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NEUROCRITICAL CARE SOCIETY 5TH ANNUAL MEETING NOVEMBER 2-NOVEMBER 3, 2007

ABSTRACTS: Oral & Poster Presentations

Rio Suite Hotel & Casino Las Vegas, Nevada

Neurocritical Care Society
5th Annual Meeting

Oral Presentations
**Friday, November 2, 2007 1:15-2:45 p.m.*
***Saturday, November 3, 2007 9:00-10:30 a.m.*

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4	Intracerebral hemorrhage and BOXes elicit a robust inflammatory response in the brain.	G. Pyne-Geithman*
5	Defining Vasospasm after Subarachnoid Hemorrhage: Clinical relevance of Symptomatic Vasospasm, Delayed Cerebral Ischemia, Angiographic Vasospasm and Transcranial Doppler Vasospasm	J. Frontera*
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9	Temperature Manipulation Alters Early EEG Bursting after Cardiac Arrest in Rats	X. Jia**
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Poster Presentations

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18	Complications of Hypertensive Hypervolemic Therapy for Symptomatic Vasospasm	J. Frontera
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22	Age of Packed Red Cells Did Not Affect Acute Outcomes After SAH	A. Naidech
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24	Human APOE 3/3 genotype offers in neuroprotection in a murine model of intracerebral hemorrhage	M. James
25	Impact Of Statins On Validation Of ICH Mortality Prediction Models	N. Naval
26	An Association Of Prior Statin Use With Decreased Perihematomal Edema	J. Carhuapoma
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131	Electroencephalography In Critically Ill Patients	S. Mittal
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145	Eligibility For The Surgical Trial In Intracerebral Hemorrhage II Study In A Population-Based Cohort	O. Adeoye
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149	Pathologic Description Of Wingspan Stent In The Setting Of Acute Ischemic Stroke	S. Samuel
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Oral Presentation 1

A COMPARISON OF THREE RADIOGRAPHIC SCALES FOR THE PREDICTION OF DELAYED ISCHEMIA AND PROGNOSIS FOLLOWING SUBARACHNOID HEMORRHAGE

Andreas Kramer[1], Bart Nathan[2], Darryl Gress[2], Michael Hehir[2], Neil Kassell[2], Aaron Dumont[2], Thomas Bleck[3]
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Introduction:
Delayed cerebral ischemia (DCI) is an important cause of morbidity and mortality following aneurysmal subarachnoid hemorrhage. Timely recognition and treatment are required for optimal outcomes to be achieved. The amount and distribution of blood seen on initial computed tomography (CT) scans, often expressed using various radiographic scales, are helpful at estimating the risk of developing DCI. We compared the commonly used Fisher scale with two newer radiographic systems for the prediction of the following complications: symptomatic vasospasm, delayed infarction and poor outcome (defined as a Glasgow Outcome Scale of 1-3).

Methods:
This was a single-center, retrospective cohort study involving 271 consecutive patients with a ruptured cerebral aneurysm. Without knowledge of subsequent events, admission CT scans were each assigned scores using three different grading schemes: the Fisher, Modified Fisher and Claassen scales. For each of the scales, the relationship between an increasing score and the risk of later complications was assessed in univariate and multiple logistic regression analysis.

Results:
With the Fisher scale, the risk of complications was relatively high when the score was 3, but not for other scores. In contrast, using the other scales, there was a more linear relationship between a rising score and the frequency of complications. This was particularly true for the Modified Fisher scale, which was the only system where each stepwise increase was associated with an escalating risk of developing each complication. Kappa scores measuring interobserver variability among four CT readers were also slightly better with the newer scales.

Conclusions:
Although the Modified Fisher and Claassen scales have yet to be prospectively validated, our findings suggest that their clinical performance is superior to that of the Fisher scale. We favour the use of the Modified Fisher Scale.

References:
1. Fisher CM, Kistler JP, Davis JM: Relation of cerebral vasospasm to subarachnoid hemorrhage visualized by computerized tomographic scanning. Neurosurgery 6: 1-9, 1980.
2. Claassen J, Bernardini GL, Kreiter K, Bates J, Du YE, Copeland D, Connolly ES, Mayer SA: Effect of cisternal and ventricular blood on risk of delayed cerebral ischemia after subarachnoid hemnorrhage: The Fisher Scale revisited. Stroke 32: 2012-2020, 2001.
3. Frontera JA, Claassen J, Schmidt JM, Wartenberg KE, Temes R, Connolly ES, Macdonald RL, Mayer SA. Prediction of symptomatic vasospasm after subarachnoid hemorrhage: the modified fisher scale. Neurosurgery 59: 21-27, 2006.
4. Klimo P Jr, Schmidt RH: Computed tomography grading schemes used to predict cerebral vasospasm after aneurysmal subarachnoid hemorrhage: a historical review. Neurosurg Focus 15: 21(3): E5, 2006.

Financial Support: None

Oral Presentation 2

CONIVAPTAN FOR EUVOLEMIC HYPONATREMIA IN THE NEUROCRITICAL CARE UNIT

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Introduction:
Conivaptan is the first vasopressin antagonist to be FDA-approved for the treatment of euvolemic hyponatremia, a common complication in neurointensive care patients.

Methods:
As part of a medication usage evaluation protocol, conivaptan was administered to 16 patients with euvolemic hyponatremia in the neurointensive care unit before allowing its addition to the formulary of a tertiary subspecialty teaching hospital. End points evaluated included time to Na increase of ≥6 meq/L; and incidences of rapid Na overcorrection, infusion site reactions, or other adverse events.

Results:
An increase of ≥6 meq/L in Na was reached in 14/16 patients studied. Among those who reached this goal, the average treatment time to goal was 14.5 hours. 5/16 patients had an infusion site reaction that required an IV change. No patients experienced a rapid overcorrection or any other adverse events.

Conclusions:
Conivaptan was safe and effective in this small series of neurointensive care patients. A series of this type has not previously been published. Further studies would be needed to compare conivaptan to hypertonic saline.

References: None

Financial Support: None

Oral Presentation 3

HYPEROSMOLAR HYPOTHERMIC NORMOGLYCEMIA (H2N) FOR PREVENTING CEREBRAL EDEMA AFTER LARGE HEMISPHERIC INFARCTION -A FEASIBILITY STUDY

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Introduction:
Large hemispheric infarction carries a mortality rate of 40–80%. Despite the introduction of decompressive hemicraniectomy, the medical management of brain edema after large hemispheric infarction is still not satisfactory.

Methods:
We treated 25 patients with large hemispheric infarctions between August 2006 and June 2007 with the combination of insulin infusion (target glucose 4.6-6.1 mmol/L), mild hypothermia (33-35˚C) using a surface cooling or intravascular heat exchange device, and hypertonic saline (3% sodium chloride acetate) at a rate 1ml/kg (goal sodium 150-155 mmol/L) within 72 hours of symptom onset. Primary outcome was progression or evolution of the midline shift on computed tomography (CT). Secondary outcome measures were modified Rankin Scale (mRS) at 3 months and complications.

Results:
Of the 25 patients 12 had right-sided infarctions and median age was 68 (range 39-83) years. Baseline NIHSS was 17.2±5.3. H2N was started on average on the day 1 of symptom onset (range 0-4 days); median duration of treatment was 9 (range 2-19) days. Mean septal midline shift was 1.8±3.2 mm on admission; peaked at 7.8±5.7 mm, and was 1.7±1.8 mm on discontinuation of the protocol. At 3 months, 14 patients had died; support had been actively withdrawn in 10 cases. The mRS was 3 in 2 patients, 4 in 4 patients and 5 in 5 patients. Complications included pulmonary edema (n=17), aspiration and ventilator-acquired pneumonia (n=8), tracheobronchitis (n=9), sepsis and septic shock (n=9), urinary tract infection (n=4), atrial fibrillation with rapid ventricular response (n=7), acute renal failure (n=5), coagulopathy (n=4), thrombocytopenia (n=10), leucopenia (n=1), anemia (n=8), and paralytic ileus (n=2).

Conclusions:
The combination of mild hypothermia (33-35˚C), infusion of hypertonic saline, and insulin infusion offers a feasible alternative strategy to minimize massive cerebral edema after large hemispheric infarction and needs to be studied in a standardized trial.

References: None

Financial Support: Stephan Mayer, MD is a consultant for and has Stock Options with Medivance, Inc.

Oral Presentation 4

INTRACEREBRAL HEMORRHAGE AND BOXES ELICIT A ROBUST INFLAMMATORY RESPONSE IN THE BRAIN.

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Introduction:

Bilirubin Oxidation products (BOXes) have been found in the CSF of patients with cerebral vasospasm after SAH, as well as in hematoma and perihematomal brain of animal models of ICH. We have found that BOXes are vasoactive, both *in vitro* and *in vivo*, and so wanted to investigate possible effects of BOXs on the inflammatory responses of the brain itself. Thromboxane B_2 is the stable metabolite of Thromboxane A_2, a potent inflammatory molecule and marker of inflammation in the brain, among other tissues.

Methods:

Control: animals were anaesthetized and perfused with 0.9% saline. The brains were removed and flash-frozen. *ICH*: 100 mL autologous blood was infused into the right cerebral cortex. The animal was allowed to recover for 24 hours, then the brain was harvested as for the control animal. *BOXes*: a cranial window was opened over the right cerebral hemisphere and 25 mL of 23 mM BOXes was applied directly to the surface of the brain. The animal was allowed to recover for 24 hours, then the brain was harvested as for the control animal. Contralateral cortex (from ICH and BOXes brains) was homogenized and assayed for Thromboxane B_2 using an EIA kit (Cayman Chemicals). Total protein was assayed using the BCA method (Pierce).

Results:

The ICH brain had significantly ($p<0.05$) increased levels (pg TXB_2/mg protein) of TXB_2 (21 ± 0.34) compared with control (17 ± 0.74), as expected from previously published data. However, the BOXes treated brain had even higher ($p<0.05$) levels of TXB_2 than either ICH or control brains (29 ± 1.6). The results indicate that BOXes induce TX production in the brain, in the absence of thrombin and other blood components normally present in hemorrhagic stroke.

Conclusions:

This suggests a role for BOXes in other complications (than vasospasm) following hemorrhagic stroke such as edema, inflammation and immune responses. It may also hint at a putative role for therapeutic use of inhibitors of TX production, such as COX-2 inhibitors.

References: None

Financial Support: None

Oral Presentation 5
DEFINING VASOSPASM AFTER SUBARACHNOID HEMORRHAGE: CLINICAL RELEVANCE OF SYMPTOMATIC VASOSPASM, DELAYED CEREBRAL ISCHEMIA, ANGIOGRAPHIC VASOSPASM AND TRANSCRANIAL DOPPLER VASOSPASM

Jennifer Frontera[1], Michael Schmidt[2], Andres Fernandez[2], Katja Wartenberg[2], Fred Rincon[2], Neeraj Badjatia[2], Augusto Parra[2], Stephan Mayer[2]
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Introduction:
Vasospasm is an important complication of subarachnoid hemorrhage (SAH) but is variably defined in the literature. We aimed to determine the clinical relevance of differing definitions of vasospasm.

Methods:
We studied spontaneous SAH patients and examined symptomatic vasospasm, defined as clinical deterioration deemed secondary to vasospasm after other causes were eliminated, delayed cerebral ischemia (DCI), defined as symptomatic vasospasm or infarct on CT due to vasospasm, angiographic spasm, as seen on digital subtraction angiography and transcranial Doppler (TCD) spasm defined as any mean flow velocity >120 cm/s. Logistic regression analysis was performed to calculate adjusted odds ratios (aOR) associated with each type of vasospasm and hospital complications (after controlling for ICU length of stay) and 3-month outcomes, as measured by the modified Rankin Scale (after adjusting for age, Hunt-Hess grade and aneurysm size).

Results:
Of 580 SAH patients, symptomatic vasospasm occurred in 16%, DCI in 21%, angiographic vasospasm in 31% and TCD spasm in 45%. DCI was associated with the most hospital complications (N=7) including arrhythmia, pulmonary edema, myocardial infarction, fever, pneumonia, blood stream infection (BSI) and cerebral edema (all $P<0.05$), compared to symptomatic spasm (N=4 complications: pulmonary edema, myocardial infarction, fever and BSI), angiographic spasm (N=1: intracerebral hemorrhage) and TCD spasm (N=1: BSI). Only DCI was independently associated with death or severe disability at 3 months (aOR 2.2, 95% CI 1.2-3.9, P=0.007).

Conclusions:
DCI is the most clinically relevant definition of vasospasm and is associated with more hospital complications and worse outcome at 3 months compared to symptomatic vasospasm, angiographic vasospasm or TCD defined vasospasm.

References: None

Financial Support: None

Oral Presentation 6

HHH THERAPY FOR TUBERCULOUS ARTERITIS: A PROSPECTIVE STUDY.

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Introduction:

Arteritis is a fairly common complication of Tuberculous Meningitis (TBM) and is associated with devastating neurologic deficits. No specific therapy is presently known for infective arteritis. Hypervolemia, hypertension and hemodilution (HHH) therapy is known to be of benefit in vasospasm secondary to subarachnoid hemorrhage.

Methods:

Patients diagnosed to have TB meningitis by clinical, CSF and imaging findings were evaluated for arteritis. Arteritis was recognized by recent focal neurologic deficits and corresponding focal hypodensities on brain CT. Patients with deficits of <96 hours were randomized after informed consent to HHH or conservative treatment. Exclusion criteria were: dense weakness (MRC 0/5); deficit duration > 4 days; tuberculoma; infarcts on CT without corresponding deficits. All patients were treated with four anti-TB drugs, Inj.dexamethasone and IV mannitol. HHH administered over 3-7 days consisted of: a) hypervolemia - with normal saline / albumin to CVP of 12-16 cm; b) hypertension – 20% higher mean blood pressure, induced by vasopressors; c) hemodilution to hematocrit 30-33%. Neurologic status and modified Rankin score at discharge were noted. Transcranial Doppler (TCD) was studied in some patients.

Results:

Six patients who received HHH were compared with 5 others who received conservative treatment (M:F::10:2; Age 25-49yrs). All had hemiparesis with power 1-3/5. GCS at admission was worse in HHH group (11 v/s 12.5). In the HHH group, 4/6 improved in motor power, and 4 in sensorium; 2 patients died. In the control group, 2/5 improved in motor power and 2 in sensorium; one died.

Conclusions:

HHH may be beneficial in infective arteritis secondary to TBM. Further studies with improved case selection and monitoring of cerebral circulation are indicated. This is the first study to attempt HHH therapy for arteritis in TBM.

References: None

Financial Support: None

Oral Presentation 7

A NOVEL APPROACH TO TREATMENT OF CEREBRAL EDEMA POST CARDIAC ARREST

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Introduction:

Neuroprotection following cardiac arrest remains an elusive goal despite advancement in resuscitation techniques. Reducing blood-brain barrier (BBB) disruption and subsequent vasogenic cerebral edema after return of spontaneous circulation (ROSC) may improve cardiac arrest outcomes. This study assessed the effect of BPZ-302 (a novel anti-edema therapeutic composed of a medium-weight, narrow, molecular range hydroxyethyl starch and Vitamin C in 7.5% hypertonic saline) on cerebral edema and BBB permeability following global hypoxia.

Methods:

Anesthetized one-week-old piglets underwent 30 min of hypoxia, 7 min of airway occlusion, and resuscitation. Cohorts were administered either BPZ-302 (n=5) loading dose of 8 ml/Kg or saline (n=5) 5 min post ROSC. A continuous infusion of BPZ-302 (0.4 ml/kg/hr) or saline (1 ml/kg/hr) was maintained for 3 hr of recovery. Two surgical sham groups received BPZ-302 (n=2) or saline (n=2). Fresh frozen specimens of striatum were obtained for determination of brain water content and BBB permeability. Wet/dry ratio and Evans blue permeability were measured. Groups were compared by ANOVA and the Newman-Keuls test.

Results:

The straitum wet-dry ratio in the treated group (4.92± 0.86) was significantly lower than the saline group (7.39±2.51) ($p=0.04$). No significant difference was found in the cortical tissue. Similarly the Evans blue extravasation in the striatal tissue was significantly lower in the treated group (26.59 ± 14.91) compared to the saline group (57.14 ± 27.34) ($p=0.04$).

Conclusions:

BPZ-302 has proven to be effective in limiting cerebral edema and reducing BBB permeability in striatal tissue following global ischemia. The combination of medium-weight, narrow, molecular range hydroxyethyl starch and Vitamin C in 7.5% hypertonic saline is novel and may lead to a new therapeutic with both anti-edema and anti-oxidant properties. Further studies will be performed to assess whether this drug has any neuroprotective effect.

References: None

Financial Support: This project is funded by an NIH grant. Dr. Torbey has no conflict of interest.Dr. Zikria, Dr. Toole, and Mr. Macolini are founders, officers, and shareholders of BioPhyZica, producer of BPZ-302.

Oral Presentation 8

NONCONVULSIVE ELECTROGRAPHIC SEIZURES AFTER HUMAN TRAUMATIC BRAIN INJURY RESULT IN A LONG-TERM HIPPOCAMPAL ATROPHY

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Introduction:

Early post-traumatic, non-convulsive seizures after traumatic brain injury result in prolonged elevations in glutamate, lactate/pyruvate ratio, and elevated intracranial pressure in the ICU (Vespa 1998; 1999; 2007). The long term effects of post-traumatic seizures on anatomic and cognitive function have not been well studied.

Methods:

Ten patients with moderate-severe traumatic brain injury underwent continuous EEG, ICP monitoring, and cerebral microdialysis for seven days. MRI (Volumetric T1 MP-RAGE, ADC, GRE) was performed twice in each subject on post-TBI day 7 and at 6 months following TBI. Comparison of changes in volumetric structure of the entire brain, including the hippocamus was made using Brainsuite (UCLA LONI).

Results:

Five patients had early post-traumatic seizures and five age, sex, GCS-matched patients did not. Elevation of ICP and microdialysis markers of cellular distress occurred in the seizure patients. Acute ADC and GRE-MRI did not show primary hippocampal injury (mean hippocampal ADC= 995 ± 108 u^2 /sec). At 6 months, all patients demonstrated global atrophy 7.9 ± 4.4%. Seizure patients demonstrated selective hipocampal atrophy ipsilateral to the seizure focus compared with non-seizure TBI patients (32.1 ± 2.4 % compared to 10.1 ± 0.6 %, respectively (Paired student's t test $p < 0.001$). In one case, repeat ADC MRI after status epilepticus shows ADC restriction coincident with the seizures.

Conclusions:

Early post-traumatic non-convulsive seizures are associated with long-term hippocampal atrophy ipisilateral to the seizure focus. Involvement of seizure activity in the secondary injury process of the hippocampus appears to be the reason, although elevations in ICP cannot be ruled out. Seizures potentially appear to be a therapeutic target to prevent long term tissue atrophy.

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Financial Support: None

Oral Presentation 9

TEMPERATURE MANIPULATION ALTERS EARLY EEG BURSTING AFTER CARDIAC ARREST IN RATS

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Introduction:
Hypothermia after cardiac arrest (CA) improves outcomes, while hyperthermia is harmful. After CA, EEG recovers through periodic bursting, the duration of which is predictive of outcome. The effect of temperature manipulation on early bursting has not been studied.

Methods:
We quantified burst frequency and the interval between CA and first burst (IBCFB) to study the effect of temperature manipulation. Twenty-four rats were evenly divided into 3 groups, based on 6 hours of hypothermia (T=33^{o}C), normothermia (T=37^{o}C), or hyperthermia (T=39^{o}C) immediately post-resuscitation from 7-minute asphyxial CA. Temperature was maintained using surface cooling and warming. Neurological recovery was defined by 72-hour Neurological Deficit Score (NDS).

Results:
There was higher burst frequency during the first 90-minute post-CA in rats treated with hypothermia (24±1.8/min) and hyperthermia (22.6±1.1/min) compared to normothermia (16.9±1.1/min) ($p<0.001$). Different patterns of burst frequency were noted in each temperature group. Within 20 minutes of resuscitation, the hypothermia group had a significantly higher burst frequency than normothermic rats which was maintained throughout 1-hour period ($p<0.01$). Although the hyperthermia group also had a significantly higher burst frequency within 20 minutes ($p<0.01$), it subsequently diminished and converged with normothermic rats by 50 minutes ($p>0.05$). Burst frequency correlated strongly with 72-hour NDS in hypothermic and normothermic rats between 20 and 90 minutes after resuscitation (Pearson correlation 0.845, $p<0.01$). The 72-hour NDS of the hypothermia group (Median:74) was significantly improved compared to normothermia (49) and hyperthermia (43) groups ($p<0.001$) supported by qualitative comparison of brain injury. No differences were noted in IBCFB between the temperature groups (p=0.127).

Conclusions:
In hypothermic and normothermic rats resuscitated from CA, early EEG burst frequency accurately predicts neurological recovery. With hyperthermia, increased bursting within the first hour – presumably due to heightened metabolic rate – may lead to falsely optimistic outcome prediction.

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Oral Presentation 10

DEEP VENOUS THROMBOSIS AMONG NICU PATIENTS IS PREVALENT AND OFTEN ASSOCIATED WITH INTRAVENOUS LINES

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Introduction:
The prevalence of DVT among critically ill patients ranges from 10-30%. Studies which prospectively evaluated patients reported higher values. The DVT rate among neurosurgical patients not receiving DVT prophylaxis has been sited at 22%. Most patients treated in the ICU have indwelling centrally placed venous catheters. The purpose of this study was to assess the prevalence of both upper and lower extremity DVTs among patients admitted to our NICU.

Methods:
Patients admitted to out NICU with a diagnosis requiring >24 hours of admission were included in this study. All subjects underwent an ultrasound of the legs as well as the upper limb on the side of the indwelling central venous line. The type of line was documented as well as the primary diagnosis. All subjects received DVT prophylaxis. Lovenox or unfractionated heparin was given to all appropriate subjects. Patients not capable of receiving heparin were treated with sequential compression devices and thigh high compression stockings. Ultrasounds were done between day 4 and 7 on all subjects unless there was a clinical indication at an earlier time.

Results:
183 patients underwent ultrasonagraphy of their legs and upper extremities. Forty six (25%) had evidence of a deep venous thrombosis; 19 in the femoral vein and 27 in the deep veins of the upper extremities, subclavian or internal jugular. Upper extremity DVTs were associated with PIC lines in 17 patients, internal jugular lines in 2 patients, subclavian lines in 6 patients and peripheral IVs in 5 (three patients had both a PIC line and a subclavian on the side of the DVT). All of the patients with DVTs were treated either with full heparin anticoagulation or with the insertion of a vena cava filter

Conclusions:
DVTs are very common among patients treated in an NICU even when they are treated with currently acceptable methods of prophylaxix (heparin or SCDs). Upper extremity DVTs are more common than lower extremity ones and are frequently associated with the use of indwelling catheters. Removal of these catheters at the earliest possible time may reduce this high rate of thrombosis.

References: None

Financial Support: None

Oral Presentation 11

USEFULNES AND COMPLICATION OF APNEA TEST FOR BRAIN DEATH DIAGNOSIS (BDD) IN 388 CASES

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Introduction:

Apnea test is required for brain death diagnosis in most of organ donation law's proceedings. There are few papers in the literature analyzing findings and complications, so we considered interesting to show them in a large population of suspected brain death patients.

Methods:

Setting: Large city Procuracion Organism (PO) that gives the official brain death certification.
Study design: Prospective database analysis from January 1/06 to June 30/07
Interventions: Apnea test according national Organ Donation and Procuration Law Proceedings. The test is performed two times with a 6 hours interval.
Data collection: Sex, age, cause of death, apnea test (AT) done by PO physicians or others, positive or negative results, reasons for not doing the test, complications, arterial blood gases, vasoactive drugs, core temperature, mean arterial pressure, sepsis instrumental certification (electroencephalogram(EEG), evoked potentials(EP), Transcranial Doppler(TCD), angiography(DA)), real donors, disconnection or cardiac arrest.
Statistical analysis: 2 tails Student test.

Results:

In the study period 388 patients were evaluated for BDD. Male 50%, mean age 39.66. AT wasn't performed in 20%: 40% due to hemodynamic instability, 33% due to drugs, 13% due to BDD for other methods and 14% for other reasons. AT performed before PO 7.5%. AT was negative in 6.2% (breathing) and indeterminate in 1.5%. Mean values: 1st evaluation (pH:7,35/7,09PCO_2:37,01/82,35PO_2:262,94/225,54) 2nd evaluation (pH:7,35/7,15PCO_2:38,75/80,30 PO_2:220,76/176,10) MAP35,58 Core Temp 35,58ºDopamine Complications: Cardiac arrest 1, desaturation 10%(6% in the 1st). EEG82%,TCD19%,EP10%,DA1%. Real donors 42%Cardiac arrest21% Disconnection38%.

Conclusions:

AT is a safe procedure. It is not routinely performed by ICU physicians before the PO evaluation (7.5%). Desaturation is the main complication and it is not associated with cardiac or hemodynamic changes. All the positive AT showed a significant difference in pH and PO_2 ($p<0.001$) but not in PO_2 (p0.15) a finding that, to our knowledge, hasn't been reported and that could be explained by a low oxygen compsuntion in BD corpses.

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Financial Support: None

Oral Presentation 12

INTRACRANIAL VOLUME ADAPTATION AND COMPLICATIONS AFTER DECOMPRESSIVE HEMICRANIECTOMY

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Introduction:
Decompressive hemicraniectomy is increasingly utilized in patients with intractable brain herniation, often as a life-saving procedure. However, there is limited data on intracranial volume changes and post-operative complications.

Methods:
Prospective case series of 28 patients who underwent decompressive hemicraniectomy with the following diagnoses:12(43%) ischemic stroke, 9(32%) subarachnoid hemorrhage (SAH), 2 (7%) intracerebral hemorrhage, 5 (18%)non-vascular. Intracranial volume changes on head CT were quantified in 15 cases using a software, *ANALYZE.* All pre- and postoperative CT images were evaluated by 2 independent neurointensivists.

Results:
Mean age of 48 years; 57% females; 57% Non-Caucasian, 43% Caucasian. Hemicraniectomy was performed within an average of 2.6 hours from onset of clinical herniation.Mean pre-operative intradural volume was 1353cc which increased to a mean of 1506 cc post-operatively(11%). Postoperative subgaleal volume increased by 10%, while the intraventricular CSF space expanded by 25%. Pre-operative ICP monitoring was available in 57% (n=16) of cases with mean ICP of 40 mmHg (range 16-90 mmHg). Post-operative ICP monitor was utilized in 82% (n=23) with mean ICP values of 16 mmHg (range 8 - 30). The average ICP reduction post-operatively was 25 mmHg. Pre-operative mean anteroseptal shift was 10 mm (range 4 - 19), while the mean pre-operative pineal shift was 5 mm (range 0 - 12). Mean reduction was 6 mm and 3 mm, for anteroseptal and pineal midline shift, respectively. Complications included: a) nonsurgical ipsilateral intraparenchymal hemorrhages 4/28 (14%) b) subdural hematoma requiring surgical evacuation (3/28, 11%) c) hydrocephalus 7/28 (25%) d) wound breakdown in 4/28 (14%) e) CSF drainage-related infection in 3/28 (11%).

Conclusions:
Decompressive hemicraniectomy reverses internal brain herniation as demonstrated by ICP normalization and detailed intracranial morphometric analyses. However, it may be associated with clinical complications including subdural and intraparenchymal hemorrhages, hydrocephalus, wound breakdown, and infections.

References: None

Financial Support: None

Oral Presentation 13

MILD HYPOTHERMIA REDUCES TISSUE PLASMINOGEN ACTIVATOR-RELATED HEMORRHAGE AND BBB DISRUPTION AFTER EXPERIMENTAL STROKE

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Introduction:
Thrombolytic therapy with rt-PA in ischemic stroke is limited by increased risks of cerebral hemorrhage and edema. Therapeutic mild hypothermia is gaining considerable interest as a neuroprotectant.

Methods:
We explored whether mild hypothermia affects detrimental aspects of rt-PA treatment such as cerebral hemorrhage and BBB breakdown. Male C57/BL6 mice were subjected to middle cerebral artery occlusion for 2 h using an intraluminal suture, followed by 24 h reperfusion. Three experimental groups included: TN: tPA treatment at normothermia (37C, n=14), TH: rt-PA treatment at hypothermia (33C, n=12), and N: no rt-PA treatment at normothermia (n=8). Rt-PA was delivered 3 h after ischemia (10 mg/kg, IV: 10% bolus, and the remainder over 30 min). BBB permeability was assessed using Evan's-blue dye. Infarct sizes were measured from cresyl violet stained sections. Hemorrhagic transformation was assessed by using a 5 point scale from gross brain slices by two observers blinded to experimental groups. Immunostains and Western blots of endogenous tPA and its inhibitor, plasminoen activator inhibitor (PAI-1) were performed in normothermic and hypothermic animals not given rt-PA.

Results:
Infarct sizes in the TH group were significantly smaller than infarcts in both the TN and N groups: TH (28.36%) versus TN (45.08%) and N (48.87%). More BBB disruption as estimated by the area of EB extravasation was seen in TN compared to TH and N. Administration of exogenous rt-PA at normothermia (TN, 57.1%) showed a higher hemorrhagic transformation rate compared to TH (25%) and N (25%). In mice exposed to ischemia but not given rt-PA, endogenous tPA expression was reduced in hypothermic mice, whereas PAI-1 levels were not.

Conclusions:
Mild hypothermia itself may reduce the incidence of hemorrhagic transformation by reducing levels of endogenous tPA. In the setting of rt-PA treatment, hypothermia can reduce the incidence and severity of hemorrhagic transformation and BBB disruption, suggesting that combination therapy with mild hypothermia and rt-PA should be safe, and may even improve outcome.

References: None

Financial Support: None

Oral Presentation 14

PREDICTORS OF OUTCOMES IN A CLOSED VERSUS SEMI-CLOSED NEUROINTENSIVE CARE UNIT

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Introduction:
Several studies have shown the benefit of a closed neurointensive care unit managed by a specialty trained neurointensivist on mortality and morbidity in a multitude of diagnoses. None of these studies have incorporated the severity of the brain injury as a potential confounder. The objective of this study is to assess whether this improved outcome is indeed present in all patients regardless of the severity of their illness.

Methods:
All ICD9 primary diagnosis of ICH were retrieved from our hospital discharge database from 2003 through 2005. Patients with any evidence of trauma, subarachnoid hemorrhage and tumor were excluded. Patient who underwent surgical evacuation were also excluded. CT of head done at admission and up to 48 hours were reviewed if available.

Results:
58 patients were included in the study. 64% were admitted to the NICU service (group A: closed unit) and 36% were admitted to neurosurgery with NICU service consulted (group B: semiclosed unit). Mean patients' age was 69±13 years (mean±SD). 51% were males. The groups were comparable in clot size, pineal shift, and septum pellucidum shift. Patient in group were more likely to go home and rehab compared to patient in group B (p=0.036). In group A 13.5% went home, 40.5% to rehab, 29.7% to nursing home, and 16.2% died in comparison to 9.5%, 23.8%, 14.3%, and 52.4% respectively in Group B. Subgroup analysis showed clearly that only two subgroups of patients had a significantly better outcome in group A compared to group B. Patient with IVH score >8 did better in Group A (p=0.025) (In group A: Home: 9%, rehab 18.2%, NH 54.5%, dead 18.2% compared to 0%, 9.1%, 9.1%, 82 % respectively in group B). Patients with clot size less ≤ 30 cc also did better when admitted to group A (p=0.014) (Group A: Home 20%, rehab 44%, NH 32%, dead 4% compared to 15.4%, 15.4%, 23.1%, 46.2% respectively in group B).

Conclusions:
The positive outcome associated with a neurointensivist run service may be specific to a subset of patients in the NICU. Institutions where neurointensivists are only consulted to manage ICH patients may need to reconsider their policies and admit at least these patients to the neurointensivist service.

References: None

Financial Support: None

Poster 15

STATIN USE WAS NOT ASSOCIATED WITH LESS VASOSPASM OR IMPROVED OUTCOMES FOLLOWING SUBARACHNOID HEMORRHAGE

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Introduction:
The development of delayed ischemia due to cerebral vasospasm remains a common cause of morbidity and mortality following aneurysmal subarachnoid hemorrhage (SAH). Preliminary studies have suggested that hydroxymethylglutaryl coenzyme A reductase inhibitors (statins) may decrease the risk of vasospasm, but further study is required.

Methods:
Beginning in May, 2006, our treatment protocol for patients presenting with SAH was altered to routinely include the use of 80 mg per day of simvastatin for 14 days. Prior to this time, only patients with other indications for statins were treated. The charts of 203 consecutive patients over 27 months were retrospectively reviewed, and 150 were included in the analysis, of which 71 received statins. These patients were compared with 79 untreated patients to determine if the use of statins was associated with a reduction in the occurrence of vasospasm, delayed infarction or poor outcome (death, vegetative state, or severe disability).

Results:
Statin-treated and untreated patients had similar baseline characteristics, although more patients in the former group were managed with endovascular coil embolization. There were no statistically significant differences in the proportion of patients developing at least moderate radiographic vasospasm (41% with statin vs. 42% without, p=0.91), symptomatic vasospasm (32% with statin vs. 25% without, p=0.34), delayed infarction (23% with statin vs. 28% without, p=0.46) or poor outcome (39% with statin vs. 35% without, p=0.61). After adjustment for differences in baseline characteristics, including the method of aneurysm treatment, statins were still not significantly protective.

Conclusions:
The addition of statins to standard care was not associated with any reduction in the development of vasospasm or improvement in outcomes following aneurysmal SAH. If there is a benefit to statin use, it may be smaller than suggested by previous studies, but further randomized controlled trials are awaited.

References: None

Financial Support: Some of the authors were co-investigators in the CONSCIOUS-I trial (clazosentan for prevention of vasospasm)

Poster 16

MULTIPLE ISCHEMIC INFARCTIONS AS A RESULT OF VASOSPASM IN A PATIENT WITH INTRAPARENCHYMAL AND INTRAVENTRICULAR HEMORRHAGE

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Introduction:
Symptomatic vasospasm has been reported in the setting of intraventricular hemorrhage (IVH) associated with a ruptured arteriovenous malformation (AVM). However, vasospasm in the setting of an IVH resulting from extension of a hypertensive intraparenchymal hemorrhage has not been previously reported.

Methods:
Single patient case report and review of literature

Results:
A 69 year-old hypertensive woman was admitted with headache, nausea and left hemiparesis. The initial head CT scan showed a right posterior corona radiata hematoma with associated intraventricular hemorrhage. No SAH was observed. The patient hospital course was complicated by multiple cerebral infarctions that resulted from diffuse vasospasm confirmed by CT angiography and transcranial dopplers studies. In the absence of SAH this was an unexpected complication.

Conclusions:
Symptomatic cerebral vasospasm and delayed ischemic deficits can occur as a complication of IVH associated with a hypertensive intraparenchymal hemorrhage. The recognition of this unusual complication requires a high level of suspicion by the clinician.

References: None

Financial Support: None

Poster 17

TENSION PNEUMOVENTRICLE RESULTING FROM DEHISCENCE OF THE TEGMEN TYMPANI

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Introduction:
Intraventricular tension pneumocephalus is not commonly reported in the neurosurgical literature. Simple pneumocephalus, on the other hand, is a common finding, and usually does not require any form of treatment. Tension pneumocephalus, on the other hand, usually occurs in the subdural area, and rarely within the ventricular system. The authors report a case of acute neurological deterioration in a 71 year old male with cerebrospinal fluid (CSF) leak. He had a prior cholesteotoma resection via a mastoidectomy. The etiology for this leak was found to be dehiscence of the tegmen tympani. A clinical diagnosis of tension pneumoventricle was made.

Methods:
A brief case review and illustration of a rare entity - tension pneumoventricle - that was treated in the neurological intensive care unit at Duke University Medical Center. Serial computed tomography (CT) scans were utilized as well as external ventricular drainage. A technetium—99m diethylenetriamene pentaacetate scan was performed for evaluation of the CSF fistula. This study confirmed the right-sided CSF leak through the dehisced tegmen tympani, which was subsequently repaired via a right temporal craniotomy.

Results:
The etiology for this leak was found to be dehiscence of the tegmen tympani. CT of the head revealed significant intraventricular pneumocephalus tracking from the mastoid air cells through a defect in the petrous temporal bone to the ipsilateral temporal horn of the lateral ventricle and throughout the ventricular system. Emergent ventriculostomy was performed and the patient's neurological status quickly improved towards normal.

Conclusions:
Though rare, tension pneumoventricle can occur, and results in rapid neurological deterioration, which requires emergent ventriculostomy followed by repair of the CSF fistula. The radiological finding of tension pneumocephalus presenting primarily as increasing intraventricular air, or pneumoventricle, should be recognized by those caring for patients in the neurological intensive care unit setting.

References: None

Financial Support: None

Poster 18

COMPLICATIONS OF HYPERTENSIVE HYPERVOLEMIC THERAPY FOR SYMPTOMATIC VASOSPASM

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Introduction:
Hypertensive Hypervolemic Therapy (HHT) has been widely used to treat symptomatic vasospasm following subarachnoid hemorrhage (SAH) though the therapy itself can cause complications.

Methods:
Among prospectively studied spontaneous SAH patients who developed symptomatic vasospasm we recorded complications related to HHT adjudicated by a team of neuro-intensivists including clinically significant arrhythmia, myocardial infarction (MI) defined by abnormal cardiac enzymes and echocardiography, pulmonary edema graded as mild, moderate or severe by an independent-blinded radiologist, new intracerebral hemorrhage and elevated intracranial pressure (ICP). Logistic regression was used to calculate odds ratios (OR) for demographic and admission risk factors related to HHT complications and to assess the effect of complications on 3-month outcomes, as measured by the modified Rankin Scale (mRS), and Telephone Interview of Cognitive Status scale (TICS).

Results:
Of 580 SAH patients, 16% (N=95) developed symptomatic vasospasm. Of these patients, 94% received volume expansion, 85% received intravenous pressors and 3% received neither. At least one complication developed in 30% and 7% had multiple complications. Pulmonary edema occurred most frequently (19%), was graded as moderate to severe in 71%. Arrhythmias or MI occurred in 5%. Elevated ICP occurred in 4% and 50% of these patients herniated. Intracranial hemorrhage occurred in 2 patients. Only female gender was a risk factor for a HHT complication (OR 3.8, 95% confidence interval [CI] 1.0-14.0, P=0.048). Though complications from HHT did not predict death, or severe disability, they did predict worse cognitive status (OR 5.3, 95%CI 1.6-18.2, P=0.008). This effect disappeared after controlling for age, Hunt-Hess, race, education and vasospasm. Hospital and ICU length of stay (LOS) were significantly longer in patients who developed complications from HHT ($p<0.001$).

Conclusions:
Complications from HHT are common and associated with longer hospital and ICU LOS, but do not independently predict poor outcome.

References: None

Financial Support: None

Poster 19

DYNAMIC CHANGES IN ECG PREDICTS POOR OUTCOME IN SUBARACHNOID HEMORRHAGE(SAH)

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Introduction:

Electrocardiographic (ECG) abnormalities following SAH have been documented. (1-2) Evidence suggests that cardiopulmonary dysfunction worsen outcome; determining which patients are at most risk is important (3-5).

Methods:

To address this issue, we prospectively studied cardiac abnormalities and clinical outcome in 20 patients (12 women and 8 men) admitted to the NeuroICU of a large academic hospital within 48 hours of SAH due to ruptured aneurysm. All patients had ECGs prior to surgical clipping, during the clipping surgery and the subsequent post-operative period.

Results:

Their ages ranged between 18 and 70 years (mean 47.21). The aneurysm was located in the anterior circulation in 17/20 patients (85%), in the posterior circulation in 3/20 patients (15%). Seven patients (35%) were Hunt and Hess grade I, 5 (25%) were grade II, 2 (10%) were grade III, 3 (15%) were grade IV and 3 (15%) were grade V. Patients were grouped according to the presence or absence of ECG abnormalities during the study period. Seven patients had normal ECGs and 13 had abnormalities at some time during the study period. Four patients (30.7%) with ECG changes showed dynamic ECG abnormalities (an abnormality that presented and disappeared during the study period or changed in character). Of the patients who had no ECG abnormalities, 6/7 had good outcome (86%) compared to 8/13 (62%) with ECG abnormalities ($p=0.277$, ns). All four patients who had fluctuating ECG changes had a poor outcome (100%) compared to 2/8 (25%) patients with fixed abnormalities ($p=0.03$).

Conclusions:

Dynamic ECG changes were a better predictor of the postoperative neurological outcome than abnormal ECG alone. Further research is needed to determine the significance of these dynamic ECG changes and the optimal treatment of cardiac injury in patients with SAH.

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Poster 20

RISK FOR HYPERGLYCEMIA AMONG NEUROLOGICALLY CRITICALLY ILL PATIENTS

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Introduction:
While hyperglycemia is common in critical illness, it is not clear whether critically ill neurological patients are at increased risk. The aim of this study was to examine the frequency of hyperglycemia in the neurological versus medical intensive care unit.

Methods:
We conducted a prospective cohort study of patients admitted to the neurological (NICU) or medical (MICU) intensive care units during a 1 month period. Admission characteristics, peak and mean glucose values, and insulin and dextrose administration in the first 24 hours of ICU stay were collected. Multivariate logistic regression analysis was used to calculated adjusted odds ratios (aOR) for the effect of neurological injury on peak glucose values >200 mg/dL .

Results:
Over 1 month, 36 patients were admitted to the NICU and 43 to the MICU. MICU patients were significantly older, more likely to be mechanically ventilated or septic, and had higher APACHE III scores (all $P<0.05$). Diabetes, body mass index, steroid, vasopressor, insulin and dextrose administration did not differ between the two ICUs. The most common diagnoses in the NICU were brain tumor, subarachnoid hemorrhage and spinal cord compression, and in the MICU, sepsis, pneumonia and gastro-intestinal bleeding. Twenty two percent of NICU and 26% of MICU patients had glucose values >200 mg/dL. After adjusting for univariate predictors of hyperglycemia (age and APACHE III scores), NICU patients were significantly more likely to have hyperglycemia >200 mg/dL within 24 hours of ICU admission compared to MICU patients (aOR 5.8, 95% confidence interval 1.1-30.8, $P=0.04$).

Conclusions:
NICU patients were significantly more likely to have severe hyperglycemia , compared to MICU patients after accounting for other predictors of hyperglycemia. Further study is needed to elucidate the mechanism by which neurological injury predisposes to hyperglycemia.

References: None

Financial Support: None

Poster 21

BIOMARKER KINETICS IN CEREBROSPINAL FLUID OF TRAUMATIC BRAIN INJURY PATIENTS

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Introduction:

Traumatic brain injury (TBI) produces αII-spectrin proteolysis breakdown products (SBDPs) that are potential biomarkers for TBI. SBDP kinetic characteristics have not previously been described. This study describes the kinetic characteristics of SBDPs in cerebrospinal fluid of adults with severe TBI and the relationship between these kinetic metrics and severity of injury.

Methods:

Kinetic analyses were conducted on 35 severe TBI patients who were enrolled in an IRB approved multicenter study. An immunoblot analysis was performed on all CSF samples and arbitrary densitometric units were used to describe the values of SBDPs observed. Non-compartmental kinetic analysis was conducted on all patients with measurable values and the kinetic metrics evaluated include AUC, MRT C_{max}, T_{max}, and $t_{½}$.

Results:

	SBDP 150 (n=34)	**SBDP 145 (n=35)**	**SBDP120 (n=25)**
AUC	317383	394749	92641
MRT [hrs]	50	55	72
T1/2 [hrs]	24	23	40
Cmax	130	139	22
Tmax [hrs]	28	30	72

As shown in the table above, SBDP120 had a smaller AUC and Cmax, but a longer MRT, T1/2, and Tmax than the SBDP150 and SBDP145. Patients experiencing hypoxia or hypotension prior to hospitalization had a greater AUC and Cmax, and a longer MRT than those patients who did not. Similar trends were seen when best day 1 GCS and severity of head injury by CT scan were evaluated. AUC for SBDP 150 and 145 was significantly greater in patients with worse GCS scores at 24 hours ($p=0.011$, $p=0.016$, respectively).

Conclusions:

Markers of calpain proteolysis primarily associated with necrosis/oncosis (SBDP145 and SBDP150) were found to have different kinetic characteristics than the markers of apoptosis produced by caspase-3 proteolysis (SBDP120) in the CSF in TBI patients. Severity of injury also appeared to influence kinetic characteristics. Further studies will be conducted to fully elucidate the relationship between SBDPs and clinical outcomes.

References: None

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Poster 22

AGE OF PACKED RED CELLS DID NOT AFFECT ACUTE OUTCOMES AFTER SAH

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Introduction:

Some data suggest that the age of packed red blood cells (PRBCs) is related to transfusion reaction, infection, fever and outcomes in ICU patients, but there are few data in patients with subarachnoid hemorrhage (SAH).

Methods:

We prospectively enrolled consecutively admitted patients with SAH. PRBC transfusions were given according to a standard ICU protocol. All units were leukocyte depleted. We prospectively ascertained transfusion-related adverse events (new fever >=100.4F, hypotension [systolic BP<100 or new vasopressor needed or increase of vasopressor dose by >25%], rash, dyspnea or radiographic pulmonary edema, infection, or elevated hepatic enzymes), vasospasm (TCD>120 or clinical diagnosis), cerebral infarction on neuroimaging, and clinical outcomes at 14 days, 28 days and 3 months.

Results:

Of 75 patients, 40 patients received one of 134 units of PRBCs in one of 103 transfusions. Sixteen (15%) transfusions had an adverse event: 12 had fever, 4 had hypotension, 1 had pulmonary edema or dyspnea, and 1 had a rash (2 transfusions had 2 adverse events). The mean PRBC age was 25.2 +/- 9.7 days. There was no relationship between PRBC age and adverse events, vasospasm, cerebral infarction, mortality, NIH Stroke Scale at 14 days, or modified Rankin Scale at 14 days, 28 days or 3 months (P>0.2 for all).

Conclusions:

PRBC age does not seem to impact acute outcomes after SAH. New fever is the most common adverse event to transfusion after SAH. Requests for newer blood are unlikely to impact SAH outcomes.

References: None

Financial Support: None

Poster 23

FACTORS INFLUENCING EARLY OR LATE MORTALITY OF NICU-ADMITTED PATIENTS

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Introduction:

We have demonstrated in the past that the presence of a neurointensivist (NI) decreases mortality in the Neuro-ICU. Factors associated with early mortality may be different than those associated with late mortality or survival after NICU admission.

Methods:

Froedtert hospital's and the University Health System Consortium (UHC) databases were used to examine early mortality (dying within the first 48 hours after admission), late mortality or survival to be discharged from the hospital after admission to the NICU in two 19-month periods, before and after the appointment of a NI. Univariate and multinomial logistic regression analyses were performed.

Results:

Eighty six patients died early (82 in the NICU and 4 on the Ward). More early deaths occurred after brain death (51% vs 23% for late deaths) and fewer after withdrawal of treatment or cardiac arrest without resuscitation (42 vs 66% and 1 vs 8%, respectively, $p < 0.001$). Early compared to late mortality was associated with younger age, admission to neurosurgery, through the emergency department and emergent admission, but not with the presence of a NI. Adjusting for co-variates, early compared to late mortality was associated with UHC expected mortality (odds ratio, 95% CI 1.01/%, 1-1.02) and hospital length of stay (0.92/day, 0.87-0.96). Early mortality compared to survival was associated with UHC expected mortality (1.06%, 1.04-1.07), UHC expected hospital length of stay (0.93/day, 0.89-0.97), neurosurgery service (19, 4.2-86.5), and diagnosis of ischemic stroke (9.6, 1.4-68) or intracerebral hemorrhage (10.5, 2.2-49).

Conclusions:

Admission to neurosurgical service, intracerebral hemorrhage and ischemic stroke are independently associated with dying within 48 hours compared to surviving after admission to the NICU. Early vs late deaths more frequently occur due to brain death and less frequently due to withdrawal of treatment or cardiac arrest with DNR orders in the NICU.

References: None

Financial Support: None

Poster 24

HUMAN APOE 3/3 GENOTYPE OFFERS IN NEUROPROTECTION IN A MURINE MODEL OF INTRACEREBRAL HEMORRHAGE

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Introduction:

Recent evidence suggests that apolipoprotein E (apoE) influences central nervous system responses to multiple mechanisms of acute brain injury and may affect overall mortality in critically ill patients. To address the mechanisms by which apoE influences functional outcomes after intracerebral hemorrhage (ICH), we test the hypothesis that targeted replacement (TR) mice expressing human apoE isoforms (apoE3 and apoE4) will react differently to a collagenase-induced intracerebral hemorrhage in a clinically relevant murine model.

Methods:

After stereotactic creation of a burr hole, high-dose clostridial collagenase (0.15 U/0.4□l) was injected into the left basal ganglia of APOE3TR (n=8) and APOE4TR (n=11) mice over 5 minutes. Once recovered, the mice were subjected to rotarod latency testing (seconds) and neuroseverity scoring (21-point scale) on postoperative days (POD) 1, 2, and 3. Additionally, cerebral edema was determined by wet-to-dry hemispheric weight measurements at 24 hours after injury. Analysis of variance was used to evaluate for functional outcomes, and student's t-test was used for edema analysis.

Results:

APOE4TR mice rotarod times were lower and neuroseverity scores were higher than their APOE3TR counterparts (p=0.05 & p=0.02, respectively) during the 72 hours post-operatively with rotarod times at 72 hours of 64 v. 108 sec (APOETR4 v. APOETR3) and neuroseverity scores of 12 v. 17. Additionally, APOE4TR exhibited increased cerebral edema when compared to APOE3TR mice (p=0.006; 0.1475+/-0.004 v. 0.1246+/-0.003 gm).

Conclusions:

Human APOE genotype appears to exhibit an influence on functional outcome after collagenase-induced ICH in mice. APOE3 genotype may confer a neuroprotective effect with increased cerebral edema formation in APOE4 genotypes being one possible mechanism. Clarification of APOE effects on ICH size and inflammatory mediators remains to be evaluated.

References: None

Financial Support: None

Poster 25

IMPACT OF STATINS ON VALIDATION OF ICH MORTALITY PREDICTION MODELS

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Introduction:

Intracerebral Hemorrhage (ICH) has the highest mortality rate of all strokes approaching 50%. Hemphill's ICH score is commonly used to predict mortality following ICH. More recently the ICH Grading Scale (ICH-GS) has been shown to improve sensitivity of 30-day mortality prediction in this patient group. The objective of this study was to assess the impact of prior statin use on 30-day mortality prediction following ICH using these contemporary mortality prediction models.

Methods:

Records of consecutive ICH patients from 1999 to 2006 were reviewed. Patients with ICH related to trauma or underlying lesions (e.g. brain tumors, aneurysms, arterio-venous malformations) and of infratentorial location were excluded. We dichotomized patients into a "predicted survival group" and "predicted death group" based on a less than or greater than 50% probability of death, respectively. The predicted mortality using ICH score and ICH-GS prediction models was calculated and was compared to the observed mortality in each of 2 patient subgroups differentiated based on prior exposure to statins. Chi-square test was used for comparison of predicted and observed outcomes.

Results:

One hundred and twenty five patients were included in the analysis. The overall observed mortality was 23.2%. In patients using statins prior to ICH, observed mortality was 38% (5/13) and 42% (5/12) of the calculated mortality using ICH-GS ($p=0.026$) and ICH score ($p=0.043$), respectively. This difference was not seen in patients not previously exposed to statins where the observed mortality was 80% of the predicted mortality (24/30) using ICH-GS ($p=0.27$) and 92% of the predicted mortality (24/26) using ICH score ($p=0.70$).

Conclusions:

The significant difference between predicted and observed mortality using ICH-GS and the ICH score in the statin cohort suggests a protective effect of statins in the setting of ICH. Such observation warrants prospective validation.

References: None

Financial Support: None

Poster 26

AN ASSOCIATION OF PRIOR STATIN USE WITH DECREASED PERIHEMATOMAL EDEMA

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Introduction:
Hematoma expansion and evolution of perihematomal edema are most commonly responsible for neurological deterioration following Intracerebral Hemorrhage (ICH). A possible role of statins in reducing perihematomal edema has been suggested based on studies in animal models. The objective of this study was to investigate the impact of statins on perihematomal edema following spontaneous supratentorial ICH.

Methods:
Records of consecutive ICH patients from 1999 to 2006 were reviewed. Patients with ICH related to trauma or underlying lesions (e.g. brain tumors, aneurysms, arterio-venous malformations) and of infratentorial location were excluded. Absolute and relative perihematomal edema were assessed on initial head CT. Using regression analysis, the impact of prior statin use on absolute and relative edema at presentation was assessed correcting for other factors possibly impacting perihematomal edema such as age, coagulopathy, aspirin use, admission mean arterial pressure and blood glucose.

Results:
One hundred twenty five consecutive ICH patients were studied. Patients with prior statin exposure had a mean edema volume of 13.2 +/- 9.2 cc compared to 22.3 +/- 18.3 cc in patients who were not using statins at the time of ICH. Following multiple linear regression analysis, we identified a statistically significant association between prior statin use with reduced early absolute perihematomal edema ($p=0.035$). Mean relative perihematomal edema was significantly lower in patients on statins at presentation (0.44) as opposed to 0.81 in patients with no prior statin use. This difference remained statistically significant ($p=0.021$) after correcting for other variables.

Conclusions:
We report the association between statin use prior to ICH and decreased absolute and relative perihematomal edema. A prospective study analyzing the role of statins in perihematomal edema reduction and the resultant effect on mortality and functional outcomes following ICH is warranted.

References: None

Financial Support: None

Poster 27

APHASIA IN A PATIENT WITH INTRAVENTRICULAR HEMORRHAGE OF UNUSUAL CAUSE

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Introduction:

Intracranial large vessel occlusion may lead to the formation of new vascular channels with greater propensity to rupture.

Methods:

We report a patient with aphasia and intraventricular hemorrhage (IVH) who was found to have middle cerebral artery (MCA) occlusion.

Results:

A 74-year-old right-handed female with hypertension and atrial fibrillation (on warfarin therapy) presented with confusion of one day duration. She was disorientated, with a non-fluent aphasia with transcortical motor features. Head CT showed isolated intraventricular hemorrhage. She became somnolent day 2 after onset, with ventricular enlargement on imaging. External ventricular drainage resulted in improvement in headache, somnolence and aphasia. Conventional angiogram revealed left MCA occlusion with reconstitution by left anterior and posterior cerebral artery collaterals. Management was conservative with improvement throughout her hospitalization.

Conclusions:

The cause of aphasia in this patient is not immediately clear. Dural and meningeal collateral flow over the cortex may have been compromised by elevated intracranial pressure from her hydrocephalus. Improvement occurred with decompression. An alternative explanation for her aphasia could be thalamic injury by irritation from blood products, or by hemorrhage originating in posterior thalamic tissue. (Injury of the dominant hemisphere thalamus produces a transcortical aphasia). Subcortical interruption of the white matter fibers due to edema associated with IVH and hydrocephalus is also a conceivable mechanism for the aphasia.
Conventional angiography remains the gold standard for the evaluation of sources of intracerebral hemorrhage, demonstrating the cause of the bleeding in 60-70% of patients with isolated IVH. Presence of concomitant focal deficits, unexpected in isolated IVH, may be a clue to suspect intracranial vessel occlusion. We believe that vascular changes in the left choroid plexus induced by redistribution of blood flow through collateral pathways compensating for the MCA occlusion may have predisposed the patient to have IVH.

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Financial Support: None

Poster 28

SURVIVAL AND LONG-TERM FUNCTIONAL OUTCOME IN 1155 CONSECUTIVE NEUROCRITICAL CARE PATIENTS

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Introduction:

Objective: To analyze survival and mortality, long-term functional disability outcome and to determine predictors of unfavorable outcome in critically ill patients admitted to a Neurologic (neuro) Intensive Care Unit (ICU).

Methods:

Design: Retrospective cohort study with post neuro ICU health related evaluation of functional long-term outcome.
Setting: A 10-bed neuro ICU in a tertiary care university hospital.
Patients: A consecutive cohort of 1155 patients being admitted to a neuro ICU during a 36-month period.

Results:

1155 consecutive patients, with 41 % being female, were enrolled in the study. The predominant reasons for neuro ICU care were cerebrovascular diseases such as intracerebral hemorrhage (20%), subarachnoid hemorrhage (16%) and complicated, malignant ischemic stroke (15%).
213 (18%) patients died in the neuro ICU. Glasgow Outcome Scale (GOS) and modified Rankin scale (mRS) were dichotomized into two groups determining unfavorable versus favorable outcome (GOS 1-3 versus GOS 4-5, mRS 2-6 versus mRS 0-1). Factors associated with unfavorable outcome in the unselected cohort according to logistic regression analysis were admission diagnosis, age ($p < .01$) and a higher score in simplified therapeutic intervention scoring system (TISS-28) at time of admission ($p < .01$). Functional long-term outcome was evaluated in a telephone interview for 662 patients after a median follow-up of approximately 2 and a half years by evaluating mRS and GOS. Factors associated with unfavorable functional long-term outcome were admission diagnosis, sex, age >70yrs (OR 8.45; 95%CI 4.52-15.83; $p<.01$), TISS-28>40 points at admission (OR 4.05; 95%CI 2.54-6.44; $p<.01$) and TISS-28>40 points at discharge from neuro ICU (OR 3.50; 95%CI 1.51-8.09; $p<.01$) and length of stay (LOS) (OR 1.01; 95%CI 1.00-1.03; $p=.02$).

Conclusions:

We found admission diagnosis, age, LOS and TISS-28 score at admission as well as discharge to be independent predictors of unfavorable long-term outcome in an unselected neurocritical care population.
Broessner et al, Crit Care Med, 2007, in press.

References: None

Financial Support: None

Poster 29

CRYPTOCOCCAL MENINGOENCEPHALITIS WITH PSEUDOCYSTS IN IMMUNOCOMPETENT PATIENT

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Introduction:

Cryptococcus is the most common cause of fungal meningitis and meningocerebral syndromes. Although immunosuppressed patients are at risk, infection can occur in immunocompetent patients as well. Meningitis is the most common neurological presentation, but cryptococcomas, cysts and psuedocysts may also occur. Raised intracranial pressure(ICP) and hydrocephalus may develop in as many as 15% of patients. CNS Cryptococcus is universally fatal if untreated. With treatment, survival improves but mortality remains as high as 46%, especially if complicated by raised ICP and hydrocephalus.[i]

Methods:

Case Report: A 52 year-old man developed fever, meningismus and rapidly decreasing level of consciousness over two days. There was no history of immunosuppression, however he had been exposed to chicken and pigeon feces at work. His GCS was 5 out of 15 and he was intubated for airway protection. A brain MRI showed bilateral pseudocystic hyperintensities involving the basal ganglia(Figure). A lumbar puncture revealed CSF opening pressure >30 cmH20 with a lymphocytic pleocytosis, increased protein, and decreased glucose. HIV serology was negative and enzyme linked immunosorbent assay and India ink stain were positive for cryptococcus. CSF cultures identified Cryptococcus neoformans var Gatti as the pathogenic species.All other infectious causes were ruled out. Subsequent lumbar puncture opening pressures were >40cmH20.

Results:

The patient was admitted to the Neurosciences Intensive Care Unit where a lumbar drain was inserted to treat the raised ICP. The patient received intravenous amphotericin B and flucytosine for six weeks and then fluconazole maintenance therapy. The lumbar drain was removed after six weeks when ICP normalized and the patient regained consciousness. He was transferred back to peripheral hospital with mild residual bradykinesia and rigidity that was treated with low dose Sinemet.

Conclusions:

This case highlights the dramatic neuroimaging of severe cryptococcal meningoencephalitis and demonstrates the possibility of good outcome with early and aggressive management.

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Poster 30

USE OF IV THROMBOLYSIS FOR ISCHEMIC STROKE ASSOCIATED WITH CRACK-COCAINE ABUSE

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Introduction:
Thrombolysis with IV rt-PA is indicated for selected patients with acute ischemic stroke. The safety of thrombolysis in patients with crack-cocaine-related strokes is unknown. There have been a few case reports of intracranial hemorrhage after thrombolysis for myocardial infarction in cocaine abusers.

Methods:
We report two patients that received thrombolysis for acute ischemic stroke associated with crack-cocaine abuse.

Results:
Case 1: 37-year-old, African American woman with no known co-morbidities, except crack-cocaine and alcohol abuse, was brought to the ER within 3 hours of onset of acute stroke. Her NIHSS score on presentation was 17 and exam was compatible with MCA distribution stroke. The patient received IV rt-PA. Her NIHSS score decreased to 15 within 24 hours but later patient had a cardiac arrest and expired. There was no hemorrhagic complication.
Case 2: 38-year-old African American male with arterial hypertension and crack-cocaine abuse, presented 1 hour 45 minutes after stroke onset. Patient was given IV rt-PA for MCA distribution stroke 1 hour later. His NIHSS score decreased from 10 to 3 within 5 days. There was no hemorrhagic complication. Patient was discharged to rehab later.

Conclusion:
Our report suggests that the use of IV rt-PA for acute ischemic stroke is safe in patients with MCA ischemic strokes associated with crack-cocaine abuse.

References: None

Financial Support: None

Poster 31

HYPOTHERMIA FOR REFRACTORY STATUS EPILEPTICUS

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Introduction:

Status epilepticus (SE) affects 150,000 Americans each year with 30-50% of cases failing initial therapy. Refractory cases requiring midazolam, pentobarbital, or propofol fail to control 8-21% of seizures. Rodent models of SE treated with hypothermia demonstrate less histopathologic evidence of neuron death, and diminished duration of epileptic discharges. We report on two patients with refractory SE (RSE) treated with therapeutic hypothermia.

Methods:

Two patients with SE refractory to conventional therapy were treated with hypothermia (32-34 ^{o}C) using an intravascular cooling system (CoolGard 3000® Thermal Regulation System and Icy™ catheter) inserted via a femoral vein.

Results:

Patient 1: A 66 year old male with RSE failed oxcarbazepine, valproate, phenytoin, levetiracetam, and lorazepam. He twice experienced generalized seizures during weaning from pentobarbital induced burst-suppression. Seizures were controlled with hypothermia at 32^{o}C. He reached the target temperature of 32^{o}C in 10h, remained there for 61h, in burst-suppression and was gradually warmed over 50h. He remained seizure-free after warming, however developed sepsis and multi-system organ failure, which was successfully managed with antibiotics and vasopressors. Currently, he is seizure free and improving.

Patient 2: A 62 year old male with a history of alcohol abuse and cirrhosis continue to have seizures despite lorazepam, levetiracetam, phenytoin, and propofol. Burst-suppression was induced with midazolam, but was complicated by hypotension. Seizure recurrence during midazolam weaning prompted institution of hypothermia. He reached 34^{o}C and burst-suppresion on EEG in 4 hours. After 20 hours he was warmed over 29 hours. This enabled midazolam and vasopressors to be discontinued. The patient developed a lower limb venous thrombosis requiring heparin and an inferior vena cava filter. He remained seizure free and was discharged to rehabilitation.

Conclusions:

Hypothermia controlled refractory seizures in two patients who failed conventional therapies. Side effects are manageable in the ICU setting. Further study is needed to evaluate its safety and efficacy.

References: None

Financial Support: None

Poster 32

BIS-AUGMENTED SEDATION ASSESSMENT IS ASSOCIATED WITH A DECREASE IN PROPOFOL USE.

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Introduction:
Sedation is frequently a component of ICU care and oversedation is common in most ICU settings. In the neurocritical care setting, the need to quickly and easily interrupt sedation infusions to obtain a neurologic exam is especially important. Determining the appropriate amount of sedation has been traditionally accomplished using subjective methods of assessment. The purpose of this study was to determine if the Bispectral index (BIS) could provide data that, when combined with subjective data, would decrease the amount of sedation used.

Methods:
Subjects were unable to provide self-consent therefore, informed consent was obtained by a legally authorized representative. Following informed consent, 51 subjects were randomized to one of two sedation assessment groups. The Ramsay-alone group received propofol adjusted to maintain a Ramsay score of 4. The BIS-augmentation group received propofol adjusted to a Ramsay score of 4 and a BIS of 60-70. The study period began at 8:00 a.m. and lasted through the 12-hour nursing shift. The total propofol volume was obtained through chart abstraction and the mean propofol rate (mcg/kg/min) was calculated using each subject's admission weight.

Results:
Two separate ANOVA models were created using SAS 9.1 for windows (Cary, NC). The difference in the mean propofol volume infused in the BIS-Augmentation group (97.51ml, SD=92.71) compared to the Ramsay-alone group (175.36ml, SD=131.72) was statistically significant (F=6.00, p=0.018) and explained 11% of the variance in scores (r^2=0.11). The difference in the mean rate of propofol infusion in the BIS-Augmentation group (15.35 mcg/kg/min, SD=12.80) compared to the Ramsay-alone group (30.19 mcg/kg/min, SD=22.23) was statistically significant (F=8.63, p=0.005) and explained 15% of the variance in scores (r^2=0.15).

Conclusions:
BIS-augmented sedation assessment is associated with an overall decrease in the amount of propofol infused over the course of a 12-hour shift for patients receiving sedation in a neurocritical care unit.

References: None

Financial Support: Research Sponsored by Aspect Medical Systems, Inc

Poster 33

MULTI-MERCI MAKES INROADS INTO THE COMMUNITY SETTING

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Introduction:

Neuro-Interventional Radiological (NIR) treatment, embolectomy with the MERCI device can be effectively used alone or in combination with thombolytics in the treatment of acute ischemic stroke in a community hospital setting.

Methods:

We reviewed our experience with acute stroke between 2005 and 2006 by chart review and abstracted all cases treated with NIR.

Results:

Out of 492 patients presenting with acute ischemic stroke during the study period, 23 (4.7%) received NIR. 20/23 (87%) presented after three hours; 3/23 (13%) received IV t-PA within the 3 hours time window. Median age was 61 (46-82), with 45% women. Patients were treated as follows: IA t-PA alone 5 (22%) MERCI alone 3 (13%), IA t-PA plus MERCI 12 (52%) and IV t-PA plus MERCI plus IA t-PA 3 (13%). Mean NIHSS on admission and discharge was 20 and 11, respectively. Complete recanalization was achieved in 12 (52%) of patients. Mean NIHSS scores on admission and discharge for the recanalization group were 19 and 6, and for the non-recanalization group were 22 and 18. No patient that recanalized died, while 4 (18%) died in the non-recanalization group. Symptomatic ICH occurred in 2/23 (8.6%) of patients. At 90 days, 11/23 (48%) of patients had mRS <= 2.

Conclusions:

In our small series of twenty three cases in a community hospital 34% achieved full and 26 % partial independence. Incidence of 8.6 % symptomatic ICH and mortality of 18% are comparable to other studies. Most of these patients were densely paralyzed (mean NIHSS 20) and had symptom onset beyond three hours. Our experience at a certified stroke center in a community setting shows that mechanical revascularization combined with thrombolytic treatment can bring some hope of independent living to even severely affected, late arriving stroke victims.

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Financial Support: None

Poster 34

MRSA COLONIZATION IN NEUROSURGERY PATIENTS CARRIES A HIGH RISK OF WOUND INFECTIONS

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Introduction:
Methicillin-resistant staphylococcal aureus (MRSA) is a major problem in hospitalized patients world-wide.

Methods:
A prospectively registered database of 493 consecutive hospitalized neurosurgical patients treated at a tertiary care community hospital from 8/06 to 2/07 was analysed. The hospital actively screened all ICU admissions (n=271).

Results:
The overall wound infection rate in the screened population was 7.4%. This was higher than the unscreened population of patients directly admitted to the floor (n=222,1.8%, $p<0.01$).
In the screened neurosurgery population, the incidence of MRSA colonization was 6.3% (17/271). For screened patients not colonized with MRSA, the wound infection incidence was 5.5% (14/254); post-operative NS infection was 3.9% (10/254) and MRSA wound infection was 1.2% (3/254). In contrast, MRSA colonized patients (n=17) had wound infection incidence of 35.3% (6/17), post-operative NS infection (23.5%, 4/17), and MRSA wound infection (23.5%,4/17).

Conclusions:
MRSA colonization is associated with a high risk of wound infection in the neurosurgical ICU population. These findings raise concerns regarding post-operative wound care and peri-operative antibiotics.

References: None

Financial Support: None

Poster 35

SAFETY OF 23.4% SODIUM CHLORIDE IN THE TREATMENT OF INTRACRANIAL HYPERTENSION

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Introduction:
Mannitol is the standard of care for patients with intracranial hypertension, but multiple administrations of mannitol risk renal toxicity and fluid accumulation in the brain parenchyma with possible worsening of brain edema. Because of this issue, there seems to be increased interest in hypertonic saline but this use may be limited by the lack extensive data regarding safety and efficacy. This study assessed the safety of small volume injections of 23.4% sodium chloride in the treatment of intracranial hypertension.

Methods:
We retrospectively reviewed the charts for adverse events of patients with documented intracranial hypertension measurements or radiographic evidence of cerebral midline tissue shifts who received mannitol and 23.4% sodium chloride over 24 consecutive months. Potential adverse events were defined as the diagnosis: central pontine mylinolysis, congestive heart failure, hypokalemia, metabolic acidemia, subdural hemorrhage, hemolysis, coagulopathy or rebound hyponatremia that was deemed to be attributable to the medication effect. All aliquots of 23.4% sodium chloride were 30 mL and given via central venous access over 5 - 30 minutes. Dosages were usually given alternating with mannitol.

Results:
Eighty one patients received a total of 417 doses of 23.4% sodium chloride. The etiology of the pathology in this patient population included: subarachnoid hemorrhage, cerebral hemorrhage, traumatic brain injury, ischemic stroke, and leucoencephalopathy. There were no adverse events attributable to 23.4% sodium chloride.

Conclusions:
Although the use of hypertonic saline may cause concern due to the potential complications, the bolus dosing of 30 mL of 23.4% sodium chloride via central access over 5-30 minutes was safe and not associated with the potential adverse events.

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Financial Support: None

Poster 36

USE OF INTRATHECAL TPA FOR THROMBOLYSIS IN INTRAVENTRICULAR HEMORRHAGES OF DIFFERENT SUBTYPES

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Introduction:
Intraventricular hemorrhage is associated with poor prognosis. Management strategies may be comprised of prompt diagnosis, etiological assessments and management. Treatment of intraventricular hemorrhage includes observation, drainage for hydrocephalus, and possibly thrombolysis and maintenance of drain patency. Unfortunately, a lack of data precludes a standardized treatment strategy. We present our experience with intrathecal tPA in ten patients with intraventricular hemorrhage.

Methods:
Patients were admitted to neurocritical care unit and diagnosed with intraventricular hemorrhage through appropriate scans and angiography. Cause of intraventricular hemorrhage was noted. An external ventricular drain was placed in all ten patients, and an early lumbar drain was inserted in one patient. tPA at 2mg/2mL was administered via external ventricular drain. Serial CT scans were done to monitor progress of thrombolysis, and 24-hour external ventricular drainage volumes were collected. Intraventricular Hemorrhage scores were also collected.

Results:
Baseline Intraventricular Hemorrhage Scale scores ranged from 4 to 12. Patients received an average of 3.2 doses of intrathecal tPA at 2mg/2mL. Intraventricular Hemorrhage Scale scores reduced an average of 79.1%. Patency of external ventricular drain was continuously maintained. Patient length of hospital stay averaged 18.9 days. 8 of 10 (80.0%) patients receiving intrathecal tPA survived. Blood pressure levels were controlled between 120-150mm Hg. INR remained between 0.9 and 1.2 for all patients.

Conclusions:
The experiences in this case series support previous reports and suggest intrathecal tPA may be appropriate for thrombolysis in selected patients and further trials are warranted.

References: None

Financial Support: None

Poster 37

FEASIBILITY OF PERCUTANEOUS TRACHEOSTOMIES BY NEUROINTENSIVISTS

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Introduction:
Improved techniques for performing tracheostomy, notably at the bedside, have made tracheostomy safer and more available than previously. Recent trends in published literature demonstrate the safety of percutaneous tracheostomy in competent hands. Little data exist on neurointensivist performed percutaneous tracheostomies.

Methods:
A retrospective chart review of 10 patients with percutaneous tracheostomies, all placed by neurointensivist, was conducted from August 2005 to June 2007. Tracheostomies were performed using the Ciaglia Blue Rhino technique. Variables collected include reason for admission, reason for tracheostomy, time of tracheostomy from admission, average time of procedure, position of tracheostomy, occurrence of immediate or delayed complications, estimated blood loss, occurrence of infections, ventilator weaning, and discharge destination.

Results:
Eight (80.0%) of percutaneous tracheostomies were performed due to failure to protect airway, two (20.0%) due to failure to wean. Tracheostomies were performed in an average 7.6 minutes (S.D. $\pm$ 1.1). Estimated blood loss was <1mL for 5 (50.0%) patients, 1mL for 3 (30.0%) patients, and 2mL for 2 (20.0%) patients. No (0.0%) patients suffered infection. Immediate complications occurred in no (0.0%) patients. Delayed complications occurred in two (20.0%) patients. Eight (80.0%) patients discharged to long-term care, 1 (10.0%) to rehab, and 1 (10.0%) did not survive.

Conclusions:
Low incidence of infection, complications, and mortality suggest trained neurointensivists may be suitable for performing percutaneous tracheostomies. Based on these supplemental results, neurointensivist performed percutaneous tracheostomies may be safe and feasible. Further research is warranted.

References: None

Financial Support: None

Poster 38

PREVALENCE OF LEFT ATRIAL THROMBUS IN CEREBRAL ISCHEMIC EVENT WITH SINUS RHYTHM AND ITS ASSOCIATION WITH LEFT VENTRICULAR SYSTOLIC DYSFUNCTION: A SINGLE CENTER EXPERIENCE

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Introduction:
Patients with cerebral ischemic events who are in atrial fibrillation (AF) are more frequently associated with left atrial thrombus (LAT). Ventricular systolic dysfunction (LVSD) is associated with LAT and ventricular clot formation in AF. The association of LVSD with LAT in sinus rhythm is not known.

Objective:
To identify the prevalence of LAT and its association with LVSD in TIA and /stroke who are in sinus rhythm (SR).

Methods:
Consecutive patients with cerebral ischemic events (SR) who were evaluated by transesophageal echocardiography (TEE) were prospectively collected from July 2000 to August 2001. Patients demographic including patient age, gender, race, cerebrovascular risk factors and stroke location were recorded.

Results:
Of 238 patients, LAT was present in 13/238 (5.5%), 6 (2.5%) in the LAA and 6 (2.5%) in left atrial cavity (LAC). LVSD was present in 25 (10%) (mild 7, moderate 8, sever 10 patients). Patient mean age was 59 ± 14 years and119 (51%) were male. In univariate analysis, LAT was associated with LVSD (OR 9.24 CI 2.8, 10.3 $p<0.001$). Additionally, male patients (OR 4.56 CI 1.6, 12.6, $p<0.003$) and history of CAD (OR 3.4 CI 1.3, 8.4, $p< 0.003$ was more likely to have LAT. There was no association of LAT with patient age, past history of hypertension, DM, HL, left atrial dilation, left atrial appendage dilation or left ventricular hypertrophy (LVH).
In multivariate analysis, LAT formation was independently associated with LVSD (OR 10.6, CI 2.2 – 51.6, $p <$ .003) controlling for gender, age, past history of CAD, DM, smoking and LVH

Conclusions:
The prevalence of LA thrombus is not uncommon in TIA and /or stroke that are in SR, especially in patients with poor left ventricular systolic functions. TEE should be considered in stroke and /or TIA patients with sinus rhythm, who have LVSD for early detection and treatment.

References: None

Financial Support: None

Poster 39

SAFETY OF RECOMBINANT FACTOR VIIA IN NEUROSURGERY

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Introduction:

Recombinant Factor VIIa (rFVIIa) is an attractive pharmacologic option in neurosurgery to assist in reversing coagulopathies. However, the efficacy remains inconclusive and safety is concerning. The aim of this study is to describe the practice and outcomes in a mixed neurosurgical population at a major academic tertiary care center.

Methods:

This case series characterizes all neurosurgery patients receiving rFVIIa. Data was collected retrospectively by means of chart review, including demographics, admitting diagnosis, rFVIIa doses and comcominant blood products, time and duration of coagulopathy reversal, and documented complications. Descriptive statistics were applied.

Results:

Twenty-five patients received rFVIIa during a 2 year period. The indications for rFVIIa included ICH with coagulopathy (8), emergent neurosurgical procedure with coagulopathy (12), spontaneous ICH (2), and other (3). The median rFVIIa first dose was 44 mcg/kg (N=25), second dose was 25 mcg/kg (N=4). Two patients received a third dose and one received a fourth dose. Those with an elevated baseline INR (N=20), the median time to and duration of reversal was 1.4 (0.95, 1.9) hours and 15.4 (8.7, 23) hours for the first dose, 0.75 (0.6, 1) hours and 10 (8, 10) hours for the second dose. Among all patients, there were 3 rebleeds (2 with clinical deterioration) and 5 new bleeds (all procedure related). One patient had a DVT and PE, detected 11 days post dose, and one patient had an isolated PE, detected 16 days post dose. Three patients had elevated cardiac enzymes, but normal wall motion on echocardiogram. Twelve of 25 patients survived, with a median GOS of 3 and mRS of 4.

Conclusions:

This data suggests that rFVIIa may be safe in neurosurgery. The optimal dose needs to be carefully evaluated with attention to pharmacoeconomics, clinical efficacy, and risk factors for complications.

References: None

Financial Support: None

Poster 40

LEFT VENTRICULAR SYSTOLIC DYSFUNCTION AND ITS PREDICTORS IN PATIENTS WITH NORMAL SINUS RHYTHM WHO EXPERIENCED A RECENT CEREBRAL ISCHEMIA

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Introduction:
Left ventricular systolic dysfunction (LVSD) is associated with increased morbidity and mortality. The prevalence of LVSD in cerebral ischemic events with normal sinus rhythm (NSR) is less known. Objectives: To identify the prevalence of LVSD in transient ischemic attack (TIA) and/or ischemic stroke with NSR

Methods:
Consecutive patients with TIA and/or ischemic stroke with NSR were selected for this study. Patients' demographic, clinical, neuroimaging, and echocardiographic characteristics were studied. SPSS 11.5 was used to perform statistical analysis (t-test, ANOVA, and Chi-square test).

Results:
Of 555 patients, LVSD was present in 41 patients (7%) (mild=15, moderate=12, and severe=14), of which 32 patients (78%) were men. Mean age was 65 +/-14 years. Prior history of stroke was present in 58/555 (10%), coronary artery disease (CAD) in 74/555 (13%), hypertension (HTN) in 270/555 (49%), diabetes mellitus (DM) in 92/555 (17%), hyperlipidemia in 242/555 (44%), and active smoking history in 152/555 (27%). Of 41 patients with LVSD, 27 (66%) have hypokinesis, 7 (17%) have dyskinesis, and 4 (10%) have akinesis as a manifestation of segmental wall motion abnormalities. LVSD was associated with male gender (p=0.001), CAD (p=0.001), and DM (p=0.004). Patients' age, history of prior stroke, HTN, hyperlipidemia, smoking history, or left ventricular hypertrophy (LVH) were not associated with LVSD. In multivariate analysis the male (OR 4.019, 95% CI 1.899~8.812) gender and CAD (OR 3.651, 95% CI 1.764~7.408) continued be a predictor of LVSD.

Conclusion:
The prevalence of LVSD in TIA and/or stroke patients with normal NSR is not uncommon, especially in those who are male and have history of a CAD. Therefore, an echocardiography to evaluate ventricular function should be considered in all TIA and /or stroke patients including patients with normal sinus rhythm

References: None

Financial Support: None

Poster 41

THE RATE OF PHLEBITIS IN 25 STROKE/ICH PATIENTS WHO RECEIVED PERIPHERAL IV DOUBLE CONCENTRATED NICARDIPINE-CARING TRIAL

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Introduction:

Nicardipine (Cardene®) is indicated for HTN control while oral treatment is not feasible. Its long term use in patients with ischemic stroke (IS) or intracerebral hemorrhage (ICH) has not been well studied. In stroke/ICH patients, standard dose of nicardipine (max 150 ml /hr) may present a challenge in managing the fluid intake in patients with brain edema. This trial is an open-label prospective study to evaluate safety and efficacy of double concentrated IV nicardipine for treatment of HTN in patients with IS, ICH or SAH.

Methods:

From 3/04 to 6/07, 25 patients who met all the criteria received double concentrated nicardipine. The IV site was checked hourly for phlebitis during the infusion and BP checked q 15 min x 4 hrs, q 30 min x 8 hrs, then hourly until 4 hrs post completion. The degree of seriousness of phlebitis was recorded according to the Infusion-Related Complications established by the Intravenous Nurses Society. Patients' demographics, stroke type, & frequencies of dose adjustments were recorded. Student *t* test was used to calculate the significance of occurrence of phlebitis with a 95% confidence interval.

Results:

Twelve male and 13 female were studied (IS 15, ICH 10). Average age was 67 (25-88) Nicardipine dose ranged 0.2-19.5 mg/hr. Average infusion duration was 40 hours 23 min. There was one (4%, $p<0.12$, CI 95%) reversible grade 3 phlebitis. Twenty two of 25 patients (88%) required < 3 dose titration to reach the target BP range.

Conclusions:

The rate of phlebitis with double concentrated nicardipine through peripheral IV is low. Such low rate was achieved by changing the peripheral IV site every 24 hours and not using IV site established in the field. Nicardipine was also well tolerated in elderly patients (24%).

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Financial Support: This trial has been partially sponsored by the PDL BioPharma

Poster 42

IMPLANT FOR PERFUSION AUGMENTATION CLINICAL TRIAL-1 (IMPACT-1). A SAFETY AND FEASIBILITY EVALUATION OF THE NEUROPATH IS SYSTEM IN THE TREATMENT OF ACUTE ISCHEMIC STROKE – A MULTI-CENTER PILOT STUDY

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Introduction:

In the rat stroke model, **sphenopalatine ganglion (SPG)** stimulation up to 24 hours after stroke onset augments cerebral blood flow, reduces the infarct volume and improves neurological deficits. We present preliminary safety and feasibility data of SPG stimulation with the NeuroPath IS System in patients with acute ischemic stroke (AIS).

Methods:

This is an ongoing multi-national open label study recruiting 70 patients with AIS, age 18-75 years, NIHSS 7-16, treatment initiated within the first 24 hours following stroke onset. The NeuroPath IS System is implanted adjacent to the SPG via the Greater Palatine Canal using a minimally invasive surgery (20 min., local anesthesia). The therapeutic regimen consists of 7 days, 3hr/d stimulation. The primary endpoint is the incidence of adverse events (AE) and serious adverse events (SAE), a secondary endpoint is the feasibility of SPG stimulation as AIS treatment. NIHSS, mRS and BI are being collected.

Results:

To date 30 patients have been enrolled, 18 (mean age 47.6 yrs, mean time from stroke onset 17.1 hr) completed the study. One patient died suddenly after being discharged after remarkable improvement. Two patients, who received 2 and 3 stimulation days only, died because of massive brain edema and cardiac failure. None was related to study treatment. One had a recurrent cardioembolic stroke. There was 1 malpositioning and 2 wound dehiscence, 1 brain edema that resolved with conservative therapy.

Conclusions:

This interim analysis suggests that NeuroPath IS System is easily and safely implanted. SPG stimulation when administered within 24hr from symptom onset appears to be safe and promising for the treatment of AIS. Further recruitment is ongoing to explore the effectiveness of the procedure.

References: None

Financial Support: Authors 7,8,9,10 and 11 are employees of Brainsgate Ltd,the sponsor for the trial.Natan Bornsetin, Marc Fisher and Werner Hacke are members of the scientific advisory board and own company's stock options

Poster 43

ROSTRO-CAUDAL HERNIATION AND TRANSIENT LOCKED-IN STATE FROM MECHANICAL COMPRESSION

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Introduction:
Locked-in syndrome (LIS) is often caused by ventral pontine ischemia involving the perforating pontine vessels of the basilar artery. Full recovery from central nervous system causes of LIS is rarely reported. We describe a patient who developed transient LIS acutely after aneurysmal subarachnoid hemorrhage and rostro-caudal herniation from hydrocephalus.

Methods:
We observed the full course and recovery of a patient with LIS. This patient was unique in both the injury mechanism, which involved mechanical buckling of the brainstem against the anteriorly located clivus, and the clinical presentation, consisting, distinctively, of a prolonged but fully reversible LIS.

Results:
The patient's clinical course and diagnostic studies support the likely mechanism of this patient's LIS to be mechanical compression of the ventral pons anteriorly against the clivus. Radiographically, there was no evidence of brainstem or spinal cord ischemia. The MRI did delineate a more anteriorly located brainstem, which can promote forward buckling of the brainstem during its downward displacement caused by a supratentorial, space-occupying process, as occurred with our patient. This, in turn, led to compressive injury of the pons at a point discernible as the shortest clivus-to-brainstem distance, causing our patient's reversible ventral pontine dysfunction. In support of this injury mechanism were the clinical examination and evoked potentials, localizing the lesion to both the corticospinal and -pontine tracts above the facial nucleus and adjacent ventro-lateral pontine fibers while sparing the more dorso-medial tegmentum.

Conclusions:
The patient's slow but full recovery allowed us to further differentiate this clinical entity from the more common LIS due to ischemic mechanisms. The recognition of herniation-associated LIS and its potential reversibility can enhance more thoughtful prognostication of long-term recovery in selected patients.

References: None

Financial Support: None

Poster 44

NEUROCRITICAL CARE OF FULMINANT DISSEMINATED ENCEPHALOMYELITIS

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Introduction:

This presentation discusses state-of-the-art management of patients with Acute Disseminated Encephalomyelitis (ADEM) and delineates the role of contemporary neurocritical care in patients with a so-called fulminant course. As epidemiological, randomized, or large-scale clinical studies are unlikely to be attained in such patients, we collected prospectively a series of ADEM patients with various clinical presentations who required admission to a neurointensive care unit. We categorize and outline the diagnostic and treatment goals with respect to the dominating mechanism of secondary injury during the acute phase.

Methods:

Case series and literature review.

Results:

The diversity and critical care spectrum of fulminant ADEM is illustrated by describing six patients with unique presentations; we added a historical patient with detailed pathological examination to complement this continuum. Each patient delineates the presentation, anticipated complications, and expected outcomes of a unique variant of fulminant ADEM. The discussion outlines the critical care approach focusing on minimizing both primary and secondary injury mechanisms. For each presentation form we propose a management algorithm next to a review of the pertinent literature on fulminant ADEM.

Conclusions:

1. Even though patients with ADEM generally have a good prognosis, there is a small but distinct clinical subgroup of patients progressing to a life-threatening variant known as fulminant ADEM. 2. Aggressive neurointensive care management of fulminant ADEM tailored at addressing the particular caveats and complications of fulminant ADEM variants provides the potential for these patients not only to survive but also for significant neurologic recovery.

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Financial Support: None

Poster 45

TOTAL AND FREE PHENYTOIN LEVEL DIFFERENCES BETWEEN MALES AND PRE- AND POSTMENOPAUSAL FEMALES IN THE NEUROINTENSIVE CARE UNIT.

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Introduction:

Phenytoin, used for the management or prevention of seizures in neurointensive care unit patients, is challenging[1] due to acute phase reactant elevations, nonlinear kinetics, metabolic genotypes[2,3], drug interactions, and hypoalbuminemia[4]. We sought to determine gender differences in free phenytoin levels based on total phenytoin levels and serum albumin.

Methods:

Retrospective chart and laboratory review of neurointensive care unit patients from November 2005 to November 2006 who were given fosphenytoin intravenously and had total phenytoin, free phenytoin, and albumin levels. Patients were excluded if they had preexisting hepatic or pancreatic impairment defined by elevated transaminases. Estimated free phenytoin was made by dividing total phenytoin by a factor of ten and correlating with serum albumin. We defined the difference between estimated and measured free phenytoin as the free phenytoin delta (Δ free).

Results:

We identified 75 patients, 38 of which were excluded due to lack of albumin and/or free phenytoin levels, and 6 patients excluded due to hepatic or pancreatic impairment. Thirty-two patients had total phenytoin, free phenytoin, and albumin levels. We found no straightforward correlation between free phenytoin, total phenytoin, and albumin levels in all patients. However, in males and postmenopausal females, we found that Δ free varied least (0-0.3 ug/mL) when serum albumin 3.5 – 5 g/dL (normal), but had increasing variability with lower serum albumin (albumin 3 – 3.4 g/dL, Δ free >0.3 – 0.6 ug/mL; when albumin 1.3 – 2.9 g/dL, Δ free 0.7 – 1.2 ug/mL). Premenopausal females had a wide variability even within normal albumin ranges (Δ free 0.2 – 0.9).

Conclusions:

In the neurointensive care unit, males and postmenopausal females free phenytoin levels correlated best with serum albumin, while premenopausal females had marked discordant free phenytoin levels despite normal albumin levels.

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Poster 46
LISTERIA RHOMBENCEPHALITIS
- A CASE OF ATYPICAL CLINICAL COMPLICATIONS AND MRI FINDINGS -
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Introduction:
Listeria Rhomboencephilitis (LR) typically presents with non-specific headache, nausea, vomiting, and fever followed by progressive cranial-nerve palsies, cerebellar signs, hemiparesis and impairment of consciousness. Respiratory failure occurs in half of the cases. If untreated, LR is fatal.

Methods:
We report a case of LR with rare complications of central apnea, paroxysmal neurogenic hypertension and reversible ischemia on MRI.

Results:
An 80-year-old woman presented following four days of nausea, vomiting and fever. She then developed an acute left facial nerve palsy, dysarthria, dysphagia and gait ataxia. Brain MRI showed an enhancing left dorso-lateral pontomedullary lesion effacing the fourth ventricle. Blood cultures grew Gram positive rods consistent with Listeria monocytogenes. Despite appropriate antibiotic therapy, she developed bilateral facial and abducens nerve palsy, left hemiparesis and respiratory failure. Once intubated, she began to experience apneas and autonomic dysfunction. When systolic blood pressure (SBP) fluctuated from 80 mm Hg to 230 mm Hg, she suddenly became quadriparetic and poorly responsive. Diffusion-weighted images (DWI) during the episode showed areas of restricted diffusion involving posterior and anterior subcortical white matter bilaterally and the splenium of the corpus callosum, with reduced apparent diffusion coefficient (ADC), suggestive of ischemia. One week later, repeat MRI showed resolution of DWI and ADC abnormalities, confirming a pattern of reversible ischemia.

Conclusions:
This case illustrates a classic initial presentation of LR, but also the first case associated with central apnea and neurogenic hypertension. Resporatory failure and autonomic dysfunction should be considered in the spectrum of complications related to LR.

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Poster 47

THERAPEUTIC HYPOTHERMIA COMBINED WITH AN EARLY GOAL-DIRECTED THERAPY ALGORITHM IMPROVES OUTCOMES AFTER RESUSCITATION FROM OUT-OF-HOSPITAL CARDIAC ARREST

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Introduction:
Neurologic injury and mortality after cardiac arrest resuscitation partially derives from a post-resuscitation inflammatory state sharing characteristics with sepsis syndrome. While therapeutic hypothermia attenuates post-resuscitation injury, further outcome improvements might be attained employing modalities known to treat sepsis physiology, such as early goal-directed therapy (EGDT). We sought to improve cardiac arrest outcomes with a post-resuscitation protocol combining therapeutic hypothermia with EGDT.

Methods:
From 5/2005 to 5/2007, we prospectively treated survivors of out-of-hospital cardiac arrest using a combined hypothermia/EGDT algorithm using a defined set of eligibility criteria. Data were collected on medical interventions, patient characteristics, physiologic parameters, survival and Cerebral Performance Category (CPC) at discharge. Outcomes were compared to matched historical control cardiac arrest patients from 1/2000 to 5/2005.

Results:
During the investigational period, 23 patients were eligible for combined hypothermia/EGDT and 15 patients were enrolled; these were compared to 140 patients from the historical cohort. Mean time to target temperature (33°C) was 4.5 hours (range 2-9 hours) from initial resuscitation; 93% (14/15) of patients achieved EGDT physiologic goals for central venous pressure and mixed venous oxygen saturation within 6 hours of initial survival. Survival to discharge before and after protocol implementation was 31% (43/140**)** and 60% (9/15), respectively; 47% (7/15) of the latter group were discharged with good neurologic outcomes (CPC 1,2).

Conclusions:
Hypothermia may be successfully combined with other therapeutic modalities in patients resuscitated from cardiac arrest. It is likely that optimal post-arrest care will require a broad array of hemodynamic and pharmacologic adjuncts to maximize neurologic benefit.

References: None

Financial Support: None

Poster 48

CEREBROSPINAL FLUID DRAINAGE CONTRALATERAL TO A HEMISPHERIC LESION DOES NOT SIGNIFICANTLY WORSEN MIDLINE SHIFT.

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Introduction:

The role of cerebrospinal fluid drainage (CSF) via an external ventricular drain (EVD) in the treatment of expanding cerebral hemispheric lesions remains controversial. Although patients with intracranial hypertension may benefit, placement of an EVD contralateral to a hemispheric lesion may augment upper brainstem lateral shift contributing to neurologic deterioration. We sought to determine the effect of contralateral EVD placement on midline shift in patients with cerebral hemispheric mass lesions.

Methods:

Ninety cases of EVD placement were retrospectively reviewed . Patients with lateralized cerebral hemispheric lesions causing midline shift who underwent contralateral EVD placement were selected. Pre and post placement midline shift was measured at the pineal gland, the midpoint of the septum pellucidum, and at the point of maximal shift. Differences were analyzed using a one-sample t test.

Results:

Thirteen of 90 cases (14.4%) met selection criteria including 6 basal ganglia, 3 thalamic, and 1 parietal lobar hemorrhage, 1 contusion, one frontal lobe hematoma associated with subarachnoid hemorrhage, and one internal carotid artery occlusion with infarct. Mean differences between pre and post EVD placement shift were: pineal gland -0.12 mm (SD $\pm$ 1.1; 95% confidence interval -0.80 to 0.55; p= 0.70); midpoint of the septum pellucidum -0.75mm (SD $\pm$ 2.6; 95%confidence interval -2.3 to 0.8; p=0.31); point of maximal shift -0.15mm (SD $\pm$ 3.3; 95% confidence interval -2.2 to 1.8; p=0.90). None of the differences were significant.

Conclusions:

In this cohort of patients, contralateral drainage of CSF via an EVD did not significantly alter midline shift at any of the measured points. Further prospective study in a larger group of patients may help further elucidate potential risks of this treatment strategy in this population of patients.

References: None

Financial Support: None

Poster 49

PHENYTOIN VS. LEVETIRACETAM: A COST ANALYSIS IN A NEUROCRITICAL CARE UNIT

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Introduction:
Phenytoin is widely available in both parenteral and oral forms, but its prophylactic use often involves frequent monitoring of serum levels and the possibility of drug interactions and adverse events. Levetiracetam is a newer antiepileptic drug associated with a more favorable safety profile which does not require monitoring of serum levels. The purpose of this analysis was to determine the cost difference associated with the use of levetiracetam versus phenytoin for routine seizure prophylaxis in acute brain injury patients in a neurocritical care unit.

Methods:
All patients with acute brain injury who received anticonvulsant prophylaxis with phenytoin, fosphenytoin, or levetiracetam admitted to the neurocritical care unit from 2/2/07 to 3/1/07 were reviewed. Using the medication administration record, the corresponding pharmacy medication system report, and the computerized physician order entry system, data were compiled on a daily basis. The costs associated with drug administration and blood level monitoring were also recorded.

Results:
Forty (40) patients received seizure prophylaxis: intracranial hemorrhage (42.5%), craniotomy (45.0%) or SAH (12.5%). There were a total of 91 patient days on phenytoin or fosphenytoin and 133 patient days on levetiracetam. Even though the direct cost of medication per patient-day was different between groups ($5.84 for phenytoin vs. $14.04 for levetiracetam), the total treatment costs were substantially lower in the levetiracetam group ($14.96 per patient-day vs. $91.23 per patient-day in the phenytoin group). The increased costs of monitoring were associated with phenytoin ($77.69 per patient-day).

Conclusions:
The major cost difference in this analysis was related to the amount spent on serum levels and monitoring for phenytoin. These data show that levetiracetam is a more cost effective option than phenytoin for seizure prophylaxis in patients with acute brain injury. With the approval of intravenous levetiracetam, there are increased options for antiepileptic drug prophylaxis in the critically ill population.

References: None

Financial Support: Dr. Daniel Laskowitz is on the speaker board and has served as a consultant for UCB Pharma.

Poster 50

TACROLIMUS (FK-506) INDUCED CEREBRAL HEMORRHAGIC POSTERIOR REVERSIBLE ENCEPHALOPATHY SYNDROME (HPRES) POST CARDIAC TRANSPLANT.

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Introduction:

Case reports describing immunosuppressant induced neurotoxicity are increasing. FK-506 is associated with a variety of neurological side effects including leukoencephalopathy and PRES. Radiological features showing white matter changes and pathological features with high FK-506 levels are documented. HPRES remains rare. There are no case reports of HPRES in patients after heart transplants.

Methods:

A 50-year-old right-handed man's case was reviewed after having HPRES with therapeutic FK-506 levels. He received a heart transplant one month prior to presentation that was indicated for severe coronary artery disease. The patient's post-operative course was uncomplicated until this neurological event. His blood pressure was well controlled postoperatively and he awoke with painless dense left hemiplegia without hemiparasthesia. At the time of the event his BP was 150/90 and FK-506 level was sub-therapeutic

Results:

The pre-operative scans were essentially normal. The post event Head CT revealed a large right fronto-parietal intracerebral hemorrhage with bilateral white matter edema. Serial MRIs showed striking bilateral subcortical changes consistent with PRES which had interval resolution after discontinuing the FK-506.

Conclusions:

FK-506 induced PRES without hemorrhage is a rare, but reported complication generally associated with high levels of FK-506. There are no cases reported of HPRES in any cardiac transplant patients, adult or pediatric, nor with low FK-506 levels. The reported cases of HPRES with FK-506 are with two stem cell transplants and a bone marrow transplant. The leukoencephalopathy we believe was due to the immunosuppressant and the cause of the hemorrhage may be secondary to the PRES itself. Hypertension cannot entirely be discounted in this case since blood pressures were not continuously monitored, however all reported vital signs had been normal for 22 days. Neurologic deficits in FK-506 transplant patients may be due to HPRES.

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Poster 51
TAKOTSUBO SYNDROME ASSOCIATED WITH SEIZURE ACTIVITY
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Introduction:
Takotsubo syndrome is a reversible neuromyocardial failure that has been reported in post menopausal women related to an acute catecholamine toxicity of the myocardium brought upon by a stressful event. Numerous case reports have reported a psychological stressor as the etiology of the phenomena; though more recently reports of the syndrome have been reported with multiple medical conditions (hypoglycemia, pneumothorax, alcohol withdrawal, neuroleptic syndrome, pheochromcytoma, sepsis, during hemodialysis and post anesthesia).

Methods/Results:
We present three episodes of Takotsubo neuromyocardial syndrome presented to our neurointensive care unit in two patients presenting with seizure disorders that had typical clinical presentation, echocardiographic and cardiac catheterization findings. All the episodes were treated conservatively with critical care support and monitoring with complete reversal of their symptoms and findings in the three episodes. Literature review and complete description of the clinical presentations, pitfalls of management and the role of diuretics and vasoactive agents, and laboratory findings are presented.

Conclusions:
Takotsubo cardiomyopathy is a phenomenon is associated with a catecholamine toxicity related to a stressful event. Past case reports addressed psychological variable as the trigger of the catecholamine surge and resultant myocardial failure though increasing reports of physical etiologies have been reported to trigger the catecholamine surge and myocardial failure.

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Poster 52

ACUTE CARE NURSE PRACTITIONERS: THE ROLE IN NEUROSCIENCE CRITICAL CARE

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Introduction:

In order to meet the needs of the high acuity population in today's critical care environment, the role of the Acute Care Nurse practitioner (ACNP) has been adopted by many intensive care units (ICU's) across the country, including specialized neurocritical care units. The purpose of this paper is to present our experience implementing an Acute Care Nurse Practitioner Service in a closed Neurocritical Care unit in a large academic medical center. Discussion includes a historical review of the ACNP, their function in various ICU settings, the details of our practice as well as future plans and challenges of the role.

Methods:

We reviewed the 4 year experience of The Johns Hopkins Hospital Neurosciences Critical Care Unit combined ACNP/MD model of proving care as well as performed an extensive review of the literature regarding the role of the ACNP in various critical care settings.

Results:

We found that ACNPs are used extensively in a variety of critical care settings and there are many benefits to integrating ACNPs into intensive care units. Our personal experience has demonstrated that it is especially advantageous to incorporate this role into the intense, exciting and ever-changing world of neurocritical care.

Conclusions:

Incorporating an ACNP service into the neurocritical care arena can have a positive impact on the way care is delivered to this complex population.

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Poster 53

THE IMPACT OF NEUROLOGIC COMPLICATIONS ON OUTCOME AFTER HEART TRANSPLANTATION

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Introduction:
Heart transplantation is a therapeutic option for end stage heart failure. Approximately 24,000 patients have undergone heart transplantation in the US over the last 10 year. In this study we investigated the prevalence of neurologic complications in heart transplant recipients.

Methods:
We retrospectively studied 313 patients from the Cardiac Transplant Program who underwent heart transplantation at the Mayo Clinic Rochester from January 1988 through June 2006.

Results:
Perioperative neurologic complications occurred in 23%: delirium or encephalopathy (9%), cerebrovascular complications (5%), and diseases of the peripheral nerves and muscles (4%); however, only perioperative cerebrovascular complications were associated with one-year mortality (HR 4.17, CI 1.04-16.76; P=0.04). The majority of these cerebrovascular complications occurred after the second postoperative day and was related to mechanical support of the circulation. Over 18 years the risk for neurologic complications was 81%: sleeping disorders (32%), polyneuropathy (26%), and cerebrovascular diseases (14%). Cause of death was neurologic in 12 of 95 patients (13%) and most common were cerebrovascular disease (n=6) and CNS infectious diseases (n=3). Adjusting for baseline predictors, CNS infection (HR 4.29, CI 1.69-10.9, p=0.0022) and depression (HR 1.81, CI 1.06-3.09, p=0.028) and seizures (HR 3.44, CI 1.33-8.85, p=0.010) were predictive for mortality.

Conclusions:
Perioperative neurologic complications are frequent in heart transplant recipients but most are transient and inconsequential. However perioperative stroke is the most important neurologic complication impacting on survival in the first year after heart transplantation. Infectious diseases of the CNS are associated with fatal outcome.

References: None

Financial Support: None

Poster 54

A COLD HAND IN RIGHT HEMISPHERIC STROKE

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Introduction:

The initial assessment is often the most critical with regards to urgent investigation and potential treatment. Not infrequently there are associated medical conditions that must be identified readily to prevent imminent danger and harm to the patient. We describe a case in which identification of a cool, discoloured upper extremity led to aggressive neurovascular intervention in a patient presenting a right middle cerebral artery (MCA) stroke.

Case Report:

A 51 year old female presented with left sided hemiplegia with a right gaze preference. The onset of her symptoms could not be accurately determined however a right middle cerebral artery ischemic stroke was diagnosed based on her presentation. Clinically, the patient was deteriorating with evidence of hemodynamic instability and a cool, pulseless right hand with limited motor and sensory function. A conventional angiogram was obtained to investigate her ischemic right hand and showed a blood clot in her brachiocephalic artery, which extended into her right carotid and subclavian arteries. The optimal treatment for such an unusual thrombus was unclear. Options discussed include anticoagulation with associated risk of hemorrhagic transformation, intraarterial catheter clot extraction and surgical embolectomy.

Results:

After careful discussion, a surgical embolectomy was successfully performed and the patient regained full function of her right hand. The etiology of the thrombus was suspected to be cardioembolic, however no cardiac source or hypercoaguable state was identified, including a search for malignancy.

Conclusions:

Stroke patients often present with other medical conditions. Careful assessment is required to identify medical conditions such as aortic dissection, carotid dissection, myocardial infarction, cardiac arrhythmia and other tissue ischemia.[1,2] In our young patient early detection of critical signs led to radiologic and surgical intervention that effectively saved her ischemic right arm and prevented bilateral upper extremity functional impairment.

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Poster 55

SERUM TROPONIN AND BRAIN NATRIURETIC PEPTIDE (BNP) IN ACUTE ISCHEMIC STROKE

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Introduction:

Acute stroke is associated with elevated serum Troponin and BNP independent of pre-existing cardiac risk factors (1, 2). Significance of this association remains unproven due to conflicting studies regarding relationship between serum Troponin and BNP and clinical outcomes in stroke (3, 4).

Methods:

Retrospective study of adult ischemic stroke patients admitted to University Hospital was done. Serum Troponin and BNP levels were measured within 24 hours after stroke. Data collected included patient age, pre-existing cardiac risk factors, and 30-day mortality.

Results:

152 patients were analyzed. 27% had high Troponin with mean of 0.39±0.81. 62% had high BNP with mean 805.11±1398.72. There was good correlation between serum Troponin and BNP (r =0.59, p=0.015). Survivors had lower BNP (601.81±11.22 vs. 2075± 2383; p=0.04) and lower CPK (139.09±237.45 vs. 692±2019; p=0.01) compared to non-survivors. Troponin was not statistically different between survivors and non-survivors. Analysis of receiver operative characteristic curve (ROC) for BNP with a cut-off value of 220 pg/ml revealed sensitivity 100 % for predicting mortality, specificity 60% with area under curve AUC of 0.82 (0.63-0.93) and p value 0.01. ROC for Troponin revealed cut-off value of 0.03 (sensitivity 55.6, specificity 68.4, AUC 0.63; p=0.2). Analysis of subgroups based on presence of risk factors such as congestive heart failure (CHF) ,diabetes and hypertension revealed no differences in serum troponin or BNP except CHF patients who demonstrated significantly higher levels of BNP($p<0.0001$)

Conclusions:

Troponin and BNP elevation is common in immediate post stroke period. BNP elevation is more common than Troponin and is associated with higher mortality. A BNP cut-off level of 220 is sensitive predictor for mortality. Our study is limited by small sample size and retrospective design. Further larger, prospective studies are necessary before adopting definitive prognostic strategy based on these markers.

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Poster 56

IMPACT OF RED BLOOD CELL TRANSFUSION ON OUTCOME AFTER SUBARACHNOID HEMORRHAGE

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Introduction:
Transfusion of packed red blood cells (PRBC) is associated with increased morbidity and mortality in selected intensive care unit populations. We sought to determine the impact of blood transfusion on outcome after subarachnoid hemorrhage (SAH) in relationship to the degree of anemia and to identify a transfusion threshold.

Methods:
Of 580 consecutive SAH patients enrolled in the Columbia University SAH Outcomes Project between 1996 and 2002 we analyzed 379 who had hemoglobin (Hb) values measured within 3 days of onset. Blood was transfused according to the preferences of the attending neurointensivist. Poor outcome was defined as death or severe disability (modified Rankin Score 4-6) at 3 months. We identified patients who developed moderate (any Hb <10 mg/dl) and severe (Hb <7 mg/dl) anemia and quantified the number of units of PRBCs transfused. The impact of anemia and blood transfusion on outcome was evaluated using multiple logistic regression after adjusting for known predictors of poor outcome.

Results:
Moderate anemia occurred in 68% and severe anemia in 26% of patients; 49% received a mean of 3.7 ± 2.9 units of PRBCs. Poor admission clinical grade (P=0.001), aneurysm clipping (P=0.001) and symptomatic vasospasm (P=0.025) were associated with moderate anemia after adjustment for baseline hemoglobin and hospital length of stay. Symptomatic vasospasm was also associated with severe anemia (P=0.002) and blood transfusion (P=0.001). The number of units of PRBCs transfused (OR 1.3, 95%CI 1.1-1.4, P=0.001), but not moderate or severe anemia, independently predicted poor outcome after adjustment for age, Hunt-Hess Grade, aneurysm size, and aneurysm rebleeding.

Conclusions:
Blood transfusions were given to half of SAH patients during their hospital stay and were associated with mortality and poor functional outcome. Transfusion guidelines directed at more restricted administration of blood products for lower hemoglobin levels may improve outcome after SAH.

References: None

Financial Support: None

Poster 57

DANTROLENE-IV IN THE TREATMENT OF CEREBRAL VASOSPASM AFTER SUBARACHNOID HEMORRHAGE – A PROSPECTIVE PHASE I STUDY

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Introduction:

Cerebral vasospasm (cVSP) after subarachnoid hemorrhage (SAH) is the major cause of disability and death[1]. Available therapies – cerebral angiography with intervention and HHH-therapy – carry significant risks, and there is controversy about the clinical utility of HHH-therapy in cVSP[2]. New therapies are needed. Dantrolene blocks ryanodine receptor-mediated intracellular calcium release from the sarco-endoplasmic reticulum. It attenuates vasoconstriction[3], and is neuroprotective in animal models[4]. We have observed vasorelaxation in three patients with vasospasm/vasoconstriction treated with dantrolene for intractable shivering during Arctic Sun® cooling [personal observation; submitted to NCC meeting 2008]. Based on these promising results, we have designed a prospective study examining the effects of dantrolene on SAH-induced cVSP.

Methods:

The study has been approved by our institutional review board. In a prospective, open-label single-blinded Phase I study in our NeuroICU, SAH patients with elevated anterior-circulation transcranial Doppler (TCD) velocities suggesting cVSP will be enrolled. After baseline TCDs, patients will receive a one-time infusion of dantrolene over 60 minutes with dose escalation (first five patients 1.25mg/kg, the following five patients 2.5mg/kg). TCDs will be repeated at 45, 60 and 135min after infusion start. All other parameters will be kept unaltered during this period, so that physiological data (BP, HR, ICP, CPP, body temperature, pH, p_aCO_2, serum Na) can be followed. LFTs before and 24hrs after the infusion will be assessed.

Results:

The primary outcome measure (mean change in peak systolic TCD flow velocity) will be compared to baseline at 45, 90, and 135min after start of the dantrolene infusion. Secondary outcome measures are mean change SBP, MAP, ICP and CPP at the same time points, and mean change in ALT, AST and alkaline phosphatase at 24 hours after the infusion. Statistical analysis of the response profile with a linear regression model for longitudinal data is planned.

Conclusions:

We expect the study to be ongoing at the time of the meeting. We anticipate to have enrolled the first five patients, and will present available data at the meeting.

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Financial Support: None

Poster 58

EVALUATION OF NORADRENERGIC INPUTS ON THE VASOPRESSIN SECRETION DURING SEPTIC SHOCK.

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Introduction:

Septic shock is the most frequent cause of death in intensive care units with a mortality ranging from 40% to 60%. The central nervous system controls a wide range of physiological functions that are crucial to maintain homeostasis and to orchestrate the host response at behavioural, neuroendocrine and autonomic levels. A relative vasopressin (AVP) deficiency is observed in about one third of septic shock patients and our work group has previously demonstrated that the content of AVP decreased specifically in the supra-optic nucleus (SON) but is preserved in the paraventricular nucleus (PVN). We decided to study the noradrenergic inputs of these structures, particularly the locus coeruleus (LC), in order to evaluate their impact on the AVP secretion.

Methods:

We performed a neuropathological study on septic rats and on patients who died from septic shock. Sections of the LC were studied by immunohistochemistry using norepinephrine, tyrosine hydroxylase (TH) and dopamine beta-hydroxylase (DBH) antibodies. Septic rats and patients were compared with non-septic rats and patients. 52 rat brains and 12 patients were evaluated.

Results:

We observed that the TH content of noradrenergic neurons in the LC increased during the septic shock in rats in comparison with non-septic rats. This could be explained by the activation of the noradrenergic pathway during sepsis in order to maintain the AVP secretion. Nevertheless, this observation is different in human where the TH content is decreased in patients with sepsis.

Conclusions:

The administration of AVP on septic patients have been already evaluated. Our study allows to better understand the mechanisms involved in the regulation of the hypothalamic nuclei which are implicated on AVP secretion. The discrepancies observed between rats and humans could be explained by the longer time course of septic shock in patients than in rats.

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Financial Support: None

Poster 59

PREDICTORS OF OUTCOME IN WARFARIN-RELATED INTRACEREBRAL HEMORRHAGE

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Introduction:
Warfarin-associated intracerebral hemorrhage (ICH) is becoming a more common problem as the use of this medication increases with the aging of the population. We performed a detailed clinical and radiological study to assess predictors of mortality and functional outcome in patients with warfarin associated ICH.

Methods:
Retrospective study of clinical and radiological information of 88 patients with warfarin-associated ICH. All CT scans were reviewed for volumetric analysis of hematoma and perihematomal edema volume. Outcome variables included hematoma enlargement, functional outcome, based on modified Rankin Scale (mRS) score, and mortality.

Results:
Seven-day mortality (rate 39.7%) was associated with lower GCS sum score, larger volume of hemorrhage, and hematoma expansion. Univariate analysis revealed that lower GCS sum score, larger initial ICH volume and higher initial and 48-hour maximum glucose levels were significantly associated with poor functional outcome. In multivariate analysis, GCS and ICH volume remained statistically significant. Conversely, level of INR at presentation, time to INR correction, initial BP, and enlargement of edema had no correlation with functional outcome. Initial systolic blood pressure was the only significant predictor of ICH expansion on multivariate analysis. Neither glucose level on admission nor highest level during the first 48 hours had any correlation with ICH or parenchymal edema enlargement. Also, neither initial INR nor time to INR correction correlated with expansion of ICH or parenchymal edema

Conclusions:
Lower level of consciousness at presentation, hematoma expansion, and possibly hyperglycemia predict poor prognosis in patients with warfarin-associated ICH. Initial systolic hypertension is the main risk factor for hematoma enlargement.

References: None

Financial Support: None

Poster 60
CENTRAL HYPOVENTILATION SYNDROME (ONDINE'S CURSE) DUE TO ACUTE DEMYELINATING ENCEPHALOMYELITIS
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Introduction:
Acquired central hypoventilation syndrome can be a challenging and deadly complication of brainstem injury. We describe a case of ADEM involving the brainstem in a young patient who made a full neurological recovery.

Methods:
We cared for a patient with ADEM involving the brainstem. Some of her manifestations were autonomic dysfunction including central neurogenic hypoventilation. We characterized her lesion by exam and MRI and her respiratory patterns and arterial blood gas changes during wakefulness and sleep. In addition, we reviewed the literature to address the mechanisms of this patient's respiratory abnormalities and developed special insights our case provides regarding management.

Results:
We documented the patient's wakeful respiratory pattern (10 breaths/min, PaCO2 51) and sleep associated hypoventilation (4 breaths/minute, PaCO2 66) characterized by decreased minute volume ventilation due to bradypnea with preservation of the tidal volume. This pattern resolved after five weeks along with her other accompanying neurological impairment. Insights will be provided regarding successful and safe management.

Conclusions:
Central hypoventilation syndrome can be associated with ADEM. Respiratory rate and partial pressure of carbon dioxide in the arterial blood should be carefully observed in a sleeping state in patients with lesions of the brainstem or high cervical cord. Anticipatory and dynamic care strategies can allow good survival in selected patients with this potentially deadly complication.

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6. Financial Support: None

Poster 61
HYPERTENSIVE THERAPY INDUCED INTRACRANIAL VASCULOPATHY: SERIES OF 2 PATIENTS.

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Introduction:
Midodrin (Proamatine) is commonly used oral drug for orthostatic hypotension and hypertensive therapy in acute ischemic stroke; with increasing use in Neuro critical care to induce hypertension and to bridge patients off the systemic vasopressor agent. The safety of its use in the NeuroICU has not been documented. This is the first report of two cases with Midodrin induce vasculoathy in NeuroICU patients.

Methods:
We report two cases of intracranial vasculopathy diagnosed by conventional DSA and CT Angiography (CTA) after treatment with midodrin to induce hypertension in patients with fibromuscular dysplasia and Takayashu arteritis.

Results:
1. 17 year old girl with diagnoses of Takayasu's arteritis that presented with occlusion of the great vessels, and treated with midodrin for hypoperfusion and pressure dependent symptoms.
2. 53 year old female with fibromuscular dysplasia presented with multiple dissections treated with midodrin for hypoperfusion and pressure dependent symptoms.
Management/Imaging/Outcome
Both patients presented with generalized seizures after approximately one week of therapy with midodrin. CTA demonstrated diffused multifocal nonocclusive intracranial narrowing of the intracranial circulation (patients 1, 2). Patient 1 was taken off midodrin without any further neurological symptoms and improvement of the diffuse narrowing on the repeat CTA. Patient 2 was tapered off to lower dose of midodrin remaining clinically asymptomatic.

Conclusions:

Our study demonstrate a potential effect of the oral vasopressor agent, Midodrin, on the intracranial vasculature with diffuse vasoconstriction and vasculopathy in one patient with FMD and a second patient with Takayasu's arteritis. We believe that in our patient, the symathomimetic effect of midodrin on the alpha receptors of the intracranial vasculature induced the intracranial vasculopathy in already pre-existing abnormal vasculature. Therefore, Caution may be exerted when using Midodrin in NeuroICU for hypertensive therapy, particularly in patients with FMD or arteritis.

References: None

Financial Support: None

Poster 62

EARLY VASOSPASM IN ANEURYSMAL SUBARACHNOID HEMORRHAGE

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Introduction:

Early vasospasm (EVSP), defined as transient arterial narrowing shortly after subarachnoid hemorrhage (SAH), is an uncommon and poorly described clinical complication and hence, little data are available delineating the occurrence and sequelae of EVSP at the time of aneurysmal rupture. Here we describe a) a patient in whom aneurysmal coiling was complicated by rupture and EVSP was demonstrated during angiography and b) discuss the possible etiologies of EVSP and its impact on patient outcome.

Methods:

A 48 year-old patient with sentinel headache days prior to admission was diagnosed with a right middle cerebral artery (MCA) aneurysm. Angiogram revealed a 5.8 by 3.9 cm MCA bifurcation aneurysm with a focal rupture point on its lateral aspect; no vasospasm was identified. After detachment of the first coil, intracranial contrast extravasation from the aneurysm dome was observed and within seconds the patient became hemodynamically unstable. Repeat carotid angiogram disclosed spasm and slow contrast clearance of the proximal and distal MCA branches, subsequent injections after immediate stabilization of the patient demonstrated complete occlusion of the aneurysm as well as normal filling and diameters of the intracranial arteries. Despite aggressive therapy the patient was declared brain dead after 24 hours.

Results:

Angiographic reconfirmation of EVSP at the time of aneurysm is obtained. Review of the literature identifies that a) previous SAH and thick subarachnoid clot are well known predisposing factor for EVSP; b) EVSP is identified in about 10% of SAH patients and c) EVSP is highly predictive of cerebral infarction, hydrocephalus, and neurological worsening as well as worsening outcome at 3-month; but d) not predictive of late vasospasm.

Conclusions:

Acute intracranial pressure changes provoked by aneurysm rupture can induce EVSP, as angiographically verified in this patient. In larger series, EVSP is not infrequently observed on initial angiogram in aneurysmal SAH patients (~10%) and it is a clinically useful predictor for increased in-hospital complications and worsening prognosis in SAH.

References: None

Financial Support: None

Poster 63

CARDIAC ARRHYTHMIAS FOLLOWING ACUTE LESIONS OF MEDULLA OBLONGATA

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Introduction:

Mesencephalic, pontine and medullary structures of the brain stem control the autonomic nervous system and involuntary somatic functions. The most common autonomic problems of lesions in these areas include abnormalities in heart rate and blood pressure regulation.
We report three patients with medullary lesions with the subsequent development of arrhythmias and cardiopulmonary arrest.

Methods:

Clinical history, neurological evaluation, and cranial imaging results were reviewed from the medical records.

Results:

Three patients, ages 39-80 with intracerebral hemorrhage (n=2) and dysmyelinating lesion (n=1) of the medulla presented with symptoms of contralateral hemiparesis, dysarthria, and in one case ptosis, diplopia, and ataxia. One patient suffered initial asystolic cardiac arrest and was successfully resuscitated and continued to recover for one month until suffering a sudden unwitnessed cardiopulmonary arrest. The remaining patients experienced no initial arrhythmias but suffered sudden cardiopulmonary arrest approximately 1-2 weeks after the incident cerebral pathology.

Conclusions:

While the occurrence of lethal arrhythmias in patients with brainstem lesions is atypical, their possibility should lower the threshold for prolonged cardiac monitoring, even after periods of stability are achieved. The utility of more extensive initial cardiac electrophysiological testing in all patients with brainstem lesions is of uncertain significance but may represent an important area of future study. Results of such study may lead to guidelines for cardiac intervention such as pacemaker and/or defibrillator implantation in patients with brainstem hemorrhages or other pathologies.

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Financial Support: None

Poster 64

TIMING OF RISE IN ICP AFTER SEVERE TRAUMATIC BRAIN INJURY.

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Introduction:

Though it is generally accepted that ICP rises 24-72 hours after TBI, our patients show a secondary or late rise in ICP. Late intracranial hypertension is rarely described in the literature.

Methods:

46 patients with severe TBI treated between 3/2006 and 7/2007 were included. Hourly ICP values for the first 10 days were reviewed. Statistical analysis was performed using Logistical Regression Analysis, Fisher's Exact Test, and Mix Effects Repeated Measures.

Results:

N=46, 93% male, age 35, GCS 5.3, 34.8% mortality, Toxicology: EtOH 34%, methamphetamine 13%, THC 21% cocaine 6.5 %. Five ICP patterns were identified: 1)no rise, 15%, 2)Early 24%- ICP elevation 0-72 hours, 3)Continuous- elevated ICP beginning 0-72 hours and remaining elevated, 17% 4)Bimodal- initial rise within first 72 hours which improves and then rises again (peak days 8.9), 24% 5)Late- >72 hours, 20%. ICP and mortality: Early(OR 0.57, CI 0.032,10.25) Late(OR 0.34, CI 0.005,8.09) Bimodal(OR 2.8, CI 0.29,42.34) Continuous(OR 2.35, CI 0.2,39.5) all P values non-significant. 54% received mannitol 0-72 hours, 46% received hypertonic saline (HS) 0-72 hours most patients received both mannitol and HS. Mannitol and pattern of ICP: Early- Hazard Ratio(HR) 0.78 p=0.62, Late- HR 3.51 p=0.03, Bimodal- HR 5.57 p=0.004, Continuous-HR 6.42 p=0.008 HS and pattern of ICP: Early- HR 0.18 p=0.004, Late- HR 0.14 p<0.001., Bimodal- HR 0.63 p=0.43 Continuous- HR 1.15 p=0.84

Conclusions:

ICP patterns differed in those who died vs.survivors(p=0.0338). Patients who died had more hours with ICP>20. Patients with a bimodal or continuous ICP rise demonstrated a 2.819 and 2.349 fold increased risk of death, respectively(p>0.05).
Mannitol correlated with late, bimodal, and continuous rises in ICP. This association does not prove causality. HS had no correlation with pattern of ICP. No correlation was found between use of alcohol or drugs and pattern of ICP. Cocaine users had a 7 fold increased likelihood of death(p=0.0321)

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Financial Support: None

Poster 65

NURSE PRACTIONER PROCEDURAL SAFETY AND EFFICACY IN THE NEUROINTENSIVE CARE UNIT

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Introduction:
Collaborative practice between advanced practice nurses, nurse practioners, and physicians is a recent trend in neurocritical care. The roles and competencies of nurse practioners in neurocritical care has heretofore not been well studied. Evaluation and benchmarking of profiency, safety and efficacy of nurse practioners is invaluable in crafting the lanscape of practioners in the future.

Methods:
A prospective study of the incidence of adverse complications for three commonly performed neurocritical care procedures was performed over a two year period for a team of nurse practioners working full time in a 19 bed academic neurocritical care unit. The three procedures were subclavian central venous access, arterial line placement, and placement of intravascular cooling devices. The procedural complications that were surveyed were: death, serious cardiac arrythmia and hypotension, pneumothorax, line sepsis, bleeding, unintended arterial cannulation or ischemia. Proctoring of 20% of procedures were performed by the attending physician, and blinded adjudication was performed by an independent physician. Rates of complications were compared with second year neurocritical care fellows for comparison. Procedures were judged to be satisfactory as judged by standard criteria (xray localization, arterial waveform, blood aspiration).

Results:
A total of 1580 procedures were performed in the two year study by a team of two nurse practioners and 2 fellows. The complication incidence rates for the entire team for each procedure was a follows: death (0%), serious cardiac arrythmia and hypotension (0%), pneumothorax (3%), line sepsis (3%), bleeding (1%), unintended arterial cannulation or ischemia (0.4%). The rates for the nurse practioners were not statistically different than that for the fellows: death (0%), serious cardiac arrythmia and hypotension (0%), pneumothorax (2%), line sepsis (1%), bleeding (0%), unintended arterial cannulation or ischemia (0.3%). Satisfactory performance of the procedures occurred in 99% of procedures.

Conclusions:
Nurse Practioners perform invasive procedures safely in the neurocritical care unit. Evaluation of overall performance of cognitive and procedural duties is presently underway.

References: None

Financial Support: None

Poster 66

SAFETY OF PARENCHYMAL ICP MONITORING IN ACUTE LIVER FAILURE

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Introduction:

One of the most deadly complications from acute liver failure (ALF) is cerebral edema (CE) and elevated intracranial pressure (ICP). ICP monitoring (ICPM) allows proactive management to optimize cerebral perfusion in these patients who are prone to hypotension and elevated ICP. However, use of ICPM is often hampered by reluctance to its insertion due to coagulopathy and other factors.

Methods:

We reviewed our experience with a standardized approach to ICPM in 17 consecutive patients with ALF. The decision for ICPM and its insertion at our center is by one of four neurocritical care physicians. Monitor type (parenchymal), insertion location (right posterior frontal) and depth, and approach to coagulopathy reversal is uniform and does not require normalization of the INR.

Results:

All of our 17 patients (6 men, 11 women) had coagulopathy (INR 1.4-9.4). Clinical factors and post ICPM imaging showed no clinically consequential bleeding complications. 1 had a small subdural hematoma without mass effect or midline shift, but his cause of death was from CE. 9 survived, and only 2of the deaths were from CE. The qualitative impact of ICPM on management will be discussed.

Conclusions:

1. Parenchymal ICPM can be performed safely in ALF patients and coagulopathy
2. More proactive use of parenchymal ICPM should become a standard both to enhance care and discover how to improve outcome from this deadly problem
3. Neurocritical care physicians (non-neurosurgeons) can safely place parenchymal ICPM and should be encouraged to learn this procedure

References: None

Financial Support: None

Poster 67

TEMPORARY PARTIAL AORTIC OCCLUSION FOR THE TREATMENT OF VASOSPASM FOLLOWING ANEURYSMAL SUBARACHNOID HEMORRHAGE

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Introduction:
A tri-lumen catheter with supra- and infra-renal aortic balloons improves cerebral perfusion by diverting blood from the lower half of the body. This device may improve symptomatic vasospasm refractory to hypertensive, hypervolemic, hemodilutional therapy (HHT) and intra-arterial vasodilators following aneurysmal SAH.

Methods:
A registry was established of patients age 18 to 85 years treated with the NeuroFlo™ catheter. After obtaining IRB-approved informed consent, seven patients who failed to neurologically improve with HHT and intra-arterial vasodilator therapy underwent intra-aortic placement of the NeuroFlo™ device. The infra-renal, then supra-renal balloons were inflated to attempt to achieve a 70% stenosis and inter-balloon gradient of 10 to 20 mm Hg for a total of 45 minutes. NIHSS, and modified Rankin Scales (mRS) were recorded.

Results:
Six women and one man ages 38 to 72 (mean 45) years were treated endovascularly in 2 patients and surgically in 5. Symptoms from vasospasm failed to improve with HHT and intra-arterial papaverine, verapamil or nicardipine. NIHSS prior to NeuroFlo™ use ranged from 10 to 32 (mean 19). NIHSS decreased 3-13 points (mean 6.7) in 6 patients and worsened by one point in one patient. Clinically significant improvement was immediately noted in 3 patients whose NIHSS improved by 8 or more. By discharge, NIHSS ranged from 0 to 23. Discharge mRS was 0 to 2 in all patients who achieved an inter-balloon gradient of $\geq$14 mm Hg. In patients with gradients of 4-9 mm Hg, mRS was 4 to 5 except in one patient (mRS 0) who improved with repeat angioplasty following NeuroFlo™.

Conclusions:
Symptomatic vasospasm refractory to HHT may improve with a novel strategy of cerebral collateral recruitment by temporary partial aortic balloon occlusion. A higher inter-balloon gradient may be essential to improve cerebral perfusion.

References: None

Financial Support: None

Poster 68

THE PACIFICA OXIMETER: A NOVEL SPECTROPHOTOMETRIC DEVICE FOR MONITORING TISSUE OXYGEN SATURATION.

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Introduction:

The Pacifica system is a novel tissue oximeter that enables noninvasive monitoring of blood oxygen saturation levels in deep tissue layers, particularly in the brain. The readings of the Pacifica system provide a universal measurement of tissue oxygen saturation, namely, the readings for subjects with similar tissue states are consistent. This study aims to show that the Pacifica system is capable of safely and accurately measuring cerebral oxygen saturation levels in anesthetized, ventilated animals during controlled manipulations of tissue oxygen levels.

Methods:

Ten young piglets were anesthetized and ventilated. Continuous measurements of tissue oxygen saturation were obtained by placing the Pacifica sensor over the forehead. Venous blood was drawn through a CVP catheter inserted into jugular bulb level on the ipsilateral side, as determined by contrast fluoroscopy. Arterial blood was drawn from the ipsilateral Carotid artery. Hypoxia was induced by lowering the inspired oxygen fraction (FiO_2). Cerebral oxygen saturation was calculated based on a 3:1 ratio of venous blood to arterial blood, as measured by direct co-oximetry.

Results:

A total of 72 blood samples were drawn during the manipulations. The Pacifica system's readings at the blood sampling times were recorded. The readings of the Pacifica system are significantly correlated with oxygen saturation levels of cerebral blood, measured by co-oximetry in all animals. The within animal correlation coefficient over the 10 animals was calculated using an analysis of covariance model. The correlation coefficient (r) for all animals was 0.85 ($p<0.001$).

Conclusions:

This study demonstrates that the Pacifica system can accurately monitor brain tissue oxygen saturation levels in anesthetized and ventilated piglets.

References: None

Financial Support: Employment (authors 1-5)

* BMI = body mass index = weight (kg)/meters^2

Poster 70

OBESITY IS ASSOCIATED WITH REDUCED BRAIN TISSUE OXYGEN TENSION AFTER TRAUMATIC BRAIN INJURY

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Introduction:
Obesity is associated with increased mortality after traumatic brain injury (TBI).(1) The underlying reasons are poorly understood. Pulmonary dysfunction, in particular, is prominent in obese patients due to reduced lung volumes and increased ventilation/perfusion mismatching.(2, 3) The impact of obesity-related pulmonary dysfunction on outcome after TBI is unknown. The brain is critically dependent on oxygen delivery after TBI, and reduced brain tissue oxygen tension ($PbtO_2$) is associated with poor outcome.(4) We tested the hypothesis that obesity is associated with reduced $PbtO_2$ after TBI.

Methods:
Consecutive obese (BMI* $\geq$ 30) and non-obese (BMI = 18.5 – 24.9) patients with severe TBI (Glasgow Coma Scale score $\leq$ 8) who underwent continuous $PbtO_2$ monitoring were identified from a prospective single-center database of TBI patients. For each group, average daily $PbtO_2$, average minimum daily $PbtO_2$, average maximum daily $PbtO_2$, and average daily fraction of inspired oxygen (FiO_2) were calculated. Student t-tests were used to compare the means for each group.

Results:
Data from each group were normally distributed. Obese patients had consistently lower $PbtO_2$ values than non-obese patients. For 16 obese and 19 non-obese patients respective means ($\pm$SD) for daily $PbtO_2$ were 24.43$\pm$10.55 and 31.71$\pm$10.24 (p=0.047), for maximum daily $PbtO_2$ were 38.47$\pm$17.09 and 51.13$\pm$14.95 (p=0.026), for minimum daily $PbtO_2$ were 15.31+8.05 and 18.77+10.85 (p=0.30), and for daily FiO_2 were 64.17$\pm$17.73 and 58.27$\pm$15.19 (p=0.30).

Conclusions:
In patients with severe TBI, obesity may be associated with reduced $PbtO_2$. Larger studies are needed to confirm this association and to determine the extent to which reduced $PbtO_2$ independently contributes to increased mortality in obese patients after TBI.

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Financial Support: None

Poster 71

VENTILATOR ASSOCIATED PNEUMONIA IN STATUS EPILEPTICUS

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Introduction:

Status Epilepticus (SE) commonly leads to endotracheal intubation and mechanical ventilation (MV). Ventilator Associated Pneumonia (VAP) affects intubated patients frequently and increases nosocomial mortality, morbidity and cost. We studied the incidence and characteristics of VAP in SE patients

Methods:

We performed a retrospective review of patients with SE admitted to two NeuroCritical Care Units from 2000 - 2005 who were endotracheally intubated and on MV greater than 48 hours. We identified those with VAP by: 1. New, persistent focal infiltrate on CXR 48hrs post initiation of MV 2. Evidence of high-risk microorganism 3. Documented high clinical suspicion 4. Sputum cultures correlating with fever and abnormal WBC

Results:

Of the 59 episodes of SE requiring endotracheal intubation and MV, 9/59 (15%) had VAP, with an incidence density of 18.3/1000 ventilator days. The overall length of stay (LOS) was longer with VAP: 40.1±1days compared to without VAP: 20.4±3days, (mean±SEM, p=0.02); similarly MV duration was longer with VAP: 12±2days than without VAP: 7±1days (p=0.02). There was no group difference in age and gender although VAP was significantly associated with SE recurrence within 24 hours (p=0.03), low antiepileptic drug (AED) level as SE etiology (p=0.02) and use of agents for pharmacologic coma (0.04) on univariate analysis but only with pharmacologic coma (p=0.04) and low AED level as etiology (p=0.008) on multivariate analysis. Micro-organisms in VAP group were gram-negative bacteria (n=5); MRSA (n=4), MSSA (n=1) and diphtheroids (n=1). Tracheostomy was performed in 4/9 with VAP and 9/50 without VAP. Mortality was 1/9 with VAP and 8/50 without VAP

Conclusions:

Patients with SE are at high risk for VAP especially those subjected to pharmacologic coma for treatment of SE and with subtherapeutic AED levels as etiology of SE. VAP is associated with longer LOS and MV duration

References: None

Financial Support: None

Poster 72

NEUROSARCOID MASQUERADING AS POSTERIOR REVERSIBLE LEUKOENCEPHALOPATHY SYNDROME (PRES)

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Introduction:

Sarcoidosis is a systemic illness with the pathologic hallmark of noncaseating granulomas.
The prevalence of nervous system involvement is between 5-15% and may involve either the peripheral and central nervous systems.[1,2]
Posterior Reversible Leukoencephalopathy Syndrome (PRES) is a radiological entity clinically characterized by a variable combination of headache, visual changes, altered mental status and seizures. PRES is diagnosed by clinical presentation and transient changes on MRI due to vasogenic edema in the parieto-temporal occipital regions. Abrupt changes in blood pressure, seizure and various medications have been reported as potential precipitants.

Methods:

We present clinical and imaging features, patient course and literature review relevant to our patient with an established diagnosis of sarcoidosis (via renal biopsy) who developed a radiographic picture masquerading as PRES.

Results:

Extensive review of current literature did not reveal any reports of neurosarcoidosis mimicking PRES on MRI imaging. Although the non-infused portion was suggestive of PRES, subsequent gadolinium enhanced imaging revealed diffuse leptomeningeal enhancement consistant with active neurosarcoidosis.

Conclusions:

- Neurosarcoidosis can present with imaging features reminiscent of PRES.
- Neurosarcoidosis can be differentiated from PRES by its rapid radiographic resolution of post-gadolinium enhancement after initiation of IV steroid therapy.

Suspected cases of PRES should include gadolinium enhanced images to aid in proper diagnosis and initiation of appropriate treatment.

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Financial Support: None

Poster 73

IMAGE GUIDED ENDOSCOPIC EVACUATION OF SPONTANEOUS INTRACEREBRAL HEMORRHAGE

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Introduction:
Spontaneous intracerebral hemorrhage (ICH) is a devastating disease with high morbidity and mortality. ICH lacks an effective medical or surgical treatment despite the acknowledged pathophysiological benefits of achieved hemostasis and clot removal. Frameless stereotactic endoscopic hematoma evacuation is a promising minimally invasive approach designed to limit operative injury and maximize hematoma removal.

Methods:
A single center randomized controlled trial was designed to assess the safety and efficacy of frameless stereotactic hematoma evacuation compared to best medical management. Patients were randomized within 24 hours of hemorrhage in a 3:2 fashion to best medical management plus endoscopic hematoma evacuation or best medical management alone. Data was collected to assess efficacy and safety of hematoma evacuation as well as to identify procedural components requiring technical improvement.

Results:
10 patients have been enrolled and randomized to treatment. Six patients underwent endoscopic evacuation with a hematoma volume reduction of 80% +/-13 at 24 hours post procedure. The medical arm demonstrated a hematoma enlargement of 78% +/- 142 during this same period. Rehemorrhage rates and deterioration rates were similar in the two groups. Mortality was 20% in the endoscopic group and 50% in the medical treatment cohort. Apparent Diffusion Coefficient abnormalities were present along the rim of two hematomas randomized to endoscopic treatment and both improved with hematoma evacuation. The endoscopic technique was shown to be effective in identification and evacuation of hematomas while reduction in the number of endoscopic passes and maintenance of hemostasis require further study.

Conclusions:
Stereotactic frameless endoscopic hematoma removal is a promising minimally invasive technique that is effective in immediate hematoma evacuation. This technique deserves further investigation to determine its role in ICH management.

References: None

Financial Support: None

Poster 74
IMPROVING OUTCOMES FOR SEVERE TBI
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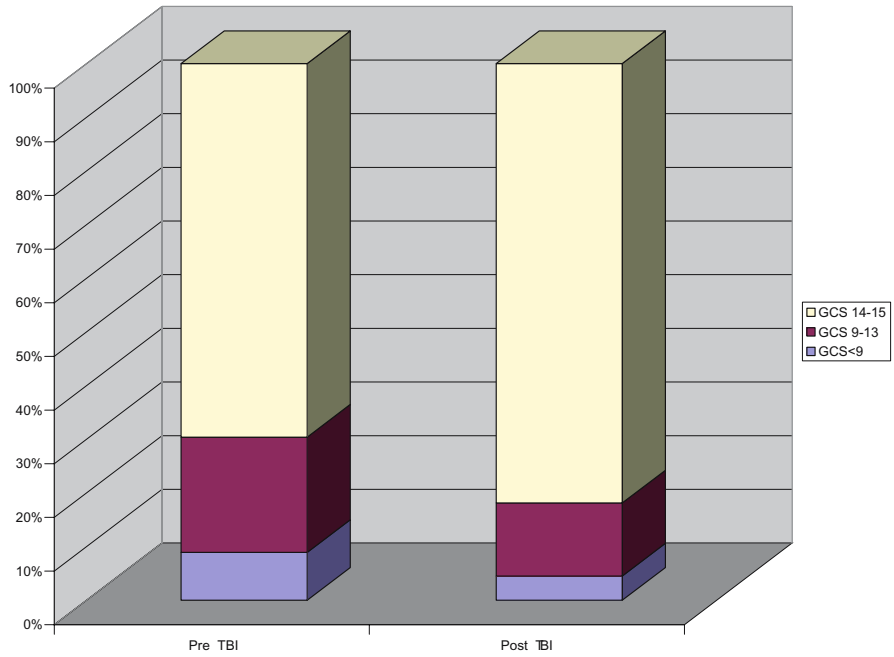
Introduction:
Brain Injuries are a primary form of death/disability after trauma. The Adam Williams Traumatic Brain Injury Initiative, an educational program, assists trauma centers in adopting the AANS/Brain Trauma Foundation (BTF) traumatic brain injury (TBI) guidelines and use of multimodality monitoring in order to improve TBI care and outcomes. Our hospital was selected for this initiative July 2005. This project was to evaluate if discharge outcomes have improved after guidelines were implemented in January 2006.

Methods:
A retrospective chart review of 148 severe TBI patients admitted from 2000-2006 (group 1) was compared to 40 prospective post-guideline (group 2) from March 2006- June 2007. Data was entered into the BTF's TBI-trac database. Variables included age, sex, admission GCS (aGCS), ICU length of stay (LOS), discharge GCS (dGCS), and mortality.

Results:
N=188 patients, ICU LOS and discharge GCS calculated on non-transferred surviving patients. Group 1/group 2: age 40/36, male 78/95%, aGCS 4.4/4.8, ICU LOS 18/17, dGCS 3-8: 9/4% , 9-13: 21/14% GCS 14-15: 70/82%, mortality 33/ 32% Discussion: aGCS, ICU LOS and mortality were not significantly changed likely due to small sample size. When adjusted for differences in gender frequencies/ age, there were statistically significant differences in: 1) dGCS (12.8 ± 3.06 vs. 13.45 ± 2.02 $p< 0.001$) and 2) aGCS to dGCS in group 2 7.35 ± 4.80 to $8.36 \pm .01$ $p<0.001$. Group 2 patients had higher dGCS, which can not be attributed to the initial difference in GCS, the difference in gender ratio or age. This difference is likely a result of increased proportion of patients with GCS >14, but can not be confirmed statistically because of a small sample size. Additional data is needed to determine statistical significance. 6-9 month post injury Glasgow Outcome scale scores would allow for better outcome measurement.

Variable	2000-2006 Pre TBI guideline development (n= 148)	2006-2007 Post TBI guideline development (n=40)
Age (mean)	40 (16-85)	36 (14-69)
Sex		
Males	78%	95%
Females	22%	5%
Admission GCS (mean)	4.4	4.8
ICU LOS, days (mean) Excludes those transferred	18 (n=101)	17 (n=37)
Monitoring device		
ICP	Not doc	95%
ICP + Brain oxygen	0%	70%
D/C status (excludes transferred and deceased)		
GCS <9	9% (7/79)	4% (1/22)
GCS 9-13	21% (17/79)	14 % (3/22)
GCS >13	70% (55/79)	82% (19/22)
Transfered	15% (22/148)	7% (3/40)
Deceased/mortality	33% (41/126)	32% (12/37)
	Missing data points for 5 pts	2 still in hospital



Conclusions:

- When adjusted for differences in gender frequencies and age, there are statistically significant ($p<0.001$) differences in the 2 groups:
  - Mean GCS at discharge for the two groups (12.80 ± 3.06 vs. 13.45 ± 2.02).
  - Change in GCS from admission to discharge in the post-TBI guideline group (7.35± 4.80 vs. 8.36 ± 2.01).
- The post-TBI group patients have a significantly higher GCS at discharge, which can not be attributed to the initial difference in GCS, to difference in male/female ratio or the age of patients. This difference is mainly a result of increased proportion of patients with GCS >14, which can not be confirmed statistically at this point because of a small sample size.
- Study limitations include small sample size for the post-TBI guideline group and inability to obtain GOS scores.

References: None

Financial Support: None

Poster 75

BILIRUBIN OXIDATION PRODUCTS ARE ABUNDANT IN THE CSF OF TBI PATIENTS.

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Introduction:

Edema remains a leading cause of morbidity and mortality following moderate to severe traumatic brain injury (TBI). Bilirubin oxidation products (BOXes) have been associated with cerebral vasospasm after aneurysmal subarachnoid hemorrhage (CV after aSAH) and edema development following intracerebral hemorrhage, both in animal models and in patients. The formation of BOXes in the CSF of SAH patients is, unsurprisingly, dependent on the presence of bilirubin (a metabolite of hemoglobin), and an oxidizing environment, which may result from immune cell-recruitment to the hematoma.

Methods:

CSF was collected from TBI patients (Mean GCS = 5, n=4). Control CSF was collected from hydrocephalus outpatients requiring therapeutic CSF. Assays for various components were performed as follows: (i) Protein (BCA method Pierce); (ii) Bilirubin, (Jendrassik-Grof); (iii) Hemoglobin (Drabkin's) ; (iv) Malondialdehyde (an indicator of oxidative stress) (commercially available kit, Calbiochem) and (v) BOXes, using a method developed by the author. In order to account for the inherent differences between patients in this small cohort, data was normalized to total protein content.

Results:

The following table summarizes the data obtained.

	Hemoglobin *mg/mg protein*	**Bilirubin** *mg/mg protein*	**MDA** *nMoles/mg protein*	**BOXes** *nMoles/mg protein*
Control (n=4)	<0.01	0.009 ± 0.0009	0.199 ± 0.017	<0.02
TBI CSF (n=4)	0.035 ± 0.02	0.063 ± 0.009	1.42 ± 0.037	28.7 ± 1.2

Conclusions:

While levels of bilirubin and oxidative stress appear to be comparable SAH CSF from non-vasospastic patients, the BOXes concentrations far exceed those seen in even vasospastic SAH patient CSF. We could postulate that bilirubin is acting as an antioxidant, thus reducing bilirubin and oxidative damage while producing BOXes. Interestingly, cell culture and *ex vivo* studies show that low BOXes levels cause potentiation of vessel constriction, whereas high BOXes concentrations (as reported here) caused vascular smooth muscle actin disorganization, cell rearrangement, and death. This could indicate a role for BOXes in TBI-related edema.

References: None

Financial Support: None

Poster 76

RESULTS OF A SALARY SURVEY OF PHYSICIAN MEMBERS OF THE NEUROCRITICAL CARE SOCIETY

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Introduction:
Neurocritical care is a relatively new discipline and its practitioners come from a variety of backgrounds and practice in a variety of settings. It would be expected that salaries differ based on variables such as geographic location, practice setting, and time spent on clinical effort. None of the features of the practice and compensation of a neurocritical care physician has been examined in any systematic fashion.

Methods:
A survey was emailed to all members of the Neurocritical Care Society. 106 members responded to the survey, and 86 members included salary information that was included in the final analysis. Information was also collected about ICU directorship, primary appointment, practice setting, hospital type, geographic location, percent effort on clinical responsibilities, sources of income including salary incentives, patient population, board certification and subspecialty training, etc.

Results:
The mean salary of all respondents (n=86) was $214,100. The mean salaries for academic vs. private practitioners were $201,400 and $258,600, respectively. The neurologists who served as ICU directors (n=31) mean salaries' were $207,900 and $247,500 depending on whether the primary department was neurology (n=25) or neurosurgery (n=6), respectively. Most respondents spent at least some time working on consults, research, or administrative duties. The majority of respondents (n=37) practice in the northeast (mean salary=$221,100; n=32), but mean salaries were highest in the southwest ($264,200; n=6).

Conclusions:
The information gathered in this survey enhances the understanding of the current practice of neurocritical care throughout the United States. These data may be valuable to hospital administrators trying to assess the feasibility of developing a neurocritical care program, for neurointensivists negotiating contracts, and to neurologists-in-training as a way of generating interest in neurocritical care as a career choice.

References: None

Financial Support: None

Poster 77

THE UTILITY OF PERFUSION CT IN PREDICTING RISK OF INTRACEREBRAL HEMORRHAGE FOLLOWING THROMBOLYSIS IN ACUTE ISCHEMIC STROKE.

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Introduction:

Intracerebral hemorrhage is the most serious complication following intravenous thrombolysis treatment with tissue plasminogen activator(tPA). Several clinical and imaging parameters have been found to help in estimating risk of hemorrhagic transformation.

The use of CT perfusion is becoming more common in evaluating acute strokes. It provides a reliable indicator of cerebral perfusion and the degree of perfusion deficit in the infarcted area. We reviewed CT perfusion hemodynamic parameters in an effort to identify factors which may be useful to estimate bleeding risk following thrombolytic treatment with IV tPA. In addition, we sought to establish guidelines for identifying the regions of interest (ROI) or area where the perfusion calculations are performed.

Methods:

We performed a retrospective review of our database between 01/01/2005 and 01/01/2006 for patients who were treated with Iv tPA within 3 hours from stroke onset and underwent perfusion CT prior to treatment.

Clinical and demographic data collected included age, sex, temperature, glucose and blood pressure. In addition, CT perfusion data included cerebral blood flow, cerebral blood volume and the time to peak.

Results:

45 patients were identified, 6 patients developed cerebral hemorrhage larger than 6 mm. diameter. Means ± SD for perfusion parameters; Cerebral blood flow, cerebral blood volume, and time to peak were 0.7±0.3, 0.8±0.2, 52.3±25.9 for the group with no bleeding and 0.7±0.3, 0.8±0.2, 39.9±24.1 for the bleeding group respectively. The difference between the two groups was not significant. Clinical and demographic data collected included age, sex, temperature, glucose and blood pressure. For the non- bleeding group: 68.3±17, 15M, 24F, 97.8±0.6, 132.8±55.8, 125.7±23.9, and for the bleeding: 80.3±7.4, 3M, 3F, 97.4±0.6, 112.7±33, 135.3±14 respectively. Only age was significantly different between the two groups.

Conclusions:

The use of CT perfusion provides important hemodynamic data during acute ischemic stroke. Data acquired from patients reviewed from 2005-2006 database was not predictive of hemmorhage transformation after IV thrombolysis.

Better methodology to define region of interest may improve specificity of data collected.

We proposed a novel method to define the region of interest ROI on perfusion CT to include the areas of major vessel distribution. Larger prospective studies using a better defined regions of interest are needed to evaluate the predictive value of CT perfusion in identifying patients who have high risk for hemorrhagic transformation.

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Financial Support: None

Poster 78

LOCALIZATION OF BRAIN INJURY FROM EXTREME HYPERAMMONEMIA

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Introduction:

Metabolic encephalopathy is associated with acute severe hyperammonemia (HA) typically due to acute liver failure (ALF). Many of these patients also develop cerebral edema due to accumulation of intra-astrocytic glutamine. Within the astrocytes, glutamine is osmotically active promoting cellular swelling, and it also causes mitochondrial dysfunction, energy failure and ultimately astrocytic necrosis. Protoplasmic astrocytes seem more selectively vulnerable to these changes.

Methods:

We present 3 patients with variable causes of extreme HA to illustrate unique, important insights about HA-associated brain injury.

Results:

Case 1: ALF from transplanted liver with acute rejection; Case 2: acute severe HA after TIPS placement for portal hypertension; Case 3: acute severe HA from newly diagnosed ornithine transcarbamylase (OTC) deficiency. All 3 developed coma and cerebral edema with mild intracranial hypertension without compromised cerebral perfusion. Acute MRI in all 3 showed bilateral, symmetric high signal changes (T2, FLAIR) involving: medial frontal, temporal and subinsular cortex, lenticular nuclei, medial thalamus and head of caudate. In one case there was also restricted diffusion in these regions.
Patient 1 died due to multiple organ failure; patient 2, while showing slow neurological improvement, life-sustaining treatments were withdrew according to previously expressed wishes and the third patient survived making a full neurological recovery.

Conclusions:

1- Hyperammonemia causes characteristic changes in specific brain regions, perhaps related to more selective vulnerability of protoplasmic astrocytes to elevated ammonia.
2- While the associated MRI changes with HA are alarming, their mechanism is likely related to astrocyte swelling and/or necrosis without obligated neuronal injury. This supports their potential reversibility and associated functional clinical recovery as illustrated in the cases 2 and 3.
3- It is best to avoid pessimistic neurological prognostic statements based on MRI changes in patients with HA who did not suffer concurrent injury from hypoperfusion or other mechanisms.

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Financial Support: None

Poster 79

AGGRESSIVE BLOOD PRESSURE REDUCTION MAY BE ASSOCIATED WITH IMPAIRED PERI-HEMATOMA TISSUE PERFUSION IN PATIENTS WITH INTRACEREBRAL HEMORRHAGE

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Introduction:

Patients with intracerebral hemorrhage frequently have markedly elevated blood pressure. The impact of hypertension on hematoma volume expansion remains controversial yet reducing markedly elevated blood pressure is the standard of care. The 2007 AHA guidelines for the management of spontaneous ICH recommend that blood pressure be reduced to MAP<110 mmHg or <160/90 mmHg. ICP monitoring is recommended in cases were elevated ICP is suspected. Although Powers et al suggested that modest blood pressure reduction does not affect peri-hematoma blood flow, studies evaluating real time tissue perfusion are few.

Methods:

Three patients with spontaneous subcortical ICH underwent multimodal monitoring in our ICU. Intracranial pressure (global) was monitored via an external ventricular drain (EVD) as well as with the use of a fibreoptic pressure catheter (Licox system) placed within 1.0 cm of the hematoma. Brian temperature and tissue oxymetry (PbtO2) was also monitored at this site. Consecutive data was collected; the sampling rate was once per hour and 287 hours of data were included in the final analysis. Data were included if the nurse recorded the ICP, MAP, and $PbtO_2$.

Results:

Models using SAS v9.1 (Cary, NC) were used to explore ICP, MAP and CPP as predictors of PbtO2. ICP ($F=5.30$, $p=.0220$) was a significant predictor of PbtO2 as was MAP ($F=20.49$, $p<.0001$) and CPP ($F=32.14$, $p<0.001$). When we adjusted the model for CPP, ICP was not an independent predictor of PbtO2. CPP was however an independent predictor of PbtO2 when the model was adjusted for ICP. Based on this model, CPP was able to predict 10% of the variance in PbtO2 ($r2 = .1014$).

Conclusions:

Tissue perfusion in the region surrounding a hematoma is directly affected by the patient's MAP and ICP. Blood pressure reduction should be guided by the effects on global as well as focal tissue perfusion. Further investigation is required to explore the ideal perfusion pressure required to maintain tissue viability while reducing the potentially harmful effects of sustained hypertension.

References: None

Financial Support: None

Poster 80

"LESSONS LEARNED," IN THE NEURO-CRITICAL CARE MANAGEMENT OF ACUTE SPINAL CORD INJURY

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Introduction:

Spinal cord injury (SCI) is a devastating illness that affects more young people than the elderly. These patients given the opportunity may attain amazing instances of rehabilitation. Identified are ten historically problematic areas in the critical are management of the patient with SCI that result in secondary injury and delays in transfer to rehabilitation facilities. These problems include: 1. Difficult airway, 2. Hypotension, 3. Hypoxemia, 4. Pneumonia, 5. Deep venous thrombosis (DVT) & pulmonary emboli, 6. Decubitus ulcers, 7. Ileus, 8. Bradycardia, 9. Anemia, 10. Spinal hardware failure. The pitfalls in managing each of these problems and the corresponding solutions are presented.

Methods:

Six selected cases of SCI cared for at a major University Neuro-Critical Care Unit over the past six years are reviewed with particular emphasis placed on the identification of problems. Iatrogenic pulmonary edema with hypoxemia, unrecognized neurogenic shock, loss of the airway, persistent pneumonia, fatal pulmonary emboli, refractory ileus, crisis intervention with respiratory failure and asystole, and the loss of three levels of neurological function due to hardware failure were reviewed.

Results:

This retrospective cohort review concludes that: early hemodynamic monitoring in the poly-trauma patient to prevent iatrogenic pulmonary edema, earlier recognition and treatment with pressers of neurogenic shock, and high tidal volume ventilation in the patient with high cervical SCI to prevent pneumonia are all under used but effective management strategies. DVT prevention with prophylactic low molecular weight heparin, medication for symptomatic bradycardia, and anticipation of respiratory failure and timely proper airway management are essential.

Conclusions:

Patients with acute SCI continue to suffer the consequences of less than ideal critical care management of well recognized problems due to the failure of acceptance or the application of known treatment interventions. University Neuro-Critical Care Units must take the lead in outlining, implementing, and educating "Best Practices" in the management of the patient with acute SCI.

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Financial Support: None

Poster 81

CATCH 22 OR DOUBLE-EDGED THERAPY? THE COMBINED USE OF RECOMBINANT FACTOR VIIA AND INTRAVENTRICULAR TPA FOR COAGULOPATHIC INTRAVENTRICULAR HEMORRHAGE.

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Introduction:
Patients with fulminant hepatic failure (FHF) may develop cerebral edema and raised intracranial pressure (ICP). Emergent or prophylactic external ventricular drain (EVD) placement is problematic due to coagulopathy. We report a patient with FHF who underwent EVD placement and developed subsequent intraventricular hemorrhage (IVH), which was treated with recombinant factor VIIa (rFVIIa) for hemostasis and intraventricular tPA for thrombolysis.

Methods:
Case Report.

Results:
We report a 50-year-old female with liver cirrhosis and coagulopathy admitted to our ICU with FHF. Emergent EVD was inserted after rFVIIa and fresh frozen plasma was administered to normalize the prothrombin time. Three days later, the patient developed an intracerebral hemorrhage with IVH along the EVD insertion site and left third nerve palsy. The patient received a subsequent dose of rFVIIa. The patient stabilized neurologically and medically to proceed with OLT. A few hours postoperatively, the patient declined neurologically had worsened IVH and hydrocephalus on CT with cerebrospinal fluid flow obstruction. We instilled 2mg rtPA via the EVD catheter and transduced intracranial pressure for 2hours before opening to drain. Subsequent CT showed reduction in hydrocephalus and IVH. The patient improved neurologically and eventually had EVD removal on postoperative day 3. The patient was discharged from the ICU with no neurological deficits except for trace partial left third nerve palsy.

Conclusions:
Coagulopathic patients with intracerebral and intraventricular hemorrhage pose a vexing management problem with a high mortality[1-3]. Coagulopathy can be rapidly corrected by rFVIIa, allowing emergency neurosurgical intervention[3-5]. Procedure-related or coagulopathic IVH is an unfortunate complication of EVD placement but may be successively treated with low dose intraventricular rTPA to expedite IVH resolution. Future studies are needed investigating combined therapies of hemostatic control with rFVIIa and intraventricular thrombolysis on clinical outcomes.

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Financial Support: None

Poster 82

THERAPUTIC HYPOTHERMIA IN SALICYLATE TOXICITY

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Introduction:

Cerebral edema is a lethal complication of salicylate toxicity that requires aggressive management. Therapeutic hypothermia may effectively control elevated ICP while awaiting removal of salicylate through hemodialysis.

Methods:

47 year old African American female admitted with a week's history of worsening headache, disorientation, slurred speech and ataxic gait. She had chronic daily headaches and self-medicated with Aspirin at doses ranging from 3250 mg to 6500 mg/day (5-10 tablets of 650mg). In the ED, the patient abruptly became unresponsive (GCS 5) with a fixed and dilated right pupil. Emergent intubation and aggressive ICP management with hypertonic saline, mannitol, and therapeutic hypothermia was initiated with successful reversal of the uncal herniation syndrome. CT scan confirmed the presence of diffuse cerebral edema. Serum Salicylate level was elevated at 55mg/dl for which hemodialysis was initiated. EEG revealed generalized slowing and the MRI revealed toxic demyelination in the deep white matter and brainstem, and cerebral edema. Successful control of ICP was achieved with the hypothermia for 24 hours. Upon rewarming, patient demonstrated neurologic recovery and was ultimately discharged without any neurologic deficits.

Results:

CNS manifestations of chronic salicylate intoxication include agitation, confusion, slurred speech, hyperventilation, seizures and cerebral edema.[1-4] The nonlinear, saturable kinetics of salicylate elimination result in repeated doses eventually accumulating in tissue[1] Hence chronic toxicity manifests at much lower salicylate levels and progresses more slowly often going unrecognized until severe complications arise. Hemodialysis is treatment of choice in life threatening acute (salicylate levels > 90-100mg/dl) and chronic (levels >40-50mg/dl) poisoning. In addition to controlling cerebral edema, therapeutic hypothermia may also provide neuroprotection against salicylate toxicity by decreasing oxygen and glucose metabolism[6, 7], improvement of ion homeostasis[8, 9], blocking of free radical production[10, 11]and preventing hyperpyrexia.

Conclusions:

Therapeutic hypothermia is efficacious in the management of Cerebral Edema in Salicylate toxicity

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Financial Support: None

Poster 83

OREXIN A AS A MARKER OF EARLY RESPONSIVENESS AFTER TRAUMATIC BRAIN INJURY

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Introduction:

Traumatic brain injury (TBI) is a significant health concern. The ability to predict outcome from TBI is an important but complicated issue. Efforts to identify biomarkers of outcome after TBI are in progress, but currently none are available. Orexin has been identified as a neurotransmitter in arousal pathways. Presently, there is limited data on orexin levels after TBI. We proposed that cerebrospinal fluid (CSF) orexin levels would correlate with injury severity and outcome.

Methods:

This was a prospective pilot study of severe TBI (GCS $\leq$8) patients who required intracranial pressure (ICP) monitoring. Cerebrospinal fluid (CSF) was collected at baseline and every 72 hours for duration of ICP monitoring. Orexin A levels were measured in CSF by ELISA. Each subjects' mean Orexin A level was compared with initial GCS, evidence of substance abuse, hospital discharge GOS, and 6-month outcome measures.

Results:

Seventeen patients were enrolled, of which 15 (88%) were male. Mean initial GCS was 6 (range 3-14). Eight subjects had evidence of substance abuse on admission. Mean discharge GOS was 3 (range 1-4) and follow up GOSE was 4.2 (range 1-8). Orexin A levels ranged between 0-207 pg (mean 49.2, normal > 200), and were higher in younger patients and those with evidence of substance abuse ($p=0.03$). Orexin levels correlated with dichotomized GOS at discharge ($p=0.043$), but this was not maintained at long-term follow-up.

Conclusions:

This small study found that measurement of Orexin A levels early after TBI is possible. The orexin level may be able to differentiate whether a decreased level of consciousness is related to intoxication versus actual brain injury. In addition, orexin may play a role as a marker of early responsiveness after TBI. Further studies need to be performed with a greater number of subjects to confirm these findings.

References: None

Financial Support: None

Poster 84

MENINGOENCEPHALITIS WITH DYSKINESIA

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Introduction:

We report five cases of meningoencephalitis with dyskinesia with a similar presentation and evolution, without causative agent found.

Methods:

Five female patients – 24 to 38 year old – had meningoencephalitis ; altered level of consciousness, change in personality, fever, seizure. Dyskinesia came early in 2 cases and at the awakening of patients in 3 cases. Involuntary movements started and remained predominant at the face, but involved all four limbs, with a choreoathetosic or myoclonic jerk aspect and required long lasting sedation.

Results:

All patients had a lymphocytic meningitis, a normal MRI, slow waves at EEG; no infectious agents was identified and no autoimmune origin was found despite extensive testing. Dyskinetic movements disappeared after month, with neuropsychiatric deficit mild to important.

Conclusions:

These meningoencephalitis cases with dyskinesia, a common manifestation of basal ganglia diseases have a presentation similar to sporadic cases of encephalitis lethargica syndrome. The acute onset with fever suggests an infectious or a dysimmune post-infectious origin; a paraneoplastic origin (opsoclonus-myoclonus) can also be suspected in some cases.

References:

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Financial Support: None

Poster 85

SUCCESSFUL THROMBECTOMY IN ACUTE MCA STROKE IN A 13 Y/O CHILD USING THE MERCI RETRIEVAL SYSTEM

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Introduction:

Acute ischemic stroke (AIS) is rare in children, but is increasingly diagnosed. There are no randomized, controlled trials addressing the treatment of AIS in children other than in patients with sickle cell disease. The utility of thrombolytic therapy for AIS in children is limited due to lack of consensus guidelines, poor reperfusion efficacy, risk of hemorrhagic complications, and short treatment time window. We report the case of a 13 y/o female with acute MCA stroke who underwent thrombectomy with the MERCI Retrieval System.

Methods:

The patient presented with a large right MCA stroke with a NIH Stroke Scale of 25. Risk factors included Down Syndrome, previously repaired endocardial cushion defect, and use of oral contraceptives. Emergent angiography revealed complete occlusion of the right MCA. Successful mechanical thrombectomy of a complete MCA occlusion was performed using the MERCI Retrieval System with removal of a large thrombus from the MCA. Angiography revealed recanalization of the MCA immediately after the procedure. The child regained left lower extremity movement within 6 hrs and had return of left upper extremity movement within 72 hrs. NIH Stroke Scale score at one month was 4.

Conclusions:

Frequently, the treatment of AIS in children is anticoagulation and supportive care, with concomitant poor outcomes. The MERCI Retrieval System has a favorable safety and efficacy profile compared to thrombolytic therapy in adults with an extended time window of eight hours. To our knowledge this is the youngest patient for whom the MERCI Retrieval System has been utilized. Pending results of larger clinical trials, mechanical thrombectomy should be considered for AIS in children. Pediatricians should be aware of this potential treatment option and interventionalists should be familiar with some of the technical challenges of thrombectomy in the pediatric population.

References: None

Financial Support: None

Poster 86

THE EARLY USE OF INTRAVENOUS NEOSTIGMINE FOR PREVENTION OF BARBITURATE-ASSOCIATED ILEUS AND NECESSITY FOR PARENTERAL NUTRITION IN NEUROSURGICAL PATIENTS IN BARBITURATE COMA: CASE SERIES AND PRELIMINARY OUTCOMES

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Introduction:

It is frequently observed that patients with severe traumatic brain injury requiring induction of barbiturate coma for intractable intracranial pressure will fail enteral feedings due to barbiturate-associated paralytic ileus[1]. Both barbiturate-associated ileus and use of parenteral nutrition are associated with significant risks. Furthermore, it has been established that early enteral nutrition in critically ill patients is associated with less complications than parenteral nutrition[2]. The objective of this study was to evaluate the combined efficacy of intravenous neostigmine infusion with an aggressive bowel protocol to prevent intolerance of enteral feedings and the necessity for parenteral nutrition

Methods:

Retrospective data were collected for patients with severe traumatic brain injury that were treated with barbiturate coma for intractable intracranial pressures, as defined by the American Association of Neurological Surgeons (AANS) guidelines. All patients were placed on a protocol with scheduled intravenous neostigmine of 2 mg q6 hours to 2 mg q1 hour supplemented with scheduled intravenous metoclopramide, scheduled enteral naloxone, along with an aggressive bowel regimen that included scheduled docusate, fleets enema, and lactulose. All patients were enterally fed via a nasoduodenal feeding tube. Patients in whom enteral feedings provided inadequate nutrition were placed on additional parenteral nutrition

Results:

Three patients were induced into barbiturate coma due to intractable intracranial pressure secondary to severe closed head injury from blunt force trauma, of which two patients also required decompressive hemicraniectomy. Two patients were male, and patient ages spanned 20-23 years. The mean length of barbiturate infusion was 9 days, with a mean length of neostigmine infusion of 9 days. The mean time to stool after initiation of neostigmine infusion was 1.7 days, and a mean time of achieving goal enteral feedings was 3 days. All patients remained on enteral nutrition. One patient required parental nutrition in addition to enteral feedings, which were held intermittently for multiple procedures. There were no significant side effects associated with neostigmine treatment.

Conclusions:

The preliminary outcomes from this case series suggest that barbiturate-induced ileus and use of parenteral nutrition may be prevented in patients induced with barbiturate coma for treatment of intractable intracranial pressure by early use of intravenous neostigmine and aggressive bowel care. A prospective study is planned for further investigation of this protocol, with emphasis on systematic nutritional assessment

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Financial Support: None

Poster 87

SAFETY OF DEXMEDETOMIDINE (DEX), FOR >24 HOURS, IN PATIENTS WITH SUBARCHNOID HEMORRHAGE (SAH) AND ICP MONITORING USING VENTRICULAR DRAINS

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Introduction:

There is little published information regarding the safety of dex in patient with SAH. Dex is an excellent agent used for sedation in SAH patients because of its properties allowing the patient to be calm, yet providing the staff the ability to do an accurate neurological exam. Known side effects of Dex are hypotension and bradycardia. Little is known about the effect of Dex on ICP. The purpose of this study was to gather information on dosage, length of treatment, ICP effect and adverse effects of dex in patients with SAH.

Methods:

Data was retrospectively collected from the records of 28, (11 women and 17 men) SAH patients in the ICU who received Dex from July 2003 to January 2007. A standard order sheet was used for the administration of Dex. Dex was titrated to a sedation scale. A loading dose was not used. Upper limit of dex was 2.5 mcg/kg/hr. The length of time on dex was not limited. Parameters for limiting dosage of Dex were, sedation level, predetermined heart rate, goal MBP, or if already on norepinephrine, the prescribed upper limit dose of norepinephrine to maintain goal MBP for cerebral perfusion

Results:

28 patients where treated for a total of 135 days of dex. The longest time for a patient on dex was 15 days. Avg number of days on dex was 4.8 days. The average dose of dex was 0.8mcg/kg/min. Maximum dose was 2.7 mcg/kg/min. During the 135 days there where 26 episodes of decreased BP and 15 episodes of slow HR that correlated with dosing of dex.

Although events were related to increasing dex, there was no max dose that was 100% associated with adverse events. All events resolved with lowering dose of dex. There was no correlation between ICP and dose of dex.

# of patients	# episodes slow HR	# of patients	# episodes decreased BP
21	0	16	0
3	1	2	1
3	2	7	2
1	9	2	3
		1	4

Conclusions:

Dex can be safely used as a sedating agent for patients with SAH and ventricular drains with ICP monitoring. Dosing in this population was greater than the FDA approved dosage of 0.7mcg/kg/hr. Patients tolerated doses up to 2.2 mcg/kg/hr without significant increase in adverse events. Patients also tolerated infusion time up to 15 days, which is longer than FDA approved 24 hrs. There appears to be no effect of dose on ICP.

References: None

Financial Support: None

Poster 88

THE USE OF SURFACE ENHANCED LASER DESORPTION IONIZATION (SELDI), IN THE SERA OF PATIENTS WITH ANEURYSMAL SUBARACHNOID HEMORRHAGE TO PREDICT VASOSPASM.

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Introduction:
Vasospasm is a complication of aneurysmal subarachnoid hemorrhage. Identifying proteins that predict the occurrence of vasospasm would improve early detection.

Methods:
We collected the serum from 20 patients with aneurysmal subarachnoid hemorrhage on day 1-3 after hemorrhage. The clinical diagnosis of vasospasm was made in ten of these patients and confirmed by angiography. We compared the early proteomic profiles from serum of those who developed vasospasm with those who did not.
All samples were thawed in 9M Urea/2% CHAPS. Each Sample was run in duplicate on 4 different Protein Chip Surfaces (Q10, CM10< IMAC30, and H50). Reproducibility was assessed by samples run on Ciphergen PBSIIc at 2 different laser intensities and instrument settings to optimize peak detection. Obtained spectra were mass-aligned, baseline-subtracted and normalized by TIC before being subject to a peak picking algorithm (Ciphergen Express software 2.1).

Results:
Analysis of the protein profiles involved complex multivariate analysis. A number of protein peaks were identified that offered potential discrimination. The mean intensity between peaks was compared using a Mann Whitney test with significance P value less than 0.001. Spikes of interest elevated in the no vasospasm group included the 9.3 kD peak from the CM10 chip as well as a 2.1 kD peak from the Q10 chip. Increased expression of proteins associated with the vasospasm group included a51.3 kD and a 3.3 kD peak from the Q10 chip, as well as a 4.15 kD and a 12.4 kD peak from the CM10 chip.

Conclusions:
The use of SELDI –MS identified candidate vasospasm biomarkers in the prevasospasm serum of patients with subarachnoid hemorrhage. Characterization may provide a means to identify patients at risk for vasospasm.

References: None

Financial Support: None

Poster 89

INTRA-AORTIC BALLOON COUNTERPULSATION (IABP) IN THE SETTING OF ANEURYSMAL SUBARACHNOID HEMORRHAGE (SAH), CEREBRAL VASOSPASM, AND CARDIAC STUNNING

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Introduction:

The management of symptomatic cerebral vasospasm after SAH can be often complicated by the presence of stunned myocardium and left ventricular failure. Vasopressors/Inotropes are commonly used to optimize MAP and CPP. We would like to add, to the limited amount of case reports, our experience with a complimentary or potentially alternative method for hemodynamic augmentation

Methods:

Case report and literature review describing the use of IABP in a patient with symptomatic vasospasm after SAH

Results:

This is a 55 year old man, admitted with an ACom Aneurysm rupture and a Hunt Hess II, Fisher grade IV SAH who developed severe cerebral vasospasm treated with volume optimization, pressors and bilateral MCA angioplasty. Subsequently developed bilateral patchy infiltrates with hypoxemic respiratory failure requiring mechanical ventilation and cardiogenic shock on multiple pressors/inotropes. Rescue therapy with an IABP was employed with improvement in shock and neurological status. No complications were encountered with IABP use, he was discharged functionally independent

Conclusions:

In the literature we identified less than 15 patients with SAH and vasospasm been treated with the assistance of an IABP. Based on this limited experience of ours and others, Intra-aortic balloon counterpulsation can be used with success for patients with hemodynamic collapse in the setting of Subarachnoid Hemorrhage complicated by cerebral vasospasm and left ventricular failure to support the circulation and maintain adequate cerebral blood flow

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2. Spann, Lang, Birch, Lamb, Neil-Dwyer: Intra-Aortic Balloon Counterpulsation: Augmentation of Cerebral Blood Flow after Aneurysmal Subarachnoid Haemorrhage Acta Neurochir (Wien) 143: 115-123, 2001

Financial Support: None

Poster 90

DIFFERENTIAL GENE EXPRESSION OF THE CEREBRAL CORTEX AFTER SHAM AND CECAL LIGATION AND PUNCTURE.

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Introduction:

Brain dysfunction associated with sepsis is poorly understood and has a wide spectrum of manifestations ranging from lethargy to overt delirium. It is common in the ICU setting and is becoming widely recognized to have serious prognostic significance. A large Veterans Administration study revealed a mortality of 49% in patients with sepsis and acute mental status changes compared to a mortality of 26% in patients with normal mental status. With numerous hypotheses postulated, it becomes obvious that this is a complex process. Because of this complexity, and lack of a basic understanding of encephalopathy associated with sepsis, we looked at whole genome expression in the brain of septic and non-septic mice as a hypothesis generating investigation.

Methods:

Eight C57 mice underwent either CLP or SHAM surgery. Animal procedures were reviewed and approved by the Institutional Animal Care and Use Committee. Before and after surgery, all mice underwent two methods of behavioral testing. After 4 hours the animals were anesthetized then euthanasia followed by craniotomy and their cerebral cortex recovered for RNA isolation. This RNA was isolated, then hybridized to the MU 74 Av2® oligonucleotide chip. The groups were compared using the Fisher's Exact test setting a p value < 0.05 with a minimal fold change of 1.5.

Results:

Animals in the CLP group had deterioration in their performance scale using the OPC (Overall Performance Scale) and in the Neuro-deficit scores compared to the SHAM group. Microarray analysis resulted in 405 genes that were up regulated in the CLP group and 221 genes that showed suppressed expression in the CLP group. Annotation of the differentially expressed genes revealed the greatest differences were in several major annotation groups: coagulation, neurotransmission, apoptosis, zinc metabolism, hormone regulation, and neuro-ligand receptor interaction pathways.

Conclusions:

The genomic expression pattern of the brain after systemic infection is complex and involves many differing pathways. The complexity of these interactions are probably further advanced by co-existing diseases, age, medications, length of illness, etc. The study identifies several pathways that warrant further investigation into the understanding of this complex syndrome such as the coagulation system, zinc metabolism and neurotransmission that may offer some therapeutic insights.

References:

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Financial Support: None

Poster 91

USE OF IV LORAZEPAM PLUS SLOW IV PUSH LEVETIRACETAM TO TERMINATE NONCONVULSIVE STATUS EPILEPTICUS

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Introduction:
Nonconvulsive status epilepticus (NCSE) is common in the neurocritical care unit. Some medications used to treat status epilepticus such as phenobarbital, phenytoin and benzodiazepines can cause hemodynamic instability and respiratory depression, which further complicate ICU management. Levetiracetam is available intravenously and is not known to cause hemodynamic instability or respiratory depression, so it appears to be safer in critically ill patients.

Methods:
A 69 y.o. woman with a right subdural hygroma was undergoing continuous digital EEG monitoring for recurrent episodes of NCSE after an initial presentation of convulsive status. Boluses of phenobarbital, phenytoin and lorazepam large enough to stop the NCSE had previously prolonged her dependence on the ventilator and caused hypotension. During a subsequent instance of NCSE, the consulting epileptologist ordered a bolus of 10mg/kg of phenobarbital. While waiting for the phenobarbital to arrive from the pharmacy, a regimen of IV lorazepam and slow IV push levetiracetam was given to try to avoid the previous complications.

Results:
Two rounds of 2mg lorazepam IV and 1000mg levetiracetam slow IV push were given before the phenobarbital bolus was administered. A review of the EEG record afterward revealed that the NCSE had stopped before the phenobarbital was infused. The slow IV push levetiracetam was well tolerated.

Conclusions:
The safety profile of IV levetiracetam makes its use in treating NCSE an attractive prospect. To our knowledge, there are no other cases presented in the medical literature of the combination of IV lorazepam and slow IV push levetiracetam terminating a case of NCSE, nor are there are any cases reporting the administration of slow IV push levetiracetam. More studies need to be done to determine if this treatment is as efficacious as standard therapies, including lorazepam alone.

References: None

Financial Support: Dr. Wright and Dr. Asbury are on the Speakers Board for UCB Pharma

Poster 92

DELAYED ISCHEMIC STROKE AFTER AORTIC ARCH/GREAT VESSEL SURGERY

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Introduction:

Late ischemic stroke in patients with a remote history of aortic arch/great vessel surgery has been rarely reported.[1,2] We describe 2 cases of ischemic stroke in young patients occurring years after aortic arch/great vessel surgery without other identifiable mechanism for stroke.

Methods:

Retrospective case series.

Results:

Case Series: Case #1: A 33 year-old male status-post aortic coarctation graft repair 18 years prior to presentation developed acute vertigo, nausea, and headache with subsequent left hemiparesis, hemisensory loss, and hemianopsia. Initial NIH stroke scale was 8. Neuroimaging was consistent with a large right posterior cerebral artery infarct and bilateral cerebellar infarcts. Evaluation for stroke was unrevealing, including hypercoagulable testing and transesophageal echocardiogram. Case #2: A 33 year-old male with a history of Ehlers Danlos syndrome type IV with multiple great vessel aneurysms status-post right common carotid-vertebral bypass graft 20 years ago and status-post left common carotid-subclavian bypass graft 2 years prior to presentation developed acute aphasia and right hemiplegia. Initial NIH stroke scale was 20. Neuroimaging was consistent with a left middle cerebral artery distribution infarction and left intracranial internal carotid artery occlusion. Further testing revealed occlusion of the left common carotid artery. We present 2 cases of late stroke in the young occurring years after great vessel surgery. Both patients presented with large volume infarcts years after uneventful aortic arch/great vessel surgery.

Conclusions:

Ischemic stroke as a late complication of great vessel/aortic arch surgery is not well described. Patients with graft-surgery of the aortic arch or great vessels should have continued long-term monitoring for potential delayed neurologic complications.

References:

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2. Matuura K, Ogino H, Matsuda H, et al. Multivariate analysis of predictors of late stroke after total aortic arch repair. Eur J Cardiothorac Surg. 3, 473-7, 2005.

Financial Support: None

Poster 93

INTRODUCING THE DESIGN OF A NEW CENTRAL LINE CATHETER WITH ABILITY TO ACCESS THE JUGULAR BULB

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Introduction:

Oxygen saturation of the jugular bulb ($SjvO_2$) is an indicator of the balance between brain oxygen delivery and consumption. In the appropriate clinical setting ($SjvO_2$) monitoring can be valuable in guiding patient management and predicting prognosis [1]. An access to the blood exiting the brain can also give information about brain metabolism and byproducts. Although ($SjvO_2$) monitors global brain oxygenation, sudden change especially after an intervention (e.g. hyperventilation, aneurysm clipping) can be indicative of brain ischemia and promote corrective measures.

Currently to access to the jugular bulb a catheter is advanced retrograde in the internal jugular vein (IJ), in opposite direction to the much accustomed central line placement. The idea of using a normal central line catheter with an additional port with the ability to access the jugular bulb encouraged the author to design this new catheter.

Methods:

The external appearance of this catheter is similar to central line catheters. One of the ports gives rise to a cannula which after making a U turn within the catheter opens at its proximal end. Placing a micro catheter or a fiberoptic catheter through this port will give access to the jugular bulb. This catheter could be placed similar to any other central line through the IJ, making it a much user friendly monitoring device. The port leading to the jugular bulb is marked to avoid inadvertent injection or fluid administration.

Results:

The new catheter is in the initial design stage. Input from neuro-critical investigators and clinicians will help in its development.

Conclusions:

Most patients in neuro-critical care setting as well as those undergoing neurovascular procedures receive a central line. The newly designed catheter will allow access to the jugular bulb for sampling and oxygen saturation monitoring.

References:

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Financial Support: The medical device company IVPSA Inc. has paid for the rights to evaluate this device for one year.

Poster 94

TREATMENT OF HYPERGLYCEMIA IN NEUROLOGICALLY CRITICALLY ILL PATIENTS: A SURVEY OF PRACTICE VARIATIONS

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Introduction:
Intensive insulin therapy targeted to glucose levels of 80-110 mg/dL has been shown to reduce morbidity and mortality in critically ill surgical and medical patients. The ideal glucose target in critically ill neurological patients is not known.

Methods:
To determine current glucose control preferences in the care of neurologically critically ill patients, we surveyed attendees at the onset of the 2007 New York Symposium of Neurological Emergencies and Neurocritical Care.

Results:
Of 363 registrants, 124 (35%) responded to the survey. Most registrants (47%) came from New York, New Jersey or Connecticut, 49% came from other U.S. states and 4% were foreign. Of responders, 29% were intensivist physicians, 33% were non-intensivist physicians and 32% were nurses. Most (49%) of responders work in a medical-surgical ICU, and 33% work in a neurosciences ICU. Though the median lower and upper acceptable limits for glucose were 80 and 140 mg/dL, 11% of responders would accept glucose levels >200 mg/dL and 26% would accept glucose values as low as 60 mg/dL. The median ideal range for glucose was 80-120 mg/dL, however, 25% preferred a higher target of 140-150 mg/dL and 7% percent of responders preferred an upper target as high as 160-180 mg/dL. Neuro-trained physicians tolerated higher glucoses: 28% tolerated levels of 160-200 mg/dL compared to 9% of intensivists (Odds ratio 5.8, 95% CI 1.5-22.6, P=0.012). The preferred method of insulin delivery was intravenous infusion in 68% and finger sticks and sliding scale subcutaneous insulin in 30%. The biggest obstacles to implementing a protocol included physician and nurse resistance (22% and 18%, respectively), while hypoglycemia presented an impediment for 10%.

Conclusions:
Practices in glucose control in critically ill neurological patients vary widely. Preferences for glucose targets differ based on physician training. More clinical studies are needed to determine ideal glucose ranges in this population.

References: None

Financial Support: None

Poster 95

CLINICAL RESPONSE TO HYPERTENSIVE HYPERVOLEMIC THERAPY PREDICTS OUTCOME IN PATIENTS WITH SYMPTOMATIC VASOSPASM AFTER SUBARACHNOID HEMORRHAGE

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Introduction:
Hypertensive Hypervolemic Therapy (HHT) has been widely used to treat symptomatic vasospasm following subarachnoid hemorrhage (SAH) though little data exists to demonstrate a relationship between early clinical response and outcomes.

Methods:
We prospectively studied spontaneous SAH patients. Patients who developed symptomatic vasospasm received volume expansion with crystalloid or 5% albumin to a goal CVP≥8 mmHg and/or hypertensive treatment with intravenous pressors for a goal SBP of 180-200 mmHg. We assessed the effect of HHT on the neurological exam during the first 2 hours of each intervention and used multivariate logistic regression analysis to calculate adjusted odds ratios (OR) assessing the effect of clinical response to HHT on 3 month outcomes, as measured by the modified Rankin Scale (mRS).

Results:
Of 580 SAH patients, 16% (N=95) developed symptomatic vasospasm and 86% of these patients received HHT. Volume expansion was used in 94% (N=89) and 43% had a clinical response, while 85% (N=81) received pressors and 68% responded. Early clinical response to pressors or volume expansion was not related to the development of infarct on CT due to vasospasm, but was independently protective against death (adjusted OR [aOR] 0.05, 95%CI [0.01-0.3], P=0.001 and aOR 0.05, 95%CI [0.01-0.4], P=0.007; respectively) and death or severe disability (mRS 4-6), (aOR 0.2, 95%CI [0.1-0.8], P=0.016 and aOR 0.2, 95%CI [0.1-0.8], P=0.016; respectively) after adjusting for age, Hunt-Hess grade and aneurysm size.

Conclusions:
SAH patients with symptomatic vasospasm who fail to demonstrate an early clinical response to volume or pressor therapy (HHT) are at long term risk for death or disability. Urgent referral for intra-arterial therapy may improve outcomes.

References: None

Financial Support: None

Poster 96

USE OF THE PORTABLE XENON CT SCANNER TO EVALUATE RCBF IN SEVERE TRAUMATIC BRAIN INJURY AND STROKE PATIENTS IN THE NEURO ICU

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Introduction:
Xenon-enhanced CT (Xe/CT) has gained wide acceptance in the diagnosis and management of patients with severe traumatic brain injury (TBI), the advantages of the method include quantitative assessment of regional cerebral blood flow (rCBF) with anatomic referencing, short investigation time, and high accuracy, even in the low-flow states. The availability of a portable scanner that can perform xenon CT images may lead to more frequent diagnosis of rCBF abnormalities. The purpose of this work is to analyze the data of patients who had a Xe/CT imaging with a portable Xe/CT scanner in the Neuro ICU

Methods:
From June 2006 to June 2007 a total of 34 Xe/CT studies have been done on 26 patients with the portable XeCT scanner in the Neuro ICU. Patients age were from 20 to 72 (mean = 35.52) years old. Primary diagnosis was: 21 patients with severe traumatic head injury, 3 with subarachnoid hemorrhage from aneurysm rupture and 2 with intraparenchymal bleeding from AVM rupture

Results:
Patients had the imaging study as early as 7.6 hrs after the injury (mean = 67.25 hrs). CBF results from slices at 4 different levels were: the average cortical CBF for the first slice was from 14.23 to 109.42 mL/100g/min (mean = 54.53), for the second slice was from 27.17 to 108.92 mL/100g/min (mean = 54.72), for the third slice was from 0 to 100.5 mL/100g/min (mean = 51.63), for the fourth slice from 7.76 to 100.5 mL/100g/min (mean = 54.61); the average of the 4 slices or the final global cortical CBF was from 17.16 to 109.47 mL/100g/min (mean = 51.63). Different therapeutic measures were taken including: increase in blood pressure to achieve a normal CPP, increase in oxygenation, increase in volume status and decompressive craniectomy to decrease ICP and increase CPP; depending on each particular case. All these therapeutic action had a common goal, maintain an adequate CBF.

Conclusions:
The advantage of this mobile portable Xe/CT scanner has the potential to alter physician practices and patient outcome. Unexpected findings on the Xe/CT scanner alters patient management or results in a therapeutic surgical procedures in some cases. We believe that portable Xe/CT could dramatically alter patters and practices on patients with pathologies where the rCBF can be compromised: severe TBI and stroke

References: None

Financial Support: None

Poster 97

SEVERE INTRACEREBRAL HEMORRHAGE AFTER VENTRICULOSTOMY DESPITE CORRECTION OF WARFARIN-ASSOCIATED ELEVATION IN INR WITH RECOMBINANT FACTOR VIIA

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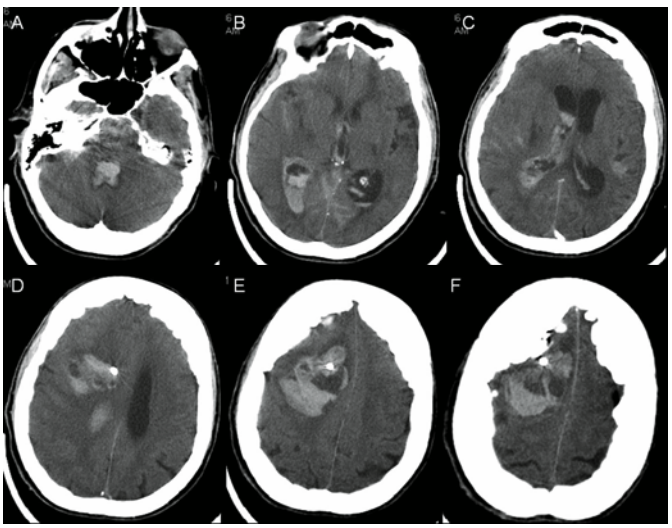
Introduction:
Management of warfarin-associated intracerebral hemorrhage (ICH) focuses on prompt and complete reversal of anticoagulation. Due to the limitations of vitamin K and fresh frozen plasma (FFP) in achieving this goal, there has been emerging enthusiasm for the use of recombinant activated factor VII (rFVIIa) as an acute hemostatic agent. While rFVIIa rapidly normalizes raised international normalized ratio (INR) values in warfarin-treated patients, it remains unclear whether it truly reverses the coagulopathy or provides superior clinical efficacy beyond that of alternative modalities.

Methods:
A single patient case report illustrating the limitations of rFVIIa use in reversing warfarin-induced coagulopathy prior to invasive neurosurgical procedures and suggesting caution in interpreting INR after rFVIIa administration.

Results:
A 79-year old woman receiving warfarin for valve replacement presented with spontaneous intraventricular hemorrhage, hydrocephalus, and declining mental status. Initial INR was 4.95 and remained elevated at 2.34 following administration of intravenous vitamin K and four units of FFP. Prior to ventriculostomy, 2.4 mg (50 µg/kg) of rFVIIa was given, lowering the INR to 0.92. A few hours after an uneventful insertion, patient suffered a fatal ICH along the catheter tract; INR at that time was still 0.90.



Conclusions:
Treatment with rFVIIa in the setting of warfarin therapy replaces only one of the multiple deficient clotting factors. It normalizes the INR by saturating the tissue factor component of the assay; this may not reflect actual reversal of underlying anticoagulation, making the INR unreliable in judging extent of residual coagulopathy in this setting. As demonstrated in this case, treatment of warfarin-associated ICH with rFVIIa does not always appear effective in preventing procedure-related bleeding, and must always be supplemented by more durable interventions.

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Financial Support: None

Poster 98

SAFETY OF PHARMACOLOGIC PROPHYLAXIS FOR VENOUS THROMBOEMBOLISM IN THE NEUROSCIENCE INTENSIVE CARE UNIT

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Introduction:

The *Chest, Brain Trauma Foundation,* and *AHA* issue guidelines to assist in VTE prophylaxis in patients with a variety of neurologic conditions. The data on which their recommendations are based is sparse, and nationwide practices are diverse. The aim of this study is to describe the practice and outcomes in a neuroscience intensive care unit (NSICU) at a major academic tertiary care center.

Methods:

This case series characterizes pharmacologic VTE prophylaxis (ppx) in patients admitted to the NSICU. Data was collected retrospectively by means of chart review, including demographics, admitting diagnosis, onset, duration and type of ppx, and documented complications. Descriptive statistics were applied.

Results:

102 patients were admitted during a 6 month period; diagnosis included ICH (41), SAH (38), TBI (19), SDH (16), SCI (9), brain tumor (12), and other (16.) Of 100 patients analyzed, 8 did not receive ppx, 38 received heparin 5000 units BID, 16 heparin 5000 units TID, 5 enoxaparin 40 mg daily, and 4 enoxaparin 30 mg BID. Twenty-nine patients received multiple regimens. Median time to ppx was 2 days (2, 5), and the median duration was 15.5 days (6.8, 28). Fifteen patients experienced a total of 23 events including CT evidence of intracranial bleed extension, GI bleed, or hematuria. Four events were clinically significant and possibly attributable to ppx, with 1 requiring discontinuation. This was an intracranial hemorrhage (ventriculostomy tract); this patient recovered completely. Seven patients developed clinically detectable VTE.

Conclusions:

This data suggests that ppx initiated very early after event onset may be safe. Despite early ppx, some patients still develop VTE. The optimal regimen and timing of initiation needs to be carefully evaluated with attention to diagnoses, adverse events related to therapy, and treatment failure.

References: None

Financial Support: None

Poster 99

BLOOD PRESSURE CONTROL WITH NICARDIPINE INFUSION IN PATIENTS WITH SUBARACHNOID HEMORRHAGE

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Introduction:
Nicardipine is a dihydropyridine derivative calcium channel blocker, which has potent vasodilatory activities. In this study we are reporting the feasibility and safety of treatment of acute hypertension with nicardipine infusion in patients with subarachnoid hemorrhage (SAH).

Methods:
A pharmacy database and chart review was performed to identify those patients with SAH admitted to the Neuro-ICU and treated with nicardipine in a 20-month period. Hourly blood pressure (BP) measurements on admission, before, during and after the infusion of the drug, failure to reach the preset BP goals, complications and premature discontinuation of the infusion were extracted. ANOVA and Bonferonni post-hoc multiple comparison tests were used to compare the BPs.

Results:
Twenty-one patients with SAH (9 men and 12 women, mean age 58 years) had 38 nicardipine infusions and 2455 extracted systolic and diastolic BP measurements. Nitroprusside was infused 15 times before the nicardipine. Nicardipine was started on the average 81 hours after admission with a mean SBP goal of 153 mm Hg (range 140-180). Duration of the infusion and mean maximum dose were 17 hours and 8.5 mg/hour, respectively. SBP during the infusion was measured 32% times above the preset goal, with a trend for higher percentage failure with lower SBP goals ($p = 0.058$). In 23 infusions more than one antihypertensive drugs were used. Five infusions were stopped prematurely. The mean pre-infusion SBP was significantly lower than the admission SBP (mean 151.5 vs 159.5 mm Hg, $p < 0.0001$) and significantly higher than the infusion and post-infusion SBP measurements (149 and 140 mm Hg, $p = 0.039$ and < 0.0001, respectively).

Conclusions:
Nicardipine infusion was a safe and effective drug to control the BP in patients with SAH. In one third of infusions, however, nicardipine failed to reach the preset goals and additional antihypertensive drugs were administered.

References: None

Financial Support: Panayiotis Varelas has been a speaker for PDL BioPharma, not currently

Poster 100
ASYMPTOMATIC BRAINSTEM EDEMA.
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Introduction:
The posterior reversible encephalopathy syndrome (PRES) has been well defined in patients with hypertensive encephalopathy, eclampsia, and in recipients of immunosuppressant drugs. Typical imaging findings are those of T2 hyperintensities, usually within the cortex and subcortical white matter of the parietal, occipital, temporal, and to a lesser degree, the posterior frontal lobes. Common neurological symptoms on presentation are seizure, altered mental status and focal neurological signs. Asymptomatic brainstem edema has not been reported previously.

Methods:
A 58 year old man with hypertension, alcohol and substance abuse complained of palpitations, shortness of breath and blurred vision. On examination, blood pressure was 260/170. pulse was 95 and respiratory rate was 16. Neurological exam revealed bilateral papilledema without any change in vision, rest of the exam was unremarkable. Labs disclosed thrombocytopenia (44K), BUN 56, creatinine 6.5 and cocaine positive urine. Sodium and serum osmolarity were normal and did not fluctuate. Head CT and MRI showed a swollen pons (34 mm ventral-dorsal, normal = 25 mm) with T2 hyper intense lesions extending into the cerebellum, midbrain, cerebral peduncles and a posterior limb of the right internal capsule. DWI sequence was normal.

Results:
Blood pressure was decreased slowly to 163/106 over a period of four days. Repeat MRI four days later revealed that pontine thickness had decreased to 29 mm. Edematous changes in the other structures had almost completely resolved as well. The patient remained neurologically stable throughout his hospital course.

Conclusions:
Brainstem involvement in hypertensive encephalopathy is not uncommon but is usually associated with supratentorial lesions. Predominant brainstem lesions without supratentorial involvement, so called brainstem hypertensive encephalopathy has been rarely reported. Unlike those reports, our patient remained alert and free of brainstem deficits despite severe edema throughout the brainstem, concluding that vasogenic edema in hypertensive encephalopathy can be asymptomatic and imaging should be consider in these patients.

References: None

Financial Support: None

Poster 101

INFLUENCE OF VARIABLE CYCLOSPORIN-A CONCENTRATIONS ON BRAIN NEUROCHEMISTRY IN SEVERE TRAUMATIC BRAIN INJURY PATIENTS

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Introduction:

Cyclosporin-A (CsA) has potential to be neuroprotective by attenuating mitochondrial dysfunction in traumatic brain injury (TBI). Our group previously reported the results of a randomized, placebo controlled trial using CsA for neuroprotection in TBI patients. To evaluate this data further, we conducted a subgroup analysis to determine the correlation between variable CsA blood exposure and brain neurochemistry from start of the CsA infusion to 24 hours post infusion.

Methods:

Patients were divided into 3 groups: 1) CsA patients with a larger CsA area under the curve (AUC) as compared to the mean CsA AUC for the entire group (n=14); 2) CsA patients with a smaller CsA AUC (n=17); and 3) those receiving placebo (n=12). The average rate of change per hour and the AUC of the brain extracellular fluid (ECF) glutamate, lactate and pyruvate in each CsA group and between the larger CsA AUC group and placebo was then compared. Statistical analysis was performed using the Welch Two sample t-test of differences of the means. The groups were trimmed to eliminate global outliers.

Results:

Patients achieving a larger CsA AUC had a greater average decrease per hour in lactate concentrations as compared to those with a smaller AUC ($p= 0.002$) and the placebo group ($p=0.049$). There was also a positive trend in the average change per hour for glutamate ($p=0.23$) and lactate/pyruvate ratio ($p=0.76$) in the larger CsA AUC group, but a negative trend was seen for pyruvate ($p=0.2$). AUC for lactate was also significantly greater for the larger CsA AUC group ($p=0.047$).

Conclusions:

These results show that greater CsA exposure does positively affect average hourly changes in ECF lactate concentrations. However, these effects may not be obvious when evaluating AUC. Larger studies are needed to confirm if greater CsA exposure positively influence brain neurochemistry in severe TBI patients.

References: None

Financial Support: None

Poster 102

CRANIAL TRAUMA AND CEREBRAL VENOUS SINUS THROMBOSIS

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Introduction:

CVST is thought to be a rare form of stroke in adults. In the largest prospective observational study on this condition, cranial trauma was observed as a potential causative factor in 1% of patients. There is a paucity of date on the clinical presentation and management of trauma-associated CVST.

Methods:

Case series and review on the association of CVST with cranio-cerebral trauma

Results:

Over a 6 month period, 3 patients were admitted to the Neurosciences Critical Care Unit with penetrating gun shot wounds to head, and were diagnosed with CVST by CT venography. Two patients were anticoagulated with intravenous heparin, and one of them developed a posterior fossa hemorrhage, requiring evacuation. The third patient did not receive any antiplatelet or anticoagulant therapy. All three did eventually well and were discharged to a rehabilitation facility

Literature review

Identifying the etiology of CVST is critical in determining appropriate management. Two small prospective randomized trials assessing the use of heparin to treat CVST, as well as one meta-analysis, suggested improved outcomes in patients receiving anticoagulation, with minimal risk of serious hemorrhage. Most of the patients in these small trials developed CVST due to etiologies other than trauma. A retrospective review included four patients with intracranial trauma as the etiology for CVST. Two patients were anticoagulated and did well. Of the two patients who did not receive anticoagulation, one did well while the other had residual deficits.

Conclusions:

Since intracranial trauma patients are at risk of hemorrhage progression, the benefits of anticoagulation in this population are unclear. The results of this small series suggests that anticoagulation provides no benefit, however, larger studies are needed to define optimal management strategies and in particular clarify risks and benefits of anticoagulation in trauma-associated CVST.

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Financial Support: None

Poster 103

PRIOR STATIN USE REDUCES MORTALITY IN SUPRATENTORIAL INTRACEREBRAL HEMORRHAGE

Neeraj Naval[1], Tamer Abdelhak[1], Paloma Zeballos[2], Nathalie Urrunaga[2], Marek Mirski[1], Juan Carhuapoma[1]
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Introduction:

ICH accounts for 10-15% of all strokes with mortality rates approaching 50%. Glasgow Coma Scale (GCS), ICH volume, age, pulse pressure, location, intraventricular hemorrhage (IVH) and hydrocephalus are known to impact 30-day survival following ICH and are included in various prediction models. The role of other clinical variables in the long-term outcome of these patients is less clear. The objective of this study was to assess the impact of blood glucose, coagulopathy, seizures and prior statin and aspirin use on clinical outcome following intracerebral hemorrhage (ICH).

Methods:

Records of consecutive ICH patients from 1999 to 2006 were reviewed. Patients with ICH related to trauma or underlying lesions (e.g. brain tumors, aneurysms, arterio-venous malformations) and of infratentorial location were excluded. Impact of admission blood glucose, coagulopathy, seizures on presentation and prior statin and aspirin use on 30 day mortality and functional outcomes at discharge was assessed using dichotomized Modified Rankin Scale (dMRS) and Glasgow Outcomes scale (dGOS). Other variables known to impact outcomes included in the multiple logistic regression analysis were age, admission GCS, pulse pressure, ICH volume, ICH location, IVH volume and hydrocephalus.

Results:

314 ICH patients were identified, 125 met inclusion criteria. Patients' age ranged from 34-90 years (mean 63.5), 57.6 % were male. Mean ICH volume was 32.09 cc (range 1-214 cc). Following multiple logistic regression analysis, prior statin use ($p=0.05$) was found to be associated with decreased mortality with a greater than 12 fold odds of survival while admission blood glucose ($p=0.023$) was associated with increased 30 day mortality. Coagulopathy, seizures on presentation and prior aspirin use had no significant impact on 30-day mortality or outcomes at discharge.

Conclusions:

This significant association of prior statin use with decreased mortality warrants prospective evaluation of the use of statins following ICH.

References: None

Financial Support: None

Poster 104

VANCOMYCIN-RESISTANT ENTEROCOCCAL MENINGITIS TREATED WITH INTRATHECAL STREPTOMYCIN

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Introduction:

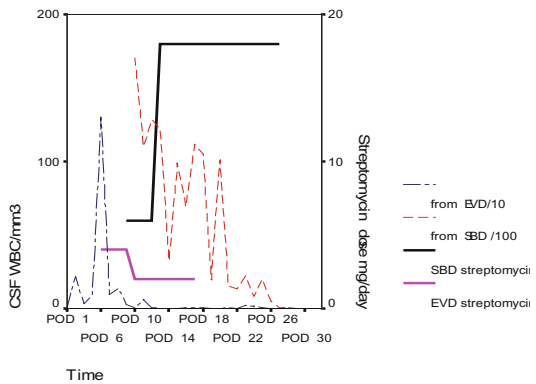
Intrathecal antibiotics have been used to treat meningitis, ventriculitis or shunt infection from Gram positive or negative, mycobacterial and fungal organisms. Because of the need for an invasive route and the significant side effects, including audiovestibular toxicity, encephalopathy, seizures and arachnoiditis, intrathecal administration is usually reserved for resistant cases. We present a patient who developed vancomycin-resistant enterococcal (VRE) ventriculitis treated successfully with intrathecal streptomycin through bilateral intracranial drains

Methods:

A 64-year-old male with metastatic melanoma presented for a surgical resection of a single lesion in the right frontal lobe. Subsequently he became comatose and an external ventricular drainage device (EVD) was placed on the left. Cerebrospinal fluid (CSF) obtained from EVD grew VRE, resistant to gentamycin. Despite the treatment with 2 mg preservative-free intrathecal streptomycin every 12 hours and intravenous linezolid since postoperative day 6, the CSF cultures continued to remain positive and on day 9 surgical bed exploration revealed purulent fluid collection that led to placement of a surgical bed drainage (SBD) catheter in the right fronto-temporal region. He was treated with 1 mg intrathecal streptomycin injected through the EVD and 3 mg through the SBD every 12 hours, followed by 4 ml of saline flush and clamping of both drains for 1 hour. Because of improvement on the EVD cultures and mental status, but persistent bacterial growth from the SBD, the SBD doses were increased to 9mg on day 12. Linezolid was stopped on day 16 and streptomycin via the EVD the next day due to improvement in the CSF through this drain only. Intrathecal streptomycin through the SBD continued until day 27 and both catheters were removed on day 33 with no subsequent complications (see figure)

Results:

Figure: Cerebrospinal fluid white blood cell count and antibiotic dose and duration



EVD = external ventricular drain, SBD = surgical bed drain, POD = post-operative day

Conclusions:

Enterococcal meningitis is an increasingly recognized complication of neurosurgical procedures. An old antibiotic, like streptomycin, may be an option, if used at lower than previously reported intrathecal doses

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Financial Support: None

Poster 105

CONTRIBUTION OF GCS IN CALCULATION OF APACHE IV SCORES TO PREDICT MORTALITY AND LENGTH OF STAY IN NICU PATIENTS

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Introduction:
Traditionally APACHE®IV scores use the lowest Glasgow Coma Score (GCS) within the first twenty-four hours.

Methods:
Three models were studied using the APACHE IV calculator, changing only the GCS parameter, to predict length of stay (LOS) and mortality for fifty patients in a neurological ICU who were in an ongoing IRB approved sedation study. In the first model, the standard APACHE IV score utilizing the lowest GCS in the first-twenty four hours was used. In the second model the GCS was removed. In the third, the highest GCS was used and a different modified APACHE IV was calculated.

Results:
Regression analyses were performed using SAS 9.1 for Windows (Cary, NC). The standard model using the lowest GCS was a significant predictor for mortality (r^2=0.078, p=0.049), but not for LOS (r^2=0.044, p=0.15). The model omitting the GCS did not significantly predict for LOS (r^2=0.073, p=0.057) or mortality (r^2=0.056, p=0.098). The model using highest GCS was a significant predictor for mortality (r^2=0.11, p=0.019), but not for LOS (r^2=0.051, p=0.12).

Conclusions:
Of the three models studied, that using the highest GCS during the first twenty-four hour period was the best predictor of mortality, explaining 11% of the variability as compared to 7.8% with the traditional method. All three models failed to significantly predict LOS. Although the sample size was small, these findings support the validity of further exploration of how GCS scores may influence APACHE IV scores in neurologically ill patients.

References: None

Financial Support: None

Poster 106

ENHANCING THERAPEUTIC HYPOTHERMIA AFTER CARDIAC ARREST WITH IMMEDIATE INITIATION AND NEUROPHYSIOLOGIC MONITORING IN A RODENT MODEL

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Introduction:
Therapeutic hypothermia (TH) after cardiac arrest (CA) improves outcomes in a fraction of patients. To enhance the administration of TH, we studied brain electrophysiologic monitoring in determining the benefit of early initiation of TH compared to conventional administration in a rat model.

Methods:
Using an asphyxial CA model, we compared the benefit of immediate hypothermia (IH, T=33^{o}C, immediately post-resuscitation, maintained 6 hours) to conventional hypothermia (CH, T=33^{o}C, starting 1 hour post-resuscitation, maintained 12 hours) via surface cooling. We tracked quantitative EEG using relative entropy (qEEG) with outcome verification by serial Neurological Deficit Score (NDS) and quantitative brain histopathological damage scoring (HDS). Thirty-two rats were divided into 4 groups based on CH/IH and 7/9-minute duration of asphyxial CA. Four sham rats were included for evaluation of the effect of hypothermia on qEEG.

Results:
The 72-hour NDS of the IH group was significantly better than the CH group for both 7-minute (74/63; Median, IH/CH, $p<0.001$) and 9-minute (54/47, $p=0.022$) groups. qEEG showed greater recovery with IH ($p<0.001$) and significantly less neuronal cortical injury by HDS (IH: 18.9±2.5% versus CH: 33.2±4.4%, $p=0.006$). The 1-hour post-resuscitation qEEG correlated well with 72-hour NDS ($p<0.05$) and 72-hour behavioral subgroup of NDS ($p<0.01$). No differences in qEEG were noted in the sham group.

Conclusions:
Immediate but shorter hypothermia compared to CH leads to better functional outcome in rats after 7- and 9- minute CA. The beneficial effect of IH was readily detected by neuro-electrophysiologic monitoring and histological changes supported the utility of this observation.

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Financial Support: None

Poster 107

ENDOVASCULAR TREATMENT OF CEREBRAL VASOSPASM: A COMPARISON OF THE EFFECTS OF INTRA-ARTERIAL NICARDIPINE AND INTRA-ARTERIAL PAPAVERINE ON CEREBRAL CIRCULATION TIME

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Introduction:
Intra-arterial papaverine (IAP) has been established as an effective method of improving cerebral blood flow in patients with cerebral vasospasm refractory to medical management. Unfortunately, the efficacy of IAP is transient and its use has been associated with elevated intracranial pressure, reduced brain tissue oxygen, and neural toxicity. Intra-arterial nicardipine (IAN) has been shown to have sustained effects on vasospasm without increases in ICP. In this study, we compare the effects of IAP and IAN, for the treatment of cerebral vasospasm, on cerebral circulation time (CCT).

Methods:
A retrospective analysis of 25 endovascular treatments for cerebral vasospasm using IAP (n=13) and IAN (n=12) was conducted. Contrast dye transit time from the arterial to the venous phase (supraclinoid internal carotid artery to parietal cortical veins) was measured to obtain CCT. Treatments preceded by angioplasty were excluded from analysis.

Results:
CCT measured before IAN was 6.99 ± 1.21 seconds and prior to IAP was 6.57 ± 1.65 seconds ($p = 0.33$). IAN improved CCT by 1.69 ± 0.84 seconds (24%; $p = 0.004$). IAP decreased CCT 1.99 ± 1.46 seconds (28%; $p < 0.001$). Both IAN and IAP significantly improved CCT. No significant difference between treatments was observed

Conclusions:
Intra-arterial nicardipine for the treatment of cerebral vasospasm, significantly improves CCT as effectively as intra-arterial papaverine. Moreover, its use may help to avoid the deleterious side effects that occur with papaverine.

References: None

Financial Support: None

Poster 108

CARDIAC TAMPONADE IN A PATIENT WITH KLIPPEL TRENAUNY SYNDROME (KTS)

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Introduction:

Klippel Trenauny syndrome is an ectomesodermal congenital abnormality initially described in 1900's usually affecting soft tissues, one extremity with hemihypertrophy, and vascular abnormalities such as varicose veins, vascular nevi's, and capillary malformation's.
Cardiac tamponade is a medical emergency. There is no report to date of cardiac tamponade as a result of vascular malformations, and in particular, KTS. The classic triad for diagnosis of cardiac tamponade is hypotension, jugular venous distension, and muffled heart sounds known as Beck's triad.

Methods:

58 years old right handed, white male with a history of dolichoectasia of the basilar artery and a right posterior cerebral artery aneurysm who presented with status epilepticus.

Results:

On day 4th the patient became tachycardic with heart rate more than 100, systolic blood pressure 110's – 130's. Patient did not respond to aggressive intravenous fluid therapy with almost 3 liters positive each day. Chest x-ray showed some enlargement of his cardiac silhouette. Immediately an echocardiogram was ordered to check his cardiac function, and was found to have moderate pericardial effusion with physiologic tamponade (collapse of his left ventricle).

Conclusions:

In our patient, he had long standing KTS but during this hospitalization course, neither jugular venous distension nor hypotension, nor muffled heart sounds were present. The only suspicious findings were tachycardia not responding to fluid boluses and widened mediastinum on the chest x-ray. The patient was diagnosed with cardiac tamponade. High clinical suspicion is essential for diagnosing Cardiac Tamponade. Echocardiogram is usually diagnostic. Electrocardiographical correlation with low QRS voltage may aide in diagnosis but not a characteristic of pericardial effusion. It is also essential to integrate the neurologic diagnosis into the overall clinical picture and look at your patient as a whole body rather then a group of functional systems.

References: None

Financial Support: None

Poster 109

A POSTERIOR CIRCULATION CAUSE FOR APHASIA

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Introduction:

The thalamus, with extensive cortical connections, acts as a relay station for higher cortical functions. We discuss a case of stroke that illustrates this interaction between deep gray matter and the cortex

Methods:

We present a case of stroke producing thalamic aphasia.

Results:

An 85yo right-handed female arrived 60 minutes from onset of language disturbance and right sided weakness. NIH Stroke Scale score was 21/42. History was significant for atrial fibrillation (INR subtherapeutic on Coumadin). Examination demonstrated a global aphasia and a dense right hemiplegia (face and arm more than leg). CT angiogram revealed occlusion of a posterior cerebral artery branch. CT perfusion studies demonstrated a region of decreased blood flow in the left thalamus, with a corresponding diffusion weighted abnormality on MRI. Intravenous tPA was administered per protocol. NIH score decreased from 21 to 9 during infusion, with further improvement later. An Aphasia Language Proficiency Scale showed moderate to severe impairment for listening and talking, moderate impairment for reading. Dominant hand weakness made writing untestable. She had jargon conversational speech output. The clinical picture was consistent with a mixed aphasia, with expressive dominance.

Conclusions:

In stroke localization aphasia with hemiparesis is typically regarded as cortical in origin and usually associated with dominant hemisphere lesions. The anatomy of language has identified a language zone the borders the perisylvian fissure. Language disturbances may also occur with thalamic pathology, usually dominant hemisphere, and typically a transcortical-form of aphasia. The pulvinar and the ventroposterior-lateral nuclei are most frequently implicated, supplied by the same posterior cerebral artery branch.

This case illustrates the importance of considering thalamic localization in an aphasic patient. This case additionally highlights aphasia resulting from posterior cerebral artery pathology, thus the necessity of imaging of all vessels in aphasia investigation, and the utility of CT perfusion acutely.

References:

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Financial Support: None

Poster 110

PREDICTIVE VALUE OF SERUM BIOMARKERS IN ACUTE TRAUMATIC BRAIN INJURY

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Introduction:
Traumatic Brain Injury (TBI) remains a leading cause of morbidity and mortality in the United States, affecting up to 1.4 million individuals annually. Following minor TBI, the rapid and non-invasive identification of patients at risk for intracranial abnormality could play an important role in the early triage and management of this patient population.

Methods:
80 patients presenting within 24 hours of minor TBI (defined as GCS 14-15) were prospectively enrolled at Duke University Medical Center. A point of care assay was used to identify a panel of serum biomarkers involved in the pathophysiology of brain injury that predicted the presence of intracranial pathology identified by head CT.

Results:
A univariate logistic analysis of 15 biomarkers involved in the pathogenesis of neuronal injury was performed. Four biomarkers were identified that were highly correlated with intracranial pathology on the initial CT. These included BNP, CRP, D-Dimer, and S100beta.

Conclusions:
A rapid screening tool utilizing serum markers of intracranial injury and/or blood-brain barrier breakdown could play an important role in the diagnosis and initial management of patients with minor TBI.

References: None

Financial Support: None

Poster 111

PERFORMANCE OF THE "FOUR SCORE" IN THE EMERGENCY DEPARTMENT

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Introduction:

The FOUR (Full Outline of UnResponsiveness) score is a new coma scale that was originally validated in the neurointensive care unit by trained staff. In this study, we sought to validate the use of FOUR score in the busy emergency department setting by non-neurologists.

Methods:

We prospectively studied the FOUR score in 81 pairs of patients with acute neurologic disease in an emergency department (ED) with an annual census of 79,000. Three different types of examiners tested the FOUR score: an ED physician, an ED resident, and an ED nurse. Each patient was rated on both scales by two different raters, who performed their examination within 10 minutes of each other without knowledge of the other's scores. The order of the evaluations was randomized to reduce bias.

Weighted κ scores were calculated to determine the degree of agreement between pairs. κ Statistics (weighted κ) of 0.4

or less are considered poor. Values between 0.4 and 0.6 are considered fair to moderate, values between 0.6 and 0.8 suggest good observer agreement, and values greater than 0.8 suggest excellent agreement.

Results:

Sensitivity Analysis		FOUR Scale				
	N	Eye	Respiration	Brainstem	Motor	Total
Kappa - Overall	64	.846	.717	.924	.827	.877
Interrater correlation coefficient		.905	.720	.943	.878	.953

Conclusions:

There is excellent interrater reliability in collection of the FOUR score even in a busy ED. The FOUR score can reliably be used by minimally trained ED staff.

References:None

Financial Support: None

Poster 112

HOW DOES THE FOUR SCORE COMPARE TO THE GCS AMONGST DIFFERENT TYPES OF EVALUATORS?

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Introduction:

Our objective was to compare overall score assigned by diverse evaluators using the FOUR Score and Glasgow Coma Scale (GCS) when evaluating patients with altered mental status (AMS).

Methods:

Patients in the Emergency Department (ED) with altered mental status were categorized as either alert, drowsy or comatose. Two evaluators (nurse, resident or staff physician) independently assigned FOUR score and GCS, yielding 6 possible permutations (nurse-nurse, nurse-resident, etc). Wilcoxon and Kruksal Wallis tests used to analyze non-normally distributed data.

Results:

There were 66 alert, 101 drowsy and 22 comatose patients. 31% of the evaluations were done by nurses, 38% by residents and 31% by physicians. There was no difference between the median FOUR score assigned by nurses, residents or physicians ($p=0.265$). There was however a statistically significant difference in the median GCS between the evaluators; nurses were above the median, residents below it, and physicians right at the median of the total GCS ($p=0.018$).

Conclusions:

Different observers give similar scores to the patients when using the FOUR score, independent of their training. FOUR score appears to be better than GCS in grading the AMS of the patients in the ED.

References: None

Financial Support: None

Poster 113

CORRELATION OF FOUR SCORE COMA SCALE AT PRESENTATION WITH FUNCTIONAL OUTCOME AT HOSPITAL DISCHARGE

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Introduction:
The FOUR (Full Outline of UnResponsiveness) score was recently developed and validated and is an alternative to the Glasgow Coma Scale (GCS). The FOUR score consists of 4 components—eye, motor, brainstem, and respiration—and each component has a maximal score of 4. Our objective was to determine whether the validated FOUR score correlates to functional outcome at hospital discharge.

Methods:
The four score was measured on adults presenting to the Emergency Department (ED) with neurologic complaints during a 12 month period. Patients were prospectively followed through hospital discharge, and their functional outcome measured via the Modified Rankin score (mRS).

Results:
The study cohort comprised 189 patients, with the following neurologic complaints: CNS infection (3%); stroke (23%); seizure (16%); subarachnoid hemorrhage (2%); altered mental status (34%); traumatic head injury (11%); encephalopathy (10%).
Linear regression analysis revealed total FOUR score to be significantly correlated with mRS (figure 2), with a higher FOUR score being associated with a lower mRS ($p<0.001$). The strength of this association was robust, with $R^2 = 0.35$. Each of the components of FOUR score was also independently associated with mRS (all $p<0.001$). The strength of association was strongest for the eye response component ($R^2 = 0.41$), and least for the brainstem reflexes component ($R^2 = 0.12$).

Conclusions:
ED FOUR score is a robust predictor of functional outcome at hospital discharge in patients seen for neurologic complaints.

References: None

Financial Support: None

Poster 114

HYPERNATREMIA INDEX INDEPENDENTLY PREDICTS OUTCOME AFTER ANEURYSMAL SUBARACHNOID HEMORRHAGE

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Introduction:
Hypernatremia has been linked to poor outcome after aneurysmal subarachnoid hemorrhage (SAH). However, the effect of transient versus persistent hypernatremia is unclear. The aim of this study was to investigate the dose-response of hypernatremia on neurological outcome.

Methods:
We analyzed prospectively collected data from all aneurysmal SAH patients admitted to our University Hospital from May 2004 to April 2006. The demographics, clinical features, and discharge Glasgow Outcome Scale scores were abstracted from the database and chart review. All of the serum sodium measurements during the hospitalization were screened for the presence of hypernatremia (serum sodium concentration of >145 mmol/L). To analyse the dose response of hypernatremia on outcome, hypernatremia index (the numbers of sodium levels >145 mmol/L divided by the total numbers of sodium measurements) was determined for each patient. The relationship between hypernatremia index (HI) and outcome was studied using univariate logistic regression and multivariate analysis.

Results:
Among the 142 aneurysmal SAH patients admitted during the study period, 17 patients (12%) were identified to have hypernatremia at least once. Five of the six patients with HI $\geq$ 0.25 died, with the surviving patient in vegetative state. Persistent hypernatremia appeared to be secondary to diabetes insipidus. The 11 patients with transient hypernatremia (HI <0.24) survived with GOS score 3 or better. There was a significant association between HI and outcome at discharge ($P < 0.001$ by analysis of variance). HI predicts outcome ($P < 0.05$ by multivariate analysis) after adjusting for age, Hunt-Hess grade, and length of ICU stay.

Conclusions:
Hypernatremia index dose-dependently predicts neurological outcome after SAH. Persistent hypernatremia (HI $\geq$ 0.25) is associated with poor outcome.

References: None

Financial Support: None

Poster 115

PARADOXICAL "DAS KLIVUSKANTENSYNDROM": A CASE OF THE BLOWING THE WRONG PUPIL

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Introduction:

Early pupillary enlargement on the side of an intracranial mass has been attributed to compression of the third nerve by the medial temporal lobe or by a caudally displaced posterior cerebral artery. We present a case of paradoxical enlargement of the opposite pupil as a result of stretching of the third nerve over the clivus on the side contralateral to a mass as the midbrain was displaced.

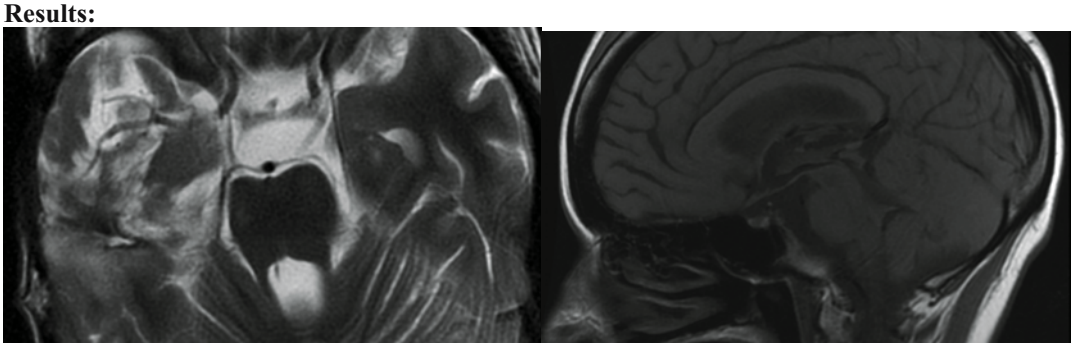
Methods:

A 23 year-old woman with a 2year history of anaplastic astrocytoma in the right temporoparietal area presented with acute cerebral herniation.

After treatment, she awakened left pupil that was slightly irregular in shape, 6mm in diameter and nonreactive. The right pupil was 4 mm and nonreactive.

Imaging obtained 8.5 hours after the above-described event revealed displacement of the midbrain to the side of the tumor and stretching of contralateral third nerve over the clivus at the entry of the nerve into the dural margin of the cavernous sinus (Fig. 1). The ipsilateral third nerve was slackened as its origin in the ventral brainstem was displaced towards the mass. There was no contact of the parahippocampal gyrus on the side of the third nerve palsy (Fig. 2)

Results:



Conclusions:

The third nerve dysfunction on the left (the side opposite the tumor) was apparently the result of stretching over the clivus ("das klivuskantensyndrom") and not from the compression by the uncus.

This case emphasizes that effects of cranial masses are not stereotyped and that the mechanism of pupillary enlargement on the side of the mass are variable despite historical notions.

References:

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Financial Support: None

Poster 116

SLIPPERY PLATELET SYNDROME IN SUBDURAL HEMATOMA SUBJECTS: PLATELET FUNCTION ASSAY RESULTS IN A SINGLE-CENTER, PROSPECTIVE CASE SERIES

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Introduction:
Coagulopathy is a known risk factor for subdural hematoma. Our hypothesis is that platelet function assays identify platelet-mediated coagulopathy in subdural hematoma subjects.

Methods:
A coagulation panel including INR, PTT, CBC, and a platelet function assay using the Dade PFA-100 analyser was obtained on 30 consecutive SDH subjects treated at the Kaiser Permanente Neurosurgery Center from May 2006 to October 2006. The collagen/epinephrine assay was used for screening with an abnormal value>172 s. The collagen/ADP assay was completed in a subset of 11 patients. Data were prospectively entered. CT scans were re-read by physicians blinded to the coagulation parameters

Results:
Eleven out of 30 patients (36.7%) had elevated platelet function assay results (collagen/epinephrine results in control group=106 s; abnormal PFA=279 s) but similar platelet values (241K vs 247K). Treatment with aspirin and clopidogrel was not more likely to predict this. Review of CT findings in patients grouped into normal coagulation results, abnormal PFA, and elevated INR demonstrated: midline shifts (4.5, 6.7, 10.9 mm); max thickness (13.9, 14.8, 16.7 mm); and blood/fluid levels (47%, 55%, 86%).

Conclusions:
'Slippery' platelets were found in about 1/3 of SDH patients. Identifying platelet dysfunction aids in determining contributing factors to the initial SDH and also the acute management.

References: None

Financial Support: None

Poster 117

ADHERENCE TO CDC GUIDELINES FOR PLACEMENT OF INTRAVASCULAR CATHETERS IN NEUROSURGICAL PATIENTS

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Introduction:

The CDC has established aseptic guidelines for placement of arterial (AL) and central (CL) catheters, which include chlorohexadine (CH), sterile gloves and gown (for CL). This study was conducted to determine anaesthesiology residents' adherence with, as well as identify factors that influence non-compliance with the guidelines.

Methods:

All neurosurgical patients requiring an intravascular catheter (IC) were enrolled over a three year period. Catheters were placed by a senior anaesthesiology resident. A knowledgeable observer, unknown to the operator, observed the insertion, and interviewed the operator to determine reasons for non-compliance, if any.

Results:

250 AL and 90 CL were observed. Compliance was observed in 217 AL (87%) and 85 CL (94%). A total of 38 violations were observed. All violations in CL (n=5) were due to failure to wear a sterile gown. AL violations (n=33) were inappropriate skin disinfectant (n=5, 15%), initial failure to wear sterile gloves (n=3, 9%), or combinations (n=3, 6%). Failure to maintain sterility occurred in the remaining 23 patients (70%). Factors that influenced non-adherence were; Lack of exact knowledge (n=15, 39.5%), technically difficult placement (n=9, 23.7%), hemodynamic instability (n=6, 15.8%), emergency surgery (n=4, 10.5%) and unavailability of supplies (n=4, 10.5%).

Conclusions:

Although all residents were previously educated about the CDC guidelines, our study demonstrated that their knowledge of the exact guidelines is still significantly lacking. These failures, along with deficient technical skills were the major reasons for non-compliance. In addition, residents had a perception that the guidelines were more important for CL than AL catheterization. Patient factors also contribute a significant portion of failure rates. We conclude that proper training in catheter placement, as well as further education endeavours are needed to improve adherence to the guidelines

References: None

Financial Support: None

Poster 118

A PILOT STUDY TO EVALUATE THE EFFECT OF CHEST PHYSIOTHERAPY ON INTRACRANIAL PRESSURE

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Introduction:
Brain injury treatment is often focused on minimizing intracranial pressure (ICP) which, when elevated may lead to secondary brain injury. Chest Physiotherapy (CPT) is a common practice used to increase the surface area of the lung thereby optimizing gas exchange. As the exchange of gas is optimized through the opening of the alveoli there is a decrease in intrathoracic pressure. This decrease is thought to lessen resistance to cerebral venous flow thereby decreasing ICP. This research addresses the impact CPT has on ICP.

Methods:
Prospective randomized control trial. The study period lasted 30-minutes. 26 Subjects were randomized to either the control (no CPT) or the intervention (10-minutes of CPT) group. ICP was recorded for 10-minutes period before, during and after a 10-minute intervention time. CPT was performed using automated beds with the HOB at 30 degrees. The mean ICP was determined and ANOVA (SAS v9.1) was used to test for group differences

Results:
For the 10-minutes prior to the intervention period there was no difference in mean ICP for the control (13.590) compared to the intervention (12.404) group (F=.30, p=.5866). For the 10-minute intervention period, there was no difference in mean ICP for the control (14.986) compared to the intervention (12.250) group (F=.56, p=.4605). For the 10-minutes following the intervention period, there was no difference in mean ICP for the control (15.509) compared to the intervention (13.327) group (F=.67, p=.4215).

Conclusions:
This pilot study provides support that CPT is safe to perform in the setting of intracranial hypertension and may in fact be beneficial in lowering ICP. Given the limited number of subjects, further studies are recommended.

References: None

Financial Support: None

Poster 119

COGNITIVE OUTCOMES FOLLOWING SEIZURE PROPHYLAXIS FOR INTRACRANIAL HEMORRHAGES WITH LEVETIRACETAM VERSUS PHENYTOIN

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Introduction:
To date, common therapy in patients with intracranial hemorrhage is treatment for the prophylaxis of seizure using antiepileptic drugs, commonly phenytoin. Phenytoin therapy is effective in seizure prevention, but is associated with a high incidence of cognitive disturbance. Levetiracetam may be a suitable alternative for seizure prophylaxis. We present results comparing cognitive outcomes in intracranial hemorrhage patients receiving seizure prophylaxis with levetiracetam versus phenytoin.

Methods:
Researchers conducted a retrospective chart review of 97 patients diagnosed with intracranial hemorrhage and received prophylactic levetiracetam or phenytoin between August 2005 and March 2007. Patients were excluded with history of seizure, history of dementia, metabolic encephalopathy, sepsis, ARDS, baseline Glasgow Coma Scale <6, or other disorders that may precipitate seizures. Variables collected include age, sex, race, type of intracranial hemorrhage, length of stay, discharge destination, dosing and level information, and cognitive outcomes.

Results:
A total of 69 reviewed patients met inclusion criteria (levetiracetam n=47, phenytoin n=22). Average Glasgow Coma Scale at baseline was 12.2 (SD $\pm$ 3.8) for levetiracetam patients and 10.3 (SD $\pm$ 3.5) for phenytoin patients. Glasgow Outcome Scale averaged 4.28 (SD $\pm$ 1.3) for levetiracetam patients and 3.4 (SD $\pm$ 1.3) for phenytoin patients. 32.5% of levetiracetam patients experienced cognitive impairment versus 66.7% of phenytoin patients (p = .032). 75.0% of levetiracetam patients were alert or cooperative versus 46.7% of phenytoin patients (p = .059).

Conclusions:
Despite similarities in disease states at onset, patients receiving seizure prophylaxis with phenytoin more frequently had poor cognitive outcome versus patients receiving levetiracetam. Because levetiracetam was also effective for seizure prophylaxis and had lower mortality rates, further research on the impact of seizure prophylaxis medication usage and cognitive outcomes is warranted.

References: None

Financial Support: None

Poster 120

FEASIBILITY OF EXTERNAL VENTRICULAR DRAIN AND INTRACRANIAL PRESSURE MONITOR PLACEMENT BY NEUROINTENSIVISTS

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Introduction:
External ventricular drains and intracranial pressure monitors are widely-used in the neurocritical care unit, and they have been placed historically by neurosurgeons. Current movements in critical care staffing have lead to a growing number of external ventricular drains and monitors being placed by neurointensivists. Little data on the safety or feasibility of such placement by neurointensivists exist. We present our experience of external ventricular drain and intracranial pressure monitor placement in the neurocritical care unit setting.

Methods:
A retrospective chart review of 27 patients with external ventricular drain and 7 patients with intracranial pressure monitor, all placed by neurointensivists, was conducted from August 2005 to June 2007. Variables collected include incidence of infection, incidence of complication, cause for and placement of drain/pressure monitor, opening pressure, and discharge destination.

Results:
For patients receiving external ventricular drain, 96.3% (n=26) remained infection-free. 100% (n=7) of patients receiving intracranial pressure monitor remained infection-free. Complications occurred in 22.2% (n=6) of patients receiving drain. Complications occurred in 0.0% (n=0) of patients receiving monitors. 55.6% (n=15) of patients receiving external ventricular drain survived their patient stay. Opening pressure was higher in patients that did not survive (28.46cm H_2O) versus patients survived (10.58cm H_2O) ($t = 2.134$, $p = .044$).

Conclusions:
Based on these supplemental results, neurointensivist placement of external ventricular drains and intracranial pressure monitors may be safe and feasible. Opening pressure of patients receiving an external ventricular drain appears to be related to mortality. Further research is warranted.

References: None

Financial Support: None

Poster 121

COMPLICATIONS OF NEUROFORM STENT IN ENDOVASCULAR TREATMENT OF INTRACRANIAL ANEURYSMS

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Introduction:
The Neuroform stent can help in the treatment of difficult, wide-necked intracranial aneurysms. The objective of our study is to report some of the challenges associated with the Neuroform stent in the treatment of intracranial aneurysms.

Methods:
From January 2003 to August 2006, consecutive patients treated with Neuroform stent for intracranial aneurysms were prospectively enrolled. Patients' demographics including cerebrovascular risk factors, aneurysms size and locations were collected. Technical and clinical complications as well as clinical outcomes were measured. Data were analyzed retrospectively using SPSS software version 11.5.

Results:
Successful deployment of the stent, in the target artery, was achieved in 65/67 (97%) patients. Stent deployment failed in two cases and the migration of stent developed in one during coiling. Postoperative thromboembolic events developed in three patients. These three patients possessed hyperactive platelets, and were treated with intravenous eptifibatide. Intraoperative rupture of aneurysm developed in one patient, which was secured by subsequent coiling. Majority of the patients had good outcomes GOS (Glasgow Outcome Score) 1 or NIHSS (National Institute of Health Stroke Scale) 0 in 63/67 (94%), GOS 2 or NIHSS 2 in 1 patient and GOS 3 or NIHSS 4 was observed in 3 cases.

Conclusion:
Despite a low rate of intraoperative complications, post procedural thromboembolic events were not uncommon in Neuroform stent treated patients, which might be associated with hyperactive platelets. Further studies were warranted to identify any potential relationship between post stent hyperactive platelets and thromboembolism

References: None

Financial Support: None

Poster 122

UTILITY OF FOUR SCORE IN PREDICTING COMPLICATIONS AND OUTCOME IN THE NEUROSURGICAL INTENSIVE CARE UNIT: A PROSPECTIVE COMPARISON WITH GLASGOW COMA SCALE

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Introduction:
The most widely used scale in acute neurological injury is the Glasgow Coma Scale (GCS) but the new FOUR Score (FS) has greater interrater reliability. We compared the ability of FS to predict complications and outcome among patients admitted to a neurosurgical intensive care unit (NSICU).

Methods:
A prospective study was performed at an academic NSICU. All patients admitted to the NSICU had GCS and FS performed by neuro-critical care nurses within 6 hours of admission. Standardized data collection form was used to collect demographics, medical history, diagnosis, in-hospital complications, neurosurgical or medical procedures, and outcome determined by Glasgow Outcome Scale. Comparison was made between FS and GCS using Fishers exact test for categorical and proportion of the means test (t-test) for continuous variables. Based on prior reports and our exploratory analysis we used a FS < 9 as cutoff value to discriminate patients.

Results:
Seventy-three patients were studied in 6-month period. Diagnosis included traumatic brain injury (23), intracranial hemorrhage (20), subarachnoid hemorrhage (16), subdural hematoma (8), and other causes (6). Median NSICU stay was 6 days. FS and GCS equally predicted intubation, intracranial pressure monitoring, tracheostomy, and gastrostomy, but FS better predicted hemicraniectomy ($p=0.005$) and pneumonia (0.049). Both scales predicted in-hospital complications and good outcome with sensitivity of 81%, but low specificity (58% FS and 50% GCS).

Conclusions:
Four Score is equivalent to the GCS in predicting complications and outcome in NSICU patients. Since it has better interrater reliability, it may be superior to evaluate NSICU patients.

References: None

Financial Support: None

Poster 123

EFFICACY OF SILVER NANOPARTICLES-IMPREGNATED EXTERNAL VENTRICULAR DRAIN CATHETERS IN PATIENTS WITH ACUTE OCCLUSIVE HYDROCEPHALUS

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Introduction:
Temporary external ventricular drainage (EVD) is a commonly used procedure in neurointensive care patients for managing acute hydrocephalus. An important complication of external cerebrospinal fluid (CSF) drainage is catheter associated ventriculitis (CAV). Recently, anti-micro-bacterial impregnated catheters were shown to decrease the infection rate, however, they are discussed to possibly induce bacterial resistance. A new option which might bypass these problems are catheters impregnated with silver nanoparticles. This pilot study addresses the efficacy of silver-impregnated EVD catheters in patients with acute occlusive hydrocephalus.

Methods:
19 consecutive patients were enrolled in the treatment arm of the study and data was prospectively recorded for these patients. The control group consisted of 20 historic patients for whom data was retrospectively assessed via patients´ hospital chart review. Silver impregnated or conventional EVD catheters by Spiegelberg where used in the treatment group or control group, respectively. CSF samples were drawn at least three times a week under sterile conditions. Routine bacterial cultures and CSF analyses were done according to standard protocols. The primary endpoint of the study was the occurrence of CAV proven by CSF culture.

Results:
In 20 control patients, 5 CAV's could be proven by positive CSF cultures. In contrast, no positive CSF cultures could be found in 19 patients in the treatment group. This difference was statistically significant ($p<0.05$). All CAV's occurred later than day 10 post catheter placement. Colonization of the catheter tip was found in 6 patients in the control group and in 5 patients in the treatment group (not significant).

Conclusions:
This pilot study indicates that EVD catheters impregnated with silver nano-particles might be a new option for preventing CAV in neurocritical care patients and should be evaluated in a large prospective randomized study.

References: None

Financial Support: None

Poster 124

CONIVAPTAN FOR THE CORRECTION OF HYPONATREMIA IN THE NEUROCRITICAL CARE UNIT

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Introduction:

Hyponatremia is common in patients with acute neurological disorders. They are at particularly high risk of secondary deterioration as hyponatremia can exacerbate cerebral edema. Current management options includes fluid restriction, hypertonic saline and loop diuretics. Conivaptan (Vaprisol®, Astellas Pharma, Deerfield, Illinois) is a non-peptide antagonist of the V_{1A} and V_2 receptors. Blocking the V_2 receptors in the renal collecting ducts enhances free water excretion, while sparing necessary electrolytes. No current studies have looked at the efficacy of conivaptan in this population.

Methods:

Consecutive patients admitted from May 2006 to June 2007 to a Neurology/Neurosurgery ICU who received conivaptan where identified. Data collected included patient demographics, diagnosis, conivaptan dose, fluid balance, and serial measurements of sodium concentration, urine output, and specific gravities before and after conivaptan administration. Patients who received more than a single dose within 72 hours were excluded from this analysis.

Results:

Seventeen doses were administered to 14 patients. Admission diagnosis included intracranial hemorrhage[5], subarachnoid hemorrhage[5], tumor[4], Guillain-Barre Syndrome[2], traumatic brain injury[2], and other[6]. Twelve patients received a bolus of 20 mg and 2 received 40 mg; none received an infusion. Intravenous fluids were isotonic saline in 10 and hypertonic (1.25-2%) in 4 patients. Average baseline serum sodium concentration [Na] (in mEq/L) was 130 ± 3.1. The mean change in [Na+] at 8, 24, 48, and 72 hours were +7.1 ± 2.8, +5.4 ± 4.2, +6.5 ± 5.1, and +4.7 ± 5.2 respectively (ANOVA $F=6.1$, $p < 0.001$). The highest [Na+] occurred at 8 hours. Urine output at 2, 4, 6, 12 and 24 hours post-dose, was 387 ± 207, 350 ± 183, 309 ± 184, 139 ± 85 and 138 ± 93 ml/hr respectively. The mean specific gravity rapidly decreased at 2 and 4 hours post-dose from a baseline of 1.010 ± .005 to 1.005 ± .006 and 1.004 ± .010 and returned to baseline by 12 hours.

Conclusions:

A single bolus dose of 20-40 mg of Conivaptan may be a useful alternative for treatment of hyponatremia in the in the Neurocritical Care Unit.

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Financial Support: None

Poster 125

INTRAVENOUS LEVETIRACETAM FOR ANTIEPILEPTIC DRUG MONOTHERAPY IN PATIENTS WITH LIVER FAILURE BEFORE AND AFTER LIVER TRANSPLANT

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Introduction:

Seizures are a recognized complication of liver failure and well described in patients who undergo liver transplant. Issues relating to medication side effects and interference with concurrent medication metabolism confound the use of traditional antiepileptic drugs (AED). Levetiracetam does not affect the cytochrome P450 and does not cause hepatotoxicity. We review our experience with levetiracetam as primary treatment and prophylaxis for seizures in patients with liver disease—before or after liver transplant.

Methods:

Retrospective consecutive chart review of patients with seizures who had liver disease or had undergone liver transplantation and were managed with levetiracetam. Clinical outcomes were assessed by recurrent seizures, death, or functional status at discharge by the Glasgow Outcome Scale (GOS).

Results:

Between 12/06 and 7/07 we identified 8 patients with seizures—4 awaiting and 4 following liver transplant. Initial benzodiazepine treatment was followed by intravenous levetiracetam loading with 1500 mg and maintenance varied (0.5g BID to 1.5g BID, IV or enteral) based on glomerular filtration rate and subsequent seizures. Seizure control was achieved in 6 of 8 patients. One patient required additional AED (fosphenytoin/phenytoin). Seizure types were generalized (n=4), epilepsia partialis continua (EPC) (2) and nonconvulsive status epilepticus (NCSE) (2). Seizure etiology was toxic-metabolic derangement (6) and cerebral edema (2). Two patients had tacrolimus-based immunosuppression. Clinical outcomes (GOS) at discharge: 2 patients had mild to moderate disability (GOS, 4-5), 3 severe disability (GOS, 3), and 3 deaths (GOS, 1). One patient required no AED and 4 received levetiracetam upon discharge. No hemodynamic instability at the time of intravenous infusion or subsequent adverse effects of levetiracetam were identified.

Conclusions:

In this small retrospective study, intravenous levetiracetam was efficacious and well-tolerated in patients with liver failure and liver transplant recipients. In particular, hemodynamic stability during infusion and a lack of interference with tacrolimus metabolism were noted.

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Financial Support: None

Poster 126

EFFICACY OF PHARMACOLOGIC DVT PROPHYLAXIS FOR PRIMARY PREVENTION OF VENOUS THROMBOEMBOLISM IN PATIENTS WITH ACUTE HEMORRHAGIC STROKE

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Introduction:
Venous thromboembolism (VTE) is a well-known complication of patients suffering from hemorrhagic strokes due to paresis, prolonged hospitalization and advanced age. Many physicians do not use pharmacologic VTE prophylaxis due to concern of hemorrhage extension. We attempt to compare the occurrence of VTE in patients with acute primary intracerebral hemorrhage (ICH) treated with low-dose unfractionated heparin (UFH) versus those treated with non-pharmacologic methods.

Methods:
A retrospective chart review was performed on all patients admitted to our service from January 2004-June 2007 with the diagnosis of acute primary ICH.

Results:
176 cases were identified. 25 patients received UFH between days 1-4 and there were no VTE in these patients. There were 14 patients with VTE among the 142 patients that did not receive UFH. One patient receiving UFH suffered a small, second area of hemorrhage. Univariant analysis using chi square did not reveal any significant results. Heparin was started later than day 2 (or not at all) in many patients due to intercurrent surgical procedures, early death and lack of consensus that chemical prophylaxis was an appropriate treatment.

Conclusions:
Additional patients with hemorrhagic strokes treated with UFH are needed to determine whether this significantly lowers the occurrence of VTE in our population. However, we feel that this study shows a trend that there is a benefit in placing these patients on prophylactic UFH. We plan to collect data from the later-half of 2007 and further analyze the results.
Only one small study from Germany showed that hemorrhagic stroke patients treated with UFH had no evidence of rebleeding and a lower incidence of PE [1, 2]. Additional studies have shown that patients with non-stroke ICH have tolerated UFH well [3, 4]. Hemorrhagic strokes have a higher incidence of VTE compared to ischemic strokes [5] and more research is needed to aid in reducing comorbid complications.

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Financial Support: None

Poster 127

PSEUDOLESIONS DUE TO MULTI-DETECTOR ROW CT ARTIFACT PREVENT APPROPRIATE USE OF TPA.

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Introduction:

Treatment of appropriate patients with tissue plasminogen activator (tPA) improves outcome in ischemic stroke. Careful patient screening minimizes associated risk of cerebral hemorrhage. Selection criteria include a non-contrast head CT that shows no hemorrhage or non-ischemic lesions that explain observed symptoms. CT artifact may prevent utilization of tPA in eligible patients. We describe a case where a patient was excluded from tPA treatment based on a CT scan showing pseudolesions incorrectly interpreted as hemorrhagic metastases.

Methods:

Case review.

Results:

A 68 year-old man sudden developed left hemiparesis and presented to a local emergency department within 30 minutes. Head CT showed several high density, round lesions surrounded by low density areas in the brainstem, thalamus, and ventricle. All abnormalities were on the left. Due to these radiographic findings, tPA was not given. After transfer to our institution, repeat CT with and without contrast failed to show the previously seen abnormalities. Unfortunately, the patient missed the window for acute recanalization therapy.

Conclusions:

Multidetector-row CT scanners utilize multiple rows rather than a single linear array of detector elements enabling simultaneous data acquisition on multiple slices. A malfunction occurring in a single detector row may produce a pseudolesion in the image. The aberrance occurs in the isocenter of the CT gantry and projects to varying locations within the brain image because of the tilted angle of the axial slices. In the present case, areas of apparent increased density with a hypoattenuated halo simulated hemorrhagic lesions with surrounding edema and led to a decision not to treat an eligible stroke patient with tPA. We believe this is the first reported case of this problem and, it is important for clinicians to recognize that this phenomenon exists.

References: None

Financial Support: None

Poster 128

GUILLAIN-BARRÉ SYNDROME FOLLOWING THORACIC SPINAL TRAUMA

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Introduction:
Guillain-Barré syndrome(GBS) is an acute immunologic attack of the peripheral nerves causing rapidly ascending weakness and areflexia. Occasionally, weakness is severe enough to leave patients paralyzed and without adequate respiratory function. In such patients, ICU admission is required. Infrequently, GBS occur in patients already admitted to ICU. When this occurs, it can be difficult to distinguish GBS from another peripheral neuropathy commonly seen in the critical care population known as critical illness neuropathy(CIN).

Case Report:
A 28-year-old man involved in a motor vehicle collision sustained multiple injuries including T6-T7 thoracic vertebrae fracture. MRI identified spinal cord compression at T6-T7, without brain or cervical cord injury. Surgical stabilization of the vertebral injury was deferred until urgent medical issues were managed. However, the patient developed marked autonomic instability with fluctuating temperatures and severe hypotension. Lower extremity weakness rapidly worsened to paraplegia and new weakness developed affecting bilateral upper extremities and face. This rapidly progressive weakness continued following posterior thoracic T6-7 decompression and T4-T10 fusion. The patient was quadriplegic with bifacial weakness and areflexia. Sensation was intact. Repeat MRI was unchanged. Electrodiagnostic studies showed severe axonal polyneuropathy, with denervation in all extremities. CSF protein was 5.03 g/L.

Results:
A presumptive diagnosis of GBS was made and a course of intravenous-immunoglobulin(IVIG) was given. One month later, plasma-exchange was administered for persisting weakness. Six months later, the patient recovered significant strength in his face and extremities, including his legs. One year later, the patient continued to improve and was walking with assistance.

Conclusions:
GBS in trauma patients is rare and limited to case reports following head trauma. [1,2] This case also highlights the similiarites and subtle differences between GBS and CIN. Ultimately, definitive diagnosis of GBS may not be possible, however an empiric course of IVIG or plasma-exchange may be warranted if GBS is a reasonable possibility.

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Financial Support: None

Poster 129

DO STATINS AND ANTIPLATELET AGENTS GIVEN PRIOR TO STROKE AFFECT ELEVATIONS OF TROPONIN AFTER ACUTE ISCHEMIC STROKE?

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Introduction:
Serum Troponin, a marker of cardiac injury, is elevated in patients with acute stroke. Statins and antiplatelet therapy are two modalities used to decrease the risk of cardiac ischemia in high risk patients. The aim of this study is to discern whether administration of these agents prior to stroke has any impact on serum Troponin and preventing neurocardiogenic injury.

Methods:
Retrospective study of adult ischemic stroke patients admitted to University Hospital was done. Data collected on 152 patients included gender, serumTroponin levels and use of antiplatelet agents and statins. Dichotomous variables were compared using Fishers exact test and chi-square tests. Odds ratios (OR) with confidence intervals were obtained using the above mentioned tests. A value of $p < 0.05$ was considered significant.

Results:
Use of anti-platelet agents had no statistically significant impact on the increase of Troponins in patients with acute stroke ($p=0.24$; OR = 0.46, 95% CI: 0.13 to 1.52). This was not significant in the subgroup analysis among men ($p = 0.69$) or women ($p = 0.28$). Statins usage did not have any statistically significant effect on the increase in Troponin ($p = 0.55$; OR = 1.48, 95% CI: 0.39 to 5.04). Men and women did not differ in serum troponin group, $p > 0.99$ and $p= 0.41$.

Conclusions:
Statins and antiplatelet agents prior to the stroke do not confer any protection against elevation of troponins in acute stroke. It may reflect that neurocardiogenic injury may occur through mechanisms that are not inhibited by antiplatelets or statins. Our study was limited by its small size and retrospective design. Future prospective trials with larger samples may be required to assess impact of antiplatelet and statins on preventing neurocardiogenic injury in stroke.

References: None

Financial Support: None

Poster 130

APOE POLYMORPHISM AFFECTS CEREBRAL EDEMA BUT NOT HEMATOMA SIZE AFTER INTRACEREBRAL HEMORRHAGE IN HUMANS

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Introduction:
To address the mechanisms by which apoE polymorphism affects functional outcome after intracerebral hemorrhage (ICH) in humans, we tested the hypothesis that the presence of the APOE4 allele results in amplified inflammatory responses and increased cerebral edema.

Methods:
We prospectively enrolled and collected data on adult patients admitted to Duke University Hospital with CT-proven ICH including hemorrhage size, midline shift (MLS), modified Rankin score (mRS), Glasgow Outcome Score (GOS), and APOE genotype. Between January 2005 and December 2006, 21 patients with supratentorial ICH were genotyped for APOE. Hemorrhage size was measured in cm^3 by CT at the time of admission and MLS at the level of the thalamus was measured in mm at >72 hours after admission; mRS, and GOS were determined at discharge. Student's t-test was used to analyze hemorrhage size, MLS, and GOS and logistical regression was used to measure allele affect on mRS. When analyzing mRS, patients were grouped by favorable outcome (mRS 0-2) or unfavorable (mRS 3-6).

Results:
Out of 21 patients, 11 possessed at least one APOE 4 allele (APOE4+). There was no difference in ICH size (25.8 v. 38.3mm for APOE4- v. APOE4+, respectively) between the groups, but there was a significant difference in MLS ($p=0.04$, 0.7 v. 4mm). Functional outcomes were worse for the patients possessing at least one APOE4 allele, as assessed by GOS ($p=0.05$; 3.7 v. 2.7) and mRS ($p=0.04$). The two patients who were homozygous for APOE4 had unfavorable outcomes.

Conclusions:
The presence of APOE4 is associated with poor functional outcomes in humans after ICH. Our data suggest that the mechanism for this may be increased cerebral edema and not larger hematoma volume. These findings are consistent with recent preclinical data demonstrating that apoE modifies the CNS inflammatory response in an isoform-specific manner.

References: None

Financial Support: None

Poster 131

ELECTROENCEPHALOGRAPHY IN CRITICALLY ILL PATIENTS

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Introduction:

Neurological evaluation of critically ill patients is important for management and prognostication. Glasgow coma scale (GCS) used in current critical care practice is an established neurological parameter as a prognostic indicator and provides no information about the underlying pathophysiology. Mechanical barriers to communication, nature of illness and widespread use of sedatives, opiates and muscle relaxants make clinical neurological examination unreliable. In these cases, the electroencephalogram (EEG) can provide additional useful information and for this reason is used frequently in critically ill patients[1]. The goal of this abstract is to provide additional information on the utility of the EEG when performed in critically ill patients

Methods:

We present EEG data from 635 records from critical care unit of a large tertiary care centre from 1/1/01 through 8/1/07. 387 records were limited to 30 minutes and 248 records ranged from 30 minutes to 24 hours, all were read by a single board certified electroencephalographer.

Results:

Only 94 records were considered to be normal. Diffuse slowing encountered in 74% records was the most common abnormality. Burst suppression and generalized suppressions were seen respectively in 15% and 14% of records. Electrocerebral silence was seen in 2% of records and 10% of records showed focal slowing, 16% demonstrated focal spikes but only 0.2% demonstrated generalized spike and wave activity. 4% of records demonstrated periodic lateralized epileptiform discharges (PLEDS). Seizures were seen in 8% of records. There was a strong association between finding epileptiform abnormalities and seizures in the same tracing. The relative risk of seizures was 3.4 when focal spikes were recorded and 5.8 when PLEDS were found.

Conclusions:

EEG recordings frequently provide important information in critically ill patients[1]. Spikes and PLEDS indicate risk of seizures[2]. More studies are required to associate abnormal EEG findings with patient outcomes.

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Financial Support: None

Poster 132

FACTOR VIIA RAPID REVERSAL PROTOCOL OF WARFARIN IN PATIENTS WITH INTRACRANIAL HEMORRHAGES

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Introduction:
There is an increase in the numbers of the general population taking warfarin. When these individuals sustain a traumatic brain injury (TBI) or spontaneous intracerebral hemorrhage (ICH), the consequences of the anticoagulated state can be devastating. Reversing warfarin with traditional methods of fresh frozen plasma (FFP) and vitamin K takes longer allowing for continuing bleeding. Rapid reversal of warfarin is desirable in this population.

Methods:
A recombinant activated factor VII/FFP protocol was instituted in March 2005 for reversal of warfarin in patients with traumatic and IC hemorrhages. This retrospective study analyzes the outcomes of reversing the international normalization ratio (INR) with a factor VII protocol in patients admitted between March 2005 and December 2006. Variables collected included the Factor VIIa dose, INR before/ after Factor VIIa, time of normalization of INR, thrombotic complications, i.e., venous thromboembolism, myocardial infarction or stroke, and outcome disposition.

Results:
35 patients (TBI=20 ICH=15) on warfarin with traumatic/intracerebral hemorrhages were reversed with the protocol. Sample included18 males and 17 females with mean age of 72.5 years. The mean INR was 2.33 on admit and 1.2 after reversal. Dose range of Factor VII was 1200 mcg-3600 mcg with a mean dose of 1600mcg. Mean units of FFP was 3.4. The mean time to INR correction verified by laboratory analysis was 6 hours. Venous thromboembolism occurred in four patients. There were no reported myocardial infarctions or ischemic strokes in the population. Outcomes at discharge included: TBI subgroup-8 home, 8 ARU, 1 SNIF, and 3 died; ICH subgroup-5 home, 3 ARU, 1 SNIF, 3 transfers and 3 died.

Conclusions:
Rapid reversal of warfarin in patients with TBI and spontaneous ICH can be achieved with the use of a protocol incorporating factor VIIa and FFP. Thrombotic complications occur but can be reduced with minimized doses of factor VIIa.

References: None

Financial Support: None

Poster 133

DANTROLENE MEDIATES VASORELAXATION IN CEREBRAL VASOCONSTRICTION – A CASE SERIES

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Introduction:

Cerebral vasospasm is the leading cause of disability and death after subarachnoid hemorrhage (SAH), and occurs in up to 45% of traumatic SAH (1,2). The only available therapies carry risks: cerebral angiography with intervention can cause stroke, vessel injury or renal failure; HHH-therapy can lead to pulmonary edema and myocardial infarction (3). New vasospasm therapies are needed in the NeuroICU. Dantrolene, a ryanodine receptor antagonist, inhibits intracellular calcium release from the sarco-endoplasmic reticulum, and is neuroprotective in animal models (4). Dantrolene inhibited vasoconstriction in an ex-vivo rat basilar artery model (5). It is used in our NeuroICU for treatment of intractable shivering during Arctic Sun® cooling. We examined the effect of dantrolene on elevated peak systolic middle cerebral artery (MCA) blood flow velocities by transcranial Doppler (TCD).

Methods:

Three consecutive patients with elevated TCD velocities receiving dantrolene (2mg/kg IV q6h) for refractory shivering while on induced normothermia for fever were reviewed. Mean middle cerebral artery (MCA) peak systolic velocities at different insonation depths before and after the dantrolene infusion were compared with a paired t-test.

Results:

Mean (±SD) MCA peak systolic velocities were 297(±8) cm/s, 248(±21) cm/s, and 268(±44) cm/s before dantrolene and 157(±19) cm/s, 169(±20) cm/s and 216(±27) cm/s after dantrolene (p=0.07). There was a trend for individual peak systolic velocities to decrease (Δ-140(±22) cm/s (-47%), Δ-79(±25) cm/s (-32%) and Δ-53(±39) cm/s (-19%)). Systemic physiological parameters (blood pressure, heart rate, central venous pressure, intracranial pressure) remained stable.

Conclusions:

Dantrolene mediated vasorelaxation in cerebral vessels and was safe. This suggests that intracellular calcium release in the smooth muscle might play a significant role in cerebral vasoconstriction. We are currently planning prospective studies to validate these observations.

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Poster 134
WARFARIN-ASSOCIATED INTRAVENTRICULAR HEMORRHAGE
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Introduction:
In this study we have reviewed our experience with anticoagulation-associated IVH. Our goal was to determine if IVH is also an independent prognosticator of fatal outcome in patients with anticoagulation-associated ICH.

Methods:
This study is a retrospective analysis of medical records and computed tomographic imaging. Eighty-eight patients with warfarin-induced intracerebral hemorrhage (ICH) were included, with eight patients with predominant intraventricular hemorrhage (IVH)

Results:
There was very low rate of extension hemorrhage in patients with predominant IVH. Despite that those patients had 50% 30-day mortality. Overall patients with ICH had 45% 30 day mortality. Ventricular extension raised mortality in ICH patients to 75%, while absence of ventricular extension carried only 23% 30-day mortality. IVH was significantly associated with 30-day mortality ($P < 0.001$). Panventricular extension was uniformly fatal in patient with ICH and carried 75% 30-day mortality in patient with predominant IVH. On a multivariate logistic regression model including age, ICH volume, and IVH, ICH volume ($P < 0.001$) and IVH ($P = 0.003$) remained independently associated with early mortality.

Conclusions:
Extension of anticoagulation-induced ICH into ventricular system caused a high mortality, especially in patients with panventricular involvement. IVH is an independent predictor of early death in these patients. In our experience, the majority of intraventricular hemorrhages do not expand over time and poor outcome appears to be related to the magnitude of the initial insult.

References: None

Financial Support: None

Poster 135

DESFERROXAMINE-MEDIATED HIF-1 ACTIVATION DECREASES CEREBROVASCULAR RESISTANCE WITHOUT ALTERING CEREBRAL AUTOREGULATION

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Introduction:
Studies in animal models of stroke have shown that Desferroxamine (DFO) activates hypoxia inducible transcription factor, HIF-1, which reduces brain damage and promotes functional recovery. Our study was designed to investigate the effects of DFO infusion on the cerebral circulation in humans.

Methods:
Fifteen volunteers were enrolled in a randomized double-blinded placebo controlled cross over study. We measured cerebral blood flow velocity in the middle cerebral artery (MCA), arterial blood pressure, end-tidal CO2, as well as HIF-1 protein concentrations in response to 8 hours of DFO versus placebo infusion. Cerebrovascular resistance (CVR) was calculated from the ratio of mean arterial blood pressure (MAP) to blood flow velocity (BFV). The transfer of spontaneous oscillations from beat-to-beat MAP to cerebral BFV were studied using "high-pass filter model" transfer function analysis in three frequency ranges: low (0.03-0.07 Hz), high (0.07-0.15 Hz), and cardiac (within beat).

Results:
Eight hours of DFO infusion resulted in 11±4% (p=0.001), 9±4% (p=0.01) and 12±5% (p=0.01) decrease in CVR at 4, 8 and 12 hours respectively. Changes in CVR were temporally correlated with increased HIF-1 protein concentration by 23 ± 18% at 4 hours, 27 ± 13% at 8 hours and remained elevated at 27 ± 15% four hours post infusion. The transfer function gains were not affected at 8 hours in all three frequency ranges: *low frequency:* placebo 1.62 ± 0.71 vs DFO 1.29 ± 0.57 (p=0.20); *high frequency:* placebo 1.87 ± 1.02 vs DFO 1.92 ± 1.02 (p=0.89); *cardiac frequency:* placebo 1.92 ± 0.69 vs DFO 1.73 ± 0.71 (p=0.56) suggesting no change in regional autoregulation.

Conclusions:
Our study shows that Desferroxamine-mediated HIF-1 activation decreases cerebrovascular resistance without altering cerebral autoregulation. HIF-1 mediated cerebral vasodilation has important clinical implications for novel HIF-1 activators in cerebral ischemic syndromes.

References: None

Financial Support: None

Poster 136

PREDICTIVE FACTORS OF ONE-MONTH MORTALITY AND NEUROLOGICAL COMPLICATIONS FOLLOWING LIVER TRANSPLANTATION.

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Introduction:
Neurological complications are common in orthotopic liver transplant (OLT) patients. Risk factors for and outcomes associated with neurological complications and mortality need to be better characterized.

Methods:
We retrospectively evaluated 227 consecutive patients before and after OLT to define the type, frequency, and risk factors for one-month post transplant neurological complications and mortality.

Results:
Neurological complications were common (n=98), most frequently encephalopathy (56.8%), tremor (26.5%), hallucinations (11.2%), and seizure (8.2%). Factors associated with neurological complications after OLT included preoperative dialysis, hepatorenal syndrome, renal insufficiency, intra-operative dialysis, preoperative encephalopathy, pre-operative mechanical ventialtion, and infection. Preoperative infection was an independent predictor of neurological complications (OR 2.83, 1.47 – 5.44). One-month mortality was 8.8% and was independently associated with urgent retransplant, preoperative intubation, intraoperative use of multiple pressors and arrhythmia.

Conclusions:
Neurological complications are common in patients undergoing OLT. Preoperative infection is an independent risk factor for neurological complications.

References: None

Financial Support: None

Poster 137

TREATMENT OF SYMPTOMATIC INTRACRANIAL AND VERTEBRAL ARTERIAL STENOSES WITH A NOVEL INTRACRANIAL STENT.

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Introduction:

Previously endovascular intracranial atherosclerotic disease (ICAD) treatment used coronary stents with high complication rates. A specifically designed intracranial stent may have fewer complications and more distal cerebral stenosis may be treated. To explore the technical feasibility and safety results of treatment of symptomatic vertebral and intracranial atherosclerotic disease (ICAD) using the first and recently available self expanding intracranial stent.

Methods:

A prospective cohort study of balloon angioplasty with stent placement in 53 patients with ICAD refractory to medical treatment. All patients received intraprocedural standard heparin and clopidogrel and ASA daily before and after the procedure. Results are reported as mean ± standard deviation.

Results:

The initial stenosis was 72.1±8.5%. Balloon angioplasty resulted in an average residual stenosis of 52±11.3% reduced further to 21.4±2.4% after stent deployment. Arterial dissection of the target artery or symptomatic distal emboli was not encountered. In one case (1.8%), the stent could not be deployed into M2 stenosis. There was one case of post procedure hyperperfusion injury (1.8%). One patient out of 10 who underwent follow-up angiogram showed re-stenosis and required re-treatment and one patient treated in two stages All patients were either stable or improved after the treatment. No patient had recurrent TIA or stroke.

Conclusions:

This study represents the largest single center experience of a specifically designed intracranial balloon and self-expandable stent for the treatment of symptomatic ICAD. Although restenosis in this cohort is less than the subgroup of WASID, the confidence intervals are overlapping. Randomized clinical trial is needed to further elucidate the role of stenting in high-risk WASID population. This approach may represent a rapid, clinically effective and technically safe treatment of ICAD when medical therapy fails.

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Poster 138
CONVULSIVE STATUS EPILEPTICUS: A UNIQUE PRESENTATION OF THE REVERSIBLE CEREBRAL VASOCONSTRICTION SYNDROME
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Introduction:
The reversible cerebral vasoconstriction syndrome (RCVS) is characterized by sudden, severe headache, variable neurological signs, and neuroimaging evidence of reversible multifocal areas of intracranial vasoconstriction (1).

Methods:
Case report of convulsive status epilepticus (CSE) as a presenting feature of RCVS.

Results:
A 46 year-old woman with a history of migraine presented with severe headache, nausea, vomiting, and behavioral changes. On arrival, she was uncooperative and combative without lateralizing neurological deficits or vital sign abnormality. Six hours after admission, she developed refractory, persistent generalized convulsive activity lasting 15 minutes. Seizures were ultimately controlled with a propofol infusion. Subsequent lumbar puncture demonstrated normal opening pressure, WBC 31, RBC 570, Protein 73, Glucose 104, and no xanthrochromia. Gram stain, cultures and HSV PCR were negative. Brain MRI 24 hours later revealed subcortical T2-signal abnormalities involving the posterior frontal, parietal, and occipital lobes bilaterally. MRA and conventional angiography demonstrated multifocal areas of concentric stenosis involving the proximal segments of the MCA's, ACA's, and PCA's bilaterally. DWI and MRV were normal. Urine toxicology, urine pregnancy test, ANA, Anti-SSA/SSB, HIV, Lyme, RPR, and Hepatitis screen results were negative. ESR peaked at 90 in the setting of an acute pneumonia. The patient's clinical condition improved without seizure recurrence. Three weeks later, MRI/MRA demonstrated near-complete resolution of the arterial abnormalities and normal brain parenchyma. Repeat lumbar puncture and ESR were normal.
The diagnosis of RCVS is supported by reversible vascular abnormalities and the absence of an alternate etiology. Although seizures have been reported as a manifestation of RCVS, convulsive status epilepticus has not been previously reported. The initial CSF abnormalities were likely explained by increased permeability of the blood-brain barrier after CSE (2).

Conclusions:
This case improves our understanding of the diverse array of neurological symptoms associated with RCVS. In cases of CSE without an identifiable precipitant, neurovascular imaging should be considered.

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Poster 139

DCD: PROBLEMS WITH ELIGIBILITY DETERMINATION AND PROCESS

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Introduction:

Solid organ donation after cardiac death (DCD) is increasingly practiced, and the JCAHO has now implemented accreditation standards that require it to be offered to families of all patients with imminent death who have a high likelihood of dying within 90 minutes of treatment withdrawal. The recommended "model elements" of DCD protocols delineates ideal DCD candidates; mainly those with disabling neurological injuries. However, the eligibility elements are too neurologically simplistic, with inappropriate assumptions, and without system safeguards to prevent against rare but destructive sensationalistic conspiratorial accusations.

Methods:

We present three neurocritical care patients who would be considered ideal DCD candidates by model protocols whose examples illustrate basic flaws in eligibility determination and process.

Results:

Our three patients had intracerebral hemorrhage (thalamic, frontal lobar, and pontine, respectively). The first two had withdrawal of treatment and would have been considered eligible for DCD with a high likelihood of death within 90 minutes after withdrawal. Both survived greater than 24 hours but would have dies in less than 30 minutes if they would have been allowed to die of airway obstruction. The third would have underwent successful DCD if treatment withdrawal had been recommended early in his course, precluding his good survival. There are key problems with the practical application of model DCD policies that refer to eligible patients with neurological injuries that are "non-recoverable and irreversible" or have "ventilator dependency." These and other flaws will be vividly highlighted with constructive suggestions.

Conclusions:

1. There are significant problems with DCD policies regarding eligibility criteria and process
2. Since many of these patients are cared for by neurocritical care physicians, we must passionately identify these problems and develop constructive strategies to address them

References: None

Financial Support: None

Poster 140

DIFFERENT SCENARIOS OF HEAD AND NECK COOLING IN LOCALE CEREBRAL HYPOTHERMIA TREATMENT

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Introduction:

Local cerebral hypothermia (LH) applied by transcranial cooling with a helmet and/or cooling the blood flow via a collar around the neck could be alternatively applied to systemic hypothermia (SH) [1, 2] in order to maximize neuroprotection whilst minimizing systemic side effects.

Methods:

A spherical head model and a neck model derived from an MR-slice including the relevant blood vessels are used to simulate different cooling strategies. First, systemic cooling was simulated using the blood temperature characteristic derived from temperature monitoring of patients with severe subarachnoid hemorrhage while suffering from brain edema with elevations of the intracranial pressure (ICP) and/or cerebral vasospasm (CVS) while treated with mild SH.. Second, cooling with a helmet was simulated by setting the boundary at the upper half of the sphere to a constant temperature of 3°C. Third, different scenarios for neck cooling have been simulated to derive optimal cooling of the carotid artery via collar. In order to derive the corresponding temperature within the brain the head and neck model were linked via the heat exchange of the blood flow.

Results:

For SH the deep brain layers reach 33°C after 3-4 hours. LH with a helmet leads to 33°C only in the upper head layers, but deep brain regions remain on 37°C for cooling periods of more then 24h, when cooling with 3°C. Cooling with a collar filled with water at 3°C leads to 36°C in the brain for dry skin and 33.7°C for wet skin. Cooling with a collar containing graphite water leads to 33.5°C in the brain.

Conclusions:

The results suggest that LH of deep brain regions by transcranial cooling alone is not feasible, while appropriate cooling using a collar can lead to LH. These preliminary results will be verified within clinical trials.

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Poster 141

ACUTE DISSEMINATED ENCEPHALOMYELITIS IN THE INTENSIVE CARE UNIT: CLINICAL FEATURES AND OUTCOME OF 20 ADULTS.

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Introduction:
Acute disseminated encephalomyelitis (ADEM) is an inflammatory disorder of the central nervous system. It typically follows a minor infection and is thought to be immune-mediated. Because it is a rare disease in adults admitted to the intensive care unit (ICU), we describe its characteristics and outcomes of patients.

Methods:
A retrospective (2000–2006), observational multicenter study was conducted in 7 medical ICU. Clinical, biological and neuroimaging features of patients diagnosed with ADEM were evaluated. Functional prognosis was graded using the modified RANKIN (mR) scale.

Results:
At ICU admission, the patients' (n=20) median [25^{th}–75^{th} percentile] Glasgow coma score (GCS) was 7 [4-13], temperature 39 [38-39] °C. Six patients (30%) had seizures, 17 (85%) had a motor deficit and 14 (70%) required mechanical ventilation. Fifteen (75%) patients had cerebrospinal fluid pleocytocis. White-matter lesions on magnetic resonance images were present in all cases. All patients received high-dose steroids. Five (25%) patients died. Fourteen (70%) patients were able to walk without assistance ($mR \leq 3$) at follow-up (7 [3-9] months). Compared to the latter, patients who died or were severely disabled at the follow-up evaluation (6 (30%) patients, $mR > 3$) had significantly lower GCS (4 [3-4] versus 12 [7-13], $p = 0.002$) and more frequent seizures (4 (67%) versus 2 (14%), $p= 0.02$) at admission.

Conclusions:
Unlike previous reports, our results showed that ADEM requiring ICU admission is a severe disease causing high mortality and 35% of the patients had persistent functional sequelae. Intensivists should be awared of ADEM clinical features to initiate appropriate immunomodulating therapy.

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Poster 142

ENDOTRACHEAL INTUBATION FOR MANAGEMENT OF ADULT STATUS EPILEPTICUS

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Introduction:

Aggressive anticonvulsant management to terminate seizure activity often causes excessive respiratory depression necessitating endotracheal intubation (EI). We determined frequency and predictive factors of endotracheal intubation in adult patients presenting with status epilepticus (SE).

Methods:

We performed a retrospective chart review of adults admitted with diagnosis of SE from an academic center over a 6-year period (2000-2005). SE was defined as continuous protracted or recurrent seizure activity without regaining pre-existing level of consciousness.

Results:

Ninety episodes of SE in 87 patients (45M/42F) with mean age 53 ± 2 (mean ± SEM) were included. Common seizure etiologies were subtherapeutic antiepileptic drug (AED) levels (31%), acute CNS disease (27%) and subacute/chronic CNS disease (22%). Of the 90 episodes of SE, 56 (66%) required EI, with mechanical ventilation for 7.8 ± 0.9 days. Fifty-three percent of patients receiving only first line (benzodiazepine) and/or non-sedating AEDs required EI. The most common indication was failure to protect airway. Five patients (6%) required reintubation for reoccurrence of SE for 18 ± 11 days and 13 patients (15%) required tracheostomy. Factors significantly associated with EI on multivariate analysis were acute CNS disease (ischemia/hemorrhage, infection, metabolic abnormality and mass lesion)($p=0.04$) and pharmacologic coma to control SE (third line) ($p=0.004$). Only seizure treatment restricted to first line and/or use of non-sedating AEDs was independently associated with intubation for < 48 hours ($p=0.02$). Nine intubated patients (15%) developed ventilator associated pneumonia (VAP) and 3 non-intubated patients (11%) developed hospital-acquired pneumonia.

Conclusions:

A high proportion of SE patients require endotracheal intubation. Although over 50% of patients receiving only first-line treatment were intubated, significant predictors for intubation were presence of acute CNS disease and requirement for pharmacologic coma to control seizures. Prolonged intubation is dependent on seizure treatment, a surrogate for SE severity.

References: None

Financial Support: None

Poster 143

PSEUDO-CENTRAL PONTINE MYELINOLYSIS IN A PATIENT WITH OLIVOPONTOCEREBELLAR ATROPHY (OPCA)

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Introduction:

Central pontine myelinolysis (CPM), one of the osmotic demyelination syndromes, has been historically linked to alcoholism, malnutrition as well as rapid correction of hyponatremia. It has also been reported that amyotrophic lateral sclerosis (ALS) patients are at increased risk for its development. It has not been reported in patients with multiple system atrophy (MSA)

Methods:

We report a patient with MSA type C who developed CPM-like syndrome after rapid changes in serum sodium and osmolarity. The clinical manifestation, patient's course and literature are reviewed.

Results:

Our patient had MSA type C, previously diagnosed with progressive ataxia, bulbar dysfunction and dysautonomia. After surgical correction of bowel perforation, she developed mild hyponatremia that rapidly over-corrected from 131 to 151meq/l in 12 hours period. Within 24 hours her neurological exam declined to complete loss of bucco-lingual function and flaccid areflexic quadriplegia with preserved eye movements. MRI of the brain and high cervical cord obtained 3 days after the insult, failed to show any acute changes. Her clinical deficits gradually improved over weeks toward her baseline disability. The rapid pace of her recovery and absence of new MRI abnormalities in light of the severity of her deficits are atypical for CPM. We hypothesize that her worsened brainstem dysfunction was due to heightened sensitivity to rapid osmotic shifts from the chronic changes associated with her OPCA.

Conclusions:

1. OPCA may predispose patients to worsened brainstem function from rapid osmotic shifts without an MRI correlate to confidently diagnose CPM.
2. The pace and extend of recovery from this "pseudo" CPM may be more rapid and complete than the "true" from of CPM

Patients with OPCA should be approached with awareness of this potential heightened sensitivity to factors that are known to cause brainstem dysfunction.

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Poster 144

LEVETIRACETAM AS ANTICONVULSANT PROPHYLAXIS IN PATIENTS WITH SUBARACHNOID HEMORRHAGE (SAH)

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Introduction:

An anticonvulsant prophylaxis in SAH with high risk for seizures remains a challenge. An efficient i.v. drug, without side effects and minimal interaction with other drugs is difficult to find.
The purpose was to compare valproate (VPA) with levetiracetam (LEV).

Methods:

Retrospective analysis of 18 severe SAH patients underwent anticonvulsant prophylaxis first line either with valproate or levetiracetam monotherapy. In occurence of severe complications or insufficient plasma concentrations (PC) (VPA<350umol/l, LEV<30umol/l) therapy was changed or added.

Results:

In 10 of 13 patients (77%) with VPA the agent had to be changed or added, compared to one change in the LEV group (9 patients,11%). The most frequent reason was insufficient PC of VPA, typically combined with meropenem (4 cases with VPA (100%), never in LEV).
LEV was first line in 5 patients and secondary monotherapy in 4 patients because of a necessary therapy change. Further changes were required in 6 cases from VPA monotherapy to a combination with LTG to elevate the PC. One patient needed a quadruple anticonvulsant because of a relapsing non-convulsive status. Another patient with LEV monotherapy developed seizures because of a low PC (application disturbances).

Conclusions:

Insufficient PC could be observed in more than the half of the patients with VPA monotherapy.
In no LEV patient treated with meropenem a loss of PC was observed, in contrast of all the VPA patients. This suggests that with LEV minimal interactions with meropenem occur.
Changes from intravenous to enteral application (liquid) LEV have to be carefully monitored, because in one third loss of PC was observed.

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Poster 145

ELIGIBILITY FOR THE SURGICAL TRIAL IN INTRACEREBRAL HEMORRHAGE II STUDY IN A POPULATION-BASED COHORT

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Introduction:
No proven treatments exist for intracerebral hemorrhage (ICH). Carefully selected patients may be benefit from surgery, and an international multicenter trial is ongoing. We sought to determine the potential eligibility for surgery in a population-based cohort of ICH patients using the Surgical Trial in Intracerebral Hemorrhage II (STICH-II) criteria.

Methods:
All patients aged ≥18 years residing in the five-county Greater Cincinnati region and hospitalized with nontraumatic ICH in 2005 were identified. ICH volume, location, and presence of intraventricular hemorrhage (IVH) or hydrocephalus were determined. Inclusion and exclusion criteria for the STICH-II trial were used to determine treatment eligibility and reasons for exclusion. Demographics and 180-day mortality were compared among eligible lobar, ineligible lobar, and nonlobar ICH patients.

Results:
During 2005, 286 ICH patients were identified (103 lobar, 126 deep, 23 brainstem, 28 cerebellar and 6 IVH); of these, 22 (7.7%) had no exclusions and presented within 48 hours. The most common reasons for exclusion (not mutually exclusive) among patients with lobar ICH were volume < 10cc or > 100cc (n=46), presence of IVH (n=27), deep extension (n=20), ICH > 1cm from the cortical surface (n=18), and hydrocephalus (n=17). There were no significant age, gender, or racial differences between groups. One of 22 STICH-II eligible patients in our population had surgery, compared with 25 of 264 ineligible patients. Survival at 180 days in STICH-II eligible patients was 64% (n=14) versus 51% (n=41) for ineligible lobar ICH patients (p=0.19).

Conclusions:
In this population-based ICH cohort, 7.7% of patients would have qualified for STICH-II enrollment. Given an estimated 67,000 patients with ICH in the United States annually, up to 5,000 patients may be STICH-II eligible. Other treatment options need to be explored for most ICH patients.

References: None

Financial Support: None

Poster 146

INDUCED NORMOTHERMIA IMPROVES BRAIN NEUROCHEMISTRY AFTER TRAUMATIC BRAIN INJURY

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Introduction:
Brain metabolism studies have documented the presence of either ischemia or metabolic distress for prolonged periods after traumatic brain injury (TBI) and can be monitored using cerebral microdialysis. The effects of temperature on disturbed brain metabolism remain uncertain. This study aims to determine if induction of normothermia (core temperature = 36.5 C) will reduce microdialysis LPR into the normal range.

Methods:
This is a case control study of induced normothermia using a catheter-based cooling method (n = 5) compared with conventional treatment using a cooling blanket to maintain temperature 37-38 C (n = 23). Age and GCS were matched between groups. Cerebral microdialysis was performed at the bedside with immediate measurement of analytes using the CMA600. Hourly values of glucose, lactate, pyruvate, glutamate, glycerol, and LPR were measured and comparisons of mean values were performed between each group. Analyte means acquired before and after induction of normothermia were also compared for each subject.

Results:
Catheter-based induction of normothermia resulted in within-subject reductions in temperature ($36.8 \pm .2$ vs. 37.5 ± 0.3, $p < 0.001$), LPR (30 ± 3 vs. 44 ± 5, $p < 0.001$). The LPR trended down over a period of 24 hours in most cases. As a group, patients with induced normothermia had a lower LPR during the initial 110 hours after TBI as compared with the conventional temperature group (time course anova $p < 0.001$), and had a lower percent time burden of elevated LPR > 40 than the conventional temperature group (16% vs. 29%, $p < 0.001$).

Conclusions:
Induced normothermia results in better temperature regulation than conventional temperature protocol, and a lower sustained LPR over time. These data suggest that normothermia may be a therapeutic target for disturbed metabolism after TBI.

References: None

Financial Support: None

Poster 147

POST-EXTUBATION DYSPHAGIA: A CAUSE OF ASPIRATION PNEUMONIA AND RESPIRATORY FAILURE IN ELDERLY ICU PATIENTS.

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Introduction:

Post-extubation dysphagia in elderly ICU patients[1] may occur from desensitization of gag and cough reflexes, impaired pharyngeal constrictor muscle function, or impairment of the deglutition reflex[2]. We sought to evaluate elderly ICU patients with post-extubation respiratory failure with documented post-extubation dysphagia and aspiration.

Methods:

We report two cases of initially abnormal modified barium swallow (MBS) studies, which improved over time, from in elderly (≥ 65 years) ICU patients with extubation failure. These patients had no primary or central nervous system (CNS) neurological disease causing dysphagia.

Results:

The first patient, age 76 years, was re-intubated 3 days after liver transplantation for aspiration pneumonia. Percutaneous tracheostomy was performed 10 days later after failure to wean from the ventilator. Subsequent speech pathology evaluations and MBS showed dysphagia and aspiration. A percutaneous gastrojejustomy (PEG) was performed. Serial MBS studies showed normalization of swallow and no aspiration after 15 months.
The second patient, age 83 years, was intubated for severe chronic obstructive lung disease (COPD) exacerbation. Initial MBS after first extubation demonstrated severe dysphagia and aspiration. Subsequent aspiration pneumonia developed which caused respiratory failure. After a second extubation, MBS showed improved swallow but silent aspiration. The patient was reintubated for the third time, and tracheostomy and PEG were performed. Three months later, MBS showed no dysphagia or aspiration.
Neurological examinations in both patients revealed normal cranial nerve function, and a critical illness neuropathy and myopathy in the first patient. MRI brain and C-spine in both patients revealed no significant CNS abnormalities.

Conclusions:

Post-extubation dysphagia in elderly ICU patients is an under-recognized phenomenon that may occur after intubation less than 24 hours, and is characterized by dysphagia and aspiration on post-extubation MBS with slow improvement often requiring temporizing tracheostomy. Prospective studies are needed to evaluate post-extubation swallow pathophysiology[3,4] and interventions[5] to prevent aspiration pneumonia.

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Poster 148

LOW 30 DAY MORTALITY IN 480 PATIENTS WITH MODERATE TO VERY LARGE PARENCHYMAL HEMORRHAGE.

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Introduction:

Intracranial hemorrhage (ICH) 30 day mortality by population studies is considerable at 44-51%. Management guidelines remain conjectural.
Aim: To evaluate our neurocritical care ICH protocol for 30 day overall mortality by composite ICH stratification score and expanded hematoma volume categories.

Methods:

Consecutive ICH patients assessed with the ICH score comprising of hemorrhage volume, GCS, intraventricular extension, infratentorial versus supratentorial location and age validity established previously by multivariate analyses [1]. The protocol included the use hypertonic saline for raised intracranial pressure control, intravascular volume optimization, early intubation and tracheostomy for mechanical ventilation, extraventricular drain or bolt intracranial pressure monitoring placement and temperature regulation with a CoolGuard™ catheter where appropriate

Results:

In 480 patients accrued over a 49 months, (mean age, 63.5 years, women 43%) the mean ICH volume was moderate at 34.2 ml (95% CI: 29.3; 39.1, range 0.5 to 255 ml) and mean ICH score was 1.8 (95% CI: 1.7; 2.0, range 0-6). The overall 30 day mortality rate from any cause including withdrawal of care was 93/480 (19%). Mortality percentages within the ICH score categories (grades 0-6) were (2.5, 7.6, 12.9, 29.6, 56.9, 77.7, 66.6). Mortality (30 day) percentage for ICH volumes in increments of 30 (0-240) ml was (7, 28, 37, 52, 69, 71,100). Pearson correlation of ICH score and hemorrhage volume was 0.9 ($p=0.005$) with 81% variance accounted for by the hemorrhage volume. Large to massive ICH (61 to >240 ml) 30 day mortality was 54%. Intraventricular hemorrhage occurred in 47%, infratentorial location in 10% and decompressive craniectomy performed in 3.6% of patients.

Conclusions:

In a moderate sized ICH patient group, our neurocritical care protocol enabled 81 % survival at 30 days and survival of 46% of patients with large to massive hemorrhages. The composite ICH score may improve critical decision making including withdrawal of care.

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Poster 149

PATHOLOGIC DESCRIPTION OF WINGSPAN STENT IN THE SETTING OF ACUTE ISCHEMIC STROKE

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Introduction:

Pathological descriptions after intracranial angioplasty and stenting in acute ischemic stroke are rare.[1,2] We present the autopsy results of a patient who died 7 days after angioplasty and stenting of the right middle cerebral artery (MCA) for acute ischemic stroke.

Methods:

Single case report.

Results:

A 75 year old woman with a history of hypertension presented to an outside hospital with left-hemiplegia and right gaze preference. Initial CT brain was normal. The patient did not receive IV-tPA for unclear reasons, and she was transferred to our institution. She arrived six hours after the onset of symptoms, at which point her NIH stroke scale was 18. She underwent a cerebral angiogram, which showed a distal right internal carotid artery bifurcation occlusion. Snare mechanical embolectomy was performed without success. We subsequently performed balloon (Gateway) angioplasty and stent (Wingspan) placement from the distal ICA to the proximal M1 segment, which established TIMI 1 antegrade flow. Repeat CT brain showed an evolving right MCA territory infarct with worsening cerebral edema. Despite maximal medical therapy, our patient died of a malignant MCA ischemic infarct with ensuing subfalcine and transtentorial herniation on post-procedure day 7. Family requested autopsy. We report the pathologic changes in an acutely occluded artery after revascularization with angioplasty and stenting (see photographs).

Conclusions:

Our case provides rare pathological description and photographs of intracranial angioplasty and stenting in the setting of acute ischemic stroke. Further studies to better understand the correlation of clinico-radiographic results with pathology in acute stroke intervention are warranted.

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Poster 150

NEUROLOGIC OUTCOME FROM OPEN DESCENDING THORACIC AND THORACOABDOMINAL AORTIC OPERATIONS IN THE ERA OF ENDOVASCULAR REPAIR

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Introduction:
Spinal cord ischemia and stroke are recognized complications of descending thoracic (DTA) and thoracoabdominal aortic (TAA) operations. However, there are limited data available on outcomes since the advent of thoracic endovascular aortic repair (TEVAR). The purpose of the study was to perform a contemporary review of the frequency of neurologic ischemic complications from these procedures and to create a model to predict risk of death or disability.

Methods:
We reviewed charts from consecutive patients who underwent open DTA and TAA repair at our high volume center from January 2000 through June 2005.

Results:
232 open DTA and TAA operations were included in the analysis. During this time period, 106 patients received TEVAR, accounting for 66% of all DTA repairs. Ischemic neurologic complications occurred in 87/232 procedures (38%): 64 patients (28%) developed spinal ischemia, 14 patients (6%) had a stroke, and 9 patients (4%) had both spinal ischemia and a stroke. The 30 day in-hospital mortality was 18%. A multivariable logistic regression incorporating characteristics known prior to surgery resulted in development of a score to stratify risk of poor outcome (defined as death or neurologic disability) based on age ≥60 (1 point), history of cerebrovascular disease (1 point), Crawford extent II or III repair (1 point), and acute rupture (1 point). The score had good discriminative function with an area under the receiver-operator characteristic curve of 0.75. Patients with score ≥ 3 had an estimated 54% risk for poor outcome, while those with score <1 had an estimated risk of 7-9%.

Conclusions:
Ischemic neurologic complications were prevalent in this contemporary review and were associated with poor outcomes after open DTA and TAA repair. Risk of death or neurologic disability can be estimated based on factors known prior to surgery.

References: None

Financial Support: None

Poster 151
HEAD AND NECK COMPUTED TOMOGRAPHIC ANGIOGRAPHY (CTA) AND THE CONFIRMATION OF BRAIN DEATH?
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Introduction:
Although the bedside "Apnea Test" continues to be the most widely performed and accepted confirmatory test for "Brain Death" in some instances it cannot be performed due to the presence of hypoxemic respiratory failure. Radionuclide brain scans, four vessel cerebral arteriography, electroencephalograms (EEG), and cerebral arterial Doppler exams have all been used for confirmation of brain death, but all suffer from serious limitations. The advantages of CTA is that it is less expensive compared to four vessel cerebral arteriography and easier to obtain, it is not operator dependent as is the arterial Doppler study, and it is not subject to artefact as is the EEG.

Methods:
This paper is a review of ten patients cared for in a major University Neuro-Critical Care Unit in 2006-2007 who clinically progressed to brain death on bedside neurological exam and then underwent evaluation by both a CTA and an "Apnea Test."

Results:
Nine of the ten patients met criteria for "Brain Death" by the "Apnea Test". One was noted to have minimal respirations during the apnea test and it was aborted. That patient by CTA although having only trace anterior circulation flow continued to demonstrate some residual posterior circulation flow. Interestingly no brainstem reflexes accept very shallow respirations persisted. Most of the others "Brain Dead" patients demonstrated no intra-cerebral blood flow, but a few patients although having no posterior circulation flow showed some residual anterior circulation flow.

Conclusions:
Head and neck CTA promises to be a useful confirmatory test for brain death. If CTA demonstrates no intra-cerebral blood flow compared to normal extra-cerebral blood flow it may ultimately be accepted as a single confirmatory test for brain death. At the present time CTA remains investigational. Validation studies are needed.

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Poster 152

BICKERSTAFF'S "GRAVE SYNDROME WITH BENIGN PROGNOSIS" IN A MEDICAL ICU

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Introduction:
We present a case report of an elderly male admitted to the ICU with weakness evolving into Bickerstaff's encephalitis.

Methods:
A 78 year old white male with antecedent respiratory illness of 2 weeks presented to ER with lower extremity weakness. He was admitted to ICU for airway monitoring, intubated on day 2, wean attempt on day 3 showed the patient was apneic in coma with signs of external ophthalmoplegia, flaccid tetraparesis, and absent reflexes. A CVA was ruled out, patient maintained on ventilator, and diagnosis was made on day 8 based on persistent clinical findings and severely abnormal nerve conduction studies. Further serology showed serum anti-GQ1b IgG antibody. IVIG treatment led to acute renal failure. Patient showed gradual improvement, often waxing and waning symptoms with plasmapheresis and supportive measures.

Results:
CSF analysis was normal except elevated protein, acetylcholine receptor antibody was negative, MRI brain showed chronic changes secondary to hypertension, and workup was negative for infectious or neoplastic etiologies. Patient developed complications of pneumonia during the hospital stay, was able to wean off ventilator on day 30 and was discharged from hospital on day 35.

Conclusions:
Bickerstaff's encephalitis should be considered in a patient with signs of brainstem involvement. We highlight the significant overlap in GBS variants. Learning points from this report also arise from review of and discussion of presentation, CSF analysis, imaging, electrophysiological and immunologic studies of GBS and its variants like Miller-Fisher syndrome and Bickerstaff's encephalitis.

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Poster 153

CATASTROPHIC HYPERAMMONEMIA: A SERIES OF 4 PATIENTS.

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Introduction:

Although a neurological emergency, management guidelines for severe hyperammonemia in adults remains unclear.

Methods:

Describing 4 patients with encephalopathy(GCS < 7) and severe hyperammonemia(>400 mg/dl)
Patients:
A: 51 y/o female with hypothyroidism and gastric bypass.
B: 47 y/o male, with ESLD, GI bleed and hepatic encephalopathy
C 31 y/o female with alcoholism and gastric bypass.
D: 27 y/o female with fulminant hepatic failure and cerebral edema.

Results:

Management:
Peak serum ammonia was greater than 400mg/dl in all patients. Delayed therapy caused sustained severe hyperammonemia for several days in patients A, B and C. Sodium benzoate and phenylacetate use (patients A,C) was less effective than continuous veno-venous hemofiltration (CVVH) use (patients A, B, D) in lowering serum ammonia. On day 1, patient D received therapeutic hypothermia and CVVH which lowered serum ammonia level to <100mg/dl in 72 hours. Amino acid levels suggested a urea cycle disorder in patient C but not in others.
Imaging & Outcome:
MRI (patients A, B, C) within the first week demonstrated restricted diffusion involving insular cortices, thalami, frontal, parietal and temporal lobes. Occipital lobes, cerebellum and brainstem were spared. Only minimal signal changes were noted in patient D. MR spectroscopy(patients B, C) showed decrease in NAA and choline peaks and increase in myoinositol and glutamine peaks. Patients A and B expired without neurological improvement. Autopsy (patient A) revealed astrocyte swelling and edema. Patient C suffered severe cognitive impairment and intractable epilepsy. Patient D returned to baseline cognitive function.

Conclusions:

Hyperammonemia causes irreversible brain injury[1-4].Hyperammonemic injury produces a distinct pattern of cytotoxic edema on MRI[5, 6] that appears to correlate with neurologic outcome. Aggressive early correction of serum ammonia irrespective of cause can prevent irreversible neurologic damage. CVVH is an effective therapy for severe hyperammonemia.[7, 8] Therapeutic hypothermia may have a role in management[9-11].

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Poster 154

SUCCESSFUL THROMBOLYSIS WITH INTRA-ARTERIAL TPA 16 HOURS AFTER ONSET OF LEFT MCA EMBOLIC STROKE

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Introduction:

Recognize the potential use of delayed intra-arterial rt-PA beyond 6 hours in a subset of patients presenting with stroke-in-evolution.

Methods:

An 82-year-old right-handed Caucasian woman developed acute onset right hemiparesis and nonfluent aphasia (NIHSS score of 10). Thrombolysis was not initiated due to resolution of symptoms within 1 hour of onset. Despite treatment with a heparin for new atrial fibrillation, intermittent weakness and aphasia continued to occur over the next 10 hours. Brain MRI at 7 hours revealed no restricted diffusion. Hypertensive therapy initiated in the neuro-ICU demonstrated improvement in strength and speech. At 16 hours, a cerebral angiogram was performed revealing a filling defect at the proximal superior division of the left MCA bifurcation. Presence of good collaterals and the absence of distal embolization maintained slow filling of the M2 despite the proximal clot. Microcatheter infusion of 5.6 mg intra-arterial rt-PA locally into the MCA clot over 30 minutes is performed.

Results:

Near complete resolution of the clot without distal embolization was achieved. Patient exhibited significant clinical recovery with no aphasia and minimal residual right hemiparesis.

Conclusions:

There is little evidence to guide management of fluctuating, reversible ischemic symptoms often referred to as stroke-in-evolution. In these cases, the window of opportunity for thrombolytic therapy is not well defined. Rare case reports of large basilar or internal carotid occlusion presenting with fluctuating symptoms over many hours or even days have shown to have benefited from delayed intra-arterial thrombolytic therapy[1-3].Our patient did not meet the criteria for thombolytic therapy, however the absence of restricted diffusion on MRI and reversibility of deficits with hypertensive therapy guided our decision to proceed with intra-arterial rt-PA. Further studies are needed in order to evaluate the use of intra-arterial rt-PA in patients with stroke-in-evolution.

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Poster 155

MANAGEMENT OF HYPERACUTE ISCHEMIC STROKE WITH URGENT SELF-EXPANDING INTRACRANIAL STENT DEPLOYMENT.

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Introduction:

Self-expanding intracranial aneurysmal and atherosclerotic stents (SEIS) allow rapid intracranial access to lesions with safe deployment. Urgent stent deployment is becoming an option in treating hyperacute ischemic stroke (AIS) that is refractory to, or with contraindication against conventional management.

Methods:

AIS patients undergoing urgent stenting with SEIS were identified. Retrospective analysis evaluated procedural protocols and clinical response to treatment. Descriptive statistics are presented. The Wilcoxon Signed Rank Test was used to compare initial and follow-up NIHSS and modified Rankin Score (mRS).

Results:

Neuroform (n=3) and Wingspan (n=2) stents were successfully deployed in the M1 (n=3), M2 and M3 segments (n=1 each) of the middle cerebral artery. Time of stent deployment from AIS onset ranged from 4 to 7.5 hours. Periprocedural oral antiplatelet, glycoprotein IIb/IIIa antagonist, and tPA use varied, depending on stroke severity, imaging findings, and time of initial presentation. All TIMI Scores before stenting were zero or one, and after stenting, two or three. Acute (<2hr) in-stent thrombosis occurred in patient 2, which was successfully treated with balloon angioplasty. Follow-up ranged between 2 and 4 months. Pre-stenting and follow-up NIHSS follow: patient 1: 17, 3; patient 2: 10, 0; patient 3: 8, 3; patient 4: 29, 6; patient 5: 19, 6 ($p=0.043$). After AIS onset, all mRS were 5, except patient 2 (4) and patient 3 (3). mRS at follow-up was 1, 0, 2, 4, and 2, respectively ($p=0.043$). Follow-up angiography in patients 1, 2, and 5 showed stent patency. Follow-up imaging was not available for patients 3 and 4.

Conclusions:

This experience with urgent stenting in hyperacute ischemic stroke is comparable to prior studies, demonstrating technical feasibility, excellent recanalization and promising clinical outcomes. Acute in-stent thrombosis, perforator occlusion, reperfusion injury, and long-term restenosis are factors that may limit widespread use.

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Poster 156

THE ROLE OF DIFFUSION AND PERFUSION WEIGHTED MRI (DWI/PWI) IN DIRECTING HYPERTENSIVE-HYPERVOLEMIC THERAPY (HHT) IN PATIENTS WITH ACUTE ISCHEMIC STROKE

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Introduction:
Induced hypertensive hypervolemic therapy (HHT) is postulated to augment cerebral blood flow and protect the ischemic penumbra after ischemic stroke. DWI and PWI can delineate areas of hypoperfusion and ischemic neuronal injury and help guide utilization HHT.

Methods:
We describe 3 patients with middle cerebral artery (MCA) territory embolic infarcts. All three patients did not fulfil the criteria for thrombolysis. Penumbra size was monitored before and after HHT using DWI/PWI. MRI with DWI/PWI was performed at initial presentation and within 72 hours of the initiation of HHT therapy. HHT initiated when lesion on PWI > DWI and was discontinued when lesion on DWI $\geq$ PWI.

Results:
Case 1. 57 year-old male with expressive aphasia and right-sided hemiparesis. Initial MRI revealed a perfusion defect larger than diffusion defect. 72 hour follow-up MRI revealed defect on DWI that was $\geq$PWI.
Case 2. 62 year-old female global aphasia and right hemiparesis. Initial MRI showed perfusion delay with no DWI restriction. MRI at 48 hours revealed larger PWI signals. MRI at 96 hours revealed decrease in the perfusion signals with matching region of abnormal diffusion.
Case 3: 32 year old female with left hemiparesis and dysarthria. MRI revealed large PWI signals with a smaller area of right frontal DWI abnormality. MRI 72 hours later revealed decrease in the PWI when compared to DWI.
In all 3 patients, HHT was halted when the lesion size on DWI $\geq$ PWI. These patients were safely discharged from the ICU in stable neurological condition.

Conclusions:
Patients with defects on PWI $\geq$DWI may benefit from HHT, but HHT should be discontinued when this mismatch reverses. This study illustrates the potential role of newer imaging techniques in guiding the management of acute ischemic stroke in the ICU.

References: None

Financial Support: None

Poster 157

PREDICTORS OF WITHDRAWAL OF CARE IN INTRACEREBRAL HEMORRHAGE PATIENTS

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Introduction:
Most studies do not report the cause of death in ICH. This is an important issue since these patients seldom die from the primary disease. Most often withdrawal of care is the cause of death. The neurosurgeons or neurointensivists often prognosticate outcome based on their experience and prejudice. Our objective was to identify predictors for withdrawal of care and assess any practice differences between neurointensivists and neurosurgeons.

Methods:
All ICD9 primary diagnosis of ICH were retrieved from our hospital discharge database between 2003-2005. Patients with trauma, SAH, tumor and ones with surgical evacuation were excluded. We noted age, gender, cause of death and discharge location. Non-contrast CT of brain at admission, at 24 and/or 48hours were reviewed. Clot size, location, IVH grade, pineal and septum pellucidum shift and hydrocephalus were noted.

Results:
58 patients were included in the study. 37 patients were admitted to the NICU service (group A) and 21 to neurosurgery with the NICU service consulted (group B). There were no statistical differences in clot size, pineal shift and septum shift between both groups. The mortality in group A was 16.2% vs 52.4% in group B ($p=0.027$). Three causes of mortality were identified: 1)withdrawal of care, 2)cardiac death and 3)brain death. A total of 14/17 patients had their care withdrawn (35.7% in group A and 64.3% in group B). Neurosurgeons were withdrawing care more often in patients with severe IVH where as neurointensivist more in patients with larger ICH. Withdrawal of care even in patients with ICH volume <30cc was higher in group B vs group A (46% vs 4%, $p=0.001$).

Conclusions:
Withdrawal of care is the major cause of mortality in ICH patients. The decision to withdraw seems to vary with the treating group of physicians. A consensus on predictors of outcome for ICH patients is needed.

References: None

Financial Support: None

Poster 158

THE YIELD OF DIGITAL SUBTRACTION ANGIOGRAPHY IN INTRACEREBRAL HEMORRHAGE

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Introduction:

The presence of specific vascular abnormalities in ICH have has important implications for management. Previous studies have shown up to 35% of an unselected group of patients have abnormalities on angiography, but the optimal diagnostic strategy to identify these subjects is still not well characterized.
The goal of our study is to determine the yield of digital subtraction angiography (DSA) in a selected group of patients with intracerebral hemorrhage.

Methods:

All ICH patients presenting to our hospital over a three year period (2003-06) were selected for a conventional angiogram if: younger than 50 years old, suffered from a lobar hemorrhage, or a pure intraventricular hemorrhage. Patients not expected to survive the hospitalization were excluded. Patients' data was collected in a retrospective fashion.

Results:

Thirteen out of 60 patients were found to have a positive angiogram with an underlying vascular abnormality related to the hemorrhage (21.6%): seven arterio-venous malformations, three aneurysms, one pseudo-aneurysm, one vasculitis and one moya-moya disease. In the negative angiogram group, 31 had lobar, 14 had basal ganglia, 7 had thalamic, 5 had cerebellar and 3 had brainstem hemorrhages respectively. In the positive angiogram group, 8 had lobar, 3 had basal ganglia, 1 had thalamic and 1 had a pure IVH respectively. There was no significant difference in age, gender, location of hemorrhage when grouped by angiogram findings.

Conclusions:

A significant proportion of patients with intracerebral hemorrhage have underlying vascular anomalies on angiography. Further studies are needed to further determine optimal patient selection and timing for DSA. Also the role of other neuroimaging modalities needs to be clarified.

References: None

Financial Support: None

Poster 159

CONCOMITANT CEREBRAL ABSCESS AND SYSTEMIC HYPOXEMIA: SUGGESTIVE OF HEREDITARY HEMORRHAGIC TELANGIECTASIA (OSLER-WEBER-RENDU SYNDROME)

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Introduction:

We present a case of cerebral abscess and concomitant hypoxemia which lead to the diagnosis of hereditary hemorrhagic telangiectasia (HHT) or Osler-Weber-Rendu syndrome. HHT is an autosomal dominant disorder which presents with multisystem vascular dysplasias. Neurologic manifestations include cerebral or spinal AVMs, which can present clinically as headache, intracerebral hemorrhage, stroke, TIA, or seizure. Our patient had pulmonary AVMs which predisposed her to the formation of cerebral abscesses.

Methods:

A 49 year old female with history of epistaxis, presented with hemiparesis and poor mental status. Brain imaging showed large contrast-enhancing mass surrounded by significant cerebral edema, pronounced mass effect and signs of brain herniation which required emergent drainage of the mass and decompressive hemicraniectomy with duroplasty. Histopathology of the mass revealed the diagnosis of cerebral abscess. In addition, she was started on IV antibiotics and steroids. The patient also had persistent systemic hypoxemia that didn't correct with increasing FiO2 in the context of normal chest X-ray.

Results:

Cultures from the abscess cavity grew Streptococcus viridans. Vascular imaging of the chest disclosed multiple bilateral pulmonary AVMs that justified the refractory hypoxemia through a right to left shunt mechanism. Based on history and physical exam findings, a diagnosis of hereditary hemorrhagic telangiectasia (Osler-Weber-Rendu syndrome) was made. Pulmonary vascular abnormalities were embolized and the right to left shunt was abolished. With appropriate medical and surgical treatment the abscess was cured. The patient made an excellent neurological recovery.

Conclusions:

1. Recognizing the combination of cerebral abscess and systemic hypoxemia is a key factor for the diagnosis of Hereditary hemorrhagic telangiectasia (HHT)
2. HHT (Osler-Weber-Rendu syndrome) is a genetic condition that can predispose for cerebral abscess given that part of the cardiac output by-passes the pulmonary capillary bed that normally acts as a filter for microorganisms.
3. Patients suffering from brain abscess and associated severe cerebral edema can have an excellent prognosis with appropriate treatment that sometimes may even include decompressive craniectomy for the treatment of intracranial hypertension and brain herniation.

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