and lower recurrence rate.⁴ Chondrosarcomas represent 0.15% of all intracranial neoplasms and approximately 6% of skull base tumors.¹ Most commonly found in a paramedian location in the petrosphenoclivus junction area, they tend to be low-grade and slow-growing.^{2,3}

These tumors are thought to arise from primitive mesenchymal cells found in cartilaginous nests at the skull base.^{4,7} Histologically, three different subtypes are identified: classical, mesenchymal, and dedifferentiated chondrosarcomas.³ Microscopically, classical chondrosarcomas contain large cells in a cartilaginous background. Three grades are defined: I, II, and III, with higher grade tumors behaving more aggressively with a higher metastatic potential. However, spinal drop metastasis has been reported in the literature after subtotal surgical resection of low-grade tumors.⁸ Mesenchymal subtypes demonstrate areas of cartilage and undifferentiated mesenchymal cells while dedifferentiated subtypes are histologically similar to anaplastic sarcomas.³ Abnormalities in chromosomes 9, 10, and 22 have been described in chondrosarcomas.³ Tumors with p53 gene overexpression and high Ki67 index show a more aggressive clinical course and a poorer prognosis.^{3,9,10}

The most common clinical presentation is referable to symptoms of headaches and diplopia (80% and 74% in one series and 48% and 45%, respectively, in another series) with cranial nerve VI palsy as the most common finding, though multiple cranial nerves can be involved.^{1,3} Radiographically, chondrosarcomas can be indistinguishable from chordomas. Both demonstrate bony erosion, are iso- or hypointense on T1 weighted images, are hyperintense on T2 images with varying heterogeneity, and show varying degrees of contrast enhancement.^{3,4,11} One distinction is that chordomas tend to be more midline and chondrosarcomas more paramedian, usually near the petro-occipital synchondrosis.³ Cerebral angiography helps to better evaluate the internal carotid artery (ICA), the basilar artery, and collateral circulation. In cases where ICA sacrifice is considered, particularly in patients with previous resections and/or radiation treatment, balloon-test occlusion of the ICA is an indicated part of

preoperative planning.³ Our patient presented with an unusual syndrome consistent with hemorrhage within the tumor, along with a history likely representative of transient cranial nerve VI palsy.

Because chondrosarcomas are rare and indolent, with high local recurrence, the best management plan remains a topic of debate. Options include biopsy, surgery, and/or radiation treatment (conventional fractionated radiation and/or radiation with charged particles, e.g., proton beam radiation, brachytherapy, and radiosurgery).³ Review of the literature reveals several institutional experiences with treatment of skull base chondrosarcomas. Most institutions advocate surgical resection, with intent to obtain gross total or near total resection especially for previously unoperated tumors. To achieve radical resection with low morbidity and mortality, attention is directed at tailoring skull base approaches to each individual tumor, based on its pattern of growth. Several surgical approaches for these tumors have been described in the literature and can be divided into anterior approaches (e.g., extended subfrontal, transthemoidal, transsphenoidal), anterolateral approaches (e.g., fronto-temporal with or without orbitozygomatic osteotomies, subtemporal, transpetrous apex, transcavernous, periauricular subtemporal-infratemporal) and posterolateral and lateral approaches (e.g., petrosal approaches, including retrolabyrinthine, partial labyrinthine, and petrous apicectomy; extreme lateral approach including transcondylar and transjugular).³ Staged resections are necessary in particular cases. In the current case, involvement of both the middle and posterior cranial vaults necessitated exposure via a combined transpetrosal approach. Though additional bone could have been removed to increase the surgical exposure, preservation of hearing for this patient was an important consideration in opting for the retrolabyrinthine exposure performed.

Though chondrosarcomas are indolent, slow-growing tumors, the local recurrence rate is high and is the eventual cause of mortality.⁴ Several series describe institutional experiences with management of these tumors and long-term outcome after treatment. In 1995, a group from France reported its combined nine-year experience and follow-up of 60 patients with skull base tumors, 14 of which were chondrosarcomas.¹² In this series, the recurrence-free survival rate for these patients was 90% at five-year follow-up and 65% toward the end of the study, with higher rates correlating with those patients who had total or near total resection and no previous surgical or radiation treatments.¹² Another series reported the long-term outcome after surgical treatment of 25 patients over 19 years.¹³ 92% of the tumors extended into the posterior fossa. Gross total resection was achieved in 19 cases, with 0% perioperative mortality. At follow-up, the average Karnofsky performance score (KPS) score was 91%, and five- and 10-year survival rates were 95%.¹³

In a larger series, a group from the University of Washington in Seattle reported its experience with aggressive microsurgical resection of chondrosarcomas.¹⁴ 47 patients were treated over a 20-year period, 14 of which had previous surgery or radiation and 24 of which required a staged operation. Follow-up ranged from two to 255 months, with an average of 86 months, and those with incomplete resections underwent post-operative radiation with proton beam radiotherapy, fractionated radiation, or radiosurgery.¹⁴ 61.7% of patients had a gross total resection, better achieved in those who had primary versus redo operations. 76.6% of patients were alive at the end of the study, of which 44.7% were disease-free. At 10 years, recurrence-free survival was 32% in all patients, though lower for those with

previous treatments. The authors report good functional status for patients at follow-up, with KPS scores > 82 before surgery and > 85 at one year and last follow-up. No significant conclusions about radiotherapy were drawn from the study.¹⁴

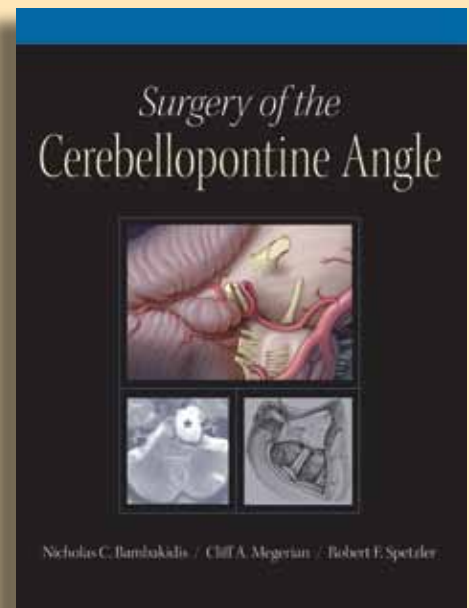
Due to the slow-growing nature of these tumors, the long-term outcome after radiation is not well-defined¹⁵ and the necessity for radiotherapy for low-grade chondrosarcomas remains unclear. Fractionated conventional external beam radiation is generally not a recommended modality because chondrosarcomas tend to be radioresistant at the standard < 70 Gy dose.³ However, treatment with charged particle/proton beam radiation is an option. It allows for increased dose delivery to the tumor while sparing critical surrounding structures.¹⁶ One series in the literature evaluated the long-term effects of proton beam radiation in 58 patients with skull base tumors, 25 of which were chondrosarcomas.¹⁷ Local control rate was obtained in 92% of these patients. Factors influencing the degree of control included tumor size (100% versus 56% for large tumors > 25 ml) and brainstem invasion (94% versus 53% for invasive tumors).¹⁷ Several recent studies addressed the efficacy of stereotactic radiosurgery (SRS) in tumor control for patients with chondrosarcomas. However, most series are small. In one study, 28 patients with skull base tumors with an average tumor volume of 9.8 cm³ were evaluated after gamma knife treatment.¹⁸ Ten of them had chondrosarcomas and, after SRS with gamma knife, five-year local tumor control was 80%.¹⁸ Another series included four patients with chondrosarcoma and showed that all four patients had tumor control at follow-up after SRS.¹⁹ In the case described in this article, we recommended SRS as adjuvant therapy with close follow-up because of the small volume of residual tumor.

Conclusions

Chondrosarcomas are indolent but recurrent tumors. The goal of treatment is radical resection, and advances in microsurgical skull base techniques have improved surgical excision. Treatment should be tailored to the individual patient; in general, however, generous surgical resection while limiting patient morbidity is warranted. In elderly patients, even subtotal resections can result in good long-term results. Radiotherapy is an attractive option, though more long-term studies are needed to determine efficacy in local tumor control with use of modalities, such as stereotactic radiosurgery.

See the surgical video at UHHospitals.org/skullbasevideo

Nicholas C. Bambakidis, MD, is a consultant for Medtronic Sofamor Danek. The other authors report no financial relationships with commercial interests relevant to the content of this article.



The cerebellopontine angle perpetually challenges the expertise of surgical teams treating pathologic conditions of the skull base. Surgery in this dense, complex area is both difficult and open to undesirable outcomes, but new diagnostic tools and treatments – endoscopy, endovascular surgery, and radiosurgery among them – offer surgeons proven, effective options.

In *Surgery of the Cerebellopontine Angle*, Drs. Nicholas C. Bambakidis, Cliff A. Megerian and Robert F. Spetzler review these options.

KEY FEATURES:

- Synthesis of anatomic, neurologic, and radiologic considerations
- Summary of surgical approaches to the skull base
- Latest advances in the treatment of acoustic neuromas
- An interactive, multimedia DVD atlas that includes full-color video clips of case samples

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Authors



Alia Hdeib, MD
Resident
Department of Neurological Surgery
UH Neurological Institute
University Hospitals Case Medical Center
216-844-3472
Alia.Hdeib@UHhospitals.org



Cliff A. Megerian, MD
Physician, Brain Tumor and Neuro-Oncology Center
UH Neurological Institute
University Hospitals Case Medical Center
Case Western Reserve University School of Medicine
216-844-5500
Cliff.Megerian@UHhospitals.org



Nicholas C. Bambakidis, MD
Director, Cerebrovascular and Skull Base Surgery
UH Neurological Institute
University Hospitals Case Medical Center
Associate Professor, Department of Neurological Surgery
Case Western Reserve University School of Medicine
216-844-8758
Nicholas.Bambakidis@UHhospitals.org

Transforming the Way We View the Brain and Its Blood Supply

By

Gurpreet S. Sandhu, MBBS

Mark Griswold, PhD

Jeffrey L. Duerk, PhD

Jeffrey L. Sunshine, MD, PhD

Magnetic resonance imaging (MRI) has become indispensable for the care of neurological patients by virtue of its vital role in planning the management for diseases. Additional impact arises from its role in the clinical research to better understand underlying detail and pathophysiology of the human nervous system. MRI permits application of different imaging components for a given patient in a single setting to reveal structural, physiological, and metabolic brain tissue analysis. The availability of such diversity to the neurologically based clinician has only been possible through joint scientific innovations often from magnetic resonance (MR) technical and clinical research personnel. The MR research team at University Hospitals Case Medical Center has made many contributions to advance the field. This group is composed of primary faculty members with their graduate students and post-doctoral fellows specializing in various areas, including pulse sequence design, coil design, MR spectroscopy, neuro-imaging, body imaging, and small animal imaging. Currently, we work on numerous projects, focusing on neurologically related projects in perfusion, vascular, and structural MRI. In addition, we review a pilot study to apply MRI criterion for triage of stroke patients with unknown time-of-onset for reperfusion therapy. In the following sections, we briefly present some of these research projects to provide an overview of our activities and a sense of their potential impact to the care of neurological patients.

Fast Dark-Blood Carotid Artery MRI: A New MRI Method at UH

Carotid artery atherosclerotic lesions are associated with approximately 15% of all ischemic stroke episodes.¹ An ischemic episode can occur as a result of rupture of the atherosclerotic plaque to release the plaque contents that move to block the distal vasculature. The rupture risk of plaque is primarily determined by the size and consistency of its atheromatous core, thickness of its fibrous cap, and ongoing inflammation.² Carotid artery screening by imaging techniques, including ultrasound, computed tomography (CT), and MR angiography, is used to identify the plaques and perhaps suggest those at higher risk of rupture.³ Limitations persist. For example, carotid MR angiography provides information regarding lumen size only and is typically unable to characterize the plaque burden.⁴ To overcome this limitation, a dark-blood MRI method has been developed at UH, where MR images of the arterial wall are acquired while the blood signal in the lumen is suppressed. Dark-blood images can be used to determine plaque composition without interference from the adjacent flowing blood signal.⁵ Such images are used to assess plaque vulnerability to rupture as well as its remodeling over time.⁶ Dark-blood images are traditionally acquired using a dual inversion-recovery fat-saturated turbo spin-echo sequence that takes a long time to produce low-resolution images.⁴ The long imaging time increases the odds of motion artifact degrading the image quality due to patient movement (e.g., swallowing). A number of alternate MRI techniques have been devised using a fast sequence that provides high-quality images in a relatively short time.⁷⁻⁹ Unfortunately, these techniques have various limitations, such as high radio frequency energy exposure, loss of contrast, and image artifacts. Using similar pulse sequence designs, we developed a novel dark-blood carotid imaging technique called Halting the Effects of Flow Enhancement with Effective Intermittent

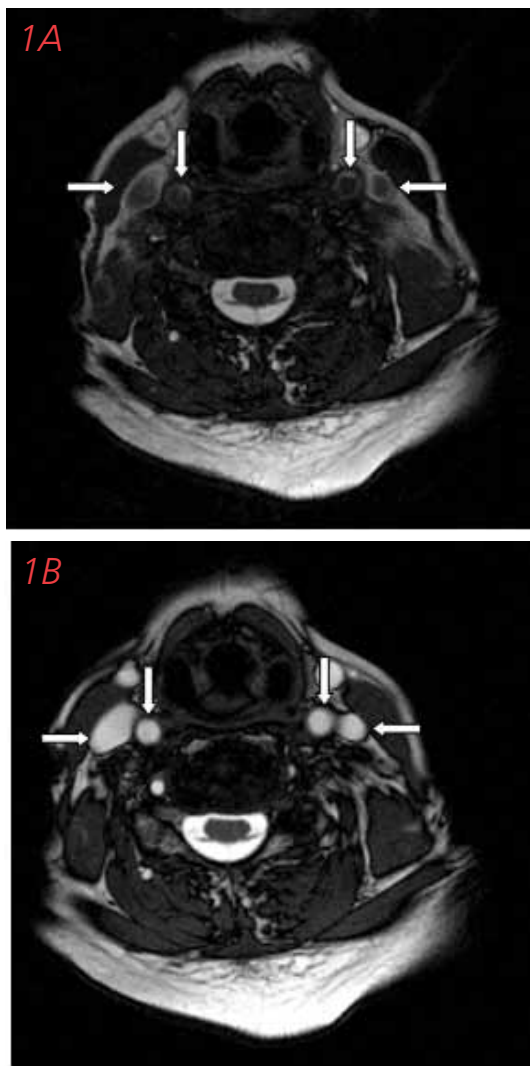


Figure 1: Carotid artery images using the HEFEWEIZEN dark-blood technique (A) and the true fast imaging with steady-state precession (TrueFISP) technique (B). Blood signal from the carotid artery (vertical arrows) and the internal jugular vein (horizontal arrows) is suppressed in the dark-blood image, which enables vessel wall visualization. Alternatively, high blood signal from the two vessels in the TrueFISP image obscures the visualization of the vessel walls.

Zeugmatographic Encoding (HEFEWEIZEN) that is under patent application.¹⁰ HEFEWEIZEN provides high-resolution carotid artery images with more than 80% blood signal suppression but with only 13% increase in the imaging time compared to our most rapid acquisition techniques (Figure 1). The parameters can be adjusted for suppression of a signal from the carotid artery or jugular vein as clinically desired.¹⁰ Currently, we are investigating the HEFEWEIZEN carotid images for clinical quality and utility in identifying plaque and its underlying stability.

Improving Structural Imaging of the Brain

We now have access to a new phased-array receive coil with 32-array elements that has been developed for head MRI and approved by the FDA for clinical use. Traditional receive coils consist of a single array element, have lower sensitivity and are incompatible with parallel imaging techniques often employed for faster imaging. Phased-array coils consist of spatially arranged elements in a desired geometry with each element separately acquiring signal that is combined to form the complete MR image.¹¹ Phased-array coils improve the image quality, enable parallel imaging, and are most commonly found with 4-, 8-, 12- or 16-array elements for clinical head MRI.¹²⁻¹⁴ Preclinical studies suggest that images acquired using such coils have a higher signal-to-noise ratio (SNR) than those acquired using the coils with a lower number of array elements. However, while SNR is a reliable quantitative parameter, an increase in the SNR value of an image does not necessarily mean improvement in its diagnostic quality.¹⁵ Therefore, to investigate the improvement in the diagnostic utility of images acquired using our 32-array head coil, we have compared images acquired using this coil with those acquired using our more common 12-array coil to demonstrate image quality and clinical utility. FLAIR, T1-, T2-, and diffusion-weighted images of 21 patients acquired on 1.5T using the two coils were qualitatively compared by neuroradiologists in terms of preferred image with better differentiation of the grey and white matter structures, smoothness of white matter tracts, visibility of cranial nerves, and differentiation of basal ganglia and internal capsule. In addition, the difference in visibility of lesions and image artifacts from the two kinds of images was also investigated. The results demonstrated a trend of preference for images acquired using this new coil in particular for FLAIR, T1-, and diffusion-weighted images. In contrast, no trend of preference for images acquired using 12-array coil was observed. There was a significant preference for images acquired using the new coil in terms of lesion visibility in the cortical and subcortical white matter and deep brain structures without any increase in the image artifacts. This has confirmed the potential image quality that can be generated at 1.5T similar in many ways to improved images seen at 3T, though with diminished cost and much greater availability.

Additional improvement in image quality has been generated with attention to patient movement during MR image acquisition that often results in image artifacts, repeat exams, delay in diagnosis, resource waste, and patient inconvenience. For motion correction, investigators have developed techniques called Periodically Rotated Overlapping Parallel Lines with Enhanced Reconstruction (PROPELLER) and similarly MRI with "rotating blade-like k-space covering" (BLADE),¹⁶ in which MR signal is collected in concentric rectangular strips that are rotated around the k-space origin. This allows any inconsistencies in the data due to head movement to be corrected (Figure 2). The BLADE method is used for reconstruction of head MR images in 15% of exams at our imaging center. We compared the quality of BLADE images with those reconstructed using the traditional technique. In this randomized single blinded study, 70 MR image sets were compared

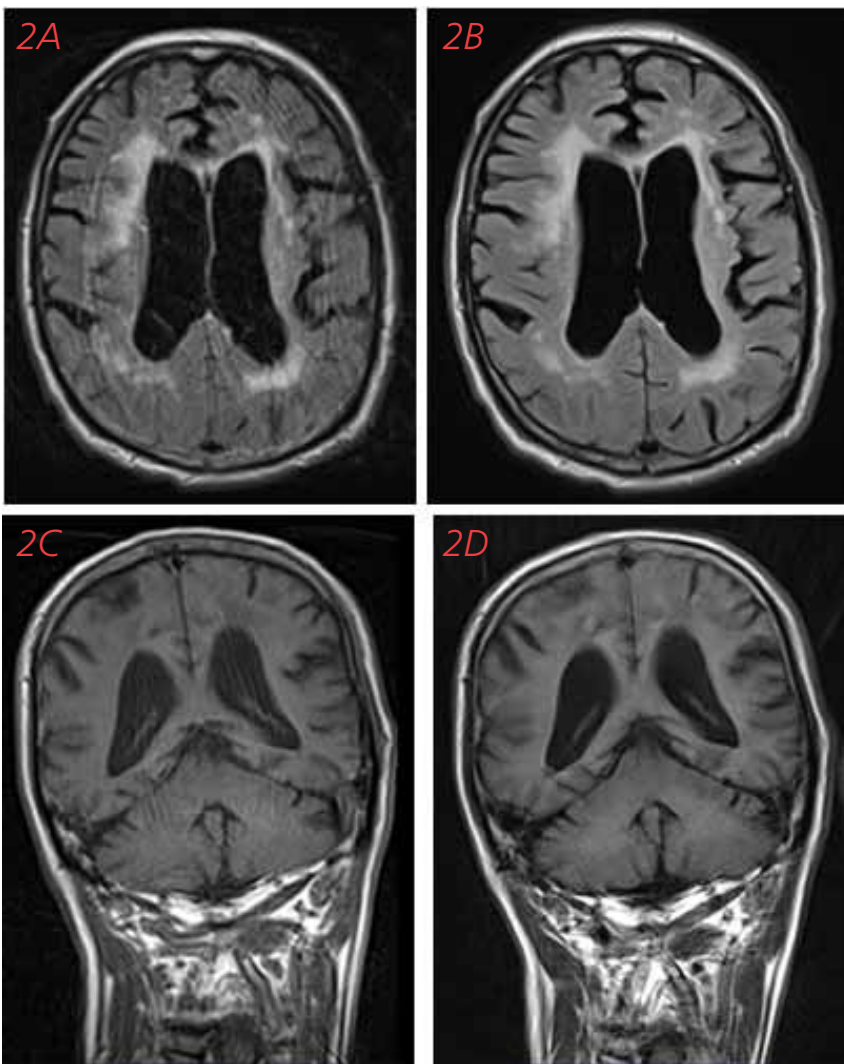


Figure 2: MR images of a patient who moved during imaging showing the utility of the BLADE technique for removal of the motion artifacts. Upper row: an axial FLAIR image reconstructed using the traditional technique (A) and an image reconstructed from the same data using BLADE technique (B). Lower row: a coronal T1-weighted image of the same patient reconstructed using the traditional technique (C) and an image reconstructed from the same data using the BLADE technique (D). Even this limited motion artifact seen in the traditional images can hinder the structural analysis of the brain while removal of the artifacts enables visualization of the anatomy and pathologies.

Quantifying Cerebral Perfusion

Perfusion imaging is used to determine regional blood flow in the brain for investigation of neurological disorders, including stroke, cerebral tumors, and epilepsy. Various imaging techniques have been employed for cerebral perfusion quantification: positron-emission tomography and nuclear single photon emission computed tomography enable accurate perfusion estimation, though the associated lengthy and complex methods can limit their clinical application. Perfusion CT can be used for quantification, but it evaluates only one or two 1 cm-thick slices of the brain after a single contrast bolus injection using repeat exposure to radiation over a single brain slab.¹⁷ Of two MRI methods for perfusion quantification, arterial spin labeling (ASL) and dynamic susceptibility contrast (DSC), ASL is limited by longer imaging time, which often limits its application particularly in stroke patients.¹⁸ DSC MRI provides a set of high-resolution imaging covering a large region of the brain (78 mm thickness) in a short time (48 images at a rate of 1.5s/image) following a gadolinium bolus injected intravenously. Differential regional change in the signal intensity from successive T1-weighted images is used to calculate various relative perfusion indices, including cerebral blood flow (CBF), cerebral blood volume (CBV), mean transit time (MTT), and time-to-peak (TTP).¹⁹ However, an accurate calculation of arterial input function (AIF) is necessary to prevent large discrepancies in perfusion results from DSC MRI.²⁰ Tim Carroll, PhD, our collaborator from Northwestern University, has developed a technique for perfusion quantification by DSC method.²¹ In this “bookend” technique, AIF analysis is not required; instead, the value of the longitudinal relaxation time of brain regions during the steady phase of the contrast (i.e., after passage of the contrast bolus) is used. Preliminary results have proven its accuracy and reliability, and we are working with Dr. Carroll toward a clinical application of this technique to assist in identifying potentially salvageable brain tissue in stroke patients.

by neuroradiologists for quality, clinical utility, lesion detection, and characterization. The image sets consisted of a pair of FLAIR, pre- and post-contrast, T1-weighted or T2-weighted images reconstructed using traditional and BLADE methods of patients who moved during imaging. The results demonstrate that the BLADE method significantly reduces motion artifacts on FLAIR, T2-weighted, and noncontrast T1-weighted images. The neuroradiologists preferred the BLADE method for T2-weighted and FLAIR images with improved lesion conspicuity in the later image type.

Applying a Diffusion-Perfusion Mismatch Hypothesis for Stroke Patients

Diffusion and perfusion MRI is used in acute ischemic stroke (AIS) to find desirable reperfusion therapy candidates beyond the window of three hours from the initial onset of stroke symptoms. Immediately following their typical arrival in emergency or onset of problems while in the hospital, our AIS patients can undergo diffusion- and perfusion-weighted MRI to calculate the volumes of the ischemic lesion. A diffusion defect approximates the irreversibly damaged region of the brain, whereas a region with cerebral hypoperfusion consists of both the irreversibly damaged tissue and the ischemic penumbra (i.e., that region of the brain where re-establishment of the blood supply will salvage the tissue and prevent progression to irreversible damage).²² The diffusion-perfusion mismatch hypothesis postulates that the difference between the volumes of apparent lesions from perfusion- and diffusion-weighted MR images of an AIS patient provides an estimate of the ischemic penumbra. Prospective multicenter studies, such as Diffusion and Perfusion Imaging Evaluation for Understanding Stroke Evolution (DEFUSE) and Echo-planar Imaging Thrombolytic Evaluation Trial (EPITHET), have shown a beneficial role of intravenous thrombolytic treatment (IVT) in AIS patients with an ischemic penumbra (identified by presence of a diffusion-perfusion mismatch) during the three- to six-hour window while acquiring MR images.^{23, 24} Alternatively, those without an ischemic penumbra (absence of a mismatch, or simply matched defect) represent a poor risk-benefit ratio for reperfusion therapy. We investigated the outcomes in the rare situations when patients with matched defects underwent intra-arterial therapy at our institute in the past 10 years. Of eight treated patients with matched defects, four had symptomatic intracranial hemorrhage, five died, and only one achieved a favorable clinical response (12.5%). This confirms that patients presenting to us with such a profile represent a very high-risk population with some similarity to the outcomes of patients with a no-mismatch profile in the DEFUSE study.

The existence of a significant region of ischemic penumbra in AIS lesions as late as 24 hours after onset of the ischemic episode has been reported, suggesting that a subset of AIS patients even with unknown time-of-onset may benefit from reperfusion therapy.²⁵ These patients form approximately one-third of all stroke patients and include those who have onset of stroke attack during sleep, those cases in which the patient is

unable to provide a reliable history, and those cases in which no other witness was present at the onset of the episode. In order to explore the utility of the mismatch hypothesis in these patients, we have designed a pilot study to establish this criterion in such a study population. MR images, including diffusion- and perfusion-weighted images, MR angiography, and T1- and T2-weighted images, will be used to identify those patients who have a diffusion-perfusion mismatch despite the unknown time of symptom onset. Under protocol, patients having a mismatch will undergo reperfusion therapy by mechanical thrombectomy and/or intra-arterial thrombolysis. The risk of reperfusion-related intracranial hemorrhage should be minimized by excluding the patients with larger irreversibly infarcted lesions from reperfusion therapy.

Laser Interstitial Thermal Therapy for Glioblastoma Multiforme

Patients suffering from a recurrent glioblastoma multiforme (GBM) have a grave clinical prognosis with a mean survival time of < 5 months.²⁶ As such, these patients have not yet achieved sufficient benefit from conventional therapeutic options, such as surgical resection, systemic chemotherapy, and radiotherapy. With our neurosurgical colleague, Andrew Sloan, MD, and other co-investigators from the company and at the Cleveland Clinic, we have studied the application of minimally invasive tumor ablation techniques for treatment of such aggressive recurrent tumors. Laser interstitial thermal therapy (LITT) is one such technique that has been applied for treatment of patients with a recurrent GBM in the past.²⁷ However, minimally invasive ablation techniques are often limited by the inability of the surgeon to monitor the exact location and extent of the ablated region. This is particularly important as one may fail to ablate the entire tumor or produce damage to the normal brain tissue that could drastically degrade the quality of life following the therapy. Thus, one desires the development of systems to monitor the ablation of a brain tumor in real time during ablation procedure. The AutoLITT system, developed by Monteris Medical Inc. (Winnipeg, Canada), enables application of LITT for GBMs with intra-operative feedback from MR thermotherapy to provide estimation of ablated tissue in real-time during the procedure. We worked with the company to implement real-time MR-guided thermotherapy to support the minimally invasive insertion of a laser probe into brain tumor followed by laser heat ablation to kill the lesion all under MRI guidance. AutoLITT was applied at UH to treat several patients with a recurrent GBM as part of a multicenter Phase 1 evaluation.

Conclusion

This review provides a sample of our neurologically important MR research projects currently ongoing in the Case Center for Imaging Research in the Department of Radiology. Other work of interest extends beyond the scope of this review, including that of our colleague, Jean Tkatch, PhD, who is evaluating novel techniques in the arena of functional MRI based on the use of an interface, similar to a modern computer game controller, and applying rapid image reconstruction methods to provide greatly improved data acquisition in functional settings using aspects of parallel imaging. With these and other contributions to the development of innovative MR imaging techniques, we are able to offer better care to the neurological patient at the UH Neurological Institute.

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Authors



Gurpreet S. Sandhu, MBBS

Department of Radiology
University Hospitals Case Medical Center
216-844-8074
Gurpreet.Sandhu@UHhospitals.org



Mark Griswold, PhD

Departments of Radiology, Physics, and
Biomedical Engineering
University Hospitals Case Medical Center
216-844-8085
Mark.Griswold@UHhospitals.org



Jeffrey L. Duerk, PhD

Departments of Radiology and
Biomedical Engineering
University Hospitals Case Medical Center
216-844-7794
Jeff.Duerk@UHhospitals.org



Jeffrey L. Sunshine, MD, PhD

Departments of Radiology, Neurology,
and Neurological Surgery
University Hospitals Case Medical Center
216-844-3061
Jeffrey.Sunshine@UHhospitals.org

The Neurological Intermediate Care Unit at UH: A New Option for Patient Care

By
Lori Mertz, CNP

Over the past decade advancement in medical care, an aging population, and an interest in providing quality patient care has led to an interest in finding better and more efficient strategies for providing inpatient care. A review of the literature addresses alternatives for intensive care unit (ICU) management of patients with less critical needs and finds intermediate care areas as one option. The advanced nursing practice at UH has seen the promising effects of offering this care to patients with neurological injury and disease.

A Change in Patient Demographics and Numbers

Over the past four years, the number of patients admitted to UH with a neurologic diagnosis has increased approximately 25% (Figures 1 and 2). This increase in volume has provided challenges, especially in acute care areas, such as the postanesthesia care unit (PACU) and the neurologic ICU known as the Neuroscience Unit (NSU). The inability to move patients with less acute needs out of the NSU can have a domino effect, preventing PACU patients from transferring out of the PACU and into the NSU, leading to overnight boarding of patients in the PACU. Additionally, a lack of ICU beds limits admissions and can result in diversion of emergency room patients.

Prior to the development of the Neurologic Intermediate Care Unit (NIU), the 14-bed NSU struggled to provide a sufficient number of monitoring beds for patients. Patients were boarded in cardiac, medical, and surgical ICUs and multiple telemetry units throughout the hospital. Members of the neurology and neurosurgery team were challenged with managing patients in multiple geographic locations as well as providing for the teaching needs of staff in multiple units less familiar with the care of neuroscience patients. With the emergence of tissue plasminogen activator (tPA) for acute stroke and the numbers of patients with more intensive monitoring needs, the challenge was magnified. To provide the specialized care these patients required, a better solution was clearly needed.

Responding to Patient Needs

A task force with representatives from the University Hospitals Neurological Institute and the Department of Medical and Surgical Nursing began to strategize to provide care for the increasing number of patients. On evaluating the care required, it was noted that many patients required more care than the nursing floor was able to provide, but many did not require the level of intensive care provided by the NSU. Determining a patient population that required an intermediate level of care and localizing these patients to a geographic area with specialized nursing care seemed to be a solution.

Design of the NIU was a collaborative venture. Integral members of the team included the nurse manager of the neurologic floor, nurse manager of the neuroscience ICU, clinical nurse specialist of the UH Neurological Institute, and administrative and physician members of the UH Neurological Institute. Additional support was provided from ancillary departments and other nursing specialties.

A patient population appropriate for the unit was determined. Patients admitted to the NIU are required to have a primary neurologic diagnosis. Patients may be admitted from the emergency room or PACU, transferred from another facility, or admitted directly. Some examples of NIU admission diagnoses are patients with acute brain attack, patients with stable stroke requiring frequent monitoring, and patients requiring frequent nursing interventions or more intensive monitoring than floor nursing is able to provide. Other neurologic patients transferred to the NIU include postoperative neurosurgery patients, such as patients who have had anterior cervical discectomy and fusion (ACDF), carotid endarterectomy, or tumor resection; patients transitioning out of the NSU after subarachnoid hemorrhage; and patients who have undergone interventional procedures, such as coiling or stenting.

Growth from Previous Year				
	2006	2007	2008	2009 Q3 Annualized
Neurology total	7.8%	0.0%	-0.9%	8.5%
Neurosurgery total	-9.5%	33.1%	29.3%	10.7%
NEUROLOGY/ NEUROSURGERY	3.8%	6.7%	6.7%	9.2%

Figure 1: Adult neurology and neurosurgery annual growth: 2006-2009 third-quarter (Q3) growth per year.

2006-2009 Q3 Annualized Growth	
Neurology total	7.5%
Neurosurgery total	90.4%
NEUROLOGY/ NEUROSURGERY	24.4%

Figure 2: Adult neurology and neurosurgery annual growth: 2006-2009 third-quarter (Q3) annualized growth.

A policy and procedure was developed that delineates the scope of practice and care of the NIU patient. Admission criteria, exclusion criteria, NIU standards, and telemetry standards were developed for the NIU. Patients may be admitted as intermediate care patients who require monitoring and assessment every two hours or as telemetry patients who have less intensive nursing care needs but require cardiac monitoring. Exclusion criteria for the NIU includes patients who require intracranial pressure monitoring, mechanical ventilation, unstable telemetry or EKG changes, mental status changes requiring hourly neurological checks, or patients in status epilepticus. These patients are better treated in the NSU.

To help facilitate the availability of beds on the neurologic floor, neurosurgery spine patients were relocated from the neurologic floor to the orthopaedic spine floor. Nurses on the orthopaedic floor had been caring for orthopaedic spine surgery patients and were familiar with the needs of these patients. The relocation of neurosurgical spine patients from the neurologic floor to the orthopaedic spine floor helped decrease the numbers of patients utilizing beds on the neurologic floor and potentially opened beds for patients awaiting transfer from the NSU, PACU, emergency room, and outside facilities as well as direct admits.

NEUROSCIENCE NURSING PRACTICE CENTER

Erin Supan, CNS
Director

The Neuroscience Nursing Practice Center is dedicated to the development and implementation of best practices in neuroscience nursing. Our mission is to improve patients' long-term outcomes by providing care according to the most advanced protocols based on the latest research findings, offering highly personalized care, and maximizing efficiencies during every stage of treatment. These strategies are implemented throughout the continuum of patient care from first diagnosis, through treatment, to follow-up visits in any location in which the patient is seen. Our program is based on three core principles:

- Evidence-based nursing practice
- Patient-centered care
- Relationship-based nursing

The Neuroscience Nursing Practice Center upholds a strong culture of patient-centered care that extends across every discipline, including rehabilitation, pharmacy, social work, nutrition, and medical services.

A physical area was designated adjacent to the neurologic floor. The area is composed of 10 private rooms, reassigned from the neurologic floor. This area can accommodate a total of nine intermediate care patients for staffing purposes or 10 patients if utilized as a combination of telemetry and step-down patient acuity. The rooms were supplied with equipment appropriate for patients with more acute needs. New monitors with the capability for telemetry and more intensive physiologic monitoring, such as arterial blood pressure monitoring and central venous pressure monitoring, were purchased as were transport monitors and emergency equipment, including a defibrillator and emergency cart designated to the NIU.

Unit staffing design allowed for 7.6 nursing full-time equivalents (FTEs) and 2.0 ancillary personnel FTEs. Patient ratios are one RN to three NIU patients, who require more intensive monitoring, or one RN to four telemetry patients, who have less acute nursing care needs. Nurses with experience working on the neurologic floor were given the opportunity to become part of the NIU staff. These nurses required additional education in cardiac monitoring and familiarity with acute patient diagnosis. An education course was developed, utilizing clinical nurse specialists from the UH Neurological Institute and nursing colleagues from the surgical and cardiac ICUs. Courses were provided in EKG, cardiomyopathy, congestive heart failure, neurologic diagnoses, and pharmacology. The design included time with a preceptor in the NSU, caring for patients who would be potential NIU patients.

The initial nursing staff included a combination of experienced neurologic floor nurses, who transitioned to the NIU, and experienced ICU nurses temporarily assigned to the NIU. The experienced ICU nurses served as a resource for the NIU nurses.

Currently, patients in the NIU are managed by the neurology and neurosurgical teams. The neurosurgical team includes a nurse practitioner model to facilitate the patient plan of care, assist with patient and family communication, and contribute to nursing staff education. This model assists hospital throughout of the neurosurgical population and facilitates communication and rapid evaluation of patient problems while the neurosurgical teams are in the operating rooms.

Looking Ahead

Future plans for the NIU include transitioning additional patient populations including endovascular interventional patients who have undergone coiling and stenting; patients with more intensive physiologic monitoring needs, such as arterial lines and central venous pressure monitoring; and patients on select medication infusions requiring frequent monitoring.

No CME credit offered for this article.



Author

Lori Mertz, CNP

Certified Nurse Practitioner
UH Neurological Institute
University Hospitals Case Medical Center
216-844-0532
Lori.Mertz@UHhospitals.org



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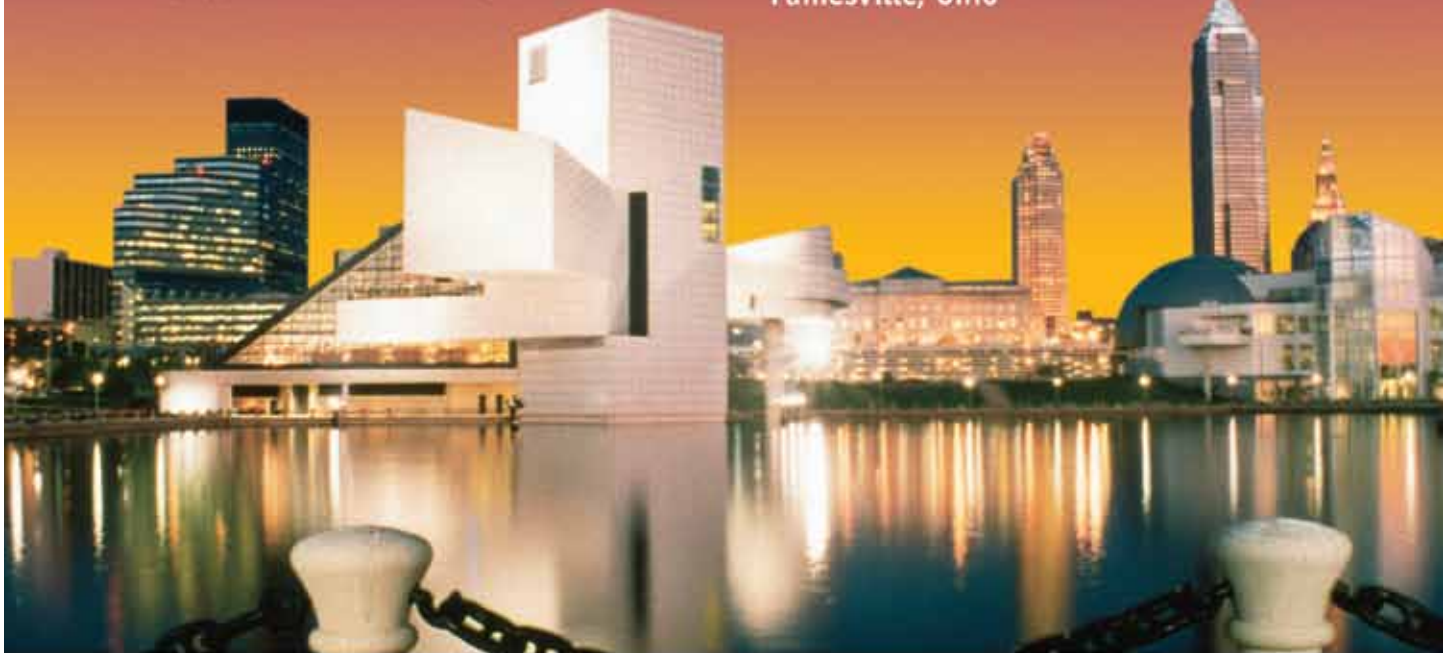
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