



# Abstracts/Poster Presentations

## ABSTRACT 1

### **HYPONATREMIA-RELATED FOCAL CEREBRAL EDEMA, A MIMIC OF WORSENING CEREBRAL EDEMA DUE TO INTRACEREBRAL HEMORRHAGE**

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**Background.** In the natural course of intracranial hematoma resolution, mass effect and edema peak at 4 to 5 days, after which there is gradual resolution and resorption of blood products over several weeks. We report the case of a patient with intracranial hemorrhage whose mass effect became worse 21 days after onset. Subsequent contrast-enhanced CT did not confirm initial suspicion of an underlying structural lesion. The persistent focal edema was related to diuretic-induced hyponatremia.

**Case report.** A 73-year-old right-handed woman presented with headache, dysarthria, and left-sided weakness. She was hypertensive on admission. She was not taking antithrombotic medication, and there was no history of preceding trauma. Examination revealed an NIH Stroke Scale score of 13. Brain CT showed a right frontoparietal hematoma. Following an initial improvement, her neurologic condition deteriorated 21 days after admission.

**Discussion.** Hyponatremia causes cerebral edema by transfer of water into brain cells across an osmolar gradient. Neurologic symptoms are more likely if this occurs acutely. While the edema usually is generalized, it may be focal in the setting of an underlying acute structural lesion. The pathogenesis, diagnosis, and management of hyponatremia in the neurologic intensive care unit is reviewed.

**Conclusion.** Hyponatremia, especially in the elderly, is prevalent in the neurologic intensive care unit and is a predictor of mortality and morbidity. Acute hyponatremia may cause focal cerebral edema that may alter the natural course of the underlying disease process and complicate the diagnosis. Care must be taken to prevent hyponatremia in at-risk groups, and to manage existing hyponatremia promptly.

## ABSTRACT 2

### **SUBOCCIPITAL CRANIECTOMY FOR ACUTE CEREBELLAR ISCHEMIC STROKE**

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**Introduction.** Suboccipital craniectomy may benefit patients with large cerebellar stroke. Patient selection and tim-

ing of surgery remain controversial. We sought to determine if surgery could improve clinical outcomes after stroke.

**Methods.** We performed a chart review of patients admitted with acute cerebellar infarction from May 1998 to July 2002 who underwent suboccipital craniectomy. Four patients were identified. Their presentation, CT findings, time to surgery, postoperative condition, and disposition were evaluated.

**Results.** Patient ages were 25, 50, 68, and 70 years. All patients had large cerebellar stroke with obstructive hydrocephalus. One patient had clinical signs of brainstem compression with nonreactive pupils; the other three had intact brainstem reflexes. Neurologic deterioration occurred in all four patients within 2 days. The mean time to surgery after stroke was  $2.5 \pm 1.7$  days. All patients underwent posterior fossa craniectomy with duroplasty and ventriculostomy. Indications for surgery were clinical deterioration with CT signs of brainstem compression. Three of the four patients had substantial improvement on their neurologic exam following surgery (initial NIH Stroke Scale [NIHSS] score of  $13 \pm 8$  vs postoperative NIHSS score of  $4 \pm 3$ ;  $P = .13$ ). The fourth patient (aged 70 years) initially presented comatose with unreactive pupils and remained unchanged postoperatively, after which the family withdrew support. Of the remaining three patients, two were discharged home and one to an acute rehabilitation facility.

**Conclusion.** Patients with neurologic deterioration after acute cerebellar stroke and findings of brainstem compression may benefit from suboccipital craniectomy. Patients with early signs of brainstem compromise may have poorer outcomes. Specific criteria are needed for the selection of patients who might benefit from this procedure.

## ABSTRACT 3

### **THE FEASIBILITY AND SAFETY OF MILD BRAIN HYPOTHERMIA OBTAINED BY LOCAL SURFACE COOLING IN ACUTE STROKE**

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**Background.** Hypothermia is known to protect the brain from ischemic injury. However, systemic surface cooling is associated with various adverse effects that can heavily burden stroke patients, most of whom are elderly. The purpose of this study is to test the feasibility, safety, and effectiveness of local surface cooling as a tool for acute stroke therapy.

**Methods.** Nine patients with acute embolic stroke and

NIH Stroke Scale scores exceeding 10 (mean age, 65 years) were subjected to local surface cooling 3 to 12 hours after stroke onset. A helmet-type apparatus was attached to the head and neck of patients, and cooling was continued for 3 to 7 days without anesthesia. Temperatures were measured at the axilla, bladder, tympanic membrane, and internal jugular bulb.

**Results.** Surface cooling was performed successfully in all patients. During the surface cooling, the axillary and bladder temperatures remained unchanged while the tympanic and jugular bulb temperatures were reduced by 1.8 and 0.8 °C, respectively, as compared with the axillary temperature. None of the patients experienced serious adverse effects, although serum CPK levels rose in all patients as a result of mild shivering, and skin erosion and infections occurred in 2 and 3 patients, respectively.

**Conclusion.** Mild brain hypothermia can be achieved feasibly and safely by local surface cooling. While the neuroprotective effect of local cooling may be less powerful than that of systemic cooling, local cooling may be clinically more useful because of its safety and feasibility.

#### ABSTRACT 4

##### THE DIVERTER, A NOVEL PERMANENT ARTERIAL DIVERSION DEVICE

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**Purpose.** A novel permanent filtering device (the Diverter™, MindGuard Ltd.), indicated for the prevention of embolic stroke in high-risk patients with proximal sources of embolism, was tested for safety and performance. Self-expandable Diverter endovascular prostheses are intended for bilateral implantation in the two carotid bifurcations from the external carotid artery (ECA) to the common carotid artery, by means of a standard percutaneous endovascular procedure. The part of the Diverter facing the internal carotid artery (ICA) orifice is designed to prevent embolic material from entering the ICA, diverting it to the ECA.

**Methods.** Sixteen Diverter devices were percutaneously implanted in the swine iliofemoral bifurcation. Harvesting was performed at 3, 10, and 17 weeks after implantation. The patency of the Diverter-guarded ostium was calculated by morphometrical software. For an additional 10 implanted Diverters, animals were injected with bromodeoxyuridine (BrdU; 40 mg/kg). Immunohistolabeling was performed and the tissue proliferation rate at the filtering part was assessed.

**Results.** No discernable stenosis was noted. Microscopy and morphometry showed 99.0% ± 1.0%, 91.8% ± 10.6%, and 93.3% ± 8.8% filtering area patency at 3, 10, and 17 weeks, respectively. The percentage of BrdU-stained cells, which corresponds to proliferation rate, was 18.7% ± 7.3%, 12.8% ± 4.6%, and 0.7% ± 0.6% after 1, 3, and 24 weeks, respectively. Proliferation thus reaches steady state within 6 months.

**Conclusion.** Permanent embolic filtration with the Diverter device is feasible and safe in the animal model.

#### ABSTRACT 5

##### FATAL ARRHYTHMIA IN AN ANXIOUS PATIENT DURING RECOVERY FROM LATERAL MEDULLARY INFARCTION

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**Background.** Sudden unexpected death has been reported in the first 2 weeks following lateral medullary infarction (LMI), during the time when the patient is recovering from the stroke and is medically stable. A number of mechanisms have been proposed, including respiratory failure and cardiac-related arrest, but a causal relationship has not been fully established. Ischemic lesions in the solitary tract nuclei were speculated to have caused autonomic instability, causing fatal cardiac arrhythmia, among patients who suffered acute heart failure from primary cardiac disorders. Patients with anxiety disorder have been shown to have abnormal autonomic flexibility.

**Case report.** A patient with generalized anxiety disorder and no previous cardiac dysfunction suffered a right LMI and died of fatal cardiac arrhythmia 21 days after the stroke. Limited autopsy did not show pulmonary embolism or acute myocardial infarction. The only abnormality on the ECG was a new prolonged QT noted on admission. There was no significant arrhythmia noted on telemetry during the acute phase of the stroke. Pre-existing autonomic abnormality due to the patient's anxiety disorder may have had additive effects with an acute lesion in the medullary autonomic centers, together predisposing the patient to arrhythmia and causing sudden unexpected death in an otherwise benign course of LMI. Further investigation is necessary to explain the time interval between the onset of stroke and the occurrence of fatal arrhythmia.

#### ABSTRACT 6

##### MOTOR, BEHAVIORAL, AND COGNITIVE CHANGES IN PATIENTS WITH THALAMIC LESIONS

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**Objective/Background.** To study the motor, behavioral, and cognitive changes in patients with thalamic lesions. The basal ganglia interacts with the motor loop and complex loop and funnels these motor, behavioral, and cognitive changes via the thalamus to the frontal cortex. The role of the thalamus requires an in-depth study.

**Design.** Cross-sectional descriptive case study.

**Methods/Results.** We analyzed 20 consecutive patients with CT/MRI-proven exclusive thalamic stroke seen from January 1998 to June 2003. The patients were 35 to 80 years old. All had undergone Folstein Mini-Mental State Examination and detailed lobar function testing. They were screened for stroke risk factors. There were 10 hemorrhages and 10 infarctions, with the mean age being 59 years. Motor signs alone occurred in 80% of the patients, with hemiplegia in 3 patients, hemiparesis in 12 patients,

and ocular signs in 1 patient. Behavioral and cognitive changes were seen in 40% of the patients, with aggression and confabulation in 2 patients (10%), speech and language disturbances in 4 patients (20%), and memory disturbance in 2 patients (10%).

**Conclusion.** Among thalamic stroke patients, 80% showed motor signs and 40% showed behavioral and cognitive changes. Behavioral and cognitive changes were not seen alone.

## ABSTRACT 7

### REVERSAL OF LOCKED-IN SYNDROME WITH ANTICOAGULATION, INDUCED HYPERTENSION, AND INTRAVENOUS t-PA

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**Background.** Spontaneous cervical artery dissection accounts for 10% to 25% of ischemic strokes in young and middle-aged patients. Anticoagulation with intravenous heparin is the currently recommended treatment for acute dissection, although the validity of this therapy has never been proven in randomized clinical trials. Acute management of posterior circulation ischemia, including anticoagulation, induction of hypertension, Trendelenburg positioning, and intravenous and intra-arterial thrombolysis, is a frequently debated area and still requires standardization.

**Case report.** We describe the case of a 39-year-old man who developed recurrent pontine ischemia due to right vertebral artery dissection producing a locked-in state that resolved with the combination of anticoagulation, Trendelenburg positioning, induced hypertension, and intravenous t-PA. None of the treatment modalities applied to our patient have been demonstrated to be safe and effective in the setting of arterial dissection. In fact, administration of intravenous t-PA outside the 3-hour window and in addition to anticoagulation with intravenous heparin and induction of hypertension (systolic blood pressure > 185 mm Hg) was contraindicated. In this scenario, the benefits outweighed the tremendous risk of hemorrhage: the patient recovered almost completely with a residual minimal left abducens palsy 2 weeks after symptom onset.

**Conclusion.** Good outcome can be achieved with the use of intravenous thrombolysis in combination with anticoagulation, induced hypertension, and Trendelenburg positioning in severe posterior circulation ischemia due to vertebral dissection.

## ABSTRACT 8

### DANTROLENE REDUCES THE THRESHOLD AND GAIN FOR SHIVERING

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**Background.** How dantrolene alters thermoregulatory control remains unknown. We aimed to determine whether

dantrolene alters the thresholds for sweating, vasoconstriction, and shivering, or the gain of shivering.

**Methods.** *Study 1*—Nine volunteers (mean age,  $25 \pm 4$  years; mean height,  $172 \pm 9$  cm; mean weight,  $66 \pm 11$  kg) were randomly assigned to control or dantrolene. On dantrolene day, they received 5 mg/kg/hr dantrolene for 30 minutes and 0.05 mg/kg/hr for 5 hours. Skin and core temperatures were increased with forced-air and circulating-water blankets to provoke sweating and were subsequently reduced to elicit vasoconstriction and shivering. *Study 2*—Seven healthy male volunteers (mean age,  $27 \pm 7$  years; mean height,  $178 \pm 10$  cm; mean weight,  $72 \pm 10$  kg) were given cold ( $3 \text{ }^{\circ}\text{C}$ ) Ringer's solution intravenously on randomly assigned control or dantrolene days. On dantrolene day, they received 5 mg/kg/hr dantrolene for 30 minutes and 0.05 mg/kg/hr for 5 hours. Cooling was started 1 hour after dantrolene. A sustained increase in oxygen consumption identified the shivering threshold. The slope of a regression between core temperature and oxygen consumption identified shivering gain. Results are presented as mean  $\pm$  SD;  $P < .05$  is the threshold for statistical significance.

**Results.** Confounding factors were comparable between the study days. Dantrolene did not alter the sweating or vasoconstriction thresholds, but it reduced the shivering threshold by  $0.3 \pm 0.3 \text{ }^{\circ}\text{C}$  and increased the vasoconstriction-to-shivering range to  $1.2 \pm 0.2 \text{ }^{\circ}\text{C}$ . In study 2, dantrolene reduced the shivering threshold by  $0.4 \pm 0.3 \text{ }^{\circ}\text{C}$  and the gain of shivering by  $130 \pm 154 \text{ mL/min/}^{\circ}\text{C}$ .

**Conclusion.** Although dantrolene reduced the threshold and gain of shivering, reductions were small and unlikely to explain the reported efficacy of this drug for treatment of potentially lethal nonspecific hyperthermia.

## ABSTRACT 9

### PREDICTING OUTCOME AFTER SUBARACHNOID HEMORRHAGE: COMPARISON OF DIFFERENT GRADING SYSTEMS

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**Introduction.** Predicting outcome after aneurysmal subarachnoid hemorrhage (SAH) is often difficult. Common grading systems include the Hunt-Hess Scale, Fisher grade, and Glasgow Coma Scale. Recently, a new 5-point scale was developed that includes patient age, aneurysm size, Hunt-Hess score, Fisher grade, and size and location of aneurysm (Ogilvy score). We previously demonstrated that the NIH Stroke Scale (NIHSS) score, originally developed for ischemic stroke, is also predictive of outcome after intracerebral hemorrhage. The NIHSS score has not been studied in SAH. In a retrospective study of 76 SAH patients, we tried to determine which grading system best predicted outcome.

**Methods.** A retrospective analysis of 76 patients with aneurysmal SAH admitted to the neurologic intensive care unit between January 2000 and July 2002 was conducted documenting demographic data, aneurysm size, and admis-

sion Hunt-Hess score, Fisher grade, Glasgow Coma Scale score, Ogilvy score, and NIHSS score. Outcomes data included Glasgow Outcome Score (GOS), modified Rankin scale score, and Functional Independence Score on discharge (dichotomized as good or poor outcome). Spearman correlation coefficient and logistic regression were used to assess relationships.

**Results.** The average patient age was  $55.3 \pm 14.2$  years. On admission, the mean Hunt-Hess score was  $2.6 \pm 1.3$ , the mean Fisher grade was  $2.8 \pm 1.1$ , the mean Glasgow Coma Scale score was  $11.8 \pm 4.1$ , and the mean Ogilvy score was  $1.8 \pm 1.2$ . The mean NIHSS score was  $7.4 \pm 10.0$ . Overall, the mortality rate was 18%. A good outcome occurred in 42 patients (55.2%). Using the Spearman correlation coefficient, the following correlations were found with discharge GOS (from best to worst correlation):

- NIHSS score,  $-0.531$  (95% CI,  $-0.73$  to  $-0.34$ ;  $P < .001$ )
- Hunt-Hess score,  $-0.523$  (95% CI,  $-0.72$  to  $-0.33$ ;  $P < .001$ )
- Ogilvy score,  $-0.431$  (95% CI,  $-0.64$  to  $-0.10$ ;  $P < .001$ )
- Fisher grade,  $-0.319$  (95% CI,  $-0.54$  to  $-0.10$ ;  $P < .001$ ).

In a subgroup of 42 patients with cerebral vasospasm, the following correlations were found:

- NIHSS score,  $-0.383$  (95% CI,  $-0.68$  to  $-0.09$ ;  $P = .012$ )
- Ogilvy score,  $-0.336$  (95% CI,  $-0.64$  to  $-0.03$ ;  $P = .03$ )
- Fisher grade,  $-0.320$  (95% CI,  $-0.64$  to  $-0.03$ ;  $P = .039$ )
- Hunt-Hess score,  $-0.306$  (95% CI,  $-0.61$  to  $-0.02$ ;  $P = .039$ ).

**Conclusion.** The admission NIHSS score is predictive of outcome following SAH and is at least as good as, if not better than, other more commonly used grading systems. The Ogilvy score is also predictive of outcome but statistically is not significantly better than the Hunt-Hess score or Fisher grade alone. Larger studies are needed to determine the scale that best predicts outcome.

## ABSTRACT 10

### INTEGRATIVE MONITORING METHODS IN NEUROCRITICAL CARE

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**Introduction.** Neurocritical care is a relatively new field of medicine focusing on the management of critically ill

patients with neurologic disease. One of the main functions of the neurologic intensive care unit (NICU) is to provide close monitoring of neurologic status. Most NICUs are equipped with a large range of sophisticated monitoring equipment that generates enormous volumes of information, mainly about cardiopulmonary status. Surprisingly, the central nervous system is largely neglected, even in patients with severe neurologic injuries in whom sedation and mechanical ventilation make clinical examination difficult. This leaves the neurointensivist with much cardiopulmonary data that is nearly impossible to interpret and analyze online, and with insufficient neurologic data. Despite advances in computer technology, an ideal bedside neuromonitoring system that collects, organizes, analyzes, and trends both cardiopulmonary and neurologic data is not available.

**Design.** At The Cleveland Clinic Foundation, we have developed a new monitoring system for the NICU that integrates cardiopulmonary monitoring modalities (blood pressure, heart rate, respiratory rate, oxygen saturation) and neuromonitoring modalities (intracranial pressure, cerebral perfusion pressure, brain tissue oxygenation, continuous EEG, and evoked potentials) into one bedside monitor. These parameters are transferred through an interface from the standard bedside Marquette monitors to a central workstation where the system extracts salient features from the raw waveforms, analyzes them, and displays them on the screen. Annotations that may affect the integrity of test results (for example, patient movement, suctioning, increasing or dosage of sedatives) can also be entered, and the system can be tailored to meet a particular patient's needs. The system can be programmed to page the clinician when a potential problem is detected and to transmit the data over a hospital network.

**Conclusion.** The data acquisition and analysis offered by this system can provide a meaningful overall assessment of a patient's condition and may give an early indication of potentially harmful secondary insults before irreversible brain damage occurs. We believe this approach of integrating multiple physiologic parameters into one user-friendly system that collects, organizes, analyzes, and trends data will revolutionize the delivery of neurocritical care.