neurocritical care society Neurocrit Care (2008) 11:S1–S224
DOI 10.1007/s12028-008-9282-0

NEUROCRITICAL CARE SOCIETY
7TH ANNUAL MEETING
November 11-14, 2009

ABSTRACT SUPPLEMENT

Hilton New Orleans Riverside
New Orleans, Louisiana

Neurocritical Care Society 7th Annual Meeting

PLATFORM SESSION I
Thursday, November 12, 2009
3:30 – 5:30 p.m.
Napoleon Ballroom

3:30 - 3:45 p.m. Michael Diringer, MD
Impact of Induced Hypertension on Cerebral Oxygen Delivery in Subarachnoid Hemorrhage

3:45 - 4:00 p.m. Jennifer Frontera, MD
National Trend in Incidence, Cost and Discharge Disposition after Subdural Hemorrhage from 1998-2007

4:00 - 4:15 p.m. William Armstead, PhD
Phenylephrine Infusion Prevents Impairment of Cerebral Autoregulation During Hypotension after Brain Injury in Female But Aggravates Impairment in Male Piglets Through Modulation of ERK MAPK Upregulation

4:15 - 4:30 p.m. Benjamin Emanuel, DO
Hypertensive Hypervolemic Vasodilatory (HHVD) Therapy with Intravenous Nicardipine for the Treatment of Cerebral Vasopsasm

4:30 - 4:45 p.m. Carmelo Graffagnino, MD
Clevidipine Rapidly and Safely Reduces Blood Pressure in Patients with Acute Intracerebral Hemorrhage: Interim Results of the ACCELERATE Trial – First Clinical Experience in ICH

4:45 - 5:00 p.m. Stefan Dupont, MD, PhD
Prediction of Angiographic Vasospasm after Aneurysmal Subarachnoid Hemorrhage: Value of the Hijdra Sum Scoring System

5:00 - 5:15 p.m. Stephan Mayer, MD
Subarachnoid Hemorrhage: Who Dies, and Why? Ten Year Experience of the Columbia University SAH Outcomes Project

5:15 - 5:30 p.m. J. Javier Provencio, MD
Behavioral Changes Of Vasospasm Reversed by Neutrophil Depletion in a Mouse Model of Subarachnoid Hemorrhage (SAH)

ORAL ABSTRACTS
(underlined author is presenting author)

3:30 – 3:45 p.m. Thursday, November 12, 2009

IMPACT OF INDUCED HYPERTENSION ON CEREBRAL OXYGEN DELIVERY IN SUBARACHNOID HEMORRHAGE

Michael Diringer, Allyson Zazulia, Raj Dhar, Tom Videen, Colin Derdeyn
Washington Univeristy, St. Louis, MO, United States

Introduction:
The most common cause of secondary neurological injury following subarachnoid hemorrhage (SAH) is delayed ischemic deficit (DID). Three processes conspire to result in a fall in cerebral blood flow (CBF): arterial vasospasm, intravascular volume depletion and impairment of autoregulatory function. Impaired autoregulation, however, may be necessary for induced hypertension to be effective in improving oxygen delivery (DO_2).

Methods:
We studied a series of 10 patients with SAH during the period of highest risk for vasospasm. These patients were part of a blinded study in which they were randomized to simvistatin 80 mg/d or placebo. All underwent early aneurysm repair and received nimodipine and aggressive hydration. DID was treated with induced hypertension and endovascular interventions. On post-hemorrhage days 7-10 each patient underwent a diagnostic angiogram and a PET study with measurement of CBF before and after raising mean arterial blood pressure (MAP) by 20% with phenylephrine. DO2 was calculated as arterial oxygen content x CBF, and autoregulatory index (AI) as %ΔMAP/%ΔCBF.

Results:
Patients were studied 8.2 ± 1.8 days after hemorrhage. Modified Fisher grade was 2 (n=2), 3 (n=6), or 4 (n=2). After phenylephrine, MAP rose from 107.70 ± 7.63 to 136.2 ± 5.25 mmHg (p=0.000), global CBF from 40.8 ± 16.2 to 44.5 ± 17.0 ml/100g/min (p=0.001) and DO_2 from 393 ± 137 to 430 ± 150 ml/100g/min (p=0.03). AI was abnormal (>0.4) in 4. In patients with normal AI, DO_2 went from 405 ± 153 to 437 ± 177 ml/100g/min (p=0.1) whereas in those with abnormal AI DO_2 went from 407 ± 279 to 495 ± 277 ml/100g/min (p=0.03).

Conclusions:
While induced hypertension improved global DO2 in most SAH patients, it was more effective in those with impaired autoregulation.

Financial Support: Michael Diringer was a consultant for Actelion.

3:45 – 4:00 p.m. Thursday, November 12, 2009

NATIONAL TREND IN INCIDENCE, COST AND DISCHARGE DISPOSITION AFTER SUBDURAL HEMORRHAGE FROM 1998-2007

Jennifer Frontera, Natalia Egorova, Alan Moskowitz
Mount Sinai School of Medicine, New York, NY, United States

Introduction:
Subdural hemorrhage (SDH) is a common type of intracranial hemorrhage, yet little has been published related to the nationwide incidence, management practices, cost and outcome over the last decade.

Methods:
The Nationwide Inpatient Sample database was used to analyze 720,297 hospital admissions for SDH across the United States from 1998-2007. The per capita increase in incidence over time, per age group and surgical group was assessed. Discharge disposition was categorized as either dead or poor (discharged to nursing home or hospice). Linear regression was used to assess national cost, number of hospital days and discharge disposition over time. Multiple logistic regression analysis was performed to adjust for other predictors of disposition including age, surgery, medical comorbidities and the severity of illness.

Results:
Hospitalizations for SDH increased from 59,373 (30 per 100,000 people) to 91,935 (40 per 100,000), constituting a 55% increase. The incidence of SDH increased with age ($P<0.001$), with the highest per capita SDH rate among those >80 years. Surgical intervention for SDH decreased from 41% to 31% ($P<0.001$). Patients who had herniated and patients who required mechanical ventilation were more likely to undergo surgery (all $P<0.001$). Coagulopathy associated SDH increased by 153% (from 6% to 9% of hospitalizations) and was a significant predictor of death or poor outcome ($P<0.001$). The number of hospital days for all SDH patients increased by 34% (from 552,903 to 738,897 days; $P=0.001$) and the national charges doubled from $2.1 to $4.5 billion ($P<0.001$). After adjusting for other predictors of outcome, mortality decreased from 15% to 12% ($P=0.001$). Conversely, poor outcome increased from 17% to 21% ($P<0.001$). Surgical intervention was protective against death, but not poor outcome.

Conclusions:
The incidence, cost and hospital stay for SDH has increased significantly in the last decade. While mortality has decreased, more patients are discharged to a nursing home or hospice.

Financial Support: None

4:00 – 4:15 p.m. Thursday, November 12, 2009

PHENYLEPHRINE INFUSION PREVENTS IMPAIRMENT OF CEREBRAL AUTOREGULATION DURING HYPOTENSION AFTER BRAIN INJURY IN FEMALE BUT AGGRAVATES IMPAIRMENT IN MALE PIGLETS THROUGH MODULATION OF ERK MAPK UPREGULATION

William Armstead[1], J. Willis Kiessling[1], W. Andrew Kofke[1], Monica Vavilala[2]
[1]University of Pennsylvania, Philadelphia,PA, United States, [2]University of Washington, Seattle, WA, United States

Introduction:
Traumatic brain injury (TBI) contributes to morbidity and mortality in children and boys are disproportionately represented. Hypotension is common and worsens outcome after TBI. ERK mitogen activated protein kinase (MAPK) is upregulated and reduces CBF after fluid percussion brain injury (FPI). We hypothesized that increased CPP via phenylephrine (Phe) sex dependently improves impairment of cerebral autoregulation during hypotension after pediatric FPI through modulation of ERK MAPK.

Methods:
CBF (microspheres), pial artery diameter, velocity (TCD), ICP and autoregulatory index were determined before and after FPI in untreated, pre- and post-injury Phe (1 μg/kg/min iv) treated male and female newborn pigs during normotension and hypotension (blood withdrawal decreased MAP by 45%). CSF ERK MAPK was determined by ELISA.

Results:
Reductions in pial artery diameter, cortical and hippocampal CBF, CPP and elevated ICP after FPI were greater in males than females, which were blunted by Phe pre- or post-FPI. During hypotension and FPI, pial artery dilation was impaired more in males than females. Phe decreased impairment of hypotensive pial artery dilation after FPI in females, but paradoxically caused vasoconstriction after FPI in males. Papaverine induced pial artery vasodilation was unchanged by FPI and Phe. Cortical and hippocampal CBF, CPP, velocity, and ARI decreased markedly during hypotension and FPI in males but less so in females. Phe prevented reductions in CBF, velocity, CPP, and ARI during hypotension in females but aggravated reductions in males. CSF ERK MAPK was increased more in males than females after FPI. Phe blunted upregulation of ERK MAPK in females, but aggravated ERK MAPK upregulation in males after FPI.

Conclusions:
These data indicate that elevation of CPP with Phe sex dependently prevents impairment of cerebral autoregulation during hypotension after FPI through modulation of ERK MAPK upregulation. These data suggest the role for sex dependent mechanisms in cerebral autoregulation after pediatric TBI.

Financial Support: None

4:15 – 4:30 p.m. Thursday, November 12, 2009

HYPERTENSIVE HYPERVOLEMIC VASODILATORY (HHVD) THERAPY WITH INTRAVENOUS NICARDIPINE FOR THE TREATMENT OF CEREBRAL VASOPSASM

Benjamin Emanuel, Monica Sapo, David McArthur, Paul Vespa
University of California, Los Angeles, Los Angeles, CA, United States

Introduction:
Cerebral vasospasm (CV) after aneurysmal subarachnoid hemorrhage (aSAH) remains a significant cause of morbidity. Intravenous nicardipine has been studied clinically as a neuroprotectant, and shown to decrease the incidence of angiographic and symptomatic vasospasm in aSAH. The present study evaluated the effectiveness of the combination of cerebral HH plus vasodilatation (VD) using continuous intravenous nicardipine, called HHVD, to treat patients that developed CV.

Methods:
This was a retrospective study of a prospective, nonrandomized crossover feasibility protocol of HHVD vs HH alone over 4 years (2005 – 2009). Patients were crossed over from HH to HHVD if CV worsened despite HH, 48 hours after starting HH. For HHVD, nicardipine was infused at 2mg/hr and hypertensive goals kept unchanged. Daily Transcranial Doppler (TCD) mean velocities (MV) of the anterior or middle cerebral arteries were completed. Data was analyzed for progressive arterial dilation between the two therapies, incidence of rescue therapy, symptomatic versus asymptomatic vasospasm, DINDS, difference in rate of norepinephrine infusion, and myocardial infarction (MI) defined as Troponin >5.0 ng/mL.

Results:
A total of 16 patients in the HHVD group and 20 patients in the HH only group were analyzed. Groups were matched for Fisher grade, GCS, age, and onset time of spasm. On average, the start of HHVD therapy showed a progressive decline in mean velocity of 4.1 days sooner then HH therapy alone (p=0.012). Fewer patients in the HHVD versus HH group needed rescue therapy during treatment, 29.3% versus 75.6% (p=0.005). There was no difference between the two groups with regard to percentage of mean velocity >180cm/sec or >200cm/sec, symptomatic or asymptomatic vasospasm, DINDS, MI, the rate of norepinephrine infusion, or hypotension.

Conclusions:
HHVD is a safe alternative to HH therapy, and may reduce the need for rescue intraaterial therapy.

Financial Support: None

4:30 – 4:45 p.m. Thursday, November 12, 2009

CLEVIDIPINE RAPIDLY AND SAFELY REDUCES BLOOD PRESSURE IN PATIENTS WITH ACUTE INTRACEREBRAL HEMORRHAGE: INTERIM RESULTS OF THE ACCELERATE TRIAL – FIRST CLINICAL EXPERIENCE IN ICH

Carmelo Graffagnino[1], Sergio Bergese[2], James Love[3], Dietmar Schneider[4], Christos Lasaridis[5], Marc Lapointe[6], Kiwon Lee[7], Gwendolyn Lynch[8]
[1]Duke University Medical Center, Durham, NC, United States, [2]Ohio State University, Columbus, OH, United States, [3]Moses H. Cone Health System, Greensboro, NC, United States, [4]University of Leipzig, Leipzig, Germany, [5]Medical University of South Carolina, Charleston, SC, United States, [6]South Carolina College of Pharmacy, Charleston, SC, United States, [7]Columbia University Medical Center, New York, NY, United States, [8]Cleveland Clinic Hospitals, Cleveland, OH, United States

Introduction:
Intracerebral hemorrhage (ICH) causes 10-15% of all first-ever strokes and has a 30-day mortality of 35-52%. Recurrent hemorrhages may cause hematoma expansion, with few mitigating therapeutic options. BP reduction may attenuate hematoma growth. Antihypertensives available in the setting of acute ICH have relatively slow onset and offset, and less-than-ideal dose-response for titration. ACCELERATE evaluated BP reduction with clevidipine (rapidly-acting, vascular-selective, L-type calcium channel blocker; lowers BP by reducing systemic vascular resistance) in patients with acute ICH.

Methods:
Patients presenting with symptoms of ICH within 6 hours and systolic BP(SBP)>160mmHg were prospectively enrolled and treated with open-label IV clevidipine, started at 2.0mg/hr and titrated every 90sec until SBP≤160mmHg was achieved, and subsequently titrated to keep SBP between 140-160mmHg.

Results:
30 patients (24 men, mean age 63.7 years) were enrolled and received clevidipine. Prior to treatment, median GCS was 14, median NIHSS 10.5, mean hematoma volume 25.1mL, and mean SBP and DBP 188mmHg and 85mmHg. The mean time-to-infusion from symptom onset was 4.7 hours.

Mean average clevidipine infusion rate (1st 30min)	8.3mg/h
Mean on-drug infusion duration	29.5 hours
Median time to target SBP (≤160mmHg to ≥140mmHg)	6.5min (95%CI 3,10)
Patients achieving SBP ≤160mmHg within 30min	100%
Patients not receiving additional or alternative IV antihypertensives during 1st 30min of clevidipine treatment	96.7%

No patients had SBP<90mmHg within 30min of clevidipine initiation. Adverse events (AEs) were consistent with previous clinical experience; the most common was pyrexia (6 patients). Three hypotension AEs were reported during treatment; BP increased promptly after clevidipine dose was reduced or the infusion stopped. One patient with normal baseline triglycerides had levels ≥300mg/dL on treatment, which resolved 6 hours post-infusion.

Conclusions:
Elevated blood pressure in patients with acute ICH was rapidly controlled using IV clevidipine, with evidence of a good safety profile consistent with previous experience in other clinical settings.

Financial Support: None

4:45 – 5:00 p.m. Thursday, November 12, 2009

PREDICTION OF ANGIOGRAPHIC VASOSPASM AFTER ANEURYSMAL SUBARACHNOID HEMORRHAGE: VALUE OF THE HIJDRA SUM SCORING SYSTEM

Stefan Dupont[1], Eelco Wijdicks[1], Edward Manno[2], Giuseppe Lanzino[1], Alejandro Rabinstein[1]
[1]Mayo Clinic, Rochester, MN, United States, [2]Cleveland Clinic, Cleveland, OH, United States

Introduction:
Vasospasm is a major complication of aneurysmal subarachnoid hemorrhage (SAH) and affects clinical outcome. The ability to predict cerebral vasospasm after SAH would allow the neuro-intensivist to institute preemptive and more aggressive therapy.

Methods:
Social, clinical, and radiological information on adult SAH patients recently admitted to our hospital were reviewed. Univariate and multivariate statistical methods were used to examine the impact of patient demographics, clinical variables, and radiologic characteristics on the development of angiographic vasospasm.

Results:
163 patients were identified (102 females, 63%). A total of 34 patients (21%) developed angiographic vasospasm. In univariate analysis, occurrence of cerebral vasospasm was associated with poor World Federation of Neurological Surgeons (WFNS 4-5, $p = 0.003$) and modified Fisher (MFS 3-4, $p = 0.02$) grades, elevated Hijdra sum score (HSS ≥ 23, $p = 0.0001$), female gender ($p = 0.04$), development of hydrocephalus ($p = 0.01$), and a history of tobacco use ($p = 0.02$). In multivariable analysis, only the HSS ≥ 23 ($p = 0.01$) and history of smoking ($p = 0.02$) predicted cerebral vasospasm. Combined history of smoking and HSS >23 had positive and negative predictive values of 37% and 88%, respectively, for prediction of cerebral vasospasm after aneurysmal hemorrhage.

Conclusions:
Hijdra sum score and a history of smoking are the strongest predictors of cerebral vasospasm on angiography. Hijdra sum score is superior to the modified Fisher scale as a radiologic grading tool to predict occurrence of angiographic vasospasm after aneurysmal subarachnoid hemorrhage.

Financial Support: None

5:00 – 5:15 p.m. Thursday, November 12, 2009

SUBARACHNOID HEMORRHAGE: WHO DIES, AND WHY? TEN YEAR EXPERIENCE OF THE COLUMBIA UNIVERSITY SAH OUTCOMES PROJECT

Stephan A. Mayer, Luis Fernandez, Andres Fernandez, Khalid Hanafy, Matthew Vibbert, Viktor Szeder, Rishi Malhotra, J. Michael Schmidt, Noeleen Ostapkovich, Kiwon Lee, Jan Claassen, Kiwon Lee, E. Sander Connolly
Columbia University, New York, NY, United States

Introduction:
Subarachnoid hemorrhage (SAH) is a devastating form of stroke. The causes and mechanisms of death after SAH in the modern era of neurocritical care remain incompletely understood.

Methods:
We studied 1015 consecutive SAH patients enrolled in the Columbia University SAH Outcomes Project between July 1996 and June 2006. The primary cause of death or neurological injury resulting in withdrawal of support was adjudicated by the study team in weekly meetings. Multivariable analysis was performed to identify predictors of in-hospital mortality.

Results:
The in-hospital mortality rate was 18% (186/1015): 3% for admission Hunt-Hess grade 1 or 2, 12% for grade 3, 24% for grade 4, and 72% for grade 5. The most common assigned causes of death were direct effect of primary hemorrhage (54%), rebleeding (15%), medical complications (15%), brain edema with ICP (5%), and infarction from vasospasm (4%). Ten of 97 patients (10%) who underwent CPR in the ICU were successfully resuscitated and survived to discharge. Of those who died 80% were DNR and 71% had life support withdrawn. Significant predictors of mortality in multivariate analysis were age, Hunt-Hess grade, aneurysm size, SAH and IVH sum scores, global cerebral edema, rebleeding, hypotension, fever >37.3 °C, anemia requiring transfusion, and non-neurogenic myocardial ischemia (all $P<0.05$). Cerebral infarction did not predict mortality.

Conclusions:
Acute resuscitation strategies directed toward minimizing the initial brain injury in poor-grade patients holds the best promise of further reducing mortality after SAH. Strategies directed at reducing early aneurysm rebleeding, initial hemorrhage burden, fever burden and anemia also deserve further study.

Financial Support: None

5:15 – 5:30 p.m. Thursday, November 12, 2009

BEHAVIORAL CHANGES OF VASOSPASM REVERSED BY NEUTROPHIL DEPLETION IN A MOUSE MODEL OF SUBARACHNOID HEMORRHAGE (SAH)

J. Javier Provencio, Shari Korday Moore, Saksith Smithason
Cleveland Clinic, Cleveland, OH, United States

Introduction:
Vasospasm after SAH often leaves long-term cognitive deficits. The mechanism of these deficits is poorly understood. Ischemic damage to cortical areas is thought to be responsible for difficulty with complex tasks reported by many patients. We investigated the mechanism of cognitive dysfunction in vasospasm using a murine model of SAH.

Methods:
C57BL/6J mice were divided into three groups: sham surgery, SAH, and mice that received neutrophil-depleting mAb RB6-8C5 one day prior to SAH. The mice underwent rotorod testing on day 1 and day 15, and Y maze testing on day 2. Over the subsequent 14 days, the mice were trained and tested on the Barnes maze. After the completion of the tests, the animals were anesthetized and perfused with formalin for immunohistochemical staining for neutrophils, microglia, and neurons.

Results:
Animals that underwent sham, SAH and neutrophil-depletion prior to SAH did similarly on rotorod testing suggesting there is no motor dysfunction associated with SAH. On Y-maze testing, SAH did worse than sham but neutrophil-depleted mice did not do better. On Barnes maze, SAH animals did worse than sham treated animals taking almost three times as long to find the goal box if they had SAH than sham (100.7 ± 17.7 sec vs. 35.6 ± 4.6, p=0.003) by day 14. In neutrophil-depleted animals with SAH, the time to find the secluded box returned to baseline (36.9± 4.6, p=0.003). There was microglial activation in the SAH group not seen in the other groups.

Conclusions:
The Y maze is a test of immediate recall memory whereas the Barnes Maze is a test of executive function. Animals with SAH had impaired immediate recall and executive function. Depletion of neutrophils rescues animals from the executive function deficits of SAH. This suggests that inflammation may play a role in the cognitive dysfunction associated with vasospasm.

Financial Support: None

Neurocritical Care Society 7th Annual Meeting

WHAT'S THE BIG IDEA? Platform Presentations
Friday, November 13, 2009
10:00 – 11:45 a.m.
Napoleon Ballroom

10:00 - 10:15 a.m. L. Keith Scott, MD, FCCM
Randomized Two-Arm Trial Comparing Differing Prophylactic Antibiotic Strategies after EDV Placement

10:15 - 10:30 a.m. Peter Le Roux, MD
The Brain Oxygen and Outcome in Severe Traumatic Brain Injury (BOOST) Study: A Multi-Center, Prospective, Phase III Randomized, Clinical Trial to Compare Brain Oxygen Based Therapy to Conventional ICP/CPP Therapy for Severe Traumatic Brain Injury

10:30 - 10:45 a.m. Neeraj Badjatia, MD, MSc
Thermoregulation after Subarachnoid Hemorrhage (THRASH)

10:45 - 11:00 a.m. Wengui Yu, MD, PhD
A Randomized Trial of Intraventricular Thrombolysis for Improved Functional Outcome in High-Grade Aneurysmal Subarachnoid Hemorrhage

11:00 - 11:15 a.m. Thorsten Steiner, MD, PhD
INR Normalization in Patients with Coumadin Related Intracranial Hemorrhages - The INCH Trial: A Randomized Controlled Trial to Compare Safety and Preliminary Efficacy of Fresh Frozen Plasma and Prothrombin Complex

11:15 - 11:30 a.m. Andrew Naidech, MD, MSPH
Improving Platelet Activity for Cerebral Hemorrhage Treatment (IMPACT)

11:30 - 11:45 a.m. Carmelo Graffagnino, MD
Optimizing Therapeutic Hypothermia after Cardiac Arrest; Evaluating Optimum Induction Methodology and Dosing of Therapeutic Hypothermia Following Cardiac Arrest

10:00 – 10:15 a.m. Friday, November 13, 2009

RANDOMIZED TWO-ARM TRIAL COMPARING DIFFERING PROPHYLACTIC ANTIBIOTIC STRATEGIES AFTER EDV PLACEMENT

L Keith Scott, Steven A Conrad, Paul McCarthy
Louisiana State University Health Sciences Center, Shreveport, LA, United States

Background:
External ventricular drains are used to divert cerebral spinal fluid out of the cranium. It has been customary to initiate antibiotics (ABX) either peri-operatively or continuously to prevent drain related ventriculitis with some advocating no ABX. Previous investigations failed to demonstrate an advantage of one over the other. Concerns about continuous use centers on the risk of developing multi-resistant CNS or other infections.

Hypothesis:
Continuous antibiotics as a strategy to prevent ventricular or menningeal infectious complications after EDV placement offer no advantage to peri-operative prophylactic antibiotics.

Study design:
Multi-center, prospective, randomized, non-blinded intervention.

Subjects:
Eligibility:
1) EDV placement for a non-infectious process.
2) No antibiotics in the past 48 hours.
3) Not immunosuppressed by underlining disease states, steroids or chemotherapy.
4) > 18 years of age

Exclusion:
1) Received ABX in the past 48 hours
2) Evidence of infection upon insertion of drain
3) HIV, on going chemotherapy, exposed to glucocorticosteroids within the past two weeks

Setting:
Neuroscience & Trauma ICU

Intervention:
Patients will be randomized to one of two groups:
1) Peri-operative antibiotics (< 24-hours)
2) Prolonged antibiotics-antibiotics administered until the drain is removed

Main outcome measure:
1) Incidence of ventriculitis among groups
2) Number of days drain in place;
3) ICU days;
4) Incidence of non-CNS infectious complications.

Secondary outcome measures:
Microbiology of cultures obtained in patients that develop ventriculitis comparing Microbial ID and resistance patterns.

Power analysis:
Infection defined as: 1) positive cultures with a supporting gram stain or, 2) pleocytosis with low glucose, high protein and strong clinical suspicion. Non-inferiority study with $p < 0.05$ with a 95% CI; β=80%, 5% increase or decrease considered inferiority/superiority. Sample size 1138.

Current status:
Planning & co-investigator recruitment.

Financial Support: None

10:15 – 10:30 a.m. Friday, November 13, 2009

THE BRAIN OXYGEN AND OUTCOME IN SEVERE TRAUMATIC BRAIN INJURY (BOOST) STUDY: A MULTI-CENTER, PROSPECTIVE, PHASE III RANDOMIZED, CLINICAL TRIAL TO COMPARE BRAIN OXYGEN BASED THERAPY TO CONVENTIONAL ICP/CPP THERAPY FOR SEVERE TRAUMATIC BRAIN INJURY

Peter Le Roux[1], Ramon Diaz - Arrastia[3], Ross Bullock[2], Sureyya Dikmen[4], Jonas Ellenberg[1], Nancy Temkin[4], Rosette Biester[1]
[1]University of Pennsylvania, Philadelphia PA, United States, [2]University of Miami, Miami, FL, United States, [3]University of Texas, Dallas TX, United States, [4]University of Washington, Seattle WA, United States

Background:
Each year about 2 million people suffer traumatic brain injury (TBI) in the US and it remains the greatest cause of death and disability among young adults. Not all neuron damage occurs at the time of injury. However, current therapies to prevent secondary injury have disappointed in the clinical environment. New therapies therefore are needed. Observational studies show that reduced brain oxygen ($PbrO_2$) is common after TBI and is an independent marker of poor outcome. In a non-randomized study $PbrO_2$ -based care was associated with better outcome after severe TBI. However, there still is clinical equipoise on its use. NINDS has acknowledged the importance of such a study by funding a Phase II trial (R01 NS 061860), designed to provide evidence of physiologic efficacy and feasibility.

Hypothesis:
$PbrO_2$ -based care was associated with better outcome after severe TBI.

Study design:
We now propose a multicenter, prospective Phase III randomized clinical trial to examine $PbrO_2$-based care.

Subjects:
Eligible severe TBI patients will be randomly assigned to one of two groups 1) ICP/CPP based care or 2) $PbrO_2$ and ICP/CPP based care. Randomization will be stratified by center and TBI severity.

Main outcome measure:
The primary objective is to determine if patients who receive $PbrO_2$ and ICP/CPP care, are more likely to have a favorable outcome at 6 months than subjects treated using ICP/CPP care alone. The trial is powered to test the hypothesis that there is an overall absolute difference of 8% in the proportion of a favorable outcome (Glasgow Outcome Score of Good or Moderate Disability) for subjects treated with $PbrO_2$ based care.

Power analysis:
The number of trial subjects is expected to be 1394.

Current status:
To date 22 research centers have agreed to participate, including the 4 Phase II centers. This group will receive administrative, statistical and data coordinating support from the University of Pennsylvania Center for Clinical Epidemiology and Biostatistics. The proposal is under review at the NIH.

Financial Support: None

10:30 – 10:45 a.m. Friday, November 13, 2009

THERMOREGULATION AFTER ANEURYSMAL SUBARACHNOID HEMORRHAGE (THRASH)

Neeraj Badjatia, Stephan Mayer
Columbia University, New York/New York, United States

Background:
Fever after aneurysmal subarachnoid hemorrhage (SAH) is common and associated with poor long-term outcome. Advanced temperature modulating devices allow for the effective elimination of fever, but carry the risks of increased metabolic stress due to shivering. Whether advanced fever control (AFC) as compared to the conventional fever control leads to improved functional recovery has not been evaluated.

Hypothesis:
Continuous AFC for the first 10 days after SAH is associated with an improved likelihood of good functional outcome at 12 months compared to conventional fever control.

Study design:
Multicenter, randomized, open-label phase II and III trials.

Subjects:
Inclusion: 1. Age 18-75 2. Poor-grade (Hunt Hess 3-5) aneurysmal SAH enrolled within 72 hours of ictus. 2. Successful aneurysm repair procedure performed. Exclusion: 1. Absent or impaired brain stem reflexes; 2. Refractory hypotension or hypoxemia, 3. Pregnancy, 4. Severe concomitant co-morbidity

Setting:
Neurological Intensive Care Unit

Intervention:
Continuous AFC with a surface or endovascular temperature modulating device (Medivance Arctic Sun or Alsius Cool Line) set to 37 °C until SAH day 10 versus to intermittent conventional water-circulating cooling blankets for acetaminophen-refractory fever.

Main outcome measure:
Phase 2: Fever burden; centrally-read blinded daily GCS scores (off sedation)
Phase 3: Modified Rankin Score at 3 and 12 months

Secondary outcome measures:
Duration of mechanical ventilation, ICU and hospital stay; VAP; CLABSI; ALI/ARDS; CRP levels; body weight; energy balance; BSAS scores; ICP/CPP

Power analysis:
Based on our pilot data, we expect AFC to lead to an absolute 10% increase in good functional recovery (defined as mRS $\leq$3) at 12 months. To achieve this goal, we will require a sample size of 266 with 90% power and a significance level of 0.05. The actual significance level achieved by this test is 0.0424 assuming that 46% of patients undergoing conventional fever therapy achieve a good functional recovery.

Current status:
This project will be submitted for independent NIH funding in January 2010.

Financial Support: Speakers Honoraria (<$10,000) for Medivance Inc.

10:45 – 11:00 a.m. Friday, November 13, 2009

A RANDOMIZED TRIAL OF INTRAVENTRICULAR THROMBOLYSIS FOR IMPROVED FUNCTIONAL OUTCOME IN HIGH-GRADE ANEURYSMAL SUBARACHNOID HEMORRHAGE

Wengui Yu
UT Southwestern Medical Center, Dallas, TX, United States

Background:
High-grade (IV-V) subarachnoid hemorrhage (SAH) portends poor functional outcome with > 60% mortality and major disability. Severe intraventricular hemorrhage (IVH) and thick SAH are the major predictors of poor outcome. Two small randomized trials of intraventricular thrombolysis in mostly grade I-III SAH patients showed inconclusive results.

Hypothesis:
Timely clearance of severe IVH and SAH with recombinant tissue plasminogen activator (t-PA) may have significantly higher outcome benefit in high-grade SAH patients.

Study design:
Multicenter, double blind, randomized, placebo-controlled trial.

Subjects:
Patient with aneurysmal SAH will be screened for enrollment based on following inclusion criteria: 1). Age >18 years old; 2). Hunt and Hess grade IV and V; 3). Initial imaging shows severe IVH and/or thick SAH; 4). Ruptured aneurysm is clipped or coiled successfully. Exclusion criteria: 1). Massive intraparenchymal hemorrhage with herniation syndrome; 2). Ruptured arteriovenous malformation; 3). History of coagulopathy or bleeding disorders; 4). Pregnancy; 5). Known allergy to t-PA.

Setting:
Neurological Intensive Care Unit.

Intervention:
Intraventricular infusion of t-PA 2 mg or placebo daily for 3 days after clipping or coiling of ruptured aneurysm.

Main outcome measure:
To determine if intraventricular administration of t-PA effectively improves functional outcome at 6 months. This will be measured using modified Rankin scale.

Secondary outcome measures:
To determine if intraventricular t-PA reduces risks of vasospasm-related infarction on MRI and shunt-dependence.

Power analysis:
Using log-rank test with a power of 90% and a hazard ratio of 0.67 (a 33% reduction in poor functional outcome), the minimal sample size will be 270.

Current status:
This project is still in the idea phase. No external funding has been applied for yet.

Financial Support: None

11:00 – 11:15 a.m. Friday, November 13, 2009

INR NORMALIZATION IN PATIENTS WITH COUMADIN RELATED INTRACRANIAL HAEMORRHAGES - THE INCH TRIAL: A RANDOMIZED CONTROLLED TRIAL TO COMPARE SAFETY AND PRELIMINARY EFFICACY OF FRESH FROZEN PLASMA AND PROTHROMBIN COMPLEX

Thorsten Steiner[1], Martin Griebe[2], Boris Ivandic[3], Rainer Kollmar[4], Thomas Pfefferkorn[5], Sven Poli[1], Katja Wartenberg[6], Christian Weimar[7], Michael Hennerici[2]
[1]Department of Neurology, University of Heidelberg, Heidelberg, Germany, [2]Department of Neurology, University of Heidelberg, Mannheim, Germany, [3]Department of Internal Medicin, University of Heidelberg, Heidelberg, Germany, [4]Department of Neurology, University of Erlangen, Erlangen, Germany, [5]Department of Neurology, University Clinic Grosshadern Munich, Munich, Germany, [6]Department of Neurology, University of Dresden, Dresden, Germany, [7]Department of Neurolopgy, University of Essen, Essen, Germany

Introduction:
Coumadin related intracranial haemorrhages (C-ICH) occur in about 10% of patients with ICH. The prognosis of C-ICH is even worse compared to spontaneous ICH (SICH). C-ICH has a mortality rate of about 50 to 60%. The higher mortality and rebleeding rate may in part be due to the higher rate of rebleeding over a longer period after symptom onset. Current recommendations for the treatment of C-ICH include fresh frozen plasma (FFP) and prothrombin complex (PCC). It is known that these drugs lower the INR, and thus it is assumed that normalization of coagulopthy may lead to haemostasis and reduction of rebleeding. However, safety and efficacy of these treatments have never been studied in a prospective trial. Our questions are: how potent are the two drugs in normalization of the INR? What is the safety profile of each of these drugs?

Methods:
We designed a prospective multicenter trial to compare safety and efficacy of FFP and PCC and C-ICH. Patients will be included if a CT scan shows intraparenchymal or subdural haematoma with and 12 hours after onset of symptoms, if the patient is on treatment with vitamin K antagonist, and the INR is above or equal to 2. Our primary endpoint is the normalization of the INR (≤ 1.2) within 3 hours after the start of infusion. Main exclusion criteria are secondary ICH, known other coagulopathies, and known acute ischemic events.

Results:
The study will start in June 2009. We will present and discuss the design of the study on the background of the current available data.

Conclusions:
Several points are of interest: this relates to the time window, the choice of the endpoint, the doses for FFP and PCC, the registration and analysis of safety issues, and rescue treatment, etc. We discuss the rational for our design on the basis of the current recommendations.

Financial Support: None

11:15 – 11:30 a.m. Friday, November 13, 2009

IMPROVING PLATELET ACTIVITY FOR CEREBRAL HEMORRHAGE TREATMENT (IMPACT)

Andrew Naidech
Northwestern University, Chicago, IL, United States

Background:
Anti-platelet medications reduce the risk ischemic stroke and myocardial infarction in primary and secondary care, but increase the risk of subsequent intracerebral hemorrhage (ICH). Platelet activity can be reliably measured in the acute setting. Reduced platelet activity on admission, but not necessarily known aspirin use, is associated with ICH volume growth, early mortality, ICH clot growth, and 3 month disability.

Hypothesis:
Platelet activity can be improved in patients with acute ICH and may reduce ICH volume growth.

Study design:
Prospective, randomized, three-armed study.

Subjects:
Acute ICH with reduced platelet activity on admission (<=550 Aspirin Reaction Units, VerifyNow-ASA, Accumetrics, CA).

Setting:
Acute care hospitals.

Intervention:
No treatment, DDAVP 0.4 mcg/kg, or platelet transfusion (one “platelet pack”); randomized treatment may be repeated once.

Main outcome measure:
Change in platelet activity after treatment

Secondary outcome measures:
Adverse effects of treatment (hypotension, respiratory distress, fever), neurologic outcomes at 3 days, 14 days or discharge, and 3 month follow-up. For patients treated within 4 hours of symptom onset, change in ICH volume by volumetric analysis.

Power analysis:
In our preliminary data on change in platelet activity with platelet transfusion, the mean change is 153 ± 74 ARU, with a correlation coefficient of 0.2. A study size of 15 patients per group total would have >95% power to detect a difference. Proof-of-concept data with DDAVP are pending.

Current status:
Planning with statistical and data management center, recruiting sites.

Financial Support: I have received past research support (July 2006 - June 2008) from NovoNordisk and the Neurocritical Care Society.

11:30 – 11:45 a.m. Friday, November 13, 2009

OPTIMIZING THERAPEUTIC HYPOTHERMIA AFTER CARDIAC ARREST; EVALUATING OPTIMUM INDUCTION METHODOLOGY AND DOSING OF THERAPEUTIC HYPOTHERMIA FOLLOWING CARDIAC ARREST

Carmelo Graffagnino
Duke University Medical Center, Durham, NC, United States

Background:
In spite of technological advances in therapeutic hypothermia, little data supports one method over another or even the optimal duration of therapy. Preclinical data supports early rapid induction and a longer duration of cooling (48 hrs) than is standard at this time.

Hypothesis:
Is there an optimal hypothermia induction method and does a longer cooling period provide additional benefit following cardiac arrest.

Study design:
Multicenter, randomized, controlled clinical trial (2x2 factorial design).

Subjects:
Witnessed, out of hospital adult cardiac arrest, comatose patients regardless of the primary rhythm. Randomization will be stratified based on initial rhythm (VT/VF vs PEA/Asystole). Exclusion criteria will be similar to the HACA study.

Setting:
Initiated in the emergency department and completed in the ICU. Only ERs and ICUs currently utilizing therapeutic hypothermia following cardiac arrest will be included.

Intervention:
Part 1: IV iced saline infusion followed by intravascular cooling (catheter) or surface cooling (blanket).
Part 2: All surviving to 18 hrs will be randomized to 24 hrs or 48 hrs of cooling followed by controlled rewarming

Main outcome measure:
Feasibility: Time to core temperature of 32^0C
Early efficacy: Survival to 18 hours
Late efficacy (6 month): Death, modified Rankin Scale

Secondary outcome measures:
Economic: acute care costs, LOS
Secondary Efficacy: Cerebral performance category (CPC) score at 6 months, Glasgow outcome score

Power analysis:
2x2 study allows us to do detect a 10% difference in outcomes between vascular and surface cooling and between 24 and 48 hours of cooling with 350 patients per arm with an 80% power at an alpha of 0.05. We are estimating a good outcome in 55% of the surface cooled patients for 24 hours.

Current status:
Planning phase; will be entering conversations with the NIH- NHLBI as well as with various device manufacturers for funding support.

Financial Support: None

Neurocritical Care Society 7th Annual Meeting

PLATFORM SESSION II
Friday, November 13, 2009
1:00 – 3:00 p.m.
Napoleon Ballroom

1:00 - 1:15 p.m. Raimund Helbok, MD
The Effect of Packed Red Blood Cell Transfusion on Cerebral Oxygenation and Metabolism after Subarachnoid Hemorrhage

1:15 - 1:30 p.m. Alan Yee, DO
Clinical Attributes Influencing Time to Death after Withdrawal of Care in Neurological ICU Patients with Possible Implications for DCD Protocols

1:30 - 1:45 p.m. Anna Finley Caulfield, MD
Comparison of Prognostic Accuracy between Neurointensivists and General Intensivists in Critically-Ill Neurological Patients

1:45 - 2:00 p.m. Salam Jarrah, DO, MPH
Use of a Commercial Surface Cooling Device for Therapeutic Hypothermia after Cardiac Arrest: Efficacy and Safety Considerations

2:00 - 2:15 p.m. Edgar Samaniego, MD, MS
Predictors of Poor Neurological Outcome in Comatose Survivors Treated with Hypothermia after Cardiopulmonary Resuscitation

2:15 - 2:30 p.m. Nitish Thakor
Neurological Recovery and Neural Spike Firing Rates with Therapeutic Hypothermia after Cardiac Arrest in Rats

2:30 - 2:45 p.m. Wendy Ziai, MD, MPH
Prediction Rules for Admission Surveillance Cultures for Methicillin-resistant Staphylococcus Aureus (MRSA) and Vancomycin-resistant Enterococci (VRE) in a Neurocritical Care Unit

2:45 - 3:00 p.m. Michael Katsnelson, MD
Are Initial Radiographic and Clinical Scales Associated with Subsequent Brain Oxygen Levels after Severe Traumatic Brain Injury?

1:00 – 1:15 p.m. Friday, November 13, 2009

THE EFFECT OF PACKED RED BLOOD CELL TRANSFUSION ON CEREBRAL OXYGENATION AND METABOLISM AFTER SUBARACHNOID HEMORRHAGE

Pedro Kurtz, Raimund Helbok, J Michael Schmidt, Jan Claassen, Luis Fernandez, R Morgan Stuart, E Sander Connoly, Kiwon Lee, Stephan Mayer, Neeraj Badjatia
Columbia University Medical Center, New York, NY, United States

Introduction:
Anemia is frequently encountered in critically ill patients and adversely affects cerebral oxygen delivery and metabolic function. However, there is limited evidence to support the use of packed red blood cell (PRBC) transfusion to optimize brain homeostasis after subarachnoid hemorrhage. The objective of this study was to investigate the effect of PRBC transfusion on cerebral oxygenation and metabolism in patients with high-grade subarachnoid hemorrhage

Methods:
Prospective observational study in a neurological intensive care unit of a university hospital. Nineteen PRBC transfusions were studied in 15 consecutive patients with subarachnoid hemorrhage that underwent multimodality monitoring (intracranial pressure, brain tissue oxygen and cerebral microdialysis). Data was collected at baseline and during 12 hours after transfusion. The relationship between Hb change and lactate/pyruvate ratio (LPR) and brain tissue oxygen (PbtO2) was tested in univariate and multivariable analyses.

Results:
PRBC transfusion was administered on median post-bleed day 8. The average Hb concentration at baseline was 8.1 g/dL and increased by 2.2 g/dL after transfusion. After transfusion initiation, PbtO2 increased between hours 2 and 4 and this rise was maintained until hour 10. LPR did not change during the 12 hours of monitoring. Multivariable analysis demonstrated that, after adjusting for peripheral oxygen saturation, cerebral perfusion pressure and LPR, change in Hb concentration was independently and positively associated with change in PbtO2 (adjusted b estimate=1.39 [95% confidence interval 0.09 – 2.69]; p=0.036). No relationship between change in LPR and change in Hb was found.

Conclusions:
Transfusion of PRBC results in PbtO2 improvement without a clear effect on cerebral metabolism after subarachnoid hemorrhage.

Financial Support: None

1:15 – 1:30 p.m. Friday, November 13, 2009

CLINICAL ATTRIBUTES INFLUENCING TIME TO DEATH AFTER WITHDRAWAL OF CARE IN NEUROLOGICAL ICU PATIENTS WITH POSSIBLE IMPLICATIONS FOR DCD PROTOCOLS

Alan Yee, Alejandro Rabinstein, Eelco Wijdicks
Mayo Clini, Rochester, MN, United States

Introduction:
Improving our ability to predict the time of death after withdrawal would have a significant impact on rates of organ donation after cardiac death (DCD) and allocation of appropriate medical resources. We sought to determine which pre-withdrawal of care clinical factors could predict earlier time to death in patients with catastrophic neurological disease.

Methods:
We retrospectively analyzed all patients who underwent care withdrawal from 2002 to 2008 in our NICU. We evaluated patients who expired within 120 minutes compared to those who died beyond this time from the point of care withdrawal. Patients declared brain dead or not intubated and cases with insufficient data were excluded. Demographic, clinical, laboratory, and radiographic data were reviewed. Statistical analysis of predictive variables was based on multivariate logistic regression analysis.

Results:
149 comatose patients satisfied our inclusion criteria. 83 patients had cardiac arrest in < 120 minutes; 58% were male and 51% were older than 66 years. Ischemic stroke (30%) and intraparenchymal hemorrhage (52%) were the most frequent diagnosis. Absent corneal response (OR= 3.52, 95% CI, 1.30 to 9.51; p=0.013), absent cough reflex (OR= 4.01, 95% CI, 1.65 to 9.74; p=0.002), extensor or absent motor response (OR= 3.78, 95% CI, 1.31 to 11.0; p=0.014), and oxygenation index greater than 4.2 (OR= 8.31, 95% CI, 2.84 to 24.4) were predictive of death within the first 120 minutes.

Conclusions:
Specific neurological signs and respiratory parameters can be predictive of earlier death after withdrawal of care in the NICU. This subset of comatose patients with irreversible neurological injury may be suitable for DCD protocols. These attributes need validation in a prospective data set.

Financial Support: None

1:30 – 1:45 p.m. Friday, November 13, 2009

COMPARISON OF PROGNOSTIC ACCURACY BETWEEN NEUROINTENSIVISTS AND GENERAL INTENSIVISTS IN CRITICALLY-ILL NEUROLOGICAL PATIENTS

Anna Finley Caulfield, Laurel Gabler, Michael Mlynash, Maarten Lansberg, Irina Eyngorn, Marion Buckwalter, Chitra Venkatasubramanian, Norman Rizk, Christine Wijman
Stanford University Medical Center, Palo Alto, CA, United States

Introduction:
At our institution intensivists with neurocritical care training (NICU) and general critical care training (ICU) are equally involved in the daily management of critically-ill neurological patients. In this prospective study we aimed to assess whether physicians' accuracy in outcome prediction differed between these two groups.

Methods:
Consecutive patients who were intubated for ≥ 72 hours with a neurological illness or complication were prospectively enrolled. NICU and ICU attending physicians were asked to predict the patient's 6-month outcome using the modified Rankin Scale (mRS). Patient outcome at 6 months was determined by a structured telephone interview. Results were dichotomized to good (mRS 0-3) and poor (mRS 4-6) outcomes.

Results:
One-hundred and forty-four patients were enrolled and 2 were lost to follow-up. The NICU had fewer years in practice than the ICU (median (IQR) 0 (0-3) vs.10 (5-24); p=0.02). Predictive accuracy of the dichotomized mRS results was similar for both groups: 80% (95% CI, 72–86%) for the NICU and 78% (95% CI, 70–85%) for the ICU. Similarly, the predictive value for good outcome (mRS 0-3) was identical between NICU and ICU (63% (95% CI, 50-74%) vs. 64% (95% CI, 49-77%). However, the NICU predictive value for poor outcome tended to be higher 94% (95% CI, 85-98%) vs. 87% (95% CI, 77-93%; p=0.16). The most notable difference between the two groups was the higher specificity in predicting poor outcome by the NICU: 89% (95% CI, 76-96%) vs. 74% (95% CI, 59-86%) (p=0.07). This difference remained exactly the same after excluding the 49 patients who were taken off life support.

Conclusions:
Neurointensivists at our institution tended to have a higher specificity in predicting poor outcomes than general intensivists, which remained after excluding patients who had care withdrawn. Further study of prognostication by neurologically and non-neurologically trained intensivists is warranted.

Financial Support: None

1:45 – 2:00 p.m. Friday, November 13, 2009

USE OF A COMMERCIAL SURFACE COOLING DEVICE FOR THERAPEUTIC HYPOTHERMIA AFTER CARDIAC ARREST: EFFICACY AND SAFETY CONSIDERATIONS

Salam Jarrah, John Dzodzio, Gilles Fraser, Christine Lord, Richard Riker, David Seder
Maine Medical Center, Portland, Maine, United States

Introduction:
We evaluated the efficacy and safety of the Arctic Sun Therapeutic Temperature Management System (AS) for routine therapeutic hypothermia in cardiac arrest survivors.

Methods:
Retrospective review of patients in a prospective therapeutic hypothermia (TH) database between January 2006 and August 2008. We collected basic demographic and clinical information, including skin and other complications, vasopressor use, left ventricular function, the rate of cooling, and neurological outcomes. Data sets were compared by standard statistical methodology.

Results:
Sixty-nine patient-events were treated with TH followed by normothermia for a total of 72h after the return of spontaneous circulation (ROSC). 83% patients achieved the goal temperature of 32-34ºC within 250 minutes; all patients achieved goal temperature within 500 minutes. Slow cooling (251-500 minutes) was associated with shorter mean downtime (14 minutes vs. 26 minutes, $p=0.04$), better admission GCS (mean 6.1 vs. 3.9, $p<0.001$), and better neurological outcome (mean discharge CPC 3.7 vs. 2.2, $p=0.014$), but not lower admission body temperature, age, or body mass index (BMI). During 1544 hours of hypothermia maintenance, patients spent 96.7% of time within the target temperature range. Skin complications occurred in 14 (20%) patients overall, of which 4 cases (2 small tears, 2 ecchymosis under cooling pads) were related to the hypothermia device. Decubitus ulcers were not associated with vasopressor use, but trended toward association with severe LV dysfunction ($p=0.051$). Other frequent complications included hypokalemia (81%), bleeding (22%), pneumonia (20%), and bacteremia (9%).

Conclusions:
The Arctic Sun device was effective and safe for induction, maintenance, and withdrawal of TH in the clinical management of cardiac arrest survivors. Slower cooling was noted in patients with shorter down-time and less severe brain injury, but was not related to BMI.

Financial Support: None

2:00 – 2:15 p.m. Friday, November 13, 2009

PREDICTORS OF POOR NEUROLOGICAL OUTCOME IN COMATOSE SURVIVORS TREATED WITH HYPOTHERMIA AFTER CARDIOPULMONARY RESUSCITATION

Edgar Samaniego, Michael Mlynash, Anna Finley-Caulfield, Christine Wijman
Stanford University, Palo Alto, CA, United States

Introduction:
It is unclear if predictors of poor outcome in comatose survivors post-cardiopulmonary resuscitation (CPR) perform accurately after treatment with hypothermia.

Methods:
Post-CPR comatose survivors were prospectively enrolled. Highly specific predictors for poor outcome were systematically recorded 72 hours after the arrest (Table). Poor outcome was defined as death or vegetative state at 3 months. Patients were considered to be sedated if they received any sedative drugs ≤ 12 hours before the 72-hour neurological assessment. False-positive rate (FPR) and 95% CIs were calculated.

Results:
Eighty-five patients were enrolled, 53 underwent therapeutic hypothermia, and 53 had a poor outcome. Baseline characteristics did not differ between groups, except for the use of sedatives: 83% of hypothermia versus 60% of normothermia patients (p=0.02). Corneal reflex, motor response and neuron specific enolase (NSE), performed sub optimally in both the hypothermia and normothermia groups. However, all predictors accurately predicted poor outcome (FPR 0%) in patients without sedation regardless of whether they received hypothermia

Table. FPR of predictors

	All* patients N=85	Hypothermia with sedation N=44	Normothermia with sedation N=18‡	All patients without sedation N=21‡
Status myoclonus	0 (0-13)	0 (0-21)	0 (0-48)	0 (0-44)
No pupillary response	0 (0-14)	0 (0-21)	0 (0-53)	0 (0-44)
No corneal reflexes	6 (1-22)	5 (0-28)	17 (0-64)	0 (0-44)
GCS motor response ≤ 2	12 (4-30)	11 (1-34)	33 (9-90)	0 (0-44)
NSE > 33 ng/ml‡	19 (7-40)	22 (7-48)	25 (1-78)	0 (0-60)
Absent SSEPs‡	0 (0-34)	0 (0-60)	0 (0-69)	0 (0-80)

**Numbers indicate percentages (95% CI).*
‡ Data not available for all patients.

Conclusions:
Sedation is a confounder in the prognostication of comatose survivors after CPR. Patients treated with hypothermia are more likely to receive sedation in proximity of their 72-hour neurological examination. Hypothermia did not affect the accuracy of predictors of poor neurological outcome in this limited data set.

Financial Support: None

2:15 – 2:30 p.m. Friday, November 13, 2009

NEUROLOGICAL RECOVERY AND NEURAL SPIKE FIRING RATES WITH THERAPEUTIC HYPOTHERMIA AFTER CARDIAC ARREST IN RATS

Xiaofeng Jia, Jai Madhok, Youngseok Choi, Romergryko Geocadin, Nitish Thakor
Johns Hopkins University School of Medicine, Baltimore, MD, United States

Introduction:
Neurological recovery in relation to therapeutic hypothermia is still not fully understood. To understand the neural response to global ischemic brain injury caused by cardiac arrest (CA) in a rodent asphyxia model of cardiac arrest, we utilize the multiple neural spike recording to investigate electrophysiologic mechanism related to arousal in the rodent cortex and ventral postero-lateral (VPL) nucleus of thalamus in relation to therapeutic hypothermia.

Methods:
Ten adult Wistar rats were evenly divided into two groups based on four-hour of immediate post-CA hypothermia (T=33^{o}C), or normothermia (T=37^{o}C). Temperature was maintained using surface cooling and warming. Multi-unit activity was recorded with two pairs of chronically implanted microelectrodes in cortex and VPL nucleus of thalamus from pre-cardiac arrest baseline and periodically during recovery after 5-minute CA. The spike firing activity was quantified by a wavelet based spike detection method.

Results:
Immediately after CA, the spike firing rate was initially reduced to silence that gradually increased over time. Comparing to normothermia group, the firing rate was significantly higher during the first 120 minutes in hypothermia group both in cortex (mean± SEM:13.8±1.4 vs. 7.7±1.1 spikes/sec, p=0.001) and in VPL thalamus (16.7± 2.0 vs. 11.4±1.4, p=0.004). Analysis at different intervals demonstrated a significant separation of spike firing rate between 2 groups at 30 minutes post-CA in VPL thalamus (p=0.029) and within the first 1-1.5 hours post-CA in cortex (p<0.01).

Conclusions:
These experiments show increased firing rate with hypothermia in the VPL thalamus and cortex. Further studies are required to establish the relationship of VPL thalamus neuron and cortical neurons spikes and how the pattern of neuronal recovery by spike activity relate to return of cortical EEG signal as well as initial arousal leading to functional outcome.

Financial Support: NIH Grant R01HL071568

2:30 – 2:45 p.m. Friday, November 13, 2009

PREDICTION RULES FOR ADMISSION SURVEILLANCE CULTURES FOR METHICILLIN-RESISTANT *STAPHYLOCOCCUS AUREUS* (MRSA) AND VANCOMYCIN-RESISTANT ENTEROCOCCI (VRE) IN A NEUROCRITICAL CARE UNIT

Paras Minhas, Alex Shangraw, Wendy Ziai
Johns Hopkins University School of Medicine, Baltimore, MD, United States

Introduction:
Hospitals are under increasing pressure to perform active surveillance cultures (ASC) for methicillin-resistant *Staphylococcus aureus* (MRSA) and vancomycin-resistant *enterococcus* (VRE). Definitive recommendations for ASC, however, cannot be made based on existing evidence. This study aimed to identify patients at low risk for positive surveillance cultures.

Methods:
A case-control study of patients admitted to a Neurocritical Care Unit (NCCU) from July 2007 – June 2008 during implementation of an admission and weekly ASC screening program for MRSA and VRE. Cases had MRSA or VRE positive surveillance cultures within 48 hours of hospital admission. We assessed the validity of readily ascertainable historical factors in defining patients at low and high risk for subsequent positive cultures with MRSA or VRE on NCCU admission.

Results:
Of 1589 patients admitted to the NCCU during the intervention period, 35 and 19 were positive for MRSA and VRE, respectively. Previous hospitalization within the past year had sensitivity 11% / 9.8%, specificity 98% / 100%, positive predictive value (PPV) 60% / 100% and negative predictive value (NPV) 79% / 77% for predicting a case of MRSA/VRE respectively. Intravenous antibiotic use within the previous year had sensitivity 24% / 23%, specificity 97% / 99%, PPV 46% / 84%, and NPV 94% / 93%. The combination of these two factors had specificity 97% / 99% and NPV 95% / 95%. No patients “admitted day of procedure” were positive for VRE.

Conclusions:
Patients with a previous hospitalization and/or intravenous antibiotic use have a high risk of positive culture for MRSA and especially VRE. In the absence of these factors routine admission surveillance cultures may not be necessary. Patients admitted from home for neurosurgical procedures may not require VRE surveillance testing.

Financial Support: None

2:45 – 3:00 p.m. Friday, November 13, 2009

ARE INITIAL RADIOGRAPHIC AND CLINICAL SCALES ASSOCIATED WITH SUBSEQUENT BRAIN OXYGEN LEVELS AFTER SEVERE TRAUMATIC BRAIN INJURY?

Michael Katsnelson[2], Larami Mackenzie[1,2], Suzanne Frangos[1], Mauro Oddo[1], Joshua M. Levine[1,2,3], Bryan Pukenas[4], Jennifer Faerber[5], Peter D. LeRoux[1]

[1]Department of Neurosurgery University of Pennsylvania Medical Center, Philadelphia, PA, United States, [2]Department of Neurology University of Pennsylvania Medical Center, Philadelphia, PA, United States, [3]Department of Anaesthesiology and Critical Care University of Pennsylvania Medical Center, Philadelphia, PA, United States, [4]Department of Radiology University of Pennsylvania Medical Center, Philadelphia, PA, United States, [5]Department of Biostatistics, Center for Clinical Epidemiology and Biostatistics University of Pennsylvania Medical Center, Philadelphia, PA, United States

Introduction:
Outcome prediction after severe traumatic brain injury (TBI) is important. In this study we examined whether clinical scales (Glasgow Coma Scale [GCS] and APACHE II) or radiographic scales based on admission CT (Marshall and Rotterdam) were associated with intracranial pressure (ICP) or brain oxygen ($PbtO_2$) during a patient's ICU course.

Methods:
In this prospective observational study, 101 patients (mean age 43.0 $\pm$ 19.3 years) with severe TBI who had ICP and $PbtO_2$ monitoring were identified. The effect of admission GCS, APACHE II, Marshall and Rotterdam scores on ICP and $PbtO_2$ and mortality was examined using mixed-effects models and logistic regression respectively.

Results:
Median (25% to 75% IQR) admission GCS and APACHE II without GCS scores were 3.0 (4.0) and 11.0 (5.0) respectively. Marshall and Rotterdam scores were 3.0 (2.0) and 4.0 (1.0) respectively. Mean ICP and mean $PbtO_2$ during the patients ICU course was 15.48 $\pm$ 10.73 mmHg and 29.93 $\pm$ 10.76 mmHg respectively. 43 (42.57%) patients had episodes of $PbtO_2$ <10mmHg; 39 (38.61%) had $PbtO_2$ between 10 - 20mmHg. Mortality at 30 days was 37.6%. Admission GCS was not associated with mortality (p=0.18). APACHE II scores (OR=1.2, 95%CI: 1.05, 1.3, p=0.003) and APACHE-non-GCS (OR=1.2, 95% CI: 1.1,1.3, p=0.006) were associated with mortality. Both Marshall (OR=2.1, 95% CI: 1.4, 3.2, p=0.0002) and Rotterdam Scores (OR=2.5, 95% CI: 1.6, 4.0, p=0.0001) were associated with mortality; the predicted odds of mortality were more than doubled for a unit increase in either score. No relationship between GCS, Marshall or Rotterdam Scores and subsequent ICP or $PbtO_2$ was observed.

Conclusions:
In patients with severe TBI admission clinical and radiographic scales predict mortality. However these scales do not appear to reliably predict ICP or $PbtO_2$. These results suggest that all patients with severe TBI should undergo intracranial monitoring.

Financial Support: None

Neurocritical Care Society 7th Annual Meeting

PLATFORM SESSION III
Saturday, November 14, 2009
7:30 – 9:30 a.m.
Napoleon Ballroom

7:30 - 7:45 a.m. **Cecil Hahn, MD, MPH**
Utility of Color Density Spectral Array vs. Amplitude-Integrated EEG for Seizure Identification in the ICU

7:45 - 8:00 a.m. **Xi Liu-DeRyke, PharmD**
Impact of Glucose Variability on In-Hospital Mortality Following Acute Spontaneous Intracerebral Hemorrhage (sICH)

8:00 - 8:15 a.m. **DaiWai Olson, PhD, RN, CCRN**
Intensive Insulin Therapy in the Neurocritical Care Setting is Associated with Poor Clinical Outcomes

8:15 - 8:30 a.m. **Lori Shutter, MD**
Prospective, Randomized, Single-Blinded Comparative Trial of IV Levetiracetam vs. Phenytoin for Seizure Prophylaxis

8:30 - 8:45 a.m. **Cigdem Akman, MD**
Seizure Detection Using Digital Trends Analysis: Rapid Interpretation in the Intensive Care Unit

8:45 - 9:00 a.m. **Deborah Green, MD**
Intensive vs. Conventional Insulin Therapy in Critically Ill Neurologic Patients

9:00 - 9:15 a.m. **Diana Greene-Chandos, MD**
The Enhancement of Organ Donor Conversion Through Diminshed Decoupling

9:15 - 9:30 a.m. **Clay Evans**
Emergent Warfarin Anticoagulation Reversal With Recombinant Factor VIIa Reduces Mortality in Acute Subdural Hematoma

7:30 – 7:45 a.m. Saturday, November 14, 2009

UTILITY OF COLOR DENSITY SPECTRAL ARRAY VS. AMPLITUDE-INTEGRATED EEG FOR SEIZURE IDENTIFICATION IN THE ICU

Cecil Hahn, Craig Stewart, Ayako Ochi, Jamie Hutchison, Hiroshi Otsubo
The Hospital for Sick Children, Toronto, Ontario, Canada

Introduction:
Continuous EEG monitoring is a valuable tool for the detection of nonconvulsive seizures in the ICU. Quantitative EEG tools may facilitate the interpretation of these prolonged EEG recordings. Our aim was to evaluate the utility of Color Density Spectral Array (CDSA) and amplitude-integrated EEG (aEEG) displays for seizure identification among critically ill children.

Methods:
27 continuous EEGs performed in a pediatric ICU were transformed into 8-channel CDSA and aEEG displays. 3 neurophysiologists and 3 EEG technologists were trained to identify seizures using CDSA and aEEG. Participants were then presented with only the CDSA or aEEG displays and asked to mark events that they suspected to be seizures. Their performance was compared to seizures previously identified using the conventional 13-21 channel EEG recording.

Results:
The 27 EEG recordings contained 553 discrete seizures over 487 hours. The sensitivity for seizure identification and false-positive rates across all recordings are shown below. Values are median (range).

CDSA		aEEG	
Sensitivity (%)	False-positive rate (# / hour)	Sensitivity (%)	False-positive rate (# / hour)
75.0 (66.7–86.7)	0.03 (0.00-0.14)	74.0 (60.3–83.9)	0.04 (0.00-0.15)

For individual recordings, however, the median sensitivity for seizure identification varied from 0% to 100%, and the median false-positive rate varied from 0/hour to 2.67/hour. Factors reducing the sensitivity included focal and low amplitude seizures. Factors increasing the false-positive rate included movement and electrode artifacts, and non-ictal EEG waveforms such as periodic epileptiform discharges and a burst-suppression pattern.

Conclusions:
CDSA and aEEG are equally sensitive and specific tools for seizure identification among critically ill children. Their performance is likely even better in a clinical context, when reviewers have access to the underlying raw EEG. These findings support the use of CDSA and aEEG as screening tools, with the caveat that low amplitude and focal seizures are liable to be missed using these techniques.

Financial Support: None

7:45 – 8:00 a.m. Saturday, November 14, 2009

IMPACT OF GLUCOSE VARIABILITY ON IN-HOSPITAL MORTALITY FOLLOWING ACUTE SPONTANEOUS INTRACEREBRAL HEMORRHAGE (SICH)

Xi Liu-DeRyke[2], Dave Collingridge[3], Jessica Crow[4], Denise Rhoney[1]
[1]Detroit Receiving Hospital, Detroit, Michigan, United States, [2]Orlando Regional Medical Center, Orlando, Florida, United States, [3]Intermountain Medical Center, Salt Lake City, Utah, United States, [4]Johns Hopkins Hospital, Baltimore, MD, United States

Introduction:
Recent studies suggest that glucose variability is an important predictor for mortality in a mixed critically ill population, however, the relationship of glucose variability with sICH remains ill defined. We sought to evaluate the relationship between glucose variability on in-hospital mortality during the acute phase of sICH.

Methods:
We performed a retrospective chart review of consecutively admitted patients with sICH with a minimum of 3 glucose readings during the first 5-ICU days. Data extracted included: patient characteristics, clinical features, glucose values/insulin use, and outcomes. Blood glucose indices assessed over the 5 days included: average glucose levels (GlucAVG), standard deviation of glucose (GlucSD), coefficient of variance (GlucCV), and peak glucose (GlucMAX). Statistical assessment of the glucose indices was assessed in relationship to in-hospital mortality.

Results:
There were a total of 2765 glucose readings in 154 patients with an overall mortality of 26%. Univariate analysis showed the only significant baseline patient characteristics were lower admission GCS and higher APACHE II in the nonsurvival group. Admission glucose (181±99 vs. 129±41; p=0.002), GlucAVG (160±74 vs. 125±24; p=0.005), GlucSD (41±22 vs. 27±16; p<0.001), GlucCV (26±11 vs. 20±9; p=0.007), and GlucMAX (244±104 vs. 179±62; p=0.001) were significantly higher in the nonsurvival compared to the survival group (mg/dL, respectively). Logistic regression analysis showed that significant predictors for in-hospital survival include admission GCS (OR 1.323; 95% CI 1.198-1.460; p<0.001), GlucSD (OR 0.962; 95% CI 0.942-0.982; p<0.001), and GlucCV (OR 0.947; 95% CI 0.913-0.983; p=0.004).

Conclusions:
Findings from this study suggest that a large variability of glucose during the acute phase of sICH is associated with higher in-hospital mortality. The variability appears to be a more important predictor for outcomes than admission glucose or average glucose levels.

Financial Support: None

8:00 – 8:15 a.m. Saturday, November 14, 2009

INTENSIVE INSULIN THERAPY IN THE NEUROCRITICAL CARE SETTING IS ASSOCIATED WITH POOR CLINICAL OUTCOMES

DaiWai Olson, Ananda Gurrram, Bradley Kolls, Carmelo Graffagnino
Duke University Medical Center, Durham, NC, United States

Introduction:
The perceived benefits of intensive insulin therapy (IIT) in critically ill patients were tempered by the results of the NICE-Sugar study which showed that morbidity and mortality was increased by such a strategy. Little data is available regarding the effects of an IIT in patients with neurological critical illness. We conducted a cohort study to evaluate the effects of SIT compared to IIT.

Methods:
We retrospectively compared the outcomes of 1,885 patients admitted to our NCCU between 2/1/2005 and 8/30/2006 during which time we used a standard insulin therapy (SIT) protocol (glucose goal <150 mg/dL) to 1871 patients admitted between 9/1/2006 and 3/30/2008 at which time we instituted an intensive insulin therapy (IIT) protocol (glucose goal 80-120 mg/dL). Groups were compared for number of hypo and hyperglycaemic episodes, length of stay and mortality.

Results:
Throughout the observational period of 4 years, the two groups remained demographically similar. The IIT group had significantly lower mean glucose levels (136.7 mg/dL, SD 47.6) than the SIT group (145.0 mg/dL, SD 46.4; p<.0001) however this group also experience more episodes of moderate (<70 mg/dL; OR = 1.8, 95%CI 1.5-2.3), severe (<40 mg/dL; OR = 2.4, 95%CI 1.5-3.8) and extreme hypoglycemia (<20 mg/dL; OR 5.36, CI 1.55-18.55). The more severe the hypoglycemia was, the higher the odds of dying (<70 mg/dL; OR 3.26, 95% CI, 2.52-4.22; <40 mg/dL; OR 3.65, 95% CI 2.21-6.02; <20 mg/dL; OR 6.25, 95%CI, 2.41-16.23). Mean length of stay in the IIT group was longer than the SIT group (9.5 vs 8.7 days; p=.046). Mortality was also higher in the IIT group (11.0% vs 7.9%, OR = 1.4, CI = 1.2-1.8, p=.001).

Conclusions:
Intensive insulin therapy may result in tighter glucose control, but the benefits do not outweigh the increased risks of hypoglycemia, mortality and length of stay.

Financial Support: None

8:15 – 8:30 a.m. Saturday, November 14, 2009

PROSPECTIVE, RANDOMIZED, SINGLE-BLINDED COMPARATIVE TRIAL OF IV LEVETIRACETAM VS. PHENYTOIN FOR SEIZURE PROPHYLAXIS

Lori Shutter, Kiranpal Sangha, Chris Lindsell, Jerzy Szaflarski
University of Cincinnati, Cincinnati, OH, United States

Introduction:
Increased awareness of seizures after neurological injury and their contribution to secondary injury has supported use of prophylactic anti-epileptic drugs (AEDs). Intravenous (IV) Levetiracetam (LEV) is a newer option, but its relative safety and efficacy in the Neuroscience ICU (NSICU) setting has not undergone a randomized study. We report results from our study comparing IV LEV with Phenytoin (PHT) for seizure prevention after neurological injury.

Methods:
This was a prospective, randomized, single-blinded comparative trial of LEV vs. PHT (2:1 ratio) for 7 days in patients with severe traumatic brain injury (sTBI) or subarachnoid hemorrhage (SAH) admitted to the NSICU of an academic urban level I trauma center. All patients underwent continuous EEG monitoring for the initial 72 hours. The study pharmacist made all medication dosage adjustments. Medication side effects and clinical outcome data were collected.

Results:
52 patients were enrolled (LEV=33; PHT=19), 73% male, and 88 % with TBI. Mean PHT level was 17.7 mcg/ml. There were no differences between groups in seizure occurrence (LEV 5/33 vs. PHT 3/19; p=1.0), mortality (LEV 14/33 vs. PHT 4/19; p=0.14), short or long term clinical outcomes. AED side effects showing no difference between groups included: occurrence of fever, increased ICP, stroke, hypotension, arrhythmia, thrombocytopenia/coagulation abnormalities, liver abnormalities, renal abnormalities, or early death (all p>0.26). LEV treated patients did show a lower frequency of worsening general neurological status (p=0.05), and gastrointestinal problems (p=0.04); there was also tendency towards lower incidence of anemia (p=0.07) compared to the PHT group.

Conclusions:
This first prospective, randomized, single-blinded study of LEV vs. PHT in the NSICU shows that the patients have similar outcomes relative to death or seizures, but LEV produces fewer undesirable side effects. This study found LEV to be a suitable alternative to PHT for seizure prevention in patients with sTBI or SAH in the NSICU setting.

Financial Support: Research was an investigator initiated study supported by UCB Pharma

8:30 - 8:45 a.m. Saturday, November 14, 2009

SEIZURE DETECTION USING DIGITAL TRENDS ANALYSIS: RAPID INTERPRETATION IN THE INTENSIVE CARE UNIT

Cigdem Akman, Vesna Micic, Anita Thompson, James Riviello
Texas Children's Hospital, Houston, United States

Introduction:
Digital trends analysis (DTA) presents EEG waveforms in graphic format that display hours of data. Several DTA software packages exist. Spectral array (SA) displays power versus frequency versus time in a three dimensional format whereas envelop trend (ET) is a time averaging technique, developed to exclude artifact. We compared these methods for seizure detection in the pediatric intensive care unit (PICU).

Methods:
Clinical and electrographic features EEG-video recordings of children in the PICU were examined. Raw EEG data was analyzed and seizures were grouped based on detection by ET and SA and were compared (t-test) based on seizure duration, EEG amplitude at baseline and during seizures.

Results:
163 seizures were reviewed in 13 patients, aged 3 days to 17 years. 131 (80.4%) were non-convulsive seizures (NCS). Seizure duration was 139.6±294 (8-1800 sec), background amplitude was 22.3±15.4 (92.2-60 uV) and maximum seizure amplitude of 113.0±47.3 (17.2-278 uV). A cyclic seizure pattern occurred in 7 children. ET identified 145 (89%) seizures and spectral trend identified 114 (69%). Both identified 100 (61%) seizures simultaneously. EEG features were similar in both groups. Background amplitude was found significant for the seizure detection by ET ($p=0.002$), whereas seizure amplitude was more significant for SA ($p=0.001$). Seizure duration was not significant for either ET or SA ($p>0.05$) and cyclic seizures were detected by both. Seizure duration was shorter in cyclic seizures (42.1±13.7 sec vs 143.3±299sec, $p<0.0001$).

Conclusion:
EEG features play a role for accurate seizure detection using DTA. Background amplitude is important for ET whereas seizure amplitude is important for spectral analysis. DTA combining ET and SA is useful and complimentary for early and accurate seizure detection, particularly for NCS, in the ICU.

Financial Support: None

8:45 – 9:00 a.m. Saturday, November 14, 2009

INTENSIVE VERSUS CONVENTIONAL INSULIN THERAPY IN CRITICALLY ILL NEUROLOGIC PATIENTS

Deborah Green[1], Kristine O'Phelan[2], Sarice Bassin[3], Cherylee Chang[4], Tracy Stern[4], Susan Asai[4]
[1]Boston University School of Medicine/Boston Medical Center, Boston, MA, United States, [2]University of Miami School of Medicine, Miami, FL, United States, [3]Northwestern University Feinberg School of Medicine, Chicago, IL, United States, [4]The Queen's Medical Center/University of Hawaii School of Medicine, Honolulu, HI, United States

Introduction:
The purpose of this prospective randomized study was to investigate whether intensive insulin therapy to keep blood glucose levels 80 to 110mg/dL or conventional treatment to keep levels less than 150mg/dL was associated with a reduction of mortality and improved neurologic outcome in critically ill neurologic patients.

Methods:
Within 24 hours of ICU admission, mechanically ventilated adult neurologic patients expected to require intensive care for ≥ three days were enrolled after written informed consent. Patients were randomized to intensive or conventional control. Primary outcome measure was death within three months. Secondary outcome measures included 90-day modified Rankin scale (mRS) score, ICU and hospital LOS, and ventilator days.

Results:
81 patients were enrolled. There were no significant baseline differences between the two groups including neurologic diagnosis, mRS, or GCS score.

The proportion of deaths was higher among patients in the intensive arm but this was not statistically significant (36% vs. 25%, p=0.34). When good versus poor outcome at 3 months was dichotomized to mRS score 0-2 versus 3-6, respectively, there was no difference in outcome between the two groups (76.2% vs. 75% had a poor three month outcome, p = 1.0). There was also no difference in ICU or hospital LOS or days on mechanical ventilation. Hypoglycemia (<60mg/dL) and severe hypoglycemia (<40mg/dL) was more common in the intensive arm (48% vs. 11%, p=0.0006) and (4% vs 0%), respectively.

Conclusions:
There was no benefit to intensive insulin therapy in this small critically ill neurologic population. Previous studies of glycemic control in non-neurologic ICU patients have shown conflicting results. This is the first glycemic control study specifically examining critically ill neurologic patients and functional outcome. Given these results and the increased resources required to implement intensive insulin therapy, it cannot be recommended over conventional control.

Financial Support: None

9:00 – 9:15 a.m. Saturday, November 14, 2009

THE ENHANCEMENT OF ORGAN DONOR CONVERSION THROUGH DIMINISHED DECOUPLING

Diana Greene-Chandos[1], Peace Anwuacha[1], Derrick Mushayamunda[2], Tacuma Johnson[2]
[1]Forsyth Medical Center, Winston-Salem, NC, United States, [2]Carolina Donor Services, Winston-Salem, NC, United States

Introduction:
To improve organ donation conversion rates, neurointensivists at a Level II trauma, community hospital and the local organ procurement organization (OPO) decided to diminish decoupling. Decoupling is when the explanation of brain death by the physician is separated from the request for organ donation by the family support coordinator (FSC). The FSC is not present for the final explanation of declaration of brain death and the physician familiar to the family, typically, is not present for the request. We were concerned that in the transition of physician to FSC, conversion was being lost.

Methods:
Data was retrospectively reviewed from 2007-2008 when decoupling was still occurring and compared to data thus far from 2009 where the physician and FSC were present together for both the final explanation of brain death (by physician) and request for organ donation (by FSC). We also compared data from two similar local hospitals that have not changed decoupling. All first person consent donors were removed from the data.

Results:
In 2007-2008, our hospital's conversion rate of eligible to actual donors was 44% and 37% respectively. In the first six months of 2009, where decoupling was diminished, the conversion rate was 84%. The conversion rates for two other local hospitals of similar potential and patient mix were 42% (2007), 59% (2008), and 43% (2009) (community based hospital); and 56% (2007), 60% (2008), and 38% (2009) (university based hospital).

Conclusions:
The presence of the physician familiar to the family during the organ donation request may enhance organ donation conversion in non-first consent potential donors. Physicians should consider working with the OPO and FSC to try this process especially if conversion rates for organ donation are low.

Financial Support: None

9:15 – 9:30 a.m. Saturday, November 14, 2009

EMERGENT WARFARIN ANTICOAGULATION REVERSAL WITH RECOMBINANT FACTOR VIIA REDUCES MORTALITY IN ACUTE SUBDURAL HEMATOMA

Clay Evans, James Meschia, Thomas Brott, William Freeman
Mayo Clinic, Jacksonville, FL, United States

Introduction:
To compare differences in clinical outcomes of patients presenting with acute subdural hematoma (SDH) during anticoagulation with warfarin compared to those without anticoagulation.

Methods:
We reviewed the records of patients presenting with acute, symptomatic subdural hematoma treated at Mayo Clinic Hospital in Jacksonville, Florida, between 2004 and 2007. SDH (SDH) was defined as patients not taking warfarin and with normal coagulation tests (i.e., international normalized ratio, INR < 1.3). Patients were classified as warfarin SDH (WSDH) if warfarin was prescribed and INR was 1.3 or greater at presentation. Outcome was assessed at death or hospital discharge by the Glasgow Outcome Scale (GOS).

Results:
17 patients had SDH (median age, 76 years; 7 women); 14 patients had WSDH (median age, 79.5 years; 6 women). All WSDH patients received rFVIIa (mean 47mcg/kg), vitamin K (median 7.5mg, 0-60) and FFP (1.5units, 0-13) and all had normalization of INR (from mean 2.3 to 1). The median presentation GCS in the WSDH group was 12 (range, 3-15) and 15 (range, 4-15) in the SDH group. 11 of 14 WSDH patients underwent surgical hematoma evacuation compared to 10 of 17 SDH patients. Glasgow Outcome Scales for the WSDH group were GOS=1, 29% (4/14); GOS=2, 0% (0/14); GOS=3, 7% (1/14); GOS=4, 21% (3/14); GOS=5, 43% (6/14)). Glasgow Outcome Scales for the SDH group were GOS=1, 23% (4/17); GOS=2, 6% (1/17); GOS=3, 0% (0/17); GOS=4, 12% (2/17); GOS=5, 59% (10/17).

Conclusions:
Compared to the historical mortality of 60% for acute warfarin subdural hematoma, we observed a 29% mortality for WSDH which was close to 23% in SDH. Emergent anticoagulation reversal may lead to earlier neurosurgical hematoma evacuation and better outcomes by reducing early hematoma growth. Larger, prospective studies are needed comparing anticoagulation reversal methods and effects on hematoma growth.

Financial Support: None

Neurocritical Care Society
7th Annual Meeting
POSTER PRESENTATIONS

Authors will be standing by their posters at the hours indicated below:
Poster Session I: Posters 1-89: Thursday, November 12, 2009 from 5:30 – 7:00 p.m.
Poster Session II: Posters 90-162: Friday, November 13, 2009 from 5:30 – 7:00 p.m.

Poster #	Title	Presenting Author
1	Autoregulation in the Posterior Circulation is Altered by the Metabolic State of the Visual Cortex	Kazuma Nakagawa, MD
2	VAP Bundle Implementation in the NeuroICU	Shahram Amini, MD
3	The Effect of Central Venous Pressure on Brain Oxygen after Aneurysmal Subarachnoid Hemorrhage	Elana Tykocinski
4	Improved Inter-Rater Reliability for SAH CT Grading Scales Through Application of a Formal Definition of Clot Thickness	William McBride, MD
5	Ventilator Associated Pneumonia in Neurosurgical Patients; Results of a 1 Year Prospective Observational Study	Julio Chalela, MD
6	Blood Pressure Decreases Due to General Anesthesia for Intra-arterial Therapy for Acute Ischemic Stroke are Associated with Decreased Functional Outcome	Elizabeth Macri, MD
7	Fever on Admission is Associated with Increased Likelihood of Developing Posttraumatic Cerebral Vasospasm	Irene M. Hutchins
8	Stunned Myocardium: Predictors, Complications and Outcome	Rishi Malhotra, MD
9	Early Non-Ischemic Oxidative Metabolic Dysfunction Leads to Brain Atrophy in Traumatic Brain Injury	Yueqiao Xu, MD
10	Association of Spreading Depolarizations with Poor Outcome after Traumatic Brain Injury: Results from a Pilot Study	Lori Shutter, MD
11	Transcranial Ultrasound Velocities in SAH Patients with ACA Infarctions	Jocelyn Cheng, MD
12	Prevalence and Etiology of Anterior Cerebral Artery Infarcts Following Spontaneous Subarachnoid Hemorrhage	Torrey Boland, MD
13	Telemedicine and Acute Neurological Care: Beyond Thrombolytic Therapy	Gustavo Ortiz, MD
14	The Effect of Episodes of Increased Intracranial Pressure on Brain Metabolism	Sharon Stoll, DO
15	Tight Glycemic Control in Traumatic Brain Injury: A Useful or Harmful Option?	Rafael Badenes, MD
16	Medical Management to Maintain Cerebral Oxygenation in Patients with Traumatic Brain Injury	Leif-Erik Bohman, MD

Poster #	Title	Presenting Author
17	Intravenous Dantrolene for the Treatment of Cerebral Vasospasm after Subarachnoid Hemorrhage – Final Results of a Prospective Phase I Study	Susanne Muehlschlegel, MD
18	Outcome of Patients with Status Epilepticus Treated with Intravenous Levetiracetam	Katja Wartenberg, MD, PhD
19	Multinuclear Array Sodium (MARS) Imaging: A Novel Multichannel Phased Array That Permits Simultaneous Hi-Speed Proton and Sodium Image Acquisition to Determine Time of Stroke Onset and Extent of Irreversible Damage	Joshua Medow, MD
20	Changes in Vasopressin Synthesis and Release in Magnocellular Neurons During Experimental and Human Septic Shock	Tarek Sharshar, MD
21	Single Brain Death Examination with Confirmatory Cerebral Blood Flow Testing as an Alternative to the Standard Dual Brain Death Examination	Chethan Venaktasubba Rao, MD
22	Diagnostic Yield of Catheter Angiography in Addition to MRI in Spontaneous Intracerebral Hemorrhage	Natasha Renda, MD
23	Risk Of Pulmonary Edema Following Intracerebral Hemorrhage	Wolfgang Leesch, MD
24	Change in the Rate of Ventilator Associated Pneumonia with Quality Improvement Interventions in a Neurointensive Care Unit	Wendy Ziai, MD, MPH
25	High Rate Of Myopathy In Aneurysmal Subarachnoid Hemorrhage Patients	Thomas Wolfe, MD
26	The Results of a Practice and Salary Survey of Physician Members of the Neurocritical Care Society	May Kim, MD
27	Serum Creatinine and Creatinine Clearance Changes Associated with Therapeutic Hypothermia	Michael Abraham, MD
28	The Relation Between the White Blood Cell Count and Hematoma Progression in Nontraumatic Supratentorial Intracerebral Hemorrhage	Catalina Ionita, MD
29	Predictors and Outcomes of Heparin-Induced Thrombocytopenia in Subarachnoid Hemorrhage Patients	Brijesh Mehta, MD
30	Thromboembolic Risk of Activated Factor VIIa Use in Acute Intracerebral Hemorrhage with Coagulopathy	Sherry Chou, MD, MMSc
31	Significance of Neurological Examination in Sedated Critically Ill Patients	Benjamin Rohaut, MD
32	Hormonal Status and ICU-Acquired Paresis in Critically Ill Patients	Tarek Sharshar, MD
33	Characteristics and Clinical Outcomes of Good Grade SAH Patients Admitted to Neurological Intermediate Care	Daniel Evans, BS
34	Evolution of Somatosensory Evoked Potentials in Post Cardiac Arrest Rats	Wei Xiong, MD

Poster #	Title	Presenting Author
35	High-dose Lipopolysaccaride (LPS) Worsens Vasospasm after Subarachnoid Hemorrhage in a Murine Model	J. Javier Provencio, MD
36	Hypoalbuminemia in Acute Ischemic and Hemorrhagic Stroke Patients Does Not Correlate with Increased Mortality	Laurie McWilliams-Dunnigan, MD
37	Brain Interstitial Fluid TNF-α after Subarachnoid Hemorrhage	Khalid Hanafy, MD, PhD
38	Relationship between Brain Interstitial Fluid Tumor Necrosis Factor-α and Cerebral Vasospasm after Aneurysmal Subarachnoid Hemorrhage	Khalid Hanafy, MD, PhD
39	Early Attained Therapeutic Hypothermia as a Predictor of Favorable Neurologic Outcome after Cardiac Arrest	Nicole Chiota, MD
40	Do Dynamic Autoregulation Tests Predict Static Autoregulation Efficiency After Traumatic Brain Injury?	Santhosh Sadasivan, MD
41	Safety of Percutaneous Gastrostomy (PEG) Tube Placement in Stroke and Critically Ill Neurological Patients on Clopidogrel Alone or in Combination with Aspirin	Benjamin Anyanwu, MD
42	Use of Levetiracetam in Status Epilepticus in Pediatric ICU	Farhan Tariq, MD
43	Clinical Evaluation of a Portable Near-Infrared Device for Detection of Traumatic Intracranial Hematomas	Claudia Robertson, MD
44	Brain Tissue Oxygen in Poor Grade SAH Patients: Clip versus Coil	Yuejie Gu, MD
45	Good Outcome Following Hunt and Hess Grade 5 Subarachnoid Hemorrhage	Matthew Vibbert, MD
46	GCS is a Powerful Predictor of 30-Day Mortality after Hypertensive Intracerebral Hemorrhage	Elias Giraldo, MD, MS
47	Oxygen Administration for the Treatment of Postcraniotomy Pneumocephalus	Panayiotis Varelas, MD, PhD
48	Does the A-a Gradient Before the Apnea Test Predict Complications?	Alan Yee, DO
49	Impaired Brain Glucose Transport and Increased Systemic Glycemic Variability Predict Cerebral Metabolic Distress and Mortality after Severe Brain Injury	Pedro Kurtz, MD, MSc
50	Cardiac Output Augmentation with Fluid Resuscitation Improves Brain Tissue Oxygenation after Severe Brain Injury	Pedro Kurtz, MD, MSc
51	Outcome and Financial Impact after Subdural Hemorrhage	Kenneth De Los Reyes, BS, MD
52	Acute Fulminant Hepatic Failure, Encephalopathy and Early CT Changes	Sathees Thayapararajah, MD
53	Pharmacoeconomic Analysis of Medications Used for Seizure Prophylaxis in Traumatic Brain Injury	Karen McAllen, PharmD

Poster #	Title	Presenting Author
54	Dissonance Between Actual DCD Eligible Patients and Required DCD Protocol "Model Elements:" Implications for Policies and Practice	Jeffrey Frank, BS, MD
55	Ventilator-Associated Pneumonia in a Neurologic Intensive Care Unit Does Not Lead to Increased Mortality	Asma Moheet, MD
56	Impact of Pattern of Admission on ICH Outcomes	Neeraj Naval, MD
57	Poor Inter-Rater Reliability for the Diagnosis of Pneumonia Limits its Utility as a Marker of Quality of Care	Isis Duran, MD
58	Financial Impact of Surgical versus Endovascular Aneurysm Repair after Subarachnoid Hemorrhage	Jennifer Frontera, MD
59	Efficacy of Repeated 14.6% Saline Boluses in Lowering Intracranial Pressure and Improving Cerebral Perfusion	Ramin Eskandari, MS, MD
60	Intraventricular Hemorrhage Secondary to Occipital Artery Pseudoaneurysm after an External Ventricular Drain Placement: A Case Report	Mona Elsayed, MD
61	Endovascular Stent-Assisted Repair of Fusiform Intracranial Aneurysm: A Single Center Experience	Veena Yashaswi
62	Hyperammonemia-Induced Brain Edema in the Setting of Otherwise Normal Liver Function Test: Report of Two Cases of Devastating but Treatable Causes of Adult-Onset Impaired Ureagenesis	Wilson Cueva, MD
63	Non-Alcoholic (NA) Wernicke's Encephalopathy (WE) as a Cause of Unexplained Coma in the Contemporary Medical Era	Wilson Cueva, MD
64	Extracorpeal Carbon Dioxide Removal to Control pH and PaCO2 in Neurotrauma	L. Keith Scott, MD, FCCM
65	Early Intracranial Pressure Changes During Continuous Renal Replacement Therapy in Patients With Acute Brain Injury	Jeff Fletcher, MD
66	Prolonged High Dose Isoflurane For Refractory Status Epilepticus: A Potential Cause Of MRI Signal Abnormalities	Jennifer Fugate, DO
67	A Bolus of Conivaptan Lowers Intracranial Pressure in a Patient with Hyponatremia after Traumatic Brain Injury	Theresa Human, PharmD, BCPS
68	Seizure-Induced Exacerbation of Delayed Cerebral Ischemia	Tomoko Sampson, MD
69	Trans-Jugular Approach for Dural Sinus Thrombectomy: A Valuable Alternative Approach	Matthew Smith, MD
70	Effect of Inhaled Nitric Oxide on Cerebral Oxygenation in Severe TBI: A Single Center Experience	Jordan Bonomo, MD
71	Cardiac Rupture After Intravenous t-PA Administration In Acute Ischemic Stroke	Amar Dhand, MD, DPhil
72	NMDA-Receptor Encephalitis: A Single Center NICU Experience	Valerie Dechant, MD

Poster #	Title	Presenting Author
73	Coma from CSF Hypovolemia: An Unrecognized Cause	Amandeep Dhillon, MBBS, MD
74	Rapid Aneurysmal Enlargement In A Patient With HIV Vasculopathy Resulting in Recurrent Subarachnoid Hemorrhage	Nancy Edwards, MD
75	New Onset Refractory Status Epilepticus (NORSE) Associated with Creutzfeld-Jakob Disease	Abhijit Lele, MD
76	Only Time Will Tell: A Cautionary Tale Of Neuron-Specific Enolase Predicting Poor Outcome after Cardiac Arrest	Anna Allred, MD
77	Cerebral Oximetry via Near Infrared Spectroscopy Mirrors Desaturations of Brain Tissue Partial Pressure of Oxygen: A Case Study	Elissa Wible, MD
78	Electroconvulsive Therapy for Refractory Status Epilepticus: A Case Series	Hooman Kamel, MD
79	Conivaptan to Induce Therapeutic Hypernatremia in the Neurocritical Care Unit	Wendy Wright, MD
80	Delayed Cerebral Ischemia In Meningitis: Cases and Review	Valerie Coon, MD
81	Opsoclonus-Myoclonus Associated with Posterior Reversible Encephalopathy Syndrome	Dolores Santamaria, MD
82	Peripartum Bilateral Simultaneous Vertebral Artery Dissections: A Description of Two Cases	Eugene Wang, MD, MS
83	Ventilatory Failure Due to Pompe's Disease: A Potentially Treatable Entity	Morgan Johnson, MD
84	The Use of Therapeutic Hypothermia for Refractory Intracranial Hypertension Following a Poor Grade Subarachnoid Hemorrhage	Patty Gessner, MSN, ACNP
85	Purtscher's Retinopathy in a Postpartum Patient with Hypertensive Encephalopathy	Dhimant Dani, MD
86	Continuous Cardiac Output Pulmonary Artery Catheters Cause Unique EEG Artifacts	William A Knight, IV, MD
87	Fulminant Guillain-Barre Syndrome Mimicking Cerebral Death: Case Reports and Literature Review	Mohammed Rehman, DO
88	Reversal of Hyperacute Hyperperfusion Syndrome Mimicking Acute Restenosis after Successful Carotid Stenting	Julius Gene Latorre, MD, MPH
89	Accurate Correlation of Brain and Core Temperatures During Intravascular Therapeutic Hypothermia	Mohammed Zaman, DO
90	Novel Intracranial Blood Flow Patterns in Patients with Implantable Long-Term Ventricular Assist Devices	Fernando Goldenberg, MD
91	Need for an Advanced Neurological Life Support Course	Gregory Kapinos, MD, MS
92	The Effect of Decompressive Hemicraniectomy on Brain Temperature after Severe Brain Injury	Kazuma Nakagawa, MD
93	Young People's Attitude Towards Stroke and Decompressive Hemicraniectomy	Kazuma Nakagawa, MD

Poster #	Title	Presenting Author
94	CSF Adrenergic Profile Among Patients with Neurogenic Cardiomyopathy	Keith Dombrowski, MD
95	Biochemical Aspirin Resistance is Associated with Recurrent Ischemic Stroke Lesions during the Acute Period	Sang-Beom Jeon, MD
96	Effect of Mannitol on Brain Metabolism and Brain Tissue Oxygenation in Patients with Severe Brain Injury and Intracranial Hypertension	Raimund Helbok, MD
97	Systemic Glucose and Brain Energy Metabolism after Subarachnoid Hemorrhage: A Microdialysis Study	Raimund Helbok, MD
98	Hyponatremia after Subarachnoid Hemorrhage: Risk Factors and Impact on Outcome	Luis Fernandez, MD
99	The Relationship between Age and Cerebral Vasospasm after Aneurysmal Subarachnoid Hemorrhage	Kiwon Lee, MD
100	Admission CT Findings Predict Risk of Posttraumatic Vasospasm	Krista Keachie, MD
101	Management of Post-Traumatic Vasospasm: Patient Selection for Intra-arterial Therapy	Krista Keachie, MD
102	Neurointensivist Performed Simultaneous Bedside Tracheostomy and Gastrostomy in Neuro-ICU Setting: Results Since Program Initiation	Pretesh Patel, MD
103	Angiopoietin I and II Levels in the CSF of Patients with and without Traumatic Brain Injury	L. Keith Scott, MD, FCCM
104	Antibiotic Use with External Ventricular Drains and Intracranial Pressure Monitoring Devices: A Survey of Current Practices	Paul McCarthy, MD
105	Coagulation Abnormalities Associated with Therapeutic Hypothermia	Muhammad Taqi, MD
106	Reversal of Transtentorial Herniation with Hypertonic Saline in Patients with Renal Failure	Karen Hirsch, MD
107	Spontaneous Hyperventilation and Vasospasm in Patients with Subarachnoid Hemorrhage: A Retrospective Cohort Study	Othman Solaiman, MD
108	Evaluation of a Checklist to Improve Content of Family Conference Discussions in the Neurocritical Care Unit	Kristi Tucker, MD
109	A Slowly Expanding Intracranial Mass Causes A Threshold Change In Somatosensory Evoked Potentials (SEPs)	H. Adrian Püttgen, MD
110	Predictors Of Increased Intracranial Pressure In Intracerebral Hemorrhage	Venkatesh Aiyagari, MBBS, DM
111	The Epidemiology and In-Hospital Outcome of Acute Kidney Injury in the Setting of Acute Stroke	Gwendolyn Lynch, MD
112	Intracortical EEG for the Detection of Vasospasm in Patients with Poor-Grade Subarachnoid Hemorrhage	Jan Claassen, MD
113	Head-of-bed Elevation In The Neurocritical Care Setting: Recognition By Health-Care Professionals	Thiago Coelho, MD

Poster #	Title	Presenting Author
114	PRES: Is It Just About The Blood Pressure?	Alejandro Rabinstein, MD
115	Monitoring Cerebral Oxygen Saturation in Acute Stroke Patients	Xiao Androulakis, MD
116	Incidence And Risk Factors For Venous Thrombo Embolism In Neuro-critical Care Patients	Abhijit Lele, MD
117	Prognostic Importance of Intraventricular Hemorrhage Volume In Patients with Ruptured Cerebral Aneurysms	Andreas Kramer, MD, MSc, FRCPC
118	Tight Glucose Control, Cerebral Infarction, and Functional Outcome after Subarachnoid Hemorrhage	Andrew Naidech, MD, MSPH
119	Anticonvulsant Use and Outcomes after Intracerebral Hemorrhage	Andrew Naidech, MD, MSPH
120	Clinical And Radiological Features of Status Epilepticus in PRES	Jennifer Fugate, DO
121	The Effects of Fluid Balance and Vasopressor Use in Patients with Severe Traumatic Brain Injury	Jeff Fletcher, MD
122	Presence and Severity of ICU-Acquired Paresis at Time of Awakening are Associated with Increased ICU and Hospital Mortality	Tarek Sharshar, MD
123	Novel Inflammatory Biomarkers in Patients with Subarachnoid Hemorrhage are Elevated in Patients without Vasospasm	Paul Nyquist, MD, MPH
124	Plasma Cortisol Levels in Guillain-Barre Syndrome	Tarek Sharshar, MD
125	Ventriculostomy and CSF Drainage In SAH	DaiWai Olson, PhD, RN, CCRN
126	Osmoregulation of Vasopressin Secretion is Altered in the Post-Acute Phase of Septic Shock	Shidasp Siami, MD
127	Non-Invasive Imaging Has Low Diagnostic Yield In Angiogram-Negative Subarachnoid Hemorrhage	Jay Joshi, MD
128	Transfer Times And Outcomes For Patients With Acute Subdural Or Extradural Haematoma	Julian Barnbrook, MB, ChB
129	Correlation Between CSF Adrenergic Levels and MCA Vasospasm on Transcranial Ultrasound	John Khoury, MD
130	Analysis of Clinical Studies Investigating Dexmedetomidine in the Neuroscience Setting 1997-2009	Marek Mirski, MD
131	Aneurysm Size is Not Related to Hemorrhage Volume Following Aneurysmal Subarachnoid Hemorrhage	Ahmed Hassan, MD
132	Intracerebral Hemorrhage Carries A Higher Likelihood Of Early Mortality Compared To Other Stroke Subtypes	Ahmed Hassan, MD
133	Endothelial Nitric Oxide Synthase Polymorphism (eNOS -786T>C) and Poor Functional Outcome after Aneurysmal Subarachnoid Hemorrhage (SAH)	Nerissa Ko, MD, MAS

Poster #	Title	Presenting Author
134	Stay Cool While You're Ahead: Prophylactic Hypothermia in Patients with Aneurysmal Subarachnoid Hemorrhage	Shirley Chen, DO
135	Neuroprotective Effect of Melatonin and Pinoline in Focal Cerebral Ischemia	Santiago Ortega Gutierrez, MD
136	Outcomes From The Use Of Pentobarbital: A Retrospective Analysis	Eric Adelman, MD
137	The Impact of Weekend Admission on In-hospital Mortality In Subarachnoid Hemorrhage	Manisha Gupte
138	ICH Score Mortality is Magnified in Warfarin Intracerebral Hemorrhage	David McCollum, MD
139	Anemia In Cerebrovascular Disease	Archana Hinduja, MD
140	Intracranial Hemorrhage Following Neuroendovascular Procedures With Abciximab is Associated with Significant Mortality	Ryan Walsh, MD
141	Initial Experience and Feasibility of a Non-invasive Brain Oxygen Monitor in Neurocritical Care	Vineeta Singh, MD
142	Hypercholesterolemia and Elevated Blood Pressure Increases the Risk of DVT in Non-ambulatory ICH Patients	Ifeanyi Iwuchukwu, MD
143	Role of Emergent Carotid Stenting in Revascularization of the Middle Cerebral Artery and Anterior Cerebral Artery in Patients with Acute Ischemic Stroke	Yahia Lodi, MD
144	Stent-Assisted Coiling of the Ruptured Intracranial Wide Neck Aneurysm	Yahia Lodi, MD
145	Effect of Mild Hypothermia on Brain Tissue Oxygenation in Patients with Severe Traumatic Brain Injury	Takashi Tokutomi, MD
146	A Strategy For Managing Venous Thrombo-embolic Disease in the Neurocritical Care Unit	Bryan Gaspard, MD
147	Heparin Induced Thrombocytopenia in Aneurysmal Subarachnoid Hemorrhage	Valerie Dechant, MD
148	Jumpstarting Mild Hypothermia in the Neuro Population: Hitting the Target of 33°C	Mary Kay Bader, RN, MSN, CCNS
149	Lung Injury, Pneumonia, and Fever Drive Duration of Mechanical Ventilation after Cerebral Hemorrhage	Storm Liebling, BA
150	Using Continuous EEG (CEEG) to Predict and Monitor Anterior Region Vasospasm in Patients with Subarachnoid Hemorrhage (SAH)	Rahul Rathakrishnan, MD, MRCP
151	Management of Subarachnoid Hemorrhage and Intracerebral Hematoma: Clipping and Clot Evacuation versus Coil Embolization Followed by Clot Evacuation	Kenneth De Los Reyes, BS, MD
152	Neuroanatomic Basis for Posturing Reflexes in Acute Head Trauma: Re-evaluating the Evidence	Hans Püttgen, MD
153	Implementation of Cerebral Microdialysis in a Community-Based Hospital Setting—A Four Year Experience	Jeff Chen, MD, PhD

Poster #	Title	Presenting Author
154	Expert vs. Non-Expert Use of Quantitative EEG Tools for Seizure Identification in the ICU	Cecil Hahn, MD, MPH
155	Thromboelastography as an Adjunct Tool to Assess Coagulation Disturbances in Isolated Traumatic Brain Injury Patients	Asaph Nini, MD, MSc
156	Differences In Resting Energy Expenditure Of Critically Ill Neurology Patients Admitted to a Neuroscience Intensive Care Unit	Christine Hartney, MS, RD, CNSC, LDN
157	Neuroscience ICU Provider Attitudes Regarding Palliative Care Consult Services	Karen Warford, RN
158	The Minimal Important Difference in Traumatic Brain Injury Research: Results of a Neurocritical Care Society Survey	Jordan Bonomo, MD
159	Quality and Sensitivity Comparison of Template and Technician Applied Electroencephalography Electrodes	Brad Kolls, MD, PhD
160	Adrenergic Markers of Clinical and Outcome Measures in Subarachnoid Hemorrhage	Scott Pello, MD
161	Quid Pro Quo: The Role of “Basic Science” and its Practitioners in Neurocritical Care Research	Gail Pyne-Geithman, CSc, DPhil
162	Effects Of Tight Glucose Control On Infection Rates Associated with External Ventricular Drains (EVD) in Neuro Intensive Care Unit	Sophie Samuel, PharmD

Poster 1

AUTOREGULATION IN THE POSTERIOR CIRCULATION IS ALTERED BY THE METABOLIC STATE OF THE VISUAL CORTEX

Kazuma Nakagawa[1], Jorge Serrador[3], Sarah LaRose[2], Fatemeh Moslehi[2], Lewis Lipsitz[3], Farzaneh Sorond[2]
[1]University of California San Francisco, San Francisco, CA, United States, [2]Brigham and Women's Hospital, Harvard Medical School, Boston, MA, United States, [3]Beth Israel Deaconess Medical Center, Harvard Medical School, Boston, MA, United States

Introduction:
Previous studies suggest that dynamic autoregulation in the posterior cerebral artery (PCA) is less efficient compared to the middle cerebral artery (MCA). We examined the role of cerebral vasodilation due to metabolic activation (i.e. visual stimulus) on autoregulatory characteristics in the two vascular territories.

Methods:
Blood flow velocity (BFV) in the PCA and MCA and mean arterial pressure (MAP) were measured continuously in 45 healthy volunteers (62±3 years) while seated with eyes open. Additional 20 subjects (60±5 years) were examined with eyes closed and open. Cerebrovascular resistance index (CVR_i) was calculated as the ratio of MAP to mean BFV in the PCA and MCA arterial territories. Autoregulation was assessed using transfer function gains in both the PCA and MCA territories in the low (0.03 – 0.07 Hz), high (0.07 – 0.15 Hz) and cardiac (~ 1 Hz) frequency ranges. The effects of vascular territory (PCA vs. MCA) or visual activation (eyes-closed vs. eyes-open) on BFV, MAP, end-tidal CO2, CVR_i, and transfer function coherence, gains, and phases were assessed by using a repeated-measures two-way ANOVA, respectively.

Results:
With eyes open, gains were significantly higher in the PCA compared to the MCA in the low (PCA: 1.41±0.09 vs. MCA: 1.18±0.07, P=0.003) and high (PCA: 2.06±0.12 vs. MCA: 1.61±0.08, P=0.0001) frequencies. Opening eyes increased BFV and reduced cerebrovascular resistance index in the PCA but not MCA. This vasodilation in the PCA was associated with increased gain in the low (autoregulatory) frequency while MCA gain did not change (PCA: 0.89±0.14 vs. 1.31±0.17, MCA: 1.24±0.16 vs. 1.16±0.11, P=0.02).

Conclusions:
Dilation of the PCA territory during visual cortex activation resulted in increased PCA transfer function gain without changing MCA gain. Thus, impaired autoregulation in the PCA reported in previous literature is likely the result of metabolic vasodilation and not an inherent difference in the autoregulatory characteristics of the posterior circulation.

Financial Support: None

Poster 2

VAP BUNDLE IMPLEMENTATION IN THE NEUROICU

Shahram Amini, J Duffy Mocco, Amin Elamin, Lennox Archibald, A. Joseph Layon
University of Florida College of Medicine, Departments of Anesthesiology, Surgery, Medicine and Neurological Surgery, Gainesville, Florida, 32610-0254, United States

Introduction:
Prevention of ventilator associated pneumonia (VAP) can decrease patient mortality, length of ICU and hospital stay, mechanical ventilation duration, and costs. We developed and evaluated a care bundle in our NeuroICU and evaluated its VAP prevention efficacy.

Methods:
A VAP bundle was implemented in our NeuroICU in July 2007. This included head of bed (HOB) elevation to 30° to 45°, daily sedation holiday, appropriate stress ulcer prophylaxis, alternating chlorhexidine and bicarbonate mouthcare, subglottic suction, periodic patient turn, DVT prophylaxis, hand hygiene, and bifurcation suction. As the bundle adherence / compliance was tracked, patients were followed for development of VAP based upon CDC criteria; the same investigator (LA) was always the diagnostic arbiter.

Results:
3,000 patients were admitted from July 2007 to June 2009. VAP rates at the beginning, after one year, and the study end were 25.3, 7.09, 3.03 per 1,000 ventilator days, respectively. Adherence to the bundle after the first and last year were, respectively, HOB elevation 93% and 99%, daily sedation holiday 83% and 73%, stress ulcer prophylaxis 100% and 99%, oral chlorhexidine mouthwash 73% and 89%, subglotic secretion suctioning 98% and 96%, periodic patient turn 94% and 96%, DVT prophylaxis 100% and 99%, and bifurcation suction 97% and 96%.

Conclusions:
As the VAP bundle implementation improved over a two year period, the VAP rates decreased dramatically. This decrement in the VAP rate appears to correlate with initiation of Chlorhexidine use. Although the bundle is important to focus attention on the issue of VAP, decreases in VAP rate may have as much to do with careful mouth care as the bundle *per se*. Over the time period studied, averted costs to our system of $ 1.8 million were attributed to VAP prevention.

Financial Support: None

Poster 3

THE EFFECT OF CENTRAL VENOUS PRESSURE ON BRAIN OXYGEN AFTER ANEURYSMAL SUBARACHNOID HEMORRHAGE

Elana Tykocinski[1], Jennifer Faerber[2], Joshua Udoetuk[1], Rohan Ramakarishna[1], Michael Stiefel[1], Gregory Heuer[1], Stephanie Bloom[1], Eileen Maloney-Wilensky[1], Peter Le Roux[1]
[1]*Department of Neurosurgery, University of Pennsylvania School of Medicine, Philadelphia, PA, United States,*
[2]*Center for Clinical Epidemiology and Biostatistics, University of Pennsylvania School of Medicine, Philadelphia, PA, United States*

Introduction:
Hypervolemia is one component of Triple-H therapy that is used to manage vasospasm after aneurysmal subarachnoid hemorrhage (SAH). This study examines the relationship between central venous pressure (CVP), as a measure of volume status, and brain tissue oxygen ($PbtO_2$), recorded during the first 48 hours of monitoring.

Methods:
Patients admitted to a Level 1 trauma center within 24 hours of SAH, and who had a Glasgow Coma scale ≤ 8, were evaluated as part of a prospective observational database. Therapy was initiated to maintain CVP > 6 cm H_2O. Brain oxygen was measured continuously in the vascular territory of interest. Patients with ICP > 20 mm Hg and/or CPP < 60 mmHg were excluded from analysis. Mixed-effects models were used to examine the longitudinal effect of CVP on physiological variables and $PbtO_2$.

Results:
Data from 38 patients (mean age of 55 ± 16 years) were examined. A total of 1,928 data points, with values for both $PbtO_2$ and CVP, were recorded during the first 48 hours post-resuscitation. Initial CVP was > 6 cmH_2O in 27 (71%) patients and during monitoring median CVP was 8 cmH_2O. Average $PbtO_2$ was 26.59 (SD=15.00) and was similar in patients with CVP ≥ 10 cmH_20) and CVP <10cm H_20 (p= 0.15). A random intercepts model showed that CVP had a small negative effect on $PbtO_2$ (estimate= -0.17, 95% CI: (-0.31, -0.037), p=0.013) and a small but significant interaction with time on $PbtO_2$ (estimate= 0.0070, 95% CI: (0.000017, 0.014), p= 0.049).

Conclusion:
The results of this study suggest that fluid therapy to increase CVP has a limited effect on $PbtO_2$ in patients after SAH.

Financial Support: None

Poster 4

IMPROVED INTER-RATER RELIABILITY FOR SAH CT GRADING SCALES THROUGH APPLICATION OF A FORMAL DEFINITION OF CLOT THICKNESS

William McBride, Valerie Dechant, Michael Moussouttas
Thomas Jefferson University Hospital, Philadelphia, PA, United States

Introduction:
Various scales have been devised for the prediction of vasospasm following aneurysmal rupture. All such tools require the measurement of SAH burden by computed tomography to predict the likelihood of symptomatic vasospasm. Especially prominent in these scales is their reliance upon a subjective assessment of clot thickness which allows for variability in grading across raters. The current study seeks to compare the inter-rater reliability of the Fisher and newer Frontera scales when a rigid definition of thick clot is used.

Methods:
50 cases of subarachnoid hemorrhage were randomly selected from our radiographic archives. Initial head CTs were independently reviewed by two raters and a score for both the Fisher and Frontera scale was assigned to each study. The following criteria were established to characterize thick clot: 1. hemorrhage in any major cistern appearing on two contiguous slices; 2. hemorrhage occupying > 50% of any major cistern on a single cut; 3. contiguous hemorrhage with a density approximating that of bone. Hemorrhage was scored as “thick” if any two of the three criteria were met. The degree of agreement in scores between raters was then assessed by way of the Spearman’s Rho and Cohen’s Kappa for inter-rater reliability.

Results:
For both the Fisher and Frontera scales, a high degree of inter-rater reliability was demonstrated with Rho values of .849 (p=.01) and .903 (p.01) respectively. When Cohen’s Kappa was employed, respective values of 0.837 and 0.723 were obtained. These kappa values, which reflect the use of a stringent definition for thick subarachnoid hemorrhage, were stronger than those previously reported by AH. Kramer et.al.

Conclusions:
With the use of a stringent definition for thick subarachnoid hemorrhage, an assessment of subarachnoid clot burden can be made that shows a high degree of reliability across observers.

Financial Support: None

Poster 5

VENTILATOR ASSOCIATED PNEUMONIA IN NEUROSURGICAL PATIENTS; RESULTS OF A 1 YEAR PROSPECTIVE OBSERVATIONAL STUDY

Julio A Chalela, Leah Ramos, Kate Anderson, Angela Hays, Christos Lazaridis, Sarah Gay, Ron Neyens
Medical University of South Carolina, Charleston, SC, United States

Introduction:
Although ventilator- associated pneumonia (VAP) carries significant mortality there is scarce data on VAP in the neurosurgical intensive care unit (NSICU). We sought to determine the clinical factors associated with VAP in the NSICU.

Methods:
We analyzed all admissions to the NSICU requiring mechanical ventilation for at least 48 hours to determine factors associated with VAP. We collected demographics, medical history, admission diagnosis, admission Glasgow and Four Coma Scale, tracheostomy need, ventilator days, length of stay, and mortality. For statistical analysis we performed Fishers exact test (categorical variables) and students t-test (continuous variables). We used the Centers for Disease Control VAP definition.

Results:
We analyzed 119 admissions to the NSICU over one year. The sample was comprised of 80 males and 49 females with a median age of 55 (20-87). The median GCS was 9 (3-15) and the median Four Scale was 10 (4-16). The median length of stay was 8 days (2-45). Diagnosis included subarachnoid hemorrhage (32%), head trauma, (17%) intracranial hemorrhage (17%), subdural hematoma (8%), spinal cord injury (10%), neoplasms (8%), and others (8%). The incidence of VAP was 22.6%. Mortality was significantly higher ($p < 0.001$) among patients with VAP (17%) than in non- VAP patients (8%). There was no difference in clinical risk factors, admission diagnosis, and need for tracheostomy. The mean age of VAP patients was 44 and that of non-VAP patients was 54. Age < 50 was associated with VAP ($p< 0.008$). The only clinical variables associated with VAP were mechanical ventilation for > 5 days and Four Score < 14 ($p = 0.03$ and 0.05 respectively).

Conclusions:
In the NSICU VAP is frequent and carries significant mortality. Duration of mechanical ventilation and Four Coma Score predict VAP.

Financial Support: None

Poster 6

BLOOD PRESSURE DECREASES DUE TO GENERAL ANESTHESIA FOR INTRA-ARTERIAL THERAPY FOR ACUTE ISCHEMIC STROKE ARE ASSOCIATED WITH DECREASED FUNCTIONAL OUTCOME

Elizabeth Macri, Jeremy Fields, Ansgar Brambrink, Stanley Barnwell, Wayne Clark, Helmi Lutsep
Oregon Health and Science University, Portland, Oregon, United States

Introduction:
Relative hypotension after large vessel stroke is associated with poor outcome. General anesthesia (GA) causes peripheral vasodilation and cardiac depression, leading to a decrease in systemic blood pressure. In initial analysis of the Merci registry—a prospective, uncontrolled cohort of patients treated with the Merci retriever—functional outcome after stroke was worse in those patients intubated for the procedure as compared with those in whom conscious sedation or deep sedation was performed. We hypothesized that the poor functional outcomes in intubated patients resulted from decreased systemic blood pressure.

Methods:
The study population consisted of all patients enrolled at our institution in the Merci registry from October 2007 to July 2009. During the study period, all IA stroke interventions were performed under GA. Data regarding demographics, stroke severity (NIHSS on presentation), recanalization (TICI grade), and functional outcome at 90 days (modified Rankin Scale; mRS) were prospectively collected. We retrospectively reviewed the blood pressures on presentation, prior to intubation and after intubation.

Results:
58 patients were identified; of these, 45 had adequate records of blood pressure before and after intubation as well as 90 day follow-up assessments. The average age was 64 and 49% were male. Compared with pre-intubation baseline, significant reductions in SBP (149.2 vs 133.3, $p<.001$) and MAP (101.2 vs 93.5, $p<.001$) were observed following intubation. Controlling for well established predictors of outcome (NIHSS, age, location of vessel occlusion, and recanalization), the first SBP and DBP recorded immediately after intubation were significantly correlated with mRS ($p=0.03$, $p=0.039$) with lower measurements associated with poor functional outcomes.

Conclusions:
Blood pressure declined significantly as a result of general anesthesia, and lower SBP and DBP following intubation were associated with worse functional outcomes. These findings suggest that blood pressure should be aggressively supported in acute stroke patients treated undergoing IA mechanical thrombectomy.

Financial Support: None

Poster 7

FEVER ON ADMISSION IS ASSOCIATED WITH INCREASED LIKELIHOOD OF DEVELOPING POSTTRAUMATIC CEREBRAL VASOSPASM

Irene M. Hutchins[1], Krista Keachie[3], Nancy Rudisill[1], Lori Madden[1], J. Paul Muizelaar[1], Kiarash Shahlaie[2]
[1]University of California, Davis, Sacramento, CA, United States, [2]University of California, San Francisco, San Francisco, CA, United States, [3]Gundersen Lutheran Hospital, La Crosse, WI, United States

Introduction:
Posttraumatic vasospasm (PTV) is an under-recognized cause of ischemic damage following traumatic brain injury (TBI), but little is known about its pathogenesis and risk factors. Although PTV significantly differs from aneurysmal vasospasm [1, 2], it shares certain characteristics [1-4] that may provide insight into its pathogenesis. In particular, the risk of aneurysmal vasospasm is increased in patients with fever [5-10] or leukocytosis [10-12], but these relationships have not been previously explored in PTV.

Methods:
A review of 394 consecutive patients with TBI yielded 46 patients with severe TBI that survived beyond 48 hours. Eight patients developed clinically significant posttraumatic vasospasm (csPTV), defined as unexplained decline in neurological function or brain tissue oxygenation with CT angiogram evidence of arterial vasospasm. Temperature and serum leukocyte counts were compared in severe TBI patients with and without PTV.

Results:
Admission temperature was significantly higher in patients that developed csPTV (37.58±0.6ºC vs. 35.67±0.3ºC, p=0.005), and fever on admission (T>38ºC) was associated with significantly increased likelihood of vasospasm (OR=22.2). csPTV did not occur in patients with hypothermia (T<36 ºC) on admission, while 75% of those with fever (T>38ºC) developed csPTV. Admission leukocyte count was significantly higher in patients that developed csPTV (20.64±2.8 K/mm^3 vs. 14.72±1.0 K/mm^3, p=0.019). 21.9% of patients with leukocytosis on admission (WBC>12K/mm^3) developed csPTV, compared to 7.14% of patients without leukocytosis.

Conclusions:
Hyperthermia on admission correlates with increased likelihood of developing clinically significant PTV. Serum leukocyte count on admission is higher in patients who subsequently develop csPTV, suggesting that activation of inflammatory pathways and/or early infection may be involved in the pathogenesis of vasospasm. The observation that csPTV did not occur in patients with admission temperatures below 36ºC suggests a possible protective role for early hypothermia.

Financial Support: None

Poster 8

STUNNED MYOCARDIUM: PREDICTORS, COMPLICATIONS AND OUTCOME

Rishi Malhotra, Raimund Helbok, Khalid Hanafy, Luis Fernandez, Jan Claassen, Kiwon Lee, Stephan Mayer, Neeraj Badjatia

Columbia University Medical Center, Division of Neurocritical Care, New York, NY, United States

Introduction:

Subarachnoid hemorrhage (SAH) frequently causes stunned myocardium (SM). The predictors of SM and its impact on clinical course and outcome are not fully defined.

Methods:

We evaluated 674 consecutive SAH patients enrolled in the SAH Outcomes Project from February 2002–June 2009. 144 patients were excluded due to history of cardiac disease, 79 were excluded due to non-aneurysmal SAH. SM was defined as wall motion abnormalities +/- elevated troponins. Demographic, clinical, and outcome data was compared between those with and without SM.

Results:

56% (n=256) of patients were Hunt & Hess (HH) grade 3-5. Modified Fisher score was >=3 in 55% (n=254). SM was diagnosed in 16% (n=71). On univariate analyses, SM was associated with loss of consciousness (LOC) at onset, HH grade, Hijdra score, posterior aneurysm location, female gender, tobacco non-use, BMI, IVH, systolic BP, heart rate (HR), glucose, and WBC count. In a logistic regression accounting for race and age, female gender ($p=0.04$), LOC at onset ($p<0.001$), posterior aneurysm location ($p=0.001$), HR ($p=0.001$), systolic BP ($p=0.002$), HH grade ($p<0.001$) & tobacco non-history ($p=0.007$) were independent predictors of SM.

SM was associated with in-hospital development of fever, hyperglycemia, pneumonia, anemia, seizures, global cerebral edema (GCE), sodium dysregulation, and arrhythmia. After accounting for age & gender, arrhythmia ($p<0.001$), fever ($p=0.002$), and GCE ($p<0.001$) were independently associated with SM.

After adjusting for gender, age and known risk factors for poor outcome, SM was an independent predictor of mRS > 3 and death at 3 months.

Conclusions:

SM in aneurysmal SAH patients may be predicted by female gender, LOC at onset, posterior aneurysm location, increased HR & systolic BP, HH grade and tobacco use. SM is associated with in-hospital development of arrhythmia, fever, and GCE. SM is an independent predictor of poor outcome or death at 3 months.

Financial Support: None

Poster 9

EARLY NON-ISCHEMIC OXIDATIVE METABOLIC DYSFUNCTION LEADS TO BRAIN ATROPHY IN TRAUMATIC BRAIN INJURY

Yueqiao Xu[3], David McArthur[1], Jeffry Alger[2], Maria Etchepare[1], David Hovda[1], Thomas Glenn[1], Paul Vespa[1]
[1]*Department of Neurosurgery, David Geffen School of Medicine at UCLA, Los Angeles, California, United States,*
[2]*Department of Neurology, David Geffen School of Medicine at UCLA, Los Angeles, California, United States,*
[3]*Department of Neurosurgery, Capital Medical University Xuan Wu Hospital, Beijing, China*

Introduction:
The occurrence of brain atrophy after traumatic brain injury (TBI) is well known, but the fundamental role of metabolism in this process has not been well studied. Our hypothesis is that early non-ischemic reduction in oxidative metabolism results in long term brain atrophy

Methods:
Patients with moderate-to-severe TBI underwent positron emission tomography within the initial week after TBI, and also underwent paired early and late (six month) volumetric magnetic resonance imaging (MRI). Regional lobar measures of brain metabolism for oxidative metabolism ($CMRO_2$, OEF, CBF) and glucose metabolism (CMRgluc) were measured for frontal, temporal, parietal and occipital lobes for each patient. Regional changes in brain volume at 6 months after TBI were compared acute MRI volumes. Regional correlations between brain metabolism and chronic atrophy were performed.

Results:
Chronic brain atrophy was greatest in the temporal lobe (10.1%), and least in the occipital lobes. Widespread reduction in $CMRO_2$, CBF, and OEF were seen overall. The extent of chronic brain atrophy correlated best with $CMRO_2$ (r= 0.35 - 0.40), CBF (r= 0.27-0.50) and in one region with CMRgluc (r=0.39) (see Table 1). OEF was not in the ischemic range in most patients, and OEF did not correlate with chronic brain atrophy.

Table 1 Correlations between metabolism and brain lobe atrophy

	Atrophy (%)	CMRO2		CBF		CMRgluc		OEF	
		r	p	r	p	r	p	r	p
Temporal lobe	10.1±7.7	0.40	0.001*	0.42	0.001*	0.08	0.541	0.03	0.805
Frontal lobe	8.4±8.6	0.46	0.031*	0.50	0.000*	0.39	0.004*	-0.13	0.335
Parietal lobe	8.1±6.6	0.35	0.006*	0.27	0.033*	0.16	0.252	0.18	0.165
Occipital lobe	4.6±4.0	0.03	0.839	0.04	0.775	0.07	0.635	-0.01	0.933

*p<0.05.

Conclusions:
Chronic brain atrophy is regionally specific and is regionally associated with reductions in oxidative brain metabolism but not ischemia. The temporal lobe exhibit the greatest extent of atrophy, which may be related to the extent of initial trauma.

Financial Support: None

Poster 10

ASSOCIATION OF SPREADING DEPOLARIZATIONS WITH POOR OUTCOME AFTER TRAUMATIC BRAIN INJURY: RESULTS FROM A PILOT STUDY

Lori Shutter[1], Anthony Strong[2], Martin Fabricius[3], Ross Bullock[4], David Okonkwo[5], Oliver Sakowitz[6], Johannes Woitzik[7], Jens Dreier[7], Jed Hartings[1]

[1]University of Cincinnati, Cincinnati, OH, United States, [2]King's College Hospital, London, United Kingdom, [3]Glostrup Hospital, Copenhagen, Denmark, [4]Medical College of Virginia, Richmond, VA, United States, [5]University of Pittsburgh, Pittsburgh, PA, United States, [6]Heidelberg University, Heidelberg, Germany, [7]Charite University Medicine, Berlin, Germany

Introduction:
Leão's spreading depression (SD) of electrocorticographic (ECoG) activity describes a propagating wave of neuronal/astroglial depolarization in cerebral grey matter. SD occurring in normally perfused cortex may be benign, but similar peri-infarct depolarizations (PID) cause ischemic lesion growth. Here we present results of a pilot study to determine the association of depolarizations with clinical outcome in traumatic brain injury (TBI).

Methods:
At five hospitals, subdural electrode strips were placed in 56 patients who required craniotomy for surgical management of TBI. SD and PID events were identified by criteria of Fabricius et al. (Brain 129:778-90, 2006) in ECoG recordings made during intensive care for a median duration of 71 hr. Six-month eGOS scores were dichotomized to good (5-8; n=26) and poor (1-4; n=30) outcomes.

Results:
In 33/56 (59%) patients, 600 depolarizations occurred. Of these, 411 were SD type, 121 were PID, and 80 were mixed. The proportion of poor outcomes was 35% (8/23) in patients with no depolarizations, compared to 57% (12/21) in patients with SD and 83% (10/12) for patients with PID. The occurrence of PID and either type of depolarization were both significantly associated with worse outcomes (Fisher exact test, p=0.02 and χ^2, p=0.02, resp.), while SD alone was not (χ^2, p=0.14). There was no association of pupil reactivity (χ^2, p=0.61), GCS motor score (χ^2, p=0.51), pre-hospital hypotension (χ^2, p=0.79), or subarachnoid hemorrhage (χ^2, p=0.56) with outcome. Ages of patients with good (40±19 S.D.) vs. poor (48±16) outcome did not significantly differ (p=0.09).

Conclusions:
These data suggest that depolarization activity is significantly associated with poor outcome, with predictive power at least as great as established outcome predictors. Prevention of depolarizations by pharmacologic or physiologic therapy may represent a novel strategy to improve TBI outcomes. An increased sample size is required for improved statistical power and to determine the independence of depolarizations from co-variates.

Financial Support: None

Poster 11

TRANSCRANIAL ULTRASOUND VELOCITIES IN SAH PATIENTS WITH ACA INFARCTIONS

Jocelyn Cheng[1], L Chang[1], M Maltefort[1], J McCourt[1], B Alberto[1], B Sergott[2], M Moussouttas[1]
[1]Thomas Jefferson University Hospital, Philadelphia, PA, United States, [2]Wills Eye, Philadelphia, PA, United States

Introduction:
Vasospasm (VS) represents a substantial source of morbidity and mortality in patients with subarachnoid hemorrhage (SAH). Transcranial ultrasound (TCUS) velocities indicating VS in the anterior cerebral artery (ACA) are not well established. The purpose of this study is to identify ACA velocities that correlate to ipsilateral ACA infarction.

Methods:
The ACA mean velocities of 250 consecutive SAH patients undergoing routine twice daily TCUS were prospectively collected. The maximum (Max), minimum (Min), and first (Fir) mean velocity value for each vessel was determined, as were the ratios for Max/Min and Max/Fir. This process was performed for the entire group, and then for only patients having at least 7 days of readings.

Determination of ACA territory infarction was made by evaluation of serial head CT scans performed up to day 21 following the ictus. Velocity comparisons were made between patients having and those not having ACA territory infarctions on CT.

Results:
For the entire group, data was available for 443 vessels, 21 of which had associated infarction. Max velocity was somewhat greater in patients with ACA infarctions (118cms/s vs 101cms/s, p=.058), and Min velocities were substantially greater (46cms/s vs 35cms/s, p=.001).

The group having at least 7 days of TCUS constituted 357 vessels, 15 of which had associated infarction. Max velocity was again somewhat greater (127cms/s vs 109cms/s, p=.069) and Min velocity was again significantly greater (45cms/s vs 35cms/s, p=.002).

No correlation was observed for Fir, Max/Min, or Max/Fir.

Conclusions:
Patients ultimately developing ACA infarctions have greater Min velocities and tend to have greater Max velocities. Since only the use of Max velocities is practical, our findings suggest that velocities between 110-120cms/s may identify those vessels at risk for infarction, which is consistent with the available literature.

Financial Support: None

Poster 12

PREVALENCE AND ETIOLOGY OF ANTERIOR CEREBRAL ARTERY INFARCTS FOLLOWING SPONTANEOUS SUBARACHNOID HEMORRHAGE

Torrey Boland, Jocelyn Cheng, Carissa Pineda, David Brock, Robert Rosenwasser, Pascal Jabbour, Michael Moussouttas

Thomas Jefferson University, Philadelphia, PA, United States

Introduction:

Cerebral infarction following subarachnoid hemorrhage (SAH) contributes to morbidity and mortality. Vasospasm (VS) has traditionally been considered the main cause, yet recent literature suggests other potential etiologies. Anterior cerebral artery (ACA) infarctions may result in permanent deficits of intellect and behavior. The purpose of this study is to document the prevalence of ACA infarctions and to characterize the etiology of these infarctions in patients with aneurysmal SAH.

Methods:

250 consecutive SAH patients underwent review of cerebral CT scans as close to 3 weeks after the ictus as possible so as to identify SAH related ACA infarctions. Earlier scans were reviewed in patients found to have ACA infarctions to determine the timing of the infarction. VS related infarctions were defined as those beginning at least 4 days after the ictus. Infarcts occurring less than 4 days after the ictus were considered to be non-VS related.

Results:

Imaging was available for 237 patients (474 ACA territories). Overall, 6.8% of patients developed ACA infarctions in 4.6% of ACA territories. Of these, only 1.6% of patients and 1.1% of territorial infarcts were deemed likely due to vasospasm. Most ACA infarct patients (56%) had ACA/AComm aneurysm ruptures. Of patients with ACA infarction and ACA/AComm aneurysms, 89% had infarcts within the first 4 days (p=.095). All bilateral ACA infarctions with ACA/AComm aneurysms had infarctions within 4 days of the ictus.

Conclusions:

ACA infarctions are not rare in patients with SAH. Patients with ACA/AComm aneurysms were more likely to have ACA infarcts in the acute phase, prior to the usual onset of VS. The etiology of these infarctions remains to be determined, but may be related to vessel thrombosis at the time of hemorrhage, procedural/operative complications, or early VS.

Financial Support: None

Poster 13

TELEMEDICINE AND ACUTE NEUROLOGICAL CARE: BEYOND THROMBOLYTIC THERAPY

Gustavo Ortiz, Colin McDonald, Leonard DaSilva
University of Miami, Miami, Florida, United States

Introduction:
Telemedicine holds promise as a technology-intensive method of providing rapid acute neurology expertise to local hospitals with available CT scanning, and has been proposed as a way to increase access to limited specialty expertise in a cost-effective manner.

We here report the experience of a multi-state telemedicine company, working in joint effort with academic hospitals, providing acute neurological consultations to community-based hospitals.

Methods:
Specialists On Call (SOC) is a California-based telemedicine company providing 24/7 specialist physicians consultations to urban, suburban and critical access hospitals via videoconferencing technology. Neurological consultations are conducted by Board-certified neurologists. Consults requests are responded within 15 minutes. Initially, the specialists discusses the case by telephone and in a second step, the video-conference is started. Tele-neurological exam is conducted following established and validated guidelines, especially for NIHSS. Recommendations and further steps are discussed with patients, family members and consulting physician.

Results:
Between January 2008 and May 2009, a total of 5825 teleneurology consults were performed, among 56 community hospitals in 6 States. Only 2 hospitals had over 300 beds (391 and 511), the rest ranged between 22 and 254 beds. Stroke was the diagnosis in 3675 cases (63.1%), of which 3491 (95%) were acute ischemic events (AIE) (Stroke or TIA) and 184 (5%), intracranial hemorrhages. 346 (9.9% of AIE) received thrombolytic therapy with intravenous tPA. Seizure was the diagnosis in 213 patients (3.65% of the total) and other diagnosis (including headache, dizziness, vertigo and chronic pain) in 1937 (33.25%) patients.

Conclusions:
Telestroke consultation can be useful in increasing the use of intravenous tPA at community hospitals without access to adequate on-site stroke expertise. Besides thrombolytic decisions, teleconsultation can improve the care of other neurocritical conditions, including seizures, or intracerebral hemorrhage and triage to centers with neurocritical-care capability.

Financial Support: Dr. Colin McDonald and Dr. Leonard DaSilva are Chief Medical Officer and Vice-Chief Medical Officer of Specialists On Call. University of Miami Neurology Department will provide neurological consultations for Specialists On Call.

Poster 14

THE EFFECT OF EPISODES OF INCREASED INTRACRANIAL PRESSURE ON BRAIN METABOLISM

Sharon S. Stoll, Daniel O'Brien, H. Isaac Chen, Mauro Oddo, Eileen Maloney-Wilenksy, Suzanne Frangos, Joshua Levine, W. Andrew Kofke, Jennifer Faerber, Peter D. LeRoux
Hospital of the University of Pennsylvania, Philadelphia, PA, United States

Introduction:
Increased intracranial pressure (ICP) is associated with poor outcome in acute brain injury. In this study we examined how episodes of increased ICP (>20mmHg; >5minutes) affected brain metabolism.

Methods:
Twenty-one patients (mean age 50.67 ± 11.41 years) with severe brain injury (GCS<8) were studied prospectively. Lactate, pyruvate, and glucose were measured each hour using cerebral microdialysis (CMA). Brain oxygen ($PbtO_2$), mean arterial pressure (MAP), ICP and cerebral perfusion pressure (CPP) were recorded continuously. Linear mixed effects models were used to examine the relationship between episodes of increased ICP and the lactate:pyruvate ratio (LPR).

Results:
There were 467 episodes of increased ICP, 298 episodes of compromised $PbtO_2$ (<20mmHg) and 137 episodes of brain hypoxia ($PbtO_2$ <10mmHg). Median ICP (25% - 75% IQR) was greater during brain hypoxia (19.5 [37.0] vs. 16.0 [12.0]; p <0.001). GEE models indicated that ICP >30mmHg was associated with more than double the odds of brain hypoxia (OR= 2.5; 95% CI: 1.1, 5.8, p=0.034) or compromised $PbtO_2$ (OR= 2.1; 95% CI: 1.2, 3.7, p=0.007). However the frequency of increased ICP (>20mmHg) was similar among patients with compromised $PbtO_2$ (p= 0.070) or brain hypoxia (p=0.73) compared to normal $PbtO_2$. Only half the patients with brain hypoxia had increased ICP. Elevated LPR (>20) was rare (n=36 [4.6%] of ICP episodes). Median LPR (25% - 75% IQR) was greater during episodes of brain hypoxia than normal $PbtO_2$ (7.7 (7.7) vs. 2.8 (0.8), p<0.0001) and only slightly greater in episodes with compromised $PbtO_2$ compared to corresponding episodes with normal $PbtO_2$ (3.3 (2.2) vs. 2.8 (0.8), p=0.016). LPR did not increase when ICP was >20mmHg.

Conclusions:
Evidence for brain energy dysfunction is very rare when ICP is >20mmHg and any ICP effect on LPR may be indirect and depend on $PbtO_2$.

Financial Support: None

Poster 15

TIGHT GLYCEMIC CONTROL IN TRAUMATIC BRAIN INJURY: A USEFUL OR HARMFUL OPTION?

Rafael Badenes, Pablo Gonzalez, Laura Alcover, Armando Maruenda, Javier Belda
Hospital Clinico Universitario, Valencia, Spain

Introduction:
This was a pilot study to compare the cerebral neurochemical changes in patients with traumatic brain injury (TBI) who underwent conventional blood glucose level (BGL) control and intensive BGL control with continuous titrated insulin.

Methods:
This prospective, randomized study was conducted in 30 traumatic brain injury patients in a surgical and trauma intensive care unit. Patients admitted over an 18-month period with TBI were prospectively divided into two groups according to the method used for BGL control: the 'intensive' group consisted of patients who underwent continuous titrated insulin infusion to maintain a lower normoglycemic level of 4-8 mmol/L, and the 'conventional' group consisted of patients whose BGL was maintained at between 8.1 and 10.0 mmol/L using conventional 'sliding scale' bolus subcutaneous insulin administration. Data on cerebral haemodynamics, interstitial brain oxygenation (PtiO(2)) and neurochemical monitoring were collected via microcatheters inserted in the penumbral region.

Results:
We analyzed 2844 cerebral microdialysis samples. In 15 patients treated with intensive insulin therapy, there was a reduction in microdialysis glucose by 77% of baseline concentration compared with a 8% reduction in 15 patients treated with a conventional blood glucose level control. Intensive insulin therapy was associated with increased incidence of microdialysis markers of cellular distress, elevated glutamate (44+/-34% vs. 8+/-14%, $p<.01$), elevated lactate/pyruvate ratio (40+/-37% vs. 14+/-22%, $p<.03$) and low glucose (26+/-17% vs. 11+/-15%, $p<.05$), and increased global oxygen extraction fraction. Cerebral microdialysis glucose was lower in nonsurvivors than in survivors (0.46 +/- 0.23 vs. 1.04 +/- 0.56 mmol/L, $p < 0.05$).

Conclusions:
Intensive glycaemic control using insulin induced a decrease of cerebral glucose and an increase in microdialysis markers of cellular distress. In patients with severe brain injury, tight systemic glucose control is associated with increased mortality.

Financial Support: None

Poster 16

MEDICAL MANAGEMENT TO MAINTAIN CEREBRAL OXYGENATION IN PATIENTS WITH TRAUMATIC BRAIN INJURY

Leif-Erik Bohman, Gregory Heuer, Eileen Maloney-Wilensky, Suzanne Frangos, Peter LeRoux, Joshua Levine, Michael Stiefel
University of Pennsylvania, Philadelphia, PA, United States

Introduction:
Brain tissue oxygen (BtO2) monitoring is used in severe traumatic brain injury (TBI) patients. How cerebral hypoxia should be treated and its response to treatment is not clearly defined. We examined which medical therapies restore normal BtO2 in TBI patients.

Methods:
Severe TBI (GCS less than 8) patients were enrolled in a prospective observational cohort study. Intracranial pressure (ICP), cerebral perfusion pressure (CPP) and BtO2 were monitored. Episodes of cerebral hypoxia (BtO2 less than 25 mmHg) and medical interventions and therapies that improved BtO2 were identified.

Results:
Three hundred seventy nine episodes of cerebral hypoxia were recorded and treated in forty nine patients (mean age 40 +/- 19 years). Medical management successfully reversed 72% of the cerebral hypoxia episodes. Ventilator manipulation, CPP augmentation, and sedation were the most frequent interventions. Increasing FiO2 restored BtO2 80% of the time. CPP augmentation and sedation were effective in 73% and 66% cerebral hypoxia episodes, respectively. ICP reduction using mannitol was effective in 73% of treated episodes. Phenylephrine was the most frequent vasopressor administered and improved BtO2 74% of the time. Other interventions including head repositioning, airway suctioning, and blood transfusions, were effective in 66%, 88%, and 50% treated episodes, respectively. Successful medical treatment of cerebral hypoxia was associated with improved outcome. Survivors had a 71% rate of response to treatment (n=38) and nonsurvivors had a 44% rate of response (n=11; p=0.01).

Conclusions:
Cerebral hypoxia occurs in TBI patients despite traditional practices to maintain CPP. Medical interventions other than those to treat ICP and CPP can improve BtO2, increasing the number of therapies for severe TBI in the ICU.

Financial Support: None

Poster 17

INTRAVENOUS DANTROLENE FOR THE TREATMENT OF CEREBRAL VASOSPASM AFTER SUBARACHNOID HEMORRHAGE – FINAL RESULTS OF A PROSPECTIVE PHASE I STUDY

Susanne Muehlschlegel[1], Guy Rordorf[2], John Sims[2]
[1]University of Massachusetts Medical School, Worcester, MA, United States, [2]Massachusetts General Hospital, Harvard Medical School, Boston, MA, United States

Introduction:
Cerebral vasospasm (cVSP) after subarachnoid hemorrhage (SAH) is the major cause of disability and death. Treatment options are limited. Dantrolene blocks ryanodine receptor-mediated intracellular calcium release from the sarco-endoplasmic reticulum. It attenuates cerebral vasoconstriction, potentates the action of nimodipine on cerebral vessels and is neuroprotective in animal models. We performed a prospective Phase I study examining the safety and effects of a single-dose of dantrolene on cVSP after SAH.

Methods:
In an IRB approved, prospective, open-label single-blinded Phase I study, SAH patients with elevated transcranial Doppler (TCD) velocities and Lindegaard Indices suggesting cVSP were enrolled. After baseline TCDs by a single, trained operator, patients 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). Infusions, ventilator and ventriculostomy settings were kept unaltered, so that physiological data could be followed. HR, BP, ICP, CPP, CVP and body temperature were recorded at infusion start ($time_0$), every 10min during the infusion and with every TCD thereafter. Serum ABG, Chem7 and osmolarity were measured at $time_0$ and 135min, and LFTs at $time_0$ and 24hrs. TCDs were repeated at 45, 60 and 135min after $time_0$. Statistical analysis was performed with repeated measures ANOVA for the physiological values and change (Δ) in systolic, mean and diastolic TCD in the vessel in cVSP from $time_0$, followed by post-test Bonferroni's Multiple Comparison Test with Bonferroni p-value adjustment for significant findings. Laboratory values were analyzed by Wilcoxon matched pairs test.

Results:
Ten patients (n=5 each group with 1.25mg/kg and 2.5mg/kg dantrolene) were enrolled. Over the entire study period, HR, MAP, DBP, ICP, CPP and body temperature remained stable, except for SBP which decreased (p= 0.015). Post-test Bonferroni's Multiple Comparison Test with p-value adjustment (p=0.001) showed a trend towards a difference between time points 40 and 90min (mean Δ-10.3mmHg), and 50 and 90min (mean Δ-10.3mmHg), although this was not significant.

Significantly different laboratory changes were Na (mean Δ-2.2 mEq/L, p= 0.0052), Cl (mean Δ -2.7 mEq/L, p= 0.0056) and Alkaline Phosphatase (mean Δ -11.4 mg/dl, p=0.039); the degree of change, however, was considered clinically insignificant. None of the other laboratory values changed. Systolic and mean TCD velocities decreased significantly over time compared to $time_0$ (systolic p= 0.0051; diastolic p= 0.0601; mean p= 0.0245). Post-ANOVA linear trend testing indicates the magnitude of change: systolic (slope -4.1, p= 0.0005); diastolic (slope -1.7, p= 0.0102) and mean TCD (slope -2.4, p= 0.0058).

Conclusions:
A one-time infusion of dantrolene appears safe, although the mild changes in Na, Cl and Alkaline Phosphatase warrant monitoring. Most importantly, transaminases did not change. Dantrolene decreases TCD velocities over time, presumably due to inhibition of cerebral vasoconstriction. We have insufficient data to comment on the duration of this effect. Our results warrant further study with repeated or continuous dantrolene dosing for treatment or prevention of cVSP after SAH or other vasoconstriction syndromes.

Financial Support: None

Poster 18

OUTCOME OF PATIENTS WITH STATUS EPILEPTICUS TREATED WITH INTRAVENOUS LEVETIRACETAM

Katja Elfriede Wartenberg[1], Christian Blume[2], Volker Puetz[2], Imanuel Dzialowski[2], Ulf Becker[2], Hjoerdis Hentschel[2]
[1]Martin-Luther-University Halle-Wittenberg, Halle, Germany, [2]University of Dresden, Dresden, Germany

Introduction:
Status epilepticus carries a mortality rate up to 77%. Newer, intravenously (IV) applicable antiepileptic agents might be powerful adjunctive therapies. We report our experiences with IV levetiracetam in a prospective patient cohort with status epilepticus.

Methods:
We treated 27 patients with convulsive status epilepticus with an institutional protocol consisting of IV lorazepam followed by IV phenytoin, IV levetiracetam as third line agent or second line agent if there were contraindications for IV phenytoin, and IV propofol and/or midazolam as fourth line agents. Primary outcome was treatment success of IV levetiracetam. Secondary outcome measures were time to treatment success, modified Rankin Scale (mRS) at 6 and 12 months, and complications.

Results:
Of the 27 patients 22 had cerebral structural abnormalities, 4 an infection, and 1 hyponatremia as underlying etiology for status epilepticus. Median age was 70 (range 30-97) years, 15 patients were male. Baseline GCS was 5 (3-12). Levetiracetam successfully treated status epilepticus in 6 patients. Median time to treatment success was 480 (10-16337) minutes in all patients. At 6 months 11 patients had died, support had been actively withdrawn in 3 patients. The mRS was 0 in 4 patients, 1 in 4 patients, 2 in 1 patient, 3 in 2 patients, 4 in 2 patients and 5 in 3 patients. At 12 months, 1 patient with a mRS of 3 had progressed to 4. The most common complications included hyperglycemia (78%), hypotension (59%), acute renal failure (26%), anemia (33%), thrombocytopenia (30%), urinary tract infection (26%), and seizure recurrence (19%).

Conclusions:
Levetiracetam offers a feasible alternative strategy to break status epilepticus as adjunctive third line therapy in a paucity of patients before administration of sedatives with a broad spectrum of adverse effects and needs to be studied in a standardized trial.

Financial Support: None

Poster 19

MULTINUCLEAR ARRAY SODIUM (MARS) IMAGING: A NOVEL MULTICHANNEL PHASED ARRAY THAT PERMITS SIMULTANEOUS HI-SPEED PROTON AND SODIUM IMAGE ACQUISITION TO DETERMINE TIME OF STROKE ONSET AND EXTENT OF IRREVERSIBLE DAMAGE

Joshua Medow, Sean Fain, Krishna Kurpad
University of Wisconsin, Madison,WI, United States

Introduction:
Stroke is the third leading cause of death in industrialized nations.The treatment pathway for ischemia is often determined by assessing the extent of permanent damage aided by imaging modalities such as MRI.Diffusion Weighted Imaging (DWI) is an accepted MRI technique that is sensitive to water diffusion.The restriction of water in damaged cells is used as a surrogate measure of cell death.Although cells may have taken on water,this surrogate measure may be inaccurate,frequently leading to overestimation of the severity of ischemia.Measures of sodium are reported to be more accurate indicators of cell death and can also be used to determine stroke onset time because of a linear relationship between ischemia and sodium concentration.We have developed a multinuclear coil and radiofrequency current source that permit simultaneous high-speed proton and sodium imaging so that treatable ischemia can be better ascertained.

Methods:
An 8-channel,broadband,radiofrequency,phased array and current source were built enabling targeted,accelerated,and simultaneous spectroscopic imaging of water,helium,and sodium on a 1.5T GE MR scanner.It was tested on phantoms of known sodium and water concentrations and in rats with known inhaled helium concentrations.

Results:
High quality imaging of protons and sodium in phantom models of known sodium concentration were obtained.Additionally, high quality helium imaging in rats demonstrated the ability of the system to image other nuclei and proved the quantitative and qualitative imaging capabilities of the array in vivo.

Conclusions:
MARS Imaging provides a new, simultaneous multinuclear approach to determine the extent of ischemia quickly and quantitatively.High quality images with this system can be obtained in humans with the same hardware and can be used with standard 1.5T MR scanners.

Financial Support: Research affiliation with General Electric Waukesha, WI

Poster 20

CHANGES IN VASOPRESSIN SYNTHESIS AND RELEASE IN MAGNOCELLULAR NEURONS DURING EXPERIMENTAL AND HUMAN SEPTIC SHOCK

Romain Sonneville[1], Céline Guidoux[1], Lucinda Barrett[2], Odile Viltart[3], Virginie Mattot[4], Andrea Polito[5], Shidasp Siami[5], Geoffroy Lorin de la Grandmaison[6], Anne Blanchard[7], Mervyn Singer[2], Djillali Annane[5], Françoise Gray[1], Jean-Philippe Brouland[1], Tarek Sharshar[5]

[1]Department of Pathology, Lariboisière Hospital, (AP-HP), 75475, Paris cedex 10, University Paris Diderot-Paris 7, Paris, France, [2]loomsbury Institute of Intensive Care Medicine, University College London, Cruciform Building, Gower Street, London, United Kingdom, [3]Laboratory of Neuroimmunoendocrinology, Pasteur Institute of Lille, IFR 142 and University of Lille, Lille, France, [4]CNRS-UMR8161, Biology Institute of Lille, Pasteur Institute of Lille, Lille, France, [5]General Intensive Care Medicine, Raymond Poincaré Hospital (AP-HP), University of Versailles SQY, Garches, France, [6]of Pathology, Raymond Poincaré Hospital (AP-HP), University of Versailles SQY, Garches, France, [7]Department of Physiology, Georges Pompidou European Hospital (AP-HP), University Paris 5, Paris, France

Introduction:
Septic shock is often associated with relative vasopressin (AVP) deficiency that may be related to impaired AVP synthesis and release by the neurohypophyseal system, which includes the neurohypophysis and magnollecular neurons of the paraventricular and supraoptic nuclei. Neurohypophyseal system has never been assessed in human septic shock and only partially in experimental sepsis. We investigated AVP synthesis and release by the neurohypophyseal system in septic rats and in human septic shock. Design: Ex vivo human and animal study. Setting: University research laboratory

Methods:
In the human study, post-mortem examination of the neurohypophyseal system was performed in patients who died from septic shock (n=9) or other causes (n=10). In the experimental study, sepsis was induced by fecal peritonitis in conscious, fluid-resuscitated Male adult wistar rats. Rats either early died spontaneously from septic shock in average at 24 hours (septic early death, n=10) or were sacrificed in average 36 hours after induction of sepsis (septic, n=10). Post-mortem examination was performed in both groups. Comparisons were made against sham operation controls (n=8). AVP protein and mRNA were assessed by immunohistochemistry and in-situ hybridization.

Results:
In both septic shock patients and septic rats with early death, the AVP content in the neurohypophyseal and supraoptic magnocellular neurones was decreased while it was increased in the paraventricular magnocellular neurones. No significant change was observed in AVP mRNA expression in either paraventricular or supraoptic magnocellular cells.

Conclusions:
In septic shock, AVP post-transcriptional synthesis and transport are altered in the supraoptic and paraventricular magnocellular neurones, respectively. This suggests that supraoptic and paraventricular nuclei are liable to distinct pathogenic mechanisms, which may account for relative AVP deficiency.

Financial Support: None

Poster 21

SINGLE BRAIN DEATH EXAMINATION WITH CONFIRMATORY CEREBRAL BLOOD FLOW TESTING AS AN ALTERNATIVE TO THE STANDARD DUAL BRAIN DEATH EXAMINATION

Chethan Venkatasubba Rao[1], Mohammed Rehman[1], Amy Barber[1], Stephanie Sommerfield[2], Aashish Patel[1], Vivek Rai[1], Jody Wellwood[1], Jesse Corry[1], Tamer Abdelhak[1], Panayiotis Varelas[1]
[1]Henry Ford Hospital, Detroit, Michigan, United States, [2]Gift of Life, Michigan, United States

Introduction:
Dual brain death (DBD) examination has been historically followed to determine irreversible brain damage. A policy was introduced in our hospital to utilize single brain death examination (SBD) including an apnoea test and a confirmatory test for cerebral blood flow in patients with catastrophic neurological injuries to determine brain death. We investigated if organ procurement would be affected by SBD.

Methods:
The database of Gift of Life (The designated organ recovery organization for Michigan), was screened for our institution patients meeting brain death criteria between Jan 2007 and July 2009. For each patient, age, sex, primary cause of mortality, number of brain death examinations performed, type of confirmatory tests used to declare brain death, medical exclusions for organ donation and number of organs procured was obtained. Continuous variables were analyzed using the student t-test and categorical variables using Fischer's exact test with p values set at ≤0.05.

Results:
Seventy patients met brain death criteria between January 2007 and July 2009 and 12 were excluded due to incomplete records. There was no difference between the age and sex composition between the two groups. Twenty seven patients were diagnosed with brain death using SBD while 31 were diagnosed using DBD. Twenty four patients with SBD and 26 with DBD were eligible for organ donation (p=0.7119). 14 each of eligible SBD and DBD patients donated organs (p=1.00); 43 organs were procured from each group (p=1.00).

Conclusions:
Single brain death examination did not preclude the rate of organ donation in our patient cohort. Dual brain death examination can be substituted by a single brain death examination along with a confirmatory test for cerebral blood flow in patients with catastrophic neurological injuries without affecting the rate of organ donation. This may result in a less time delay before declaring death and minimize physician workload.

Financial Support: None

Poster 22

DIAGNOSTIC YIELD OF CATHETER ANGIOGRAPHY IN ADDITION TO MRI IN SPONTANEOUS INTRACEREBRAL HEMORRHAGE

Natasha Renda, Rashmi Narayana, Ryan Snider, Demi Thai, Anna Finley Caulfield, Chitra Venkatasubramanian, Marion Buckwalter, Nancy Fischbein, Michael Marks, Christine Wijman
Stanford University, Stanford, CA, United States

Introduction:
Etiologies for spontaneous intracerebral hemorrhage (ICH) or intraventricular hemorrhage (IVH) vary. MRI can often identify underlying vascular lesions, but conventional catheter angiography remains the gold standard. Guidelines for the use of catheter angiography are non-specific. We aimed to determine the diagnostic yield of catheter angiography in addition to MRI in patients with ICH or IVH who met pre-defined criteria.

Methods:
Consecutive patients with spontaneous ICH or IVH were enrolled. In addition to non-contrast brain CT and laboratory testing, all patients underwent gadolinium enhanced MRI/MRA. Catheter angiography was pursued if the following criteria were met: 1. Lobar ICH or isolated IVH and age ≤ 65 years or 2. Deep ICH *and* no history of hypertension *and* age ≤ 45 years or 3. Any other indication based on the opinion of the treating neurointensivist.

Results:
Of 160 prospectively enrolled patients, 58 (36%) met criteria for catheter angiography. Seven were excluded from angiography because a definitive ICH cause was established by MRI and 2 because of a coagulopathy explaining the ICH. Forty-four (28%) patients underwent catheter angiography, which identified the ICH etiology in 13 (30%). In 12 of these, the diagnosis was already suspected based on MRI, but in 4 cases catheter angiography increased the diagnostic confidence. In one patient a small AVM was diagnosed by angiography alone.

Thirteen patients (8%) had both contrast angiography and pathology. Of these, 8 had a vascular abnormality as the cause of the ICH. Five of these were diagnosed by angiography. In one patient the pathology showed an AVM while the angiogram and the MRI were negative. Two patients with cavernous malformations were diagnosed by MRI alone.

Conclusions:
The diagnostic yield of catheter angiography in spontaneous ICH or IVH is limited if patients also undergo gadolinium enhanced MRI/MRA.

Financial Support: None

Poster 23

RISK OF PULMONARY EDEMA FOLLOWING INTRACEREBRAL HEMORRHAGE

Wolfgang Leesch, Lauren Wendell, Louis Fazen, Alexandra Oleinik, Patrick Lenehan, Natalia Rost, Kristin Schwab, Steven Greenberg, Jonathan Rosand, Joshua Goldstein
Massachusetts General Hospital, Harvard Medical School, Boston, MA, United States

Introduction:
Patients with anticoagulation-related intracerebral hemorrhage (ICH) commonly are treated with fresh frozen plasma (FFP) for anticoagulation reversal. One risk of FFP is thought to be related to volume overload and Pulmonary Edema (PEd). However, this has neither been validated, nor quantified compared with the natural risk following ICH. We hypothesized that patients with anticoagulation-related ICH are at higher risk of PEd in-hospital, and that this increased risk would be related to dose of FFP used.

Methods:
Retrospective review of a prospectively collected cohort of consecutive patients with primary ICH presenting to a single center between August 1994-May 2006.

Results:
Of 989 included patients, 53% were male, and mean age was 73 +/- 12 years. 22% were on warfarin at presentation, with a median INR of 2.7 (IQR 2.1-3.6). Of these patients, 81% received FFP, at a median dose of 5 (IQR 3-8) units. Overall, 78 patients (7.9%) developed PEd, at a median time of 4 (IQR 1-8) days after presentation, and these patients showed a longer hospital length of stay [median 17 (IQR 11-31) days vs. 7 (IQR 4-12) days, p=0.001]. Anticoagulated patients were at higher risk of developing PEd during hospitalization (14% vs. 6%, p=0.001). Patients receiving FFP were also at higher risk than those who did not (15% vs. 5%, p<0.001). In multivariable analysis with a Cox proportional hazards model, use of FFP was an independent predictor of developing PEd (HR 1.08 per unit given, 95% CI 1.04-1.13), and the effect of warfarin fell out of the analysis when FFP was included.

Conclusions:
Patients with anticoagulation-related ICH are at increased risk of PEd during hospitalization, accounted for by FFP use. Each additional unit of FFP confers an approximately 8% increased risk of this complication.

Financial Support: Dr Joshua Goldstein received consulting fees from CSL Behring

Poster 24

CHANGE IN THE RATE OF VENTILATOR ASSOCIATED PNEUMONIA WITH QUALITY IMPROVEMENT INTERVENTIONS IN A NEUROINTENSIVE CARE UNIT

Alexander Papangelou, Laura Desnoo, Donna Fellerman, Elizabeth Zink, Paul Nyquist, Wendy Ziai
Johns Hopkins University School of Medicine, Baltimore, MD, United States

Introduction:
Ventilator Associated Pneumonia (VAP) is an infrequently studied morbidity in neurointensive care, despite high historical rates ranging from12.8 to 27.4 infections per 1000 device days. In comparison, mean rates in medical ICU's have been substantially lower: 7.3-7.5. In our neurocritical care unit (NCCU), VAP incidence was initially near the National Nosocomial Infection Surveillance (NNIS) 75th percentile for Neurosurgical ICUs (31.9 infections per 1000 ventilator days) necessitating an evidence-based performance improvement initiative to reduce VAP rates.

Methods:
Prospective surveillance study of VAP incidence in a 22-bed NCCU over a 10 month period. VAP rates were defined by National Healthcare Safety Network (NHSN) criteria. Interventions included an aggressive hand hygiene campaign, use of the ventilator bundle, oral care every 4 hours, and introduction of the Hi/Lo endotracheal tube. Ventilator bundle compliance was assessed.

Results:
5281 patient days with a total of 2235 ventilator days were monitored. In September 2008, concomitant to an acinetobacter outbreak, VAP rates were 30 infections per 1000 ventilator days. By January 2009, hand hygiene compliance had increased from 36 to 80% and rates were 25. Following institution of frequent oral care in combination with the emphasized VAP bundle, by May 2009, rates had decreased to 16. At this point, all new intubations performed in the unit employed the Hi/Lo ETT. By June 2009, VAP incidence decreased to 11.7 infections per 1000 ventilator days (< 10th NNIS percentile). Compliance with components of the ventilator bundle ranged from 55% to 97%.

Conclusions:
Despite caring for patients at high risk of VAP, concerted efforts with multiple evidence-based performance measures and interventions can significantly reduce the incidence of infection. Feedback with compliance may be essential to maintain low VAP rates.

Financial Support: None

Poster 25

HIGH RATE OF MYOPATHY IN ANEURYSMAL SUBARACHNOID HEMORRHAGE PATIENTS

Thomas Wolfe, Denise Miller-Niklasch, Dhimant Dani, Michael Abraham, Muhammad Taqi, John Lynch, Michel Torbey
Medical College of Wisconsin, Milwaukee, WI, United States

Introduction:
The use of HMG-CoA reductase inhibitors (statins) has increased among subarachnoid hemorrhage (SAH) patients for cerebral vasospasm prophylaxis. Statins increase risk of myopathy, but additional factors may also be causative. Myopathy rates in this group of critically ill patients are not well characterized.

Methods:
SAH patients were prospectively entered into an institutional database; those with myopathy were retrospectively identified. Serum creatine kinase (CK), aldolase (at time of suspected myopathy diagnosis), and catecholamines (at admission), muscle biopsy results, and medication administration records were evaluated.

Results:
Four (3.7%) of 109 aneurysmal SAH patients treated between Jan 1, 2008 through July 31, 2009 were newly diagnosed with myopathy. Two were Hunt/Hess 3, and 1 each: grade 4 and 5. One was Fisher 3, others were Fisher 4. All had symptomatic hydrocephalus, were treated endovascularly with paralysis paraprocedurally, given simvastatin 80mg daily within 24 hours of SAH (none had prior statin use). All were insulin resistant, requiring high dose sliding scale insulin. Two received steroids. All had elevated catecholamines. Maximum CK levels (U/L) were 91, 208, 370, and 714, respectively. Aldolase was checked and elevated in 3 patients. The forth underwent muscle biopsy revealing necrotic fibers; this patient had the lowest maximum CK.

Conclusions:
The myopathy rate in this cohort is 30 times higher than that reported in healthy patients treated with statins. Since all SAH patients did not receive statins, this rate is underestimated and much higher than reported in other trials evaluating statins in SAH, assuming risk is solely attributable to statins. The contribution of other etiologic variables (critical illness, paralytic, steroid) is not clear, and potentially are additive. Aldolase may be an additional means of identifying subclinical cases. These findings need to be confirmed and pathogenic factors better elucidated.

Financial Support: None

Poster 26

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

May Kim[1], Wendy Wright[2], Gene Sung[1]
[1]University of Southern California/Keck School of Medicine, Los Angeles, CA, United States, [2]Emory University, Atlanta, GA, United States

Introduction:
Neurocritical care is a relatively new discipline and its practitioners come from a variety of backgrounds. Salaries are likely to differ based on primary appointment, geographic location, practice setting, and time spent on clinical effort. It is not known how many neurointensivists practice full-time critical care, and it is likely that many also have responsibilities as consultants, researchers and administrators.

Methods:
A survey will be emailed to all members of the Neurocritical Care Society. Information that will be collected regarding salary, 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 results will be reported at the meeting and compared to the previous survey done by the authors.

Conclusions:
The information gathered in this survey enhances the understanding of the current practice of neurocritical care throughout the United States. This data may be valuable to neurointensivists during contract negotiations, to hospital administrators trying to assess the feasibility of hiring a neurointensivist, and to neurologists-in-training as a way of generating interest in neurocritical care as a career choice.

Financial Support: None

Poster 27

SERUM CREATININE AND CREATININE CLEARANCE CHANGES ASSOCIATED WITH THERAPEUTIC HYPOTHERMIA

Michael Abraham, Thomas Wolfe, Muhammad Taqi, Dhimanti Dani, Denise Miller-Niklasch, John Lynch, Michel Torbey
Medical College of Wisconsin, Milwaukee, Wisconsin, United States

Introduction:
Therapeutic hypothermia (TH) is being implemented with an increase for multiple indications in the neuro-ICU. The risk of developing renal dysfunction with TH is thought to be low, but is not clearly defined in neurologic patients.

Methods:
Retrospective chart review of prospectively identified patients. Per institutional TH protocol, patient's goal temperature was 33^0 C. Baseline serum creatinine (Cr) level and Creatinine clearance (CrCl) were obtained and followed at least daily during TH and rewarming. Data was evaluated for changes in Cr and CrCl during and following TH induction and any impact these changes had on treatment.

Results:
Thirty five patients received TH (for post-cardiac arrest and intracranial pressure control related to subarachnoid haemorrhage, intracerebral haemorrhage or traumatic brain injury). Maintenance of goal temperature varied from 4 to 120 hours. Nine (26%) had an increase in Cr and CrCl within normal limits; of these, 3 occurred following induction, 5 occurred during TH maintenance, and 1 during rewarming. Three (9%) patients had an elevation in Cr above normal limits; all of these elevations arose after beginning rewarming, and none led to chronic renal failure. There was a direct relationship in the change in Cr and CrCl ($p<0.0001$). Of the 3 patients who had an abnormal change in Cr, 1 had an abnormal change in CrCl. Overall, 34% of the patients demonstrated some form of elevation in Cr and decrease in CrCl. No patient experienced clinically significant changes in renal function requiring changes in therapy.

Conclusions:
This cohort experienced changes in renal function that were not associated with clinical relevance. The majority of changes occurred during TH or rewarming and were not chronic. Any contribution of TH induced muscle injury to Cr changes would need to be assessed with future study.

Financial Support: None

Poster 28

THE RELATION BETWEEN THE WHITE BLOOD CELL COUNT AND HEMATOMA PROGRESSION IN NONTRAUMATIC SUPRATENTORIAL INTRACEREBRAL HEMORRHAGE

Catalina C. Ionita, Jitendra Sharma, John G. Baker, Sachin Agrawal, Frederick Vincent, Lee R. Guterman
Catholic Health System, Buffalo, NY, United States

Introduction:
Initial hematoma size, coagulopathy, and hypertension are recognized predictors of hematoma progression in intracerebral hemorrhage (ICH). We aimed in our study to assess if the absolute number of WBC and/or increase in the WBC number within 24 hours of progression can predict hematoma progression.

Methods:
Data of consecutive patients with primary, supratentorial ICH, admitted within 24 h of onset were reviewed, identifying patients with progression (WP) and no progression (NP). Hematoma progression was defined as 30% increase of hematoma size, subsequent intraventricular bleeding or increase of the preexistent amount of intraventricular blood. We compared the two groups for demographic data, risk factors, admission neurological status, neurological deterioration occurence, and WBC, coagulation profile, and blood pressure (BP) at admission or within 24 hours of hematoma progression, using univariate and multivariate analysis.

Results:
We identified 221 cases (NP) and 45 (WP). Baseline variables were similar, except for the systolic BP that was higher in WP than in NP group (192 $\pm$ 32 mmHg versus 181 $\pm$ 28 mmHg, p=0.01). Neither WBC at admission (9. 9 $\pm$ 0.7 x 10^3 /mm3 versus 12. 3 $\pm$ 1.6 x10^3 /mm^3, p=0.17) nor the variation of the WBC admission - within 24 h of progression (0.78 $\pm$ 1.87 x10^3 /mm3 versus 1.77 $\pm$ 2.47 x 10^3 /mm3, p <0.15) was significantly different between the NP and WP groups.

Neurological deterioration and mortality were more frequent in the WP than NP group (60% versus 23%, p<0.0001; 38% versus 24 %, p=0.05 respectively). Logistic regression showed that the change in WBC from admission to within 24 hours of progression and systolic BP were associated with hematoma progression (wald statistic 5.72, p<0.02; wald statistic 4.60, p <0.03).

Conclusions:
The variation of WBC within 24 hours of progression and systolic BP seem to be independent predictors of hematoma progression.

Financial Support: None

Poster 29

PREDICTORS AND OUTCOMES OF HEPARIN-INDUCED THROMBOCYTOPENIA IN SUBARACHNOID HEMORRHAGE PATIENTS

Brijesh P Mehta[1], John R Sims[1], Jennifer D Robinson[1], Carlos E Baccin[2], Thabele M Leslie-Mazwi[1], Johnny C Pryor[2], Christopher S Ogilvy[3], Raul G Nogueira[1]
[1]*Department of Neurology, Massachusetts General Hospital, Harvard Medical School, Boston, MA, United States,*
[2]*Department of Radiology, Massachusetts General Hospital, Harvard Medical School, Boston, MA, United States,*
[3]*Department of Neurosurgery, Massachusetts General Hospital, Harvard Medical School, Boston, MA, United States*

Introduction:
Heparin-induced thrombocytopenia (HIT) is a dreaded complication of heparin related products. We analyzed the risk factors and outcomes of subarachnoid hemorrhage (SAH) patients in whom HIT was suspected and either confirmed as present or absent by platelet factor 4 (PF4) antibody test.

Methods:
All patients with presumed aneurysmal, non-traumatic SAH and a PF4 test were identified through the Massachusetts General Hospital's Research Patient Database. Charts, laboratory values and images were analyzed retrospectively.

Results:
We identified 166 patients with SAH who were tested for HIT. Of these patients, 42 (25%) had a positive antibody test. There was no difference between mean platelet nadirs of HIT+ and HIT- patients, 147±93 vs. 153±86 th/mm^3, respectively. Univariate analysis identified gender, magnesium prophylaxis, Fisher group 3, clipping vs. coiling, presence of angiographic spasm, number of vasospasm treatments and day of HIT testing as potential risk factors associated with HIT. A multivariate analysis showed that female gender (OR 8.2, 95%CI 2.0–33.2), greater number of vasospasm treatments (OR 1.5, 95%CI 1.2–2.0), later day of HIT testing (OR 1.2, 95% 1.1–1.3) increased the risk of HIT and coiling reduced the risk compared to clipping (OR 0.2, 95%CI 0.1–0.7). Those patients in whom HIT was present had more infarcts on CT, longer ICU and hospital stays and worse modified Rankin scores on discharge.

Conclusions:
The presence of HIT in SAH has adverse consequences and is more likely in female patients, who have undergone aneurysm clipping and require more than one endovascular vasospasm treatment.

Financial Support: None

Poster 30

THROMBOEMBOLIC RISK OF ACTIVATED FACTOR VIIA USE IN ACUTE INTRACEREBRAL HEMORRHAGE WITH COAGULOPATHY

Sherry H-Y Chou, Xuemei Cai, Rachael Konigsberg, Galen V. Henderson, Farzaneh A. Sorond, Allan H. Ropper, Steven K. Feske
Brigham and Women's Hospital, Boston, MA, United States

Introduction:
Coagulopathy-associated intracerebral hemorrhage (cICH) leads to over 50% mortality and is associated with secondary thromboembolic (TE) complications. Rapid coagulopathy reversal improves cICH outcome. Activated factor VIIa (FVIIa) rapidly reverses coagulopathy and causes local hemostasis, but is associated increased TE. We examine a large case series of cICH patients treated with FVIIa to determine TE rates in this population

Methods:
All cICH patients are treated with standardized protocol with emergent intravenous vitamin K, fresh frozen plasma (FFP), and are eligible for FVIIa 40 mcg/kg. We identified consecutive FVIIa-treated cICH patients from database from 2003-2008 and identified 80 patients. 12 were excluded for no identifiable coagulopathy or FVIIa use for severe trauma. 68 were analyzed. We collect data on diagnosis, coagulopathy etiology, history of ischemic heart disease (CAD) and TE. We examined the incidence of troponin elevation, EKG changes, symptomatic coronary ischemia, venous thrombosis, and stroke following FVIIa use.

Results:
Subjects had average age 67.4 years. Over 80% had abnormal EKG on presentation. 18% FVIIa-treated cICH patients had history of venous thrombosis (DVT) or pulmonary embolism (PE), 25% had CAD, 51% had atrial fibrillation, and 18% stroke. Troponin elevation above 1.0ng/mL developed in 16% patients. Only 1/68 patients developed clinically symptomatic cardiac ischemia. 6% developed DVT/PE, and 1/68 (1.5%) developed ischemic stroke. There is a trend towards correlation of CAD history with degree of troponin elevation (p=0.55).

Conclusions:
Coagulopathy-associated ICH patients have high burden of prior ischemic heart disease and venous thromboembolism. Though low level troponin elevations occur, incidence of FVIIa-related symptomatic cardiac ischemia, stroke, or venous thrombosis is low in FVIIa-treated cICH patients. This low incidence justifies a prospective controlled study to evaluate risk versus benefit of FVIIa use for emergent coagulopathy reversal in cICH.

Financial Support: None

Poster 31

SIGNIFICANCE OF NEUROLOGICAL EXAMINATION IN SEDATED CRITICALLY ILL PATIENTS

Benjamin Rohaut[1], Raphaël Porcher[2], Shidasp Siami[3], Andrea Polito[1], Juliette Bailly-Salin[1], Céline Guidoux[4], Romain Sonneville[5], Emanuele Iacobo[1], Nicholas S Hopkinson[7], Olivier Morla[1], Djillali Annane[1], Tarek Sharshar[1]
[1]General Intensive Care Unit, Raymond Poincaré Teaching Hospital (AP-HP), University of Versailles Saint-Quentin en Yvelines, Garches, France, [2]Biostatistics and Medical Computer Science Department, Saint-Louis Teaching Hospital (AP-HP), Paris, France, [3]General Intensive Care Unit, General Hospital, Etampes, France, [4]General Intensive Care Unit, Teaching Hospital (AP-HP), University of Versailles Saint-Quentin en Yvelines, Boulogne-Billancourt, France, [5]General Intensive Care Unit, Bichat Claude-Bernard Teaching Hospital (AP-HP), Paris, France, [6]General Intensive Care Unit, General Hospital, Ancona, Italy, [7]Respiratory Muscle Laboratory, Royal Brompton and Harefield NHS Trust, London, United Kingdom

Introduction:
Assessing neurological function is important in critical illness, but in sedated patients neurological examination is considered to be non interpretable.

Methods:
This prospective multicentre observational study assessed neurological responses in critically ill patients who required to be sedated with midazolam (± subfentanyl). Their relationship with 28-day mortality and altered mental status (delirium or coma within three days after sedation discontinuation) was also assessed. Daily neurological examination included the Glasgow Coma Scale, the Assessment to Intensive Care Environment score (ATICE), eye position and movement, pupil size and response to light, corneal reflex, oculocephalic response, grimace to noxious stimuli and cough reflex. At awakening, mental status was assessed with using ATICE or Confusion Assessment Method for the ICU (CAM-ICU). Predictive value of neurological responses were assessed in half (2005-2007, Fitting Group, FG) and validated in the other half (2008-2009, Validating Group, VG) of total sample.

Results:
144 patients were included (Women: 52 (36%), SAPSII 54 ± 20). The presence or absence of brainstem responses, assessed within the first 24 hours of sedation, was not dependent on sedative dose. Forty-three (30%) patients died within 28 days of inclusion. Multivariate analysis showed that in both groups, SAPS-II (FG - adjusted OR: 1.06, 95% CI:[1.02–1.10], p=0.0001; VG - adjusted OR: 1.03, 95% CI:[1.00–1.07], p=0.051) and absent cough reflex (FG - adjusted OR: 7.80, 95%CI:[2.00-30.4], p=0.003; VG - adjusted OR: 4.07, 95% CI:[1.07–15.40], p=0.039) were independent predictors of mortality. Fifty (35%) and 25 (17%) patients developed delirium or coma, respectively. Absent oculocephalic response was significantly associated with subsequent occurrence of altered mental status in both groups (FG – 8 (31%) versus 22 (65%), p = 0.005; VG - 7 (30%) versus 23 (68%), p= 0.004).

Conclusions:
Neurological examination is interpretable and may be useful for prediction of outcome of critically ill sedated patients.

Financial Support: None

Poster 32

HORMONAL STATUS AND ICU-ACQUIRED PARESIS IN CRITICALLY ILL PATIENTS

Tarek Sharshar[1], Sylvie Bastuji-Garin[2], Bernard De Jonghe[6], Robert D Stevens[5], Andrea Polito[1], Virginie Maxime[1], Pablo Rodriguez[3], Charles Cerf[4], Hervé Outin[6], Philippe Touraine[7], Kathleen Labord[8]

[1]Department of Intensive Care Medicine,AP-HP, Raymond Poincaré Hospital University Versailles Saint-Quentin en Yvelines, Garche, France, [2]Department of Clinical Research and Public Health, AP-HP, Albert Chenevier-Henri Mondor Hospital, Université Paris 12, Faculty of Medicine, Créteil, France, [3]Department of Medical Intensive Care Medicine, AP-HP, Albert Chenevier-Henri Mondor Hospital, Université Paris 12, Faculty of Medicine, Créteil, France, [4]Department of Surgical Intensive Care Medicine, AP-HP, Albert Chenevier-Henri Mondor Hospital, Université Paris 12, Faculty of Medicine, Créteil, France, [5]Departments of Anesthesiology and Critical Care Medicine; Neurology; and Neurosurgery Johns Hopkins University School of Medicine, Baltimore, United States, [6]Department of Intensive Care Medicine Poissy-Saint-Germain en Laye Hospital, Poissy, France, [7]Department of Endocrinology and Reproductive Medicine, AP-HP, Pitié-Salpêtrière Hospital University Paris Pierre Marie Curie, Paris, France, [8]Department of Physiology, AP-HP, Necker Enfants-Malades Hospital, University Paris Descartes, Paris, France

Introduction:
Pathogenesis of acquired paresis in intensive care unit (ICUAP), a frequent and severe complication of critical illness, is poorly understood. Since critical illness is associated with endocrine dysfunction and ICUAP with female gender in some studies, we hypothesized that hormonal dysfunction might contribute to ICUAP. To determine the relationship between hormonal status and ICUAP in patients with protracted critical illness.

Methods:
Prospective observational study in four medical and surgical ICUs with mechanically ventilated patients for > 7 days. Plasma levels of IgF1, prolactin, TSH, FSH, LH, estradiol, progesterone, testosterone, DHEA, DHEAS and cortisol were measured on the first day patients were awake and cooperative (day 1). Mean blood glucose from admission to day 1 was calculated. ICUAP was defined as a Medical Research Council score < 48/60 on day 7.

Conclusions:
We studied 102 patients, 65 men and 37 women (29 postmenopausal). Among the 86 patients tested at day 7, 39 (49%) had ICUAP, which was more frequent in women (63% vs 36%, p=0.02). 24 (24%) patients died in hospital. Mean blood glucose was higher in patients with ICUAP. Estradiol/testosterone ratio was greater in men with ICUAP.

Financial Support: None

Poster 33

CHARACTERISTICS AND CLINICAL OUTCOMES OF GOOD GRADE SAH PATIENTS ADMITTED TO NEUROLOGICAL INTERMEDIATE CARE

Daniel Evans[3], Gail Tudor[2], Deborah Cushing[1], Jeffrey Florman[1], David Seder[1]
[1]Maine Medical Center, Portland, ME, United States, [2]Husson College, Bangor, ME, United States, [3]university of New England, Biddeford, ME, United States

Introduction:
We evaluated complication rates, outcomes, and the cost of care of patients with good-grade (Hunt and Hess grades I-III) aneurismal subarachnoid hemorrhage (GGSAH) admitted directly to an intermediate care unit (IMC).

Methods:
Retrospective chart review of all GGSAH admitted to a tertiary referral center from 2000 to 2008. We recorded demographics, vital signs, and pertinent aspects of the hospital course. A multivariate logistic regression model including Hunt and Hess grade was employed to evaluate for association between admission location and radiographic or clinical vasospasm or infarction.

Results:
Among 139 GGSAH admissions to IMC or the intensive care unit (ICU), mortality was 6.5%. Thirty-three Grade I patients (60%), 15 Grade II patients (31%), and 4 Grade III patients (11%) were admitted directly to IMC. None of these patients died, and 2 (4%) suffered cerebral infarction. Factors associated with IMC admission were lower HH Grade ($p<0.001$), GCS of 15 ($p<0.001$), and no ventricular drain placement ($p=.013$). Age, medical comorbidities, and clipping vs. coiling were not associated with admission location. Eight patients (15%) admitted to IMC were subsequently transferred to ICU. Patients admitted to ICU were more likely to die (10% vs. 0%, $p=.015$), to suffer respiratory failure (24% vs 4%, $p=.002$), and fever (47% vs 19%, $p=.001$). In multivariate logistic regression, IMC admission was unrelated to vasospasm or infarction. Admission to ICU was associated with higher median patient charges ($92,346.47 vs. $64,286.04, $p<0.001$).

Conclusions:
We found no evidence that IMC admission (primarily among Hunt and Hess I and II patients) was associated with increased morbidity, and the in-hospital mortality rate of IMC admissions over 8 years was zero. Given the higher cost of care among patients admitted to ICU, it may be appropriate to consider IMC admission for selected patients.

Financial Support: None

Poster 34

EVOLUTION OF SOMATOSENSORY EVOKED POTENTIALS IN POST CARDIAC ARREST RATS

Wei Xiong, Matthew Koenig, Xiaoxu Kang, Xiaofeng Jia, Adrian Puttgen, Nitish Thakor, Romeryko Geocadin
Johns Hopkins University, Baltimore, MD, United States

Introduction:
Neurologic injury from cardiac arrest (CA) continues to be a significant problem, in part due to the lack of real-time monitoring of brain injury and recovery. Somatosensory evoked potentials (SEP) are a reliable marker of poor outcome because they are relatively resistant to physiologic and therapeutic perturbations. We tested the hypothesis that early recovery of cortical SEP would be associated with better outcome after resuscitation from CA.

Methods:
Sixteen adult male Wistar rats were subjected to asphyxial cardiac arrest. Half underwent 7 mins of asphyxia (Group CA7) and half underwent 9 mins (Group CA9). Continuous SEPs from median nerve stimulation were recorded from these rats for 4 hours immediately following CA. Additional serial SEPs were recorded at 24, 48, and 72 hours after CA. Clinical recovery was evaluated using the Neurologic Deficit Scale (0 – 80, normal = 80), which was performed at 24, 28, and 72 hours after CA (primary outcome measure).

Results:
All rats in group CA7 survived to 72 hours, while only 4 rats in group CA9 survived to that time. Mean NDS values in the CA7 group at 24, 48, and 72 hours after CA were 58.6, 61.6, and 63.5; while in group CA9, they were 41.3 (p-value 0.001), 51.0 (p-value 0.024), and 54.5 (p-value 0.101), respectively. The N10 (first negative peak at approximately 10 ms) amplitude differed significantly between the two groups within 1 hour after CA. Rats that suffered longer CA durations showed later recovery of N10. The N10 latency was similar between the two groups. Although early recovery of N10 showed a trend towards better 72-hour NDS scores, this was not significant. A smaller N7 peak was consistently observed to recover earlier in all rats, which may represent the thalamic component of SEP.

Conclusions:
The delayed recovery of N10 is associated with longer CA times in rats. Early recovery of N10 shows a trend towards better outcomes. N7, which may represent thalamic activity, reappears much earlier than cortical responses (N10), suggesting thalamocortical desynchrony in early recovery. SEP after CA is a dynamic and promising tool to monitor early neurologic recovery after CA.

Financial Support: None

Poster 35

HIGH-DOSE LIPOPOLYSACCARIDE (LPS) WORSENS VASOSPASM AFTER SUBARACHNOID HEMORRHAGE IN A MURINE MODEL

J. Javier Provencio, Saksith Smithason, Shari Korday Moore
Cleveland Clinic, Cleveland, OH, United States

Introduction:
Evidence suggests a role for inflammation in vasospasm after subarachnoid hemorrhage (SAH). Recent studies suggest that systemic inflammation may lead to vasospasm. To test the hypothesis that systemic inflammation worsens vasospasm we evaluated the effect of LPS on vasospasm.

Methods:
C57BL/6J mice received either 150 ug/animal LPS i.p., or saline. 24 hours later, animals had either SAH induction or sham surgery. In a separate group, neutrophils were depleted prior to LPS administration. To test whether neutrophils in the CSF from the SAH are required for vasospasm, we injected blood from LPS-sensitized, neutrophil-depleted mice to the CSF of LPS-sensitized, non-depleted mice and the converse (adoption studies). At 24 hours post injection, animals were perfused with saline, formalin and India ink, and the brains were removed for quantitative evaluation of basal cerebral vasculature for vasospasm. The mean differences in diameter of MCA segments at 1 mm distal to bifurcation were compared. A separate set of animals were perfused with saline and formalin for immunohistochemical staining of neutrophils and microglia.

Results:
In saline-injected animals with SAH, the mean vessel diameter was significantly smaller compared to the saline-injected sham group. There was no difference in the means of vessel diameter between saline- or LPS-injected sham groups. LPS injection in the animals with SAH exacerbated the vasospasm. Neutrophil depletion prior to LPS ameliorated vasospasm. Neutrophil extravasation into the brain and microglial activation was increased in the LPS group compared to controls but was reversed by neutrophil depletion. In the adoption studies, depletion of neutrophils in the CSF blood ameliorates vasospasm but neutrophil depletion in the systemic circulation did not.

Conclusions:
Systemic inflammation induced by LPS exacerbates vasospasm. The effect is reversed by neutrophil depletion in the CSF. This suggests that inflammation in the brain is a more important contributor than systemic inflammation in vasospasm.

Financial Support: None

Poster 36

HYPOALBUMINEMIA IN ACUTE ISCHEMIC AND HEMORRHAGIC STROKE PATIENTS DOES NOT CORRELATE WITH INCREASED MORTALITY

Laurie McWilliams-Dunnigan, MD, Lindsay Maurath, Eric Novak, Gwendolyn Lynch, Javier Provencio
Cleveland Clinic, Cleveland, OH, United States

Introduction:
Malnutrition in the intensive care setting is associated with increase mortality presumably secondary to increased infections. Acute ischemic and hemorrhagic stroke patients in the ICU often experience a delay of enteral nutrition due to delays in swallowing evaluation and diagnostic procedures that require a period of food abstinence. Serum albumin levels are often used as markers for malnutrition.

Methods:
This study was retrospective analysis of patients admitted from January/December 2006 with the diagnosis of ischemic or hemorrhagic stroke. The goal of the study was to determine the association between albumin levels less than 3.5mg/L during the first 48 hours of ICU admission and mortality. T-tests were used to identify significant difference between means. Chi-square tests were used to examine the distribution of categorical variables across discharge statuses. After identifying variables that were significantly different, a logistic model was built to determine if admission day albumin levels are independently associated with mortality.

Results:
There was no difference in mean serum albumin levels between non-survivors (3.6mg/L) or survivors (3.7mg/L (p=0.1089)). There was no difference between non-survivors and survivors in day 3 albumin levels or in the change from day 1 to day 3. A logistic model controlling for age and dyslipidemia (factors significantly or marginally significantly elevated in non-survivors) showed that admission day albumin was not an independent predictor of outcome.

Conclusions:
In our study, there was no correlation between serum albumin levels and mortality. We did not analyze the incidence of infections in this study population. This study validates the May 2009 Critical Care Medicine guidelines on nutrition support in the ICU. They concluded that albumin was not a valid nutrition assessment tool in the ICU. Future studies should examine the relationship between hypoalbuminemia, prealbumin levels and the incidence of infections in the stroke patient population.

Financial Support: None

Poster 37

BRAIN INTERSTITIAL FLUID TNF-α AFTER SUBARACHNOID HEMORRHAGE

Khalid Hanafy[1], Bartosz Grobelny[2], ES Connolly[2], Stephan A. Mayer[1], Christian Schindler[3], Neeraj Badjatia[1]
[1]Columbia University Medical Center Div. of Neurocritical Care, New York, NY, United States, [2]Columbia University Medical Center, Dept. of Neurosurgery, New York, NY, United States, [3]Columbia University Medical Center, Dept. of Medicine, New York, NY, United States

Introduction:
TNF-α is an inflammatory cytokine that plays a central role in promoting the cascade of events leading to an inflammatory response. Recent studies have suggested that TNF-α may play a key role in the formation and rupture of cerebral aneurysms, and that the underlying cerebral inflammatory response is a major determinate of outcome following subrarachnoid hemorrhage (SAH).

Methods:
We studied 14 comatose SAH patients who underwent multimodality neuromonitoring with intracranial pressure (ICP), cerebral microdialysis, and brain tissue oxygen ($PbtO_2$) as part of their clinical care. Continuous physiological variables were time-locked every 8 hours and recorded at the same point that brain interstitial fluid TNF-α was measured in brain microdialysis samples. Significant associations were determined using generalized estimation equations.

Results:
Each patient had a mean of 9 brain tissue TNF-α measurements obtained over an average of 72 hours of monitoring. TNF-α levels rose progressively over time. Predictors of elevated brain interstitial TNF-α included higher brain interstitial fluid glucose levels (β=0.066, P<0.02), intraventricular hemorrhage (β=0.085, P<0.021), and aneurysm size >6 mm (β=0.14, p<0.001). There was no relationship between TNF-α levels and the burden of cisternal SAH; concurrent measurements of serum glucose, or lactate-pyruvate ratio.

Conclusions:
Brain interstitial TNF-α levels are elevated after SAH, and are associated with large aneurysm size, the burden of intraventricular blood, and elevation brain interstitial glucose levels.

Financial Support: None

Poster 38

RELATIONSHIP BETWEEN BRAIN INTERSTITIAL FLUID TUMOR NECROSIS FACTOR-α AND CEREBRAL VASOSPASM AFTER ANEURYSMAL SUBARACHNOID HEMORRHAGE

Khalid Hanafy[1], Morgan Stuart[2], Alexander Khandji[4], ES Connolly[2], Neeraj Badjatia[1], Stephan A. Mayer[1], Christian Schindler[3]

[1]Columbia University Medical Center, Div. of Neurocritical Care, New York, NY, United States, [2]Columbia University Medical Center, Dept. of Neurosurgery, New York, NY, United States, [3]Columbia University Medical Center, Dept. of Medicine, New York, NY, United States, [4]Columbia University Medical Center, Dept. of Radiology, New York, NY, United States

Introduction:

Experimental studies have demonstrated that tumor necrosis factor-α (TNF-α) plays a crucial role in the onset of hemolysis-induced vascular injury and cerebral vasoconstriction [1]. We hypothesized that TNF-α measured from brain interstitial fluid would correlate with the severity of vasospasm following aneurysmal subarachnoid hemorrhage (aSAH).

Methods:

From a consecutive series of 10 aSAH patients who underwent cerebral microdialysis (MD) and evaluation of vasospasm by computed tomographic angiogram (CTA) or digital subtraction angiography (DSA), TNF-α levels from MD were measured at 8 hour intervals from SAH days 4-6 using enzyme-linked immunosorbent assay (ELISA). A blinded attending neuroradiologist independently evaluated each CTA and DSA and assigned a vasospasm index (VI).

Results:

Five patients had VI<2 and 5 patients had a VI>2, where the median VI was 2 (range 0-18). Median log TNF-α area under the curve (AUC) was 1.64 (pg/mL)*day (interquartile range 1.48-1.71) for the VI<2 group, and 2.11 (pg/mL)*day (interquartile range 1.95-2.47) for the >=2 group ($p<0.01$).

Conclusions:

In this small series of poor-grade aSAH patients, the area under the curve of TNF-α levels from SAH days 4-6 correlates with severity of radiographic vasospasm. Further analysis in a larger population is warranted based on our preliminary findings.

Financial Support: None

Poster 39

EARLY ATTAINED THERAPEUTIC HYPOTHERMIA AS A PREDICTOR OF FAVORABLE NEUROLOGIC OUTCOME AFTER CARDIAC ARREST

Nicole Chiota, William Freeman, Kevin Barrett
Mayo Clinic Florida, Jacksonville, Florida, United States

Introduction:
Mild therapeutic hypothermia (TH, 32-34ºC) reduces mortality and improves neurologic outcomes after ventricular fibrillation cardiac arrest (CA). The relationship between time to achieve TH and outcomes remains undefined. We hypothesized that a shorter interval from CA to achieve TH would be associated with improved neurologic outcome.

Methods:
We retrospectively reviewed all subjects with in- or out-of-hospital CA treated with TH between November 2006 and April 2009 at our institution. The time to target temperature was defined as the interval between witnessed CA and first measurement of hypothermia (≤ 34 ºC) and further categorized as early (< 6 hours) or delayed (> 6 hours). Outcomes were assessed at the time of death or discharge by the Cerebral Performance Category Score (CPC). Good neurologic outcome was defined as CPC 1 or 2. Fisher's Exact test was used to assess the univariate relationship between time to target temperature and neurological outcome.

Results:
33 patients were treated with TH after in-hospital (39%) and out-of-hospital (61%) CA. Subjects that did not reach target temperature or with unwitnessed CA were excluded. Of the remaining patients, 42% (11/26) survived to discharge and 35% (9/26) achieved a good neurologic outcome. Five patients (5/26) reached early target temperature; 80% (4/5) of those had a good neurological outcome. 24% (5/21) of subjects with delayed target temperature achieved a good neurological outcome. The univariate relationship between time to target temperature and neurological outcome was statistically significant (p=0.034).

Conclusions:
Attaining TH within 6 hours of in- or out-of-hospital CA is associated with a greater likelihood of a good neurological outcome at discharge. Time from CA to achieved TH should be included as a clinically important covariate in future studies of predictors of outcome after CA.

Financial Support: None

Poster 40

DO DYNAMIC AUTOREGULATION TESTS PREDICT STATIC AUTOREGULATION EFFICIENCY AFTER TRAUMATIC BRAIN INJURY?

Santhosh Sadasivan, Shankar Gopinath, Claudia Robertson
Baylor College of Medicine, Houston, Texas, United States

Introduction:
Cerebral autoregulation tests have gained importance for the assessment of patients with a variety of brain disorders. In critically ill patients, testing of dynamic autoregulation is safe and practical, but the ability to respond to steady state change in blood pressure is probably more clinically relevant. The purpose of this study was to compare static autoregulation testing with two different dynamic autoregulation tests (cuff deflation and carotid compression tests) in patients with severe traumatic brain injury.

Methods:
Twenty-two studies were performed in 21 TBI patients. Changes in middle cerebral artery flow velocity (mcaFV) were observed by transcranial Doppler. Static autoregulatory index (sARI) was determined from the steady-state response of mcaFV to phenylephrine-induced rise in blood pressure. Dynamic autoregulatory index (dARI) was determined by the cuff deflation method as described by Aaslid (1989) and the transient hyperemic response ratio (THRR) was calculated as described by Smielewski (1996). These dynamic tests were performed in triplicate at baseline prior to inducing hypertension with phenylephrine, and the 3 values were averaged to give a single index value. Since the anatomy of the brain injury varied from patient to patient, the autoregulatory indices were summarized for the worst and best sides of the brain based on the appearance of the initial CT scan.

Results:
The sARI averaged 36.7$\pm$23.2 on the side of the brain that was more injured, and 35.9$\pm$14.8 on the less injured side. THRR was closely correlated with the sARI, both on the side that was more injured (r=.637, p=.011) and on the less injured side (r=.647, p=.005). The dARI was significantly correlated with sARI only on the side that was more injured (r=.615, p=.004).

Conclusions:
These data suggest the ability of dynamic autoregulation to predict static autoregulation may vary with the type of test chosen.

Financial Support: None

Poster 41

SAFETY OF PERCUTANEOUS GASTROSTOMY (PEG) TUBE PLACEMENT IN STROKE AND CRITICALLY ILL NEUROLOGICAL PATIENTS ON CLOPIDOGREL ALONE OR IN COMBINATION WITH ASPIRIN

Benjamin Anyanwu, Sean Giese
Forsyth Medical Center, Winston-Salem, NC, United States

Introduction:
Published guidelines from the American Society for Gastrointestinal Endoscopy considers PEG tube placement a high-risk procedure for bleeding and recommends discontinuation of Clopidogrel, 7 to 10 days before PEG placement. Unfortunately the perioperative time period is associated with increased ischemic events, length of stay (LOS) in the hospital and resource consumption.

Methods:
This is a retrospective review of prospectively collected data that sought to examine the safety of PEG tube placement in patients while on Clopidogrel alone or in combination with Aspirin.

Results:
10 patients admitted into our Neuro-ICU who met the set criteria during the period January 2008 to July 2009 were included in the study. Mean duration on antiplatelet therapy prior to PEG placement was 7 days. One patient had a new stroke during hospitalization, unrelated to the procedure. No post-operative complications, bleeding or neurologic changes were noted in any of the patients. Relevant blood indices remained largely unchanged.

Conclusions:
Although PEG tube placement is considered a high-risk procedure for bleeding, in the absence of pre-existing bleeding disorder it may be safe to perform this procedure in patients taking Clopidogrel. There may be no need to consider reversion to Aspirin alone, in those on combination therapy. Timely placement of PEG tubes in this sub-group of patients may reduce their LOS and decrease the risk of new ischemic events.

Financial Support: None

Poster 42

USE OF LEVETIRACETAM IN STATUS EPILEPTICUS IN PEDIATRIC ICU

Farhan Tariq, Farida Abid, Ankur Garg, Ahmed Bilal
Oklahoma Health Science Center, OKC,OK, United States

Introduction:
Treatment of status epilepticus (SE) has changed a little over last decades. SE in intensive care unit (ICU) is treated with intravenous benzodiazepines or conventional antiepileptic drugs (AEDs), such as phenytoin and phenobarbital. Levetiracetam (LVM) is a novel AED that does not undergo extensive liver metabolism, does not require drug level monitoring and easy to escalate dose in SE .There is thus a need for alternative/adjuvant therapy. However little data exist regarding use in pediatric population. We retrospectively analyzed the use and safety of LVM in pediatric ICU.

Methods:
Data was collected in pediatric ICU from January to June 2009 included age, sex, therapy indication, duration, ICU admission diagnoses, length of stay, documented seizure clinically and with EEG, concomitant use of other AEDs, and adverse events.

Results:
Thirty six patients were identified (12 males, 24 females; age range day1 to 4 years).The loading dose of LVM was 20 mg/kg intravenous and maintenance dose was 20 mg/kg/day with max escalating dose was 80 mg/kg/day. The most common ICU diagnosis was anoxic hypoxic brain injury (12out of 36 patients). Three patients had impaired renal dose for which LVM was adjusted. All of the patients were initially treated with benzodiazepines and phenytoin for SE. Seventy two percent of the patients treated with LVM for SE after benzodiazepine and phenytoin had no subsequent seizure. The remaining (28%) requiring additional AEDs. The mean ICU stay was 10 days. No adverse event reported with LVM.

Conclusions:
LVM appears to be a good alternative /adjuvant agent in pediatric ICU for SE

Financial Support: None

Poster 43

CLINICAL EVALUATION OF A PORTABLE NEAR-INFRARED DEVICE FOR DETECTION OF TRAUMATIC INTRACRANIAL HEMATOMAS

Claudia Robertson[1], Eric Zager[2], Raj Narayan[5], Neal Handly[3], Alok Sharma[4], Daniel Handley[6], Homero Garza[1], Eileen Maloney-Wilensky[2], Justin Plaum[2], Carolyn Koenig[5], Anne Johnson[3], Timothy Morgan[6]
[1]Baylor College of Medicine, Houston, TX, United States, [2]The University of Pennsylvania School of Medicine, Philadelphia, PA, United States, [3]Drexel University College of Medicine, and Hahnemann Hospital, Philadelphia, PA, United States, [4]Lokmanya Tilak Municipal General Hospital, Mumbal, India, [5]University of Cincinnati, Cincinnati, Ohio, United States, [6]Johns Hopkins University, Baltimore, MD, United States

Introduction:
Early identification of intracranial hematomas is an important management principle for traumatic brain injury (TBI) patients. The purpose of this multicenter clinical study was to evaluate the performance of a near infrared (NIR) based, non-invasive and portable device (Infrascanner by InfraScan, Inc.) to detect traumatic intracranial hematomas.

Methods:
Clinical personnel in five trauma centers collected data using the portable NIR device at the time of a computed tomography (CT) scan of the head performed to evaluate a suspected TBI. All of the CT scans were read by a neuroradiologist from an independent site who was blinded to the measurements of the NIR device.

Results:
Of 431 patients, across a wide range of demographics, 365 patients were included in the per protocol population which was analyzed. In the per protocol population, 96 patients were determined by CT scan to have intracranial hemorrhage of various sizes, depths and anatomical locations. The portable NIR device demonstrated high sensitivity (88%) and specificity (90.7%) in detecting intracranial hematomas larger than 3.5 mL in volume, and less than 2.5 cm from the surface of the brain.

Conclusions:
These results suggest that the NIR based portable device may be useful to supplement clinical information such as symptoms and neurological examination in determining need for and urgency of further imaging studies. Because of the high specificity for detecting an intracranial hematoma, a positive NIR exam would suggest the need for a more definitive imaging study, even in an otherwise low risk patient.

Financial Support: Infrascanner, Inc. funded the study.

Poster 44

BRAIN TISSUE OXYGEN IN POOR GRADE SAH PATIENTS: CLIP VERSUS COIL

Yuejie Gu[1], Andrew Kofke[1], Bryan Pukenas[3], Suzanne Frangos[1], Eileen Maloney-Wilensky[1], Peter LeRoux[2], Joshua Levine[1], Michael Stiefel[2]

[1]University of Pennsylvania Medical Center, Division of Neurocritical Care, Philadelphia, PA, 19104, United States,
[2]University of Pennsylvania Medical Center, Department of Neurosurgery, Philadelphia, PA, 19104, United States,
[3]University of Pennsylvania Medical Center, Division of Interventional Neuroradiology, Philadelphia, PA, 19104, United States

Introduction:

ISAT suggests that patients with ruptured intracranial aneurysms have a better outcome when treated with endovascular therapy.[1] Studies on the use of continuous brain tissue oxygen (BtO2) monitoring support that prolonged episodes of BtO2 < 10 mmHg are associated with poor outcome.[2] We sought to determine whether there is a difference in BtO2 in patients undergoing coil versus clipping that may help to explain the differences in clinical outcome.

Methods:

Patients admitted with subarachnoid hemorrhage (SAH), Hunt and Hess 3-5, that underwent continuous brain tissue oxygen monitoring for a minimum of 2 days during the week immediately after the subarachnoid hemorrhage were considered for analysis. Comparison of minimum daily BtO2, average daily BtO2 and mortality was performed.

Results:

Twenty-eight patients were included in the analysis, 16 clip and 12 coil. No significant difference in minimum (daily) BtO2 was observed between groups; ranged from 11 mmHg – 21 mmHg and 12 mmHg – 19 mmHg on days 2-7 in endovascular and craniotomy groups, respectively. No difference was observed for the average (daily) BtO2 between days in each group; 25, 26, 29, 27, 33, 33 mmHg and 23, 26, 27, 28, 24, 27 mmHg on days 2 – 7 in endovascular and craniotomy groups, respectively. No difference was observed for mortality rate in each group; 14/16 and 4/12 respectively.

Conclusions:

Despite studies, which suggest that patients with ruptured aneurysms have a better outcome when undergoing endovascular therapy, variations in BtO2 do not explain this difference.

Financial Support: None

Poster 45

GOOD OUTCOME FOLLOWING HUNT AND HESS GRADE 5 SUBARACHNOID HEMORRHAGE

Matthew Vibbert, Raimond Helbock, Luis Fernandez, Rishi Malhotra, Khalid Hanafy, Santiago Ortega, Stephan Mayer
Columbia Presbyterian Medical Center, New York, NY, United States

Introduction:
Hunt and Hess grade 5 SAH is accompanied by high rates of mortality and severe disability. Mortality in these patients is driven by withdrawal or limitation of care. Some patients do enjoy good functional outcomes. We seek to describe the frequency and predictive factors for good outcome following grade 5 SAH. We will also describe risk factors for limitation of care following grade 5 SAH.

Methods:
We identified 171 consecutive patients with SAH and worst Hunt and Hess grade of 5 within 24 hours of admission (mean age 58 years; 67% female) in a prospectively collected registry of 1154 aneurysmal SAH patients. The frequency of good outcome (modified Rankin 0 through 3) at 12 months was calculated. We performed univariate analysis of pre-admission and admission characteristics to identify associations with good outcome. Independent risk factors were identified through multiple logistic regression analyses. We performed a multivariate analysis of risk factors for limitation of care.

Results:
Good functional outcome occurred in 25 (15%) of patients. White ethnicity (OR5.1 95%CI1.5-17.2), employment at the time of SAH (OR4.5 95%CI1.1-19.1), lack of limitation of care (OR40.5 95%CI4.9-337.2), and normal papillary reactivity on admission (OR5.8 95%CI1.5-23.1) were independently associated with good outcome. In a subgroup of 77 patients in whom care was not limited, 24 (31%) had good outcomes; white ethnicity(OR6.3 95%CI1.8-21.8), employment status(OR13 95%CI2.7-63.1), and absence of fever(OR6.5 95%CI1.4-30.7) independently predicted good outcome. Among patient characteristics analyzed, admission GCS($p<0.01$) predicted limitation in care.

Conclusions:
A substantial proportion of patients with grade 5 SAH who receive full medical support enjoy a good recovery at 12 months. Age, pre-morbid co-morbidity, and clinical and radiographic measures of hemorrhage severity do not predict good outcome in our study, but socioeconomic factors may.

Financial Support: None

Poster 46

GCS IS A POWERFUL PREDICTOR OF 30-DAY MORTALITY AFTER HYPERTENSIVE INTRACEREBRAL HEMORRHAGE

Elias A. Giraldo, Mohamad A. Haykal
University of Tennessee, Memphis, United States

Introduction:
Intracerebral hemorrhage (ICH) is the most lethal type of stroke. Hypertensive ICH (HICH) is the most frequent ICH subtype. We aimed to evaluate predictors of 30-day mortality after HICH.

Methods:
Retrospective cohort. This study was approved by our IRB.

Results:
We found 64 patients with HICH amongst patients admitted to our hospital from July 2004 to June 2007. Mortality was 18% (n = 12). Thirty-two patients (50%) were male, and 53 (82%) were black. Mean age was 52 ± 12 years (4 patients were ≥ 80 years). Initial pulse pressure 79 ± 28 mm Hg, and mean GCS score was 11 ± 4. Mean ICH volume was 30 ml (range, 0.1 to 253 ml) measured on first head CT scan with the use of the ABC/2 method, and 26 patients (40%) had intraventricular hemorrhage (IVH). Fifty-five patients (86%) had supra- and 9 patients had infra-tentorial ICH. The mean ICH Score (Hemphill, et al. Stroke. 2001;32:891-897) was 1.3 points (range, 0 to 5 points). One of 25 patients with ICH Score of 0, and 7 patients with scores of 4 or 5 died. In univariable logistic regression modeling, all independent predictors used to develop the ICH Score, except age ≥ 80 years ($p = 0.7419$), were associated with 30-day mortality: initial GCS ($p = 0.0001$), ICH volume ($p = 0.0014$), IVH ($p = 0.0036$), and infra-tentorial ICH ($p = 0.0197$), and the ICH Score ($p = 0.0003$) accurately predicted mortality at 30 days. In multivariable logistic regression analysis, only GCS alone was predictive of 30-day mortality. The ROC/AUC analysis demonstrated that GCS was a powerful predictor of mortality with an AUC = 0.91. GCS ≤ 8 was the most powerful predictor of mortality.

Conclusions:
Our study suggests that GCS ≤ 8 is a powerful predictor of 30-day mortality after HICH. Further research is warranted.

Financial Support: None

Poster 47

OXYGEN ADMINISTRATION FOR THE TREATMENT OF POSTCRANIOTOMY PNEUMOCEPHALUS

Panayiotis Varelas, Tamer Abdelhak, Jody Wellwood, Mohammed Rehman, Lonni Schultz, Sandeep Bhangoo
Henry Ford Hospital, Detroit, MI, United States

Introduction:
Pneumocephalus is found in 66% of postcraniotomy computed tomographies of the head (CTOH) and is considered a benign complication of surgery. Occasionally, however, it may lead to lethargy, headache and, if under tension, signs of elevated intracranial pressure or brain herniation. High percentage supplemental oxygen is frequently used as a treatment, but data regarding its effectiveness are very limited.

Methods:
Postcraniotomy patients admitted to the Neuro-Intensive Care Unit with pneumocephalus received 100% FiO2 on-off every 3 hours for at least 24 hours (treatment subgroup). During the off period, this subgroup, as well as the controls remained on room air (or 30% FiO2 if mechanically ventilated). The assignment to each subgroup was based on the neurosurgeon's preference. The intracranial air volume on the CTOH was measured before and after the intervention via an Image J analysis package.

Results:
Twenty-two treated patients and 11 controls (mean age 62 and 60 years, 41 and 55% women, respectively) were identified. The most common diagnoses were subdural hematoma (in 32% vs 27% for the treatment and control subgroups, respectively) and tumor (27% in both). There was no difference in the number of ventilated patients or in those with external ventricular drainages, in the lapsed period between the initial and final CTOH and the initial and final volume of air between the two subgroups. The percentage of air volume change (after adjustment for the lapsed time) and the rate of air absorption were significantly higher in the treated group ($71 \pm 23\%$ vs $45 \pm 35\%$, $p = 0.013$ and $2.6 \pm 1.97\%$/hour vs $1.25 \pm 0.8\%$/hour, $p = 0.007$, respectively).

Conclusions:
This pilot study suggests that intermittent oxygen administration in patients with craniotomy decreases the pneumocephalus volume and increases the rate of intracranial air absorption.

Financial Support: None

Poster 48

DOES THE A-A GRADIENT BEFORE THE APNEA TEST PREDICT COMPLICATIONS?

Alan Yee, Alejandro Rabinstein, Eelco Wijdicks
Mayo Clinic, Rochester, MN, United States

Introduction:
In a recent publication (Wijdicks et al. *Neurology*. 2008 Oct 14;71(16):1240-4), the safety of apnea testing in the declaration of brain death was evaluated at a single tertiary care center. One major conclusion was that apnea testing was safe in hemodynamically compromised patients in most circumstances and rarely aborted. Determinants of apnea test completion failure are unknown.

Methods:
We calculated the alveolar-arterial oxygenation gradients (A-a gradient) in the previously studied cohort. Arterial blood gas values were obtained prior to the initiation of apnea testing. Patients that completed the procedure during the declaration of brain death were compared to those whose studies were aborted. Statistical analysis was performed using nonparametric Wilcoxon rank-sum test.

Results:
Of the original 228 patients studied, A-a gradients were calculated for 205 patients. Seven of these patients had aborted apnea testing because of hypoxemia and/or hypotension. Seventy-nine percent of patients that completed apnea testing had gradients larger than 100 mm Hg compared to 86% in those whose study was aborted, 21% versus 71% with gradients greater than 300 mm Hg, and 9% versus 43% with gradients greater than 400 mm Hg. The A-a gradient median values for completed and aborted apnea tests were 183 mm Hg (range: -150 - 620) and 325 mm Hg (range: 80 - 561), respectively (p value= 0.038).

Conclusions:
The apnea test can be performed safely in most hemodynamically compromised individuals with large A-a gradients undergoing brain death evaluation. A larger percentage of patients that failed completion of apnea testing had significantly greater A-a gradients. Predicting apnea test failure with this respiratory parameter warrants further validation in a larger population.

Financial Support: None

Poster 49

IMPAIRED BRAIN GLUCOSE TRANSPORT AND INCREASED SYSTEMIC GLYCEMIC VARIABILITY PREDICT CEREBRAL METABOLIC DISTRESS AND MORTALITY AFTER SEVERE BRAIN INJURY

Pedro Kurtz, Jan Claassen, J Michael Schmidt, Raimund Helbok, Khalid A. Hanafy, Luis Fernadez, Kiwon Lee, Neeraj Badjatia, Stephan A. Mayer
Columbia University Medical Center, New York, NY, United States

Introduction:
Cerebral glucose metabolism and energy production are affected by serum glucose levels. The objective of this study was to assess whether serum glucose variability and the ratio of cerebral-to-serum glucose are associated with cerebral metabolic distress and outcome after severe brain injury

Methods:
Retrospective cohort study conducted in a neurological intensive care unit of a university hospital. We studied 46 consecutive comatose patients with subarachnoid or intracerebral hemorrhage, traumatic brain injury, or cardiac arrest who underwent cerebral microdialysis and intracranial pressure monitoring.Continuous insulin infusion was used to maintain target serum glucose levels of 80-120 mg/dl. General linear models of logistic function utilizing generalized estimating equations were used to relate these predictor variables to cerebral metabolic distress (defined as a lactate/pyruvate ratio [LPR] ≥40) and mortality.

Results:
The ratio of brain-to-serum glucose was calculated every 1 to 2 hours. Daily serum glucose variability was expressed as the standard deviation (SD), mean amplitude glycemic excursion (MAGE), and glycemic lability index (GLI) of all serum glucose measurements. A total of 5187 neuromonitoring hours and 300 days were analyzed. After adjustment for Glasgow Coma Scale scores, cerebral perfusion pressure, and serum glucose levels, brain/serum glucose ratios below the median (0.12) were independently associated with increased risk of metabolic distress (adjusted OR=1.4 [1.2 – 1.7], P<0.001). Increased serum glucose variability was also independently associated with higher risk of cerebral metabolic distress (adjusted OR=1.7 [1.3 – 2.4], P<0.001 for SD and adjusted OR=1.2 [1.02 – 1.4], P=0.03 for MAGE). Low brain/serum glucose ratios and all three measures of increased serum glucose variability were also independently associated with in hospital mortality after adjusting for age and APACHE-II scores (all P≤0.03)

Conclusions:
Reduced brain/serum glucose ratios and increased serum glucose variability are associated with cerebral metabolic distress and increased hospital mortality after severe brain injury.

Financial Support: None

Poster 50

CARDIAC OUTPUT AUGMENTATION WITH FLUID RESUSCITATION IMPROVES BRAIN TISSUE OXYGENATION AFTER SEVERE BRAIN INJURY

Pedro Kurtz, Raimund Helbok, Jan Claassen, J Michael Schmidt, Luis Fernandez, Mathew Vibbert, R Morgan Stewart, E Sander Connoly, Neeraj Badjatia, Stephan Mayer, Kiwon Lee
Columbia University Medical Center, New York, NY, United States

Introduction:
In critically-ill neurological patients, cerebral perfusion may be optimized by manipulating cerebral perfusion pressure and cardiac output. The objective of this study was to investigate the relationship between cardiac output (CO) response to a fluid challenge and changes in brain tissue oxygen pressure (PbtO2) in patients with severe brain injury

Methods:
Prospective observational study conducted in a neurological intensive care unit of a university hospital. Normal saline (500mL) or albumin 5% (250mL) boluses were given according to a standardized fluid management protocol. The relationship between CO and PbtO2 was analyzed using generalized estimating equations with an exchangeable correlation structure

Results:
We studied 78 fluid challenges administered to 17 consecutive comatose patients that underwent multimodality monitoring with CO, intracranial pressure (ICP), and PbtO2. Diagnoses included subarachnoid hemorrhage (N=10), intracerebral hemorrhage (N=3), cardiac arrest (N=2), traumatic brain injury and status epilepticus (N=1 each). Of the 78 fluid boluses analyzed, 34 (44%) resulted in a ≥10% increase in CO. Median absolute (+5.4vs+0.7 mmHg) and percent (20% vs 3%) changes in PbtO2 were greater in CO responders than in non-responders. In a multivariable model, a CO response was independently associated with PbtO2 response (adjusted OR 15.4, 95%CI 1.9–122.0, P=0.01) after adjusting for mean arterial pressure, ICP and end-tidal CO2. Stroke volume variation showed a good ability to predict CO response with an area under the ROC curve of 0.85 and a best cutoff value of 8%.

Conclusions:
Bolus fluid resuscitation resulting in augmentation of CO can improve cerebral oxygenation after severe brain injury

Financial Support: None

Poster 51

OUTCOME AND FINANCIAL IMPACT AFTER SUBDURAL HEMORRHAGE

Kenneth De Los Reyes, Erin Biro, Arjun Gowda, Christina Grilo, Errol Gordon, Aman Patel, HR Winn, Joshua Bederson, Jennifer Frontera
Mount Sinai School of Medicine, New York, NY, United States

Introduction:
Little current data exists regarding outcome, cost and length of stay after subdural hemorrhage (SDH). We sought to examine predictors of discharge disposition, ICU and hospital length of stay (LOS) and direct, indirect, ICU, surgical and imaging charges for SDH.

Methods:
A retrospective review was conducted of acute, chronic and subacute SDH patients, aged >18 years admitted to our hospital between 2001-2008. Disposition was characterized as dead or poor (discharged to a nursing home, hospice, subacute or chronic care facility). Multivariable logistic regression analysis was performed to identify predictors of each outcome variable.

Results:
Of 216 SDH patients, the median age was 73.5 (19-95), and the median admission Glasgow Coma Scale (GCS) was 14 (3-15). The SDH was characterized as acute in 65 (36%), subacute in 77 (44%), chronic in 22 (13%) and acute, subacute and chronic in 12 (7%). Craniotomy was performed in 108 (50%) of patients, burrhole drainage in 22 (10%) of patients and both in 9 (4%) of patients. Death occurred in 29 (13%) of patients and poor outcome in 66 (31%). Significant predictors of death or poor outcome included age, admission GCS and hospital LOS (all $P<0.05$). Surgery was protective against poor outcome (Odds ratio [OR] 0.3, 95% confidence interval [CI] 0.1-0.8, $p=0.010$). Median hospital LOS was 8 (1-99) days and median ICU LOS was 3 (0-70) days. Both were associated with GCS (all $P<0.05$). Median total direct charges for hospitalization were $11,106 ($686-$172,556). ICU and hospital LOS were significant predictors of direct charges, overhead, imaging and surgical charges (all $P<0.001$). Herniation, SDH thickness, type of SDH, type of surgery and gender did not predict discharge disposition, cost or LOS.

Conclusions:
Despite good admission neurological status, death or poor discharge outcome is common after SDH. Though surgery mitigates against poor discharge disposition, LOS and charges remain high.

Financial Support: None

Poster 52
ACUTE FULMINANT HEPATIC FAILURE, ENCEPHALOPATHY AND EARLY CT CHANGES
Sathees ThayaparaRajah, Bryan Young, Irene Gulka, Ahmed Al-Amri, Sujit Das
LHSC, University of Western Ontario, London, ON, Canada

Introduction:
Acute fulminant hepatic failure (AFHF) is common in tertiary care centers with transplant facilities. Cerebral edema frequently threatens the lives of such patients. We reviewed 25 cases of AFHF in the NeuroICU, noting the incidence of cerebral edema with CT scans and factors associated with mortality.

Methods:
Patients were captured through HMRI classification of acute liver/hepatic failure. Chart review included tabulation of: demographics, INR; serum bilirubin, creatinine, albumin; in-hospital mortality. CT scans were re-read with blinding to clinical information and catalogued for changes in sulcal markings, ventricular size and gray-white differentiation (GWD). Inclusion criteria: age greater than 12 years, encephalopathy, hepatic failure within 8 weeks of onset of liver disease, CT scans of head performed.

Results:
Acetaminophen toxicity was the most common etiology (9 cases). Twelve patients had cerebral edema on CT, including 8 of the 9 with acetaminophen toxicity. Decreases in sulcal markings and ventricular size preceded conspicuous alterations in GWD. Fourteen died, including all 12 with cerebral edema. None of the hematological or biochemical variables correlated significantly with mortality.

Conclusions:
Acetaminophen toxicity is a common cause of AFHF; this combination has a strong association with cerebral edema. Early development of cerebral edema occurs in almost all the AFHF cases with Acetaminophen overdose and can be detected in its early stages. This facilitates management for prevention of fatal brain herniation. AFHF patients develop the changes on brain parenchyma within 24 hours of onset of symptoms, during Grade I-II encephalopathy, most strikingly with AOD.

Financial Support: None

Poster 53

PHARMACOECONOMIC ANALYSIS OF MEDICATIONS USED FOR SEIZURE PROPHYLAXIS IN TRAUMATIC BRAIN INJURY

Karen McAllen, Jeffrey Barletta
Spectrum Health, Grand Rapids, MI, United States

Introduction:
Levetiracetam is increasingly being considered for seizure prophylaxis following TBI. Although its acquisition cost is higher than phenytoin, the complete cost of therapy remains unknown as levetiracetam does not require therapeutic drug monitoring and has less pharmacokinetic variability. We developed a cost-minimization model to compare total costs associated with phenytoin and levetiracetam when used for seizure prophylaxis following TBI.

Methods:
Five scenarios were tested based on drug, initial method of administration (IV vs. PO) and whether or not PO conversion occurred. Factors considered in the analysis were drug costs, monitoring costs and likelihood of achieving a therapeutic concentration. Treatment duration consisted of 7 days. For arms that included PO transition, PO therapy began after day 2. Decision trees were developed and a single-payoff method was used to identify the least costly scenario. Hospital acquisition costs using 2008 US dollars were used to assess all costs.

Results:
The scenario associated with the lowest cost was IV phenytoin followed by PO levetiracetam ($109/patient). This was followed by IV levetiracetam transitioned to PO levetiracetam ($142), IV phenytoin transitioned to PO phenytoin ($152), IV phenytoin ($161) and IV levetiracetam ($397). The factor associated with the most variability in the model was timing of PO transition. A two-way sensitivity analysis which altered timing of PO transition revealed IV phenytoin followed by PO levetiracetam as the least costly scenario except when IV to PO transition occurred after day 1. In this scenario, IV levetiracetam followed by PO levetiracetam was preferred.

Conclusions:
IV phenytoin followed by PO levetiracetam will result in the lowest overall cost when used for a total 7-day course in patients with TBI. This illustrates the importance of considering all costs associated with a therapy when evaluating the total cost of medication therapy.

Financial Support: None

Poster 54

DISSONANCE BETWEEN ACTUAL DCD ELIGIBLE PATIENTS AND REQUIRED DCD PROTOCOL "MODEL ELEMENTS:" IMPLICATIONS FOR POLICIES AND PRACTICE

Jacqueline Kehler, Jeffrey Frank, Fernando Goldenberg, Agnieszka Ardelt
University of Chicago, Chicago, United States

Introduction:
The Joint Commission accreditation standards require hospitals to develop policies which address donation after cardiac death (DCD). OPTN/UNOS published the "model elements" for DCD protocols that describe suitable DCD candidates to primarily have "non-recoverable and irreversible neurological injury" resulting in ventilator dependency; a description we suspected to be highly inaccurate. We sought to more accurately clinically characterize those considered DCD eligible to facilitate constructive improvement of relevant policies and processes.

Methods:
Local OPO quarterly audits over 27 months identified 49 patients who were considered eligible for DCD and died within the requisite 90 minutes of treatment withdrawal (DCDEP). All cases were reviewed to determine the frequency/nature of any neurological abnormalities and whether they were "non-recoverable and irreversible." We also characterized the mechanism of respiratory failure and death.

Results:
27 (55%) of DCDEP had an identified neurological injury. Only 10/49 (20%) had "non-recoverable and irreversible neurological injuries. 25/49 (51%) of DCDEP were seen by a neuro-specialist, and 23/49 (47%) had brain imaging. 9/49 (18%) had a neurological injury that could compromise ventilatory drive, and at least 6/49 (12%) died of airway compromise with variable approaches to palliative care regarding sedation and oral airway usage.

Conclusions:
1. 45% of DCDEP had no neurologic injury.
2. Only 9 patients (18%) had neurologic injuries that could be correctly characterized as "non recoverable and irreversible" leading to "ventilatory dependency."
3. Airway compromise is an important cause of death in DCDEP and demands better uniformity of palliative care to assure equivalent treatment of dying patients independent of "donor status."
4. The published "model elements" for DCD protocols do not accurately represent the patient population.

Financial Support: None

Poster 55

VENTILATOR-ASSOCIATED PNEUMONIA IN A NEUROLOGIC INTENSIVE CARE UNIT DOES NOT LEAD TO INCREASED MORTALITY

Asma M. Moheet, S. Andrew Josephson, Wade S. Smith
University of California, San Francisco, San Francisco, CA, United States

Introduction:
Ventilator-associated pneumonia (VAP) is the most common nosocomial infection among medical intensive care unit (ICU) patients and associated with increased mortality and length of stay (LOS). Neurologic disease is a risk factor for VAP development, but the relationship between VAP and outcomes in neurologic patients remains largely unknown.

Methods:
All mechanically-ventilated patients over a two-year period with neurovascular disease were included. Data collected included patient demographics, dates of admission and discharge, LOS, and ventilator hours. VAP was defined using standard published criteria. Comparisons between neurologic patients who did and did not develop VAP were made using univariate and multivariate analysis.

Results:
Of 585 intubated neurovascular patients, 24 (4.1%) developed VAP. Compared with those who did not develop VAP, those with VAP were younger (51.8 ± 13.9 versus 58.8 ±15.9, $p=0.03$), had increased LOS (32.6 ± 29.2 days versus 14.5 ± 7.8, $p<0.001$), and more ventilator hours (272 ± 257 versus 85.9 ±140, $p<0.001$). There was no difference in mortality between patients with and without VAP (25.0% versus 28.3%, $p=0.72$). VAP was not an independent predictor of mortality in a multivariate model (OR 1.11, $p=0.855$). Subsequent case-control analysis of patients with and without VAP demonstrated an increase in transports for cross-sectional head imaging (6.8 transports versus 4.9, $p=0.03$).

Conclusions:
VAP in neurocritical care patients is associated with increased LOS and ventilator hours, but does not lead to increased mortality, contrary to prior studies in medical ICU patients. The significance and frequency of VAP in neurologic patients is different from patients in other ICUs because reasons for intubation vary. Neurologic patients with VAP have more imaging-related transports compared to controls, suggesting an association with ventilator disconnections.

Financial Support: None

Poster 56

IMPACT OF PATTERN OF ADMISSION ON ICH OUTCOMES

Neeraj Naval, Shannon Ledroux, Juan Carhuapoma
[1]Johns Hopkins University School of Medicine, Baltimore, MD, United States, [2]Johns Hopkins University School of Medicine, Baltimore, MD, United States, [3]Johns Hopkins University School of Medicine, Baltimore, MD, United States

Introduction:
ICH causes the highest mortality of all strokes. Admission to a Neuro-ICU has been associated with reduced mortality following ICH. This is leading to several hospitals routinely transferring ICH patients to hospitals with Neuro-ICUs. However, delays in optimizing management prior to and during transfer often leads to deleterious consequences. Our objective was to compare functional outcomes in ICH patients admitted to our Neuro-ICU directly from our ED with inter-hospital transfer admissions.

Methods:
Records of consecutive spontaneous supratentorial ICH patients admitted to our Neuro-ICU were reviewed. Patients with ICH related to trauma or underlying lesions (brain tumors, aneurysms, AVM) were excluded. We compared outcomes at discharge in patients admitted directly from our ED and inter-hospital transfers (IHT) using dichotomized modified Rankin Scale. Other factors potentially impacting outcomes such as age, ICH volume, IVH volume and admission GCS were included in the multiple logistic regression analysis.

Results:
125 patients were included in the analysis (ED 61.6%; IHT 38.4%). There were no significant differences between the 2 groups in mean age (ED 63.4 +/- 13.1; IHT 63.4 +/- 15.2, p 0.96), ICH volume (ED 31.4 +/- 37.6; IHT 33.5 +/- 42.8, p 0.76), IVH volume (ED 6.0 +/- 11.2; IHT 8.0 +/- 14.5, p 0.38) and GCS (ED 11.3 +/-3.7, IHT 10.9 +/-3.5; p 0.44). 57.2% ED patients had good outcomes at discharge compared to 37.5% IHT. This difference was statistically significant following univariate (p=0.034, 95% CI=0.2151-0.9416) and multivariate analysis (p=0.028, 95% CI=0.1338-0.8896). Odds (adjusted) of ED admissions having good outcomes was 3 times higher than inter-hospital transfers.

Conclusions:
ICH patients brought to the Neuro-ICU directly from our ED had significantly better outcomes than inter-hospital transfers. Although this could possibly be caused by delays in optimizing ICH management, other equally plausible hypotheses need to be prospectively tested.

Financial Support: None

Poster 57

POOR INTER-RATER RELIABILITY FOR THE DIAGNOSIS OF PNEUMONIA LIMITS ITS UTILITY AS A MARKER OF QUALITY OF CARE

Isis Duran, Storm Liebling, Michael Moore, Andrew Naidech
Northwestern University, Chicago, United States

Introduction:
Hospital acquired pneumonia (HAP) is a significant cause of morbidity and mortality. HAP increases costs, impacts quality metrics and will soon be designated as a Medicare “never event”. The US Centers for Disease Control have published standard guidelines for the diagnosis of pneumonia, but few confirmatory data exist. We sought to determine the inter-rater reliability of diagnosing pneumonia by CDC criteria in patients admitted for brain hemorrhage.

Methods:
Patients with intraparenchymal or subarachnoid hemorrhage admitted to our Neuro/Spine ICU in 2007 were included in this IRB-approved study. Utilizing CDC criteria, pneumonia was diagnosed prospectively by a neurointensivist and institutional infection control (IC) personnel. Following a thorough review of the electronic medical records, chest radiographs, and microbiology results, a neurocritical care fellow and a pulmonary critical care attending physician made an independent retrospective assessment of the diagnosis. Analysis of the inter-rater reliability of the diagnosis of pneumonia was performed using kappa statistics.

Results:
One hundred three patients were identified. The male:female ratio was 42:61. Pneumonia was diagnosed in 5 patients by IC personnel, 10 by the neurointensivist, 24 by the fellow, and 26 by the pulmonologist. Overall inter-rater reliability was poor, with a median kappa value of 0.22 [0.13-0.44]. The highest inter-rater agreement was between the fellow and the pulmonologist (kappa=0.58), while the lowest was between the pulmonologist and IC personnel (kappa=0.05).

Conclusions:
The diagnosis of HAP by CDC criteria, despite highly trained reviewers and clear diagnostic criteria, had poor inter-rater reliability in a sample of high risk patients. The diagnosis of HAP should not be a measure of quality of care, nor should it be used as a determinant of payment unless the inter-rater reliability can be markedly improved.

Financial Support: None

Poster 58

FINANCIAL IMPACT OF SURGICAL VERSUS ENDOVASCULAR ANEURYSM REPAIR AFTER SUBARACHNOID HEMORRHAGE

Jennifer Frontera, Kenneth De Los Reyes, Errol Gordon, Arjun Gowda, HR Winn, Joshua Bederson, Aman Patel
Mount Sinai, New York, United States

Introduction:
Recent studies have reported excess hospitalization costs for aneurysm coiling compared to clipping after subarachnoid hemorrhage (SAH). We aimed to compare categories of charges, length of stay (LOS), and discharge disposition in patients who underwent surgical versus endovascular aneurysm repair.

Methods:
A retrospective review was conducted of spontaneous SAH patients between 7/2001-3/2009. Charges captured in the hospital database and were categorized as direct, overhead, ICU, surgical and radiographic/angiographic. Analysis was adjusted for age, Hunt-Hess grade, aneurysm size, aneurysm location and LOS. Discharge disposition and LOS were compared between clipped and coiled patients using logistic regression or Mann Whitney U-nonparametric test.

Results:
Of 264 SAH patients, 90 (34%) were clipped and 131 (50%) were coiled. Coiled patients were significantly older (58 versus 54 years; P=0.008), and had larger aneurysms (6 versus 5mm; P=0.005). There were no differences in Hunt-Hess grade, aneurysm location, or modified Fisher Score. Compared to coiled patients, median radiographic/angiographic charges were lower in the clipped group ($5063 versus $11,803, adjusted OR [aOR] 0.04, 95% CI 0.01-0.2, P<0.001), but median surgical charges were higher ($4,686 versus $0, aOR 5398, 95% CI 139-208,870, P<0.001). Total median direct charges were similar ($29,247 for clipped versus $32,056 for coiled patients, P=0.209), as were ICU direct charges ($12,364 versus $12,581, P=0.263) and overhead ($10,847 versus $12,240, P=0.093). Median ICU LOS (11 days for each group) and hospital LOS (16 days for each group) were similar as were discharge dispositions after adjusting for age, Hunt-Hess grade and aneurysm size: 9% of clipped patients died versus 14% of coiled patients (P=0.719) and 20% versus 30% had a poor discharge disposition (p=0.581).

Conclusions:
Though surgical and radiographic/angiographic charges differed between SAH patients who had surgical versus endovascular repair, ICU charges, overhead and total direct charges were similar as were ICU and hospital LOS and discharge disposition.

Financial Support: None

Poster 59

EFFICACY OF REPEATED 14.6% SALINE BOLUSES IN LOWERING INTRACRANIAL PRESSURE AND IMPROVING CEREBRAL PERFUSION

Ramin Eskandari, Elaine Skalabrin
University of Utah, Salt Lake City, UT, United States

Introduction:
Osmotic diuretics and hypertonic saline (HS) are commonly used to treat traumatic brain injury (TBI). The untoward effects of mannitol, including hypotension, rebound intracranial hypertension, decreased potency and effect duration have lead to research of alternative treatments. Hypertonic saline has been increasingly used to treat cerebral edema, however, efficacy and safety of repeated boluses has not been established. This preliminary prospective trial assesses the ability of single 50 ml 14.6% saline bolus to lower ICP without losing potency while maintaining hemodynamic stability.

Methods:
Thirty-five individual boluses of 14.6% saline were given in 5 TBI patients (aged 21-53) during a 4-month period. Included TBI patients sustained ICP elevation (>30mmHg x 30 minutes) despite full sedation, paralytics, temperature control and minimal stimulation. Starting at bolus initiation, ICP, cerebral perfusion pressure (CPP), heart rate (HR), systolic blood pressure (SBP), sodium level (Na), and serum osmolality (SOsm) were recorded regularly for 12 hours. If repeated boluses were given in the same patient (ICP re-elevated >30mmHg x 30 minutes), recording of parameters was restarted at a new zero time-point to assess the effect of each bolus individually. Statistical analysis included power analysis, normalization testing, ANOVA (analysis of variance) and Scheffe test.

Results:
Within 10 minutes of administration a statistically significant decrease in ICP was sustained up to 12 hours (power >90%, $p<0.05$). Mean ICP at initiation declined from 43 mmHg to <20 mmHg by 25 minutes (>50% reduction, $p<0.05$). The mean CPP before treatment increased from 58 mmHg to 81 mmHg by 20 minutes (40% rise, $p<0.05$). Mean HR and SBP remained constant. Sodium levels ranged from 134 to 175 and SOsm from 297 to 362.

Conclusions:
Small volume 14.6% saline boluses can be used repeatedly in patients with TBI to significantly lower ICP and improve cerebral perfusion. Repeated boluses resulted in a sustained magnitude and duration of ICP reduction up to 12 hours.

Financial Support: None

Poster 60

INTRAVENTRICULAR HEMORRHAGE SECONDARY TO OCCIPITAL ARTERY PSEUDOANEURYSM AFTER AN EXTERNAL VENTRICULAR DRAIN PLACEMENT. A CASE REPORT

Mona Elsayed, Panayiotis Varelas, Tamer Abdelhak
Henry Ford Hospital, Detroit, Michigan, United States

Introduction:
Intraventricular hemorrhage (IVH) can result from different etiologies all are intracranial in location. In this unique case we describe a case of IVH secondary to an extracranial vascular source.

Methods:
Retrospective chart analysis for a patient that was taken care of at our institution's NeuroCritical Care Unit with cerebellar hemorrhage.

Results:
Patient was a 55 year old male who presented with severe headache and imbalance. CT scan revealed a cerebellar hemorrhage and minimal Intraventricular hemorrhage. Patient underwent suboccipital craniectomy with evacuation of the hematoma. Patient had an external ventricular drain (EVD) placed in the OR through the occipital horn. Patient recovered over the following few days with minimal neurological deficits. Suddenly patient suffered from a profuse bleeding from the scalp site of the EVD. Bleeding was controlled by pressure and suture. Next day patient suffered from a similar episode. During the control of the bleeding the patient deteriorated neurologically and had to be intubated. Patient CT scan showed massive Intraventricular hemorrhage. Angiography revealed an occipital artery pseudoaneurysm that was the cause of the bleeding and probably resulted from the EVD insertion. The aneurysm was coiled without complication and patient was discharged later to long term care facility.

Conclusions:
Pseudoaneurysms of the external carotid artery branches could result from trauma induced during EVD insertion. In the presence of EVD tract the hemorrhage that occurs from these pseudoaneurysms could track along under pressure to cause intracranial hemorrhage. This is an unusual and unfortunate experience that we wanted to raise awareness about.

Financial Support: None

Poster 61

ENDOVASCULAR STENT-ASSISTED REPAIR OF FUSIFORM INTRACRANIAL ANEURYSM: A SINGLE CENTER EXPERIENCE

Veena Yashaswi, Ravi Patel, Adham Kamel, Jonathan Naysan, Juliuse Gene Latorre, Tara Ramachandran, Ziad El-Zammar, Yahia Lodi
Upstate Medical University, Syracuse, NY, United States

Introduction:
Surgical treatment of fusiform intracranial aneurysm is extremely difficult and associated with poor outcome. Endovascular stent-assisted treatment of fusiform intracranial aneurysm without sacrificing the parent artery has been introduced into clinical practice recently as an alternative option. Objective: The objective of our study is report our experience of stent-assisted treatment of fusiform intracranial aneurysm.

Methods:
Consecutive patients who underwent stent-assisted treatment for fusiform intracranial aneurysm were enrolled from 2004 to 2006. Patient's demographics including the Hunt & Hess grade, Fished scale, location and size of aneurysm including the rate of radiographic evidence of aneurysm occlusion were collected. Additionally a 90 days outcome measurement was obtained using Glasgow Outcome Scale (GOS).

Results:
Five female patients, median age 40 years (ranges 10 to 67) with five unruptured symptomatic intracranial fusiform aneurysms were treated with Neuroform stent. Four of which required staged coiling in addition to stenting and one required stenting only. Three aneurysms were located at the internal carotid artery (two at the carotid bifurcation, one at the origin of ophthalmic artery) one at the middle cerebral artery and one at the vertebral artery. There was no intraoperative or post operative complication related to the stent-assisted treatment. Immediate near complete occlusion was observed in one and subtotal occlusion in 4 cases. In 12 months follow-up angiography, complete occlusion of aneurysm was observed in 2 patients (vertebral artery 1 and carotid bifurcation 1), near complete occlusion in two (Carotid ophthalmic one and carotid bifurcation one) and subtotal in one (middle cerebral artery). Good outcome was observed in all 5 cases (GOS 5).

Conclusions:
Endovascular stent-assisted repair not only provides a safe alternative option for the treatment of intracranial fusiform aneurysm, but also improve progressive occlusion of aneurysm with good outcome.

Financial Support: None

Poster 62

HYPERAMMONEMIA-INDUCED BRAIN EDEMA IN THE SETTING OF OTHERWISE NORMAL LIVER FUNCTION TEST: REPORT OF TWO CASES OF DEVASTATING BUT TREATABLE CAUSES OF ADULT-ONSET IMPAIRED UREAGENESIS.

Wilson Cueva, Obi Iwuchukwu, Fernando Goldenberg, Agnieszka Ardelt, Jeffrey Frank
University of Chicago Medical Center, Chicago, IL, United States

Introduction:
Hyperammonemia is a well recognized precipitant of cerebral edema (CE) and an important cause of death in acute liver failure. However, isolated hyperammonemia can occur in patients with enzymatic deficiencies important for ureagenesis. Extreme hyperammonemia from newly diagnosed ureadysgenesis in adults has been reported, most often leading to disabled outcome or death from CE. We present the clinical, therapeutic and outcome details of two patients with newly symptomatic ureadysgenesis-induced hyperammonemia who developed profound CE and intracranial hypertension (ICH). Both are the only survivors ever reported with their degree of extreme hyperammonemia (peak 1608 and 881 mcg/dL) with normal neurological outcome.

Methods:
Case-1: A healthy 49 year-old male developed seizures followed by profound encephalopathy associated with ammonia level of 881 mcg/dL without liver failure.

Case-2: After a successful lung transplant, a 36 year-old man developed severe encephalopathy associated with ammonia level of 1608 mcg/dL without liver failure.

Results:
Ornithine transcarbamylase deficiency was discovered in case 1 and acquired glutamine synthetase deficiency was suspected in case 2. Steroids provoked symptomatology in both cases leading to severe CE and ICH. Both required intracranial pressure monitoring, cerebral perfusion pressures directed therapy, promotion of ammonia clearance (CVVHD, lactulose), catabolism limiting treatments (hypothermia, insulin administration, infection control, nourishment), protein restriction, and the use of alternative pathway therapy. Both patients fully recovered.

Conclusions:
- Hyperammonemia should be suspected in patients presenting with unexplained CE even in the absence of liver failure.

- Multidimensional contemporary neurocritical care strategies can optimize survival and improve functional outcome from this historically disabling and deadly condition.

- Extreme hyperammonemia should not deter aggressive proactive management in these patients now that we report normal neurological outcome in these unique survivors.

Financial Support: None

Poster 63

NON-ALCOHOLIC (NA) WERNICKE'S ENCEPHALOPATHY (WE) AS A CAUSE OF UNEXPLAINED COMA IN THE CONTEMPORARY MEDICAL ERA

Wilson Cueva, Obi Iwuchukwu, Fernando Goldenberg, Agnieszka Ardelt, Jeffrey Frank
University of Chicago Medical Center, Chicago, IL, United States

Introduction:
WE is a known neurological complication of thiamine deficiency. Although it usually manifests among alcoholics, NA patients with either malabsorption, poor dietary intake, severe vomiting or increased metabolic demands are prone to develop WE. We present three clinical cases in whom typical Brain MRI and pathology findings led to the diagnosis of NA-WE.

Methods:
Case-1: 65 year-old NA female became comatose after a two months history of severe vomiting secondary to a gastrointestinal disease.

Case-2: 53 year-old NA female with breast cancer became comatose in the setting of 3 weeks history of severe vomiting after chemotherapy.

Case-3: 68 year-old NA female became comatose status-post cardiac arrest of very short duration, not enough to explain the severity of the encephalopathy.

Results:
Severe alteration of the level of consciousness without focal deficits was the prominent clinical finding in all patients; nystagmus was present in Case 2. Brain MRI showed T2/FLAIR signal abnormalities in bilateral mamillary bodies, thalamus and periaqueductal area in cases 1 and 2 and an autopsy in case 3 revealed findings consistent with WE. Despite intravenous thiamine supplementation, cases 1 and 3 did not improve clinically and eventually expired. Case 2 had complete neurological recovery within the first 48 hours of treatment with intravenous thiamine.

Conclusions:
- WE should be considered in all patients with unexplained confusion or deteriorating mental status even in the absence of a prior history of alcohol abuse.

- In the presence of atypical or incomplete clinical picture of WE, appropriate Brain MRI findings can help establishing the diagnosis.

- Early diagnosis of WE is critical given that the success of the treatment depends on the urgent thiamine supplementation.

- Failure to recognize and treat WE may result in devastating neurological outcome.

Financial Support: None

Poster 64

EXTRACORPEAL CARBON DIOXIDE REMOVAL TO CONTROL pH AND PaCO2 IN NEUROTRAUMA

Paul McCarthy, Shashikant Patil, Laurie Grier, L Keith Scott
Louisiana State University Health Sciences Center, Shreveport, LA, United States

Introduction:

Arterial Venous Carbon Dioxide Removal (AVCO2R) is a technique that uses a pumpless extracorpeal circuit for carbon dioxide removal. AVCO2R has been used in adult and pediatric patients with severe hypercapnea. The system is placed at bedside using the Seldinger technique to cannulate the femoral vessels. Normally this system requires anticoagulation but can be performed without anticoagulation.

Methods:

Case reports of AVCO2R used to control pH and PCO2 after neurotrauma.

Results:

Case 1

A male suffered extensive head injury after MVC progressing to brain death. Organ harvest was planned. Because of extensive lung injury complicated by hypercapnea and academia, donor viability was in jepody with PaCO2 rising to 88 and pH of 7.18.

AVCO2R was placed for pH and carbon dioxide control. The PaCO2 and pH promptly corrected to 42 and 7.38. After a brain death exanimation he remained on AVCO2R until organ harvest.

Case 2

A male presented with a cervical injury at the C-5 level. Imagining showed cervical fractures, disc herniation and cord contusion involving C-4 to T-1.The patient developed respiratory distress. Chest X-ray revealed ARDS. In APRV the pH was 6.8 and PCO2 of 125. The patient had a subsequent cardiopulmonary arrest. After successful resuscitation AVCO2R was started. Within 3 hours the pH and PCO2 were 7.21 and 57 without manipulation of the ventilator settings. He was on ACO2R for 6 days and eventually weaned off mechanical ventilation after surgical decompression and fusion and discharged to rehabilitation.

Neither case required anticoagulation.

Conclusions:

1. AVCO2R is a simple extracorpeal technique that can be used to manage life threatening hypercapnea in patients with critical neurologic illness or injury
2. The technique can be inserted at bedside and used without anticoagulation

Financial Support: None

Poster 65

EARLY INTRACRANIAL PRESSURE CHANGES DURING CONTINUOUS RENAL REPLACEMENT THERAPY IN PATIENTS WITH ACUTE BRAIN INJURY

Jeff Fletcher MD, Karen Bergman RN, Glenn Carlson MSN, CCRN
[1]Bronson Methodist Hospital / Michigan State University, Kalamazoo, MI, United States, [2]Michigan State University, Kalamazoo, MI, United States

Introduction:
Continuous renal replacement therapy (CRRT) is preferred over intermittent hemodialysis (IHD) in patients with acute brain injury (ABI) due to increased intracranial pressure (ICP) seen during IHD [1,2]. Despite the preference for CRRT in this patient population limited data is available on ICP changes during therapy. There is some support for the early stability of ICP for patients with fulminant hepatic failure that underwent continuous arteriovenous hemofiltration [3,4].

Methods:
Retrospective observational study (over a 3 year period) of patients with ABI and ICP monitoring whom also underwent CRRT. ICP and fluid volumes were analyzed for the 12 hours before and after initiation of CRRT.

Results:
Four patients met criteria. Table-1 describes the sample population. Three patients had developed refractory intracranial hypertension (RIH) prior to initiation of CRRT (2 in pharmacologic coma) and 1 patient developed intracranial hypertension on IHD that resolved with CRRT. No changes in medications were made in the 12 hours prior to starting CRRT except pentobarbital coma was initiated one patient six hours prior to CRRT without lowering of ICP. No attempts were made to lower ICP in the 12 hours following CRRT. A decline in ICP was seen at 1, 4, and 12 hours following initiation of CRRT in RIH patients (Table-2)

Patient	Age / sex	Diagnosis / (GCS)	Indication	ICP management	Mode / BFR / TEF rate / Anticoagulant / Vascular access / filter changes during therapy	Discharge / LOS
1	28 / M	Severe TBI (6) Polytrauma MOF ARDS	RIH / Hypervolemia	EVD, sedation, Osmotic therapy, hypothermia (33deg), 23.4% saline, pharmacologic coma	CVVH / 80-180cc/min /20-25cc/kg/hr / Heparin / Femoral vascular catheter / 1	LTCF / 39
2	30 / M	Severe TBI (3) Polytrauma MSOF ARDS	Hypervolemia (RIH present)	Sedation, osmotic therapy, paralytics, mild hypothermia	SCUF / 150cc.min / 150-400cc/hour / Heparin / Femoral vascular catheter / 0	LTCF / 34
3	51 / F	SAH (Treated with coil embolization)	RIH	EVD, sedation, Osmotic therapy, hypothermia (33deg), 23.4% saline, pentobarbital coma	CVVH / 150cc/min / 20cc/kg/hr / Heparin / Femoral vascular catheter / 0	Death / 4
4	63 / M	SDH (10) Status epilepticus	CRF with increased ICP during IHD	EVD, mild sedation	CVVHD /250cc/min / 20cc/kg/hr / Sodium Citrate / Femoral vascular catheter / 0	Hospice / 18

GCS = Glasgow Coma Scale Score; RIH = Refractory intracranial hypertension defined as failure of first tier therapy as defined but the brain trauma foundation; BFR = blood flow rate; TEF = total effluent flow rate; CVVH = hemofiltration; SCUF = slow continuous ultrafiltration; CVVHDF = Hemodialfiltration; IHD = intermittent hemodialysis; ICP = intracranial pressure management ***Prior to*** CRRT; EVD = external ventricular drain; MSOF = multi-system organ failure; acute respiratory distress syndrome; LOS = length of stay; LTCF= long term care facility; TBI = traumatic brain in jury; SAH = subarachnoid hemorrhage

Patient	12 hours prior (mmHg) / P value	4 hours after (mmHg)	12 hours after (mmHg)	Change 1 hour (mmHg)	Change Volume 1 hr (L)	Change volume 12 hr (L)	% FB 12 hr
1	35.5	24.3	21.3	-9	-0.4	- 4.1	-16%
2	23.4	11.5	13.8	-13	-0.16	-2.56	-6%
3	36.7	32.5	29.8	-17	-0.02	-0.62	-41%
4	13.1	14.3	15.0	+3	+0.57	0	0%
RIH	31.9	22.8	21.6	-13	-0.19	-2.43	-8%
All	27.2	20.7	20	-9	-0.01	-1.83	-10%

L = liters; hr = hours

Conclusions:
Given the decrease in ICP at one hour and relatively small percentage of total fluid balance removed, it seems unlikely that fluid removal or improved systemic oxygenation decreased ICP. Early improvement in ICP may be due to removal of cytokines and myocardinal depressants seen with ultrafiltration and membrane absorption which is maximal during the first hour of filter use due to filter charge [5]. Given the mortality and morbidity associated with RIH, further research is warranted.

References:

1. Davenport A. Renal replacement therapy in the patient with acute brain injury. Am J Kidney Dis 2001;31(3)457-66.
2. Davenport A. Renal replacement therapy for the patient with acute traumatic brain injury and severe acute kidney injury. Contrib Nephrol 2007;156:333-9.
3. Davenport A, Will EJ, Davison AM. Early changes in intracranial pressure during haemofiltration treatment in patients with grade 4 hepatic encephalopathy and acute oliguric renal failure. Nephrol Dial Transplant 1990;5(3):192-8.
4. Davenport A, Will EJ, Losowski MS, Swindells S. Continuous arteriovenous hemofiltration in patients with hepatic encephalopathy and renal failure. Br Med J 1987;295:10985.
5. De Vriese AS, Colardyn FA, Philippé JJ, Vanholder RC, De Sutter JH, Lameire NH: Cytokine removal during continuous hemofiltration in septic patients. J Am Soc Nephrol;(10): 846-53

Financial Support: None

Poster 66

PROLONGED HIGH DOSE ISOFLURANE FOR REFRACTORY STATUS EPILEPTICUS: A POTENTIAL CAUSE OF MRI SIGNAL ABNORMALITIES

Jennifer Fugate, Joseph Burns, Eelco Wijdicks, Alejandro Rabinstein
Mayo Clinic, Rochester, MN, United States

Introduction:
Isoflurane, an inhalational anesthetic, is an alternative treatment for refractory status epilepticus (RSE). It is effective, has rapid onset of action, and is easily titrated to produce burst-suppression patterns on the electroencephalogram. Little is known regarding potential human toxicities caused by isoflurane. We present two cases of prolonged RSE treated with prolonged high dose isoflurane who developed abnormal T2 hyperintensity lesions on magnetic resonance imaging (MRI), which improved after taper or discontinuation of isoflurane.

Methods:
We report two patients with prolonged refractory status epilepticus who were treated with prolonged high-dose isoflurane (defined as an average end tidal concentration > 1.5% for seven or more days) and developed new changes on brain MRI. We collected demographic information, daily dosing of all antiepileptic medications and anesthetics received. We reviewed and analyzed the results of serial MRI scans.

Results:
Patient one had prolonged RSE for 140 days and was treated with isoflurane for 85 days with 1975.2 % concentration-hours. Patient two, currently hospitalized, has had RSE for at least 210 days and was treated with isoflurane for 34 days with 1382.4 % concentration-hours. In both patients, serial brain MRIs showed progressive T2 signal hyperintensity involving bilateral thalami, cerebellar hemispheres, and cerebellar vermis after treatment with 2-3 weeks of high dose isoflurane. These findings improved following taper and/or discontinuation of isoflurane.

Conclusions:
These cases raise the possibility that isoflurane is neurotoxic when used in high doses for prolonged time periods. Though we cannot be certain of the exact cause of brain lesions, the timing of their appearance after isoflurane initiation and subsequent improvement after taper or discontinuation suggest a possible association with isoflurane. Further studies are needed to clarify the safety of prolonged isoflurane use in RSE

Financial Support: None

Poster 67

A BOLUS OF CONIVAPTAN LOWERS INTRACRANIAL PRESSURE IN A PATIENT WITH HYPONATREMIA AFTER TRAUMATIC BRAIN INJURY

Theresa Human, Rajat Dhar
[1]*Barnes-Jewish Hospital, Saint Louis, Missouri, United States,* [2]*Washington University School of Medicine, Saint Louis, Missorui, United States*

Introduction:
Cerebral edema is common in severe brain injury and can lead to harmful elevations in intracranial pressure (ICP). Hyponatremia, typically associated with excess levels of vasopressin (ADH), frequently complicates acute brain injury and can worsen edema and ICP. Conivaptan, a vasopressin-receptor antagonist, has been shown to correct hyponatremia in these high-risk patients by inducing loss of free water (aquaresis). It is unknown whether raising sodium with a bolus of conivaptan can also acutely reduce ICP.

Methods:
We prospectively assessed the change in serum sodium (Na^+), ICP, and cerebral perfusion pressure (CPP) after a bolus of conivaptan was given for the treatment of hyponatremia in a patient with cerebral edema associated with traumatic brain injury (TBI).

Results:
A 22-year old suffered severe TBI with left carotid dissection, complicated by hemispheric infarcts and worsening edema. Conivaptan 20 mg IV was given as a bolus when Na^+ rapidly dropped to 128mEq/L. Its aquaretic effect peaked between 3 and 5 hours after the dose, with hourly urine outputs of 1100 ml/hour. Eight hours post-administration, Na^+ had risen to 146 mEq/L. ICP had been stable at 13-15 mm Hg for several hours prior and remained in this range for the first 2 hours after conivaptan, but then fell to 3 mm Hg at 4 hours, remaining 5 mm Hg after 8 hours. CPP, initially stable at 60-70 mm Hg, rose to 94 mm Hg after 4 hours.

Conclusions:
A single bolus of conivaptan not only resulted in rapid correction of hyponatremia but also a significant fall in ICP temporally associated with peak aquaresis. Confirmation of this novel osmotic effect is required, as is further delineation of the role of such agents in the management of brain edema.

Financial Support: The authors have received speaking honoraria from Astellas Pharma.

Poster 68

SEIZURE-INDUCED EXACERBATION OF DELAYED CEREBRAL ISCHEMIA

Tomoko Sampson, Rajat Dhar, Gregory Zipfel
Washington University School of Medicine, Saint Louis, Missouri, United States

Introduction:
Seizures are a known complication of aneurysmal subarachnoid hemorrhage (SAH). They can increase cerebral metabolic demand and lead to cardiopulmonary compromise. This could be detrimental in the setting of delayed cerebral ischemia (DCI), when brain tissue is vulnerable to further reductions in oxygen delivery or increases in demand. An association between seizures and worsening ischemia could influence the decision to use antiepileptic drug (AED) prophylaxis in patients with vasospasm.

Methods:
Case report of a patient who developed irreversible neurological deficits and cerebral infarction immediately after a seizure in the setting of initially stable vasospasm with DCI.

Results:
A 64 year-old woman developed confusion, aphasia, and right hemiparesis on day 7 after SAH. Angiography confirmed severe anterior circulation vasospasm. The patient responded to hypertensive therapy with almost complete resolution of her ischemic neurological deficits. On day 10, however, she had a single generalized seizure and required intubation after brief oxygen desaturation. She had a concurrent drop in blood pressure, necessitating an increase in previously stable dose of vasopressors Post-ictally she developed recurrent aphasia and worsening hemiparesis which did not resolve despite further hemodynamic augmentation. Subsequent head CTs revealed new infarcts in the left anterior and middle cerebral artery territories. She had received prophylactic phenytoin for only the first 3 days of her ICU stay per our SAH protocol.

Conclusions:
AED prophylaxis is typically used early after SAH when risk is high and a seizure may precipitate aneurysmal rebleeding. This case illustrates how a seizure occurring later, in the setting of vasospasm, can lead to decompensation of DCI with potential for irreversible infarction. Therefore, patients with vasospasm may benefit from extended duration of prophylaxis to prevent such complications.

Financial Support: None

Poster 69

TRANS-JUGULAR APPROACH FOR DURAL SINUS THROMBECTOMY: A VALUABLE ALTERNATIVE APPROACH

Matthew Smith[1], Mohammed Zaman[1], Glen Pollock[1], David Decker[1], Karen Wilson[2], Eric Sauvageau[1], Reza Behrouz[1], James Lefler[2]
[1]University of South Florida College of Medicine, Tampa, FL, United States, [2]Tampa General Hospital, Tampa, FL, United States

Introduction:
Dural sinus thrombosis is a rare cause of stroke. Anticoagulation is the preferred treatment; however, some patients experience rapidly progressive neurological deficits and poor outcomes despite adequate anticoagulation. Mechanical thrombectomy via a trans-femoral approach is an effective alternative treatment, but technical limitations can make this approach impossible in some patients.

Methods:
We report two cases in which Angiojet® (Medrad-Interventional/Possis) thrombectomy was performed via a trans-jugular approach. In the first patient, trans-jugular access was preferred due to the presence of bilateral deep vein thromboses in the femoral and iliac veins and an inferior vena cava filter. In the second patient, the trans-femoral approach was impossible due to the length of the catheter (135cm XMI), which was insufficient to access to the anterior two thirds of the superior sagittal sinus via a trans-femoral approach. In both patients, the trans-jugular access was obtained utilizing direct ultrasound and fluoroscopic guidance. A stabilizing guidewire was placed to deliver the Angiojet® catheter to the superior sagittal sinus.

Results:
The first patient was a 67 year-old woman with heparin-induced thrombocytopenia, a large intracranial hemorrhage and refractory increased intracranial pressure. The second patient was a 32 year-old man who presented with dehydration and a rapidly declining neurological exam. In both patients, antegrade blood flow was restored within the dural sinuses after mechanical thrombectomy via the trans-jugular approach. Computed tomography scans after thrombectomy did not show evidence of increased hemorrhage and there were no complications from the procedure. The first patient died despite successful thrombectomy from other complications of her underlying disease. The second patient made a full recovery.

Conclusions:
Mechanical thrombectomy has been shown to be a successful treatment for dural sinus thrombosis for patients with progressive symptoms despite adequate anticoagulation. The trans-jugular approach is a valuable alternative variation of mechanical thrombectomy in patients who have contraindications to the trans-femoral approach.

Financial Support: None

Poster 70

EFFECT OF INHALED NITRIC OXIDE ON CEREBRAL OXYGENATION IN SEVERE TBI: A SINGLE CENTER EXPERIENCE

Jordan Bonomo, Erin Grise, William Knight, Rhonda Cadena, Krishna Mohan, Opeolu Adeoye, James Hamilton, Lori Shutter
University of Cincinnati, Cincinnati, OH, United States

Introduction:
We retrospectively reviewed data from patients with sTBI admitted to the Neuroscience ICU (NSICU) of an urban tertiary care level 1 trauma center who had a cerebral oxygen monitor (Licox®) in place and were administered inhaled nitric oxide (iNO) per institutional protocol. Data were collected from bedside flow sheets.

Methods:
Two patients met inclusion criteria. Patient #1 was admitted after a motor vehicle collision with sTBI and pulmonary contusions. She developed adult respiratory distress syndrome (ARDS) on hospital day #3 requiring iNO at 20 ppm. Prior to iNO therapy, PbtO2 was 36.8 mm Hg, PaO2 = 63 mm Hg and ICP = 8 cm H2O; within 3 hours of iNO initiation, PbtO2 climbed to 76 mm Hg (+106%), PaO2 rose to 266 (+322%), and ICP remained 8 cm H2O (+0%). Patient #2 sustained a sTBI and pulmonary contusions after a motorcycle collision. He developed ARDS on hospital day #3 requiring iNO at 20 ppm. Prior to iNO therapy PbtO2 was 19.3 mm Hg, PaO2 = 67 mm Hg and ICP = 9 cm H20; within 3 hours of iNO initiation, PbtO2 rose to 43mm Hg (+122%), PaO2 rose to 181 mm Hg (+170%) and ICP climbed to 18 cm H2O (+200%).

Results:
Patients admitted to the NSICU after sTBI may develop complex physiologic derangements, including ARDS. The use of iNO may benefit both cerebral and pulmonary dysfunction, and may warrant further investigation.

Financial Support: None

Poster 71

CARDIAC RUPTURE AFTER INTRAVENOUS t-PA ADMINISTRATION IN ACUTE ISCHEMIC STROKE

Amar Dhand, Kazuma Nakagawa, Wade Smith, Tarik Tihan
University of California, San Francisco, San Francisco, CA, United States

Introduction:
Ventricular free wall rupture is a fatal complication of myocardial infarction (MI). Although described in MI patients who receive thrombolytic therapy, this complication is not well known in ischemic stroke patients who receive intravenous (IV) t-PA. Here, we report a patient who had cardiac rupture and hemopericardium immediately following IV t-PA administration.

Methods:
Case report.

Results:
A 93-year-old woman with history of coronary artery disease presented with acute onset of left hemiparesis and right gaze preference (NIHSS 13). CT angiography showed right middle cerebral artery (MCA) occlusion at the bifurication, a filling defect in the left atrial appendage suggestive of left atrial thrombus, and right segmental pulmonary embolism. An electrocardiogram showed ST elevations in the V5-V6 distribution with initial troponin I level of 8.08 ug/L. IV t-PA was administered 1 hour from symptom onset. One hour after completing t-PA infusion, the patient suddenly became unresponsive, bradycardiac, and rapidly demonstrated an asystolic arrest. Given the established DNR/DNI status, she was not resuscitated. Autopsy study showed subacute myocardial infarction (2-4 days old), rupture of the anterolateral wall of the left ventricle, and 200cc of hemopericardium. Pathological study of the brain showed an old hemorrhagic infarction in the left occipital lobe, evidence of remote hypoxic/ischemic injury and 30% occlusion of the basilar artery, but no evidence of intracranial hemorrhage.

Conclusions:
This case report illustrates a fatal cardiac complication of IV thrombolytic therapy that was used for acute ischemic stroke treatment in the setting of subacute myocardial infarction. The speculated mechanism of this phenomenon is alteration of collagen metabolism by thrombolytic therapy. Although MI is not an absolute contraindication for IV t-PA administration, clinicians should be aware that cardiac rupture may occur when IV t-PA is given to patients with concomitant stroke and MI.

Financial Support: None

Poster 72

NMDA-RECEPTOR ENCEPHALITIS: A SINGLE CENTER NICU EXPERIENCE

Valerie Dechant, William McBride, Diana Tzeng, Michael Moussouttas, Carissa Pineda, Michael Moussouttas, Rodney Bell, Monisha Kumar
Thomas Jefferson University Hospital, Philadelphia, PA, United States

Introduction:
Anti-NMDA-receptor encephalitis (NMDARE) is a rare autoimmune encephalitis associated with antibodies that antagonize NMDA receptors. Although NMDARE is an uncommon disorder, we present 3 confirmed cases treated in our Neuro-ICU over 6 months.

Methods:
We report three cases of NMDARE including the first reported case during pregnancy.

Results:
All patients were women between 19 and 39 years of age. All had a prodrome of psychiatric symptoms and had orofacial and limb dyskinesias at presentation. Each developed progressive unresponsiveness, required mechanical ventilation secondary to hypoventilation and had autonomic instability. One required a transvenous pacer for symptomatic bradycardia. All were evaluated for ovarian teratoma. All underwent oopherectomy. Two were found to have ovarian teratomas by pathology. Patients were treated with a combination of IVIG, steroids and plasma exchange. All patients improved and were discharged from the hospital to inpatient rehabilitation. One patient was 14 weeks pregnant at admission. She delivered via Cesearean section at 37 weeks gestation. The neonate had mildly increased tone but appeared otherwise healthy and was discharged home at 9 days. The CSF of the neonate was negative for NMDA antibodies.

Conclusions:
NMDARE is a reportedly rare cause of encephalitis which may be more common than reported. It has characteristic features that should not go unrecognized. High suspicion for ovarian teratoma is appropriate. Imaging may not accurately differentiate between benign cysts and teratoma. Oopherectomy for any ovarian abnormality may be reasonable, given poor correlation of pathologically confirmed teratoma with radiographic imaging. This is the first reported case of NMDARE during pregnancy. NMDA receptors play an important role during fetal development and the long-term sequelae for children exposed to NMDAR antibodies in utero are unknown.

Financial Support: None

Poster 73
COMA FROM CSF HYPOVOLEMIA: AN UNRECOGNIZED CAUSE
Amandeep Dhillon, Jasvinder Nangiana, Rabinstein Alejandro, Wijidicks EElco
Mayo clinic, Rochester MN, United States

Introduction:
CSF hypovolemia is typically diagnosed in patients presenting with positional headaches. However, severe intracranial hypotension and brain sagging may cause orthostatic coma. We present a case that illustrates this uncommon presentation.

Methods:
Case report

Results:
A 50yr old male who presented with acute onset of headache, nausea, vomiting and disequilibrium. Extensive diagnostic work-up, including head CT, MRI/MRA and LP, was initially unremarkable. His headaches became progressively worse with a prominent postural component. A CSF leak was suspected at the lumbar level diagnosed with CT myelogram and treated with a blood patch at the outside hospital with temporary resolution of his symptoms. A repeat MRI/MRA revealed bilateral subdural hematomas without mass effect and diffuse dural enhancement, consistent with decreased CSF pressure. Due to worsening level of consciousness, the patient underwent urgent evacuation of the left subdural without any change in his mental status subsequent imaging showed reaccumulation of the hematoma. Over the next few days, the patient became increasingly stuporous and had an acute respiratory decline requiring intubation.
The patient was subsequently transferred to our institution. Head MRI revealed dramatic sagging of the brain showing the pontomedullary junction at the opening of the foramen magnum. There was reproducible improvement in his cognitive status and Cheyne stokes breathing with Trendelenburg positioning. He underwent a repeat CT myelogram which showed a CSF leak at T3 and possibly at C5-C6. The CSF leaks were repaired with localized blood patches with significant improvement in his neurological exam.

Conclusions:
CSF hypovolemia may cause coma from distortion and downward displacement of the thalamus-brainstem structures. Evacuation of subdural fluid collections- typically without mass effect- may be detrimental in these patients contributing to further reduction of CSF volume. Instead, identification and treatment of the responsible CSF leak is curative.

Financial Support: None

Poster 74

RAPID ANEURYSMAL ENLARGEMENT IN A PATIENT WITH HIV VASCULOPATHY RESULTING IN RECURRENT SUBARACHNOID HEMORRHAGE

Nancy Edwards, Kazuma Nakagawa, Wade Smith
University of California at San Francisco, San Francisco, CA, United States

Introduction:
Infection with human immunodeficiency virus (HIV) has been associated with the development of intracranial aneurysms. Although the pathogenesis of aneurysm formation in HIV infected patients is unclear, one purported mechanism is direct invasion of cerebral vessels by the virus itself. Here, we report a unique case of an HIV infected patient whose intracranial aneurysm rapidly enlarged during a period of anti-retroviral therapy non-adherence.

Methods:
Case report.

Results:
A 39 year-old HIV infected female (CD4 count 181 cells/mL) was admitted with a subarachnoid hemorrhage. Cerebral angiography revealed a dilating vasculopathy of multiple large intracranial vessels along with fusiform aneurysms of the right and left proximal anterior cerebral arteries (ACA). A saccular aneurysm measuring 4 x 2.5 mm arose from the right fusiform ACA aneurysm and was successfully treated with endovascular coiling. Four weeks later, the patient was re-admitted with a decreased level of consciousness. Head CT revealed recurrent subarachnoid hemorrhage. The patient had not adhered to her anti-retroviral therapy and her CD4 count upon re-admission was 108 cells/mL (HIV load 671 copies/mL). Cerebral angiography revealed enlargement of the previously coiled aneurysm which now measured 6 x 5 x 3.5mm. Infectious vasculitides were excluded with serum and cerebrospinal fluid (CSF) testing, including negative blood and CSF cultures, negative serum and CSF antibodies for syphilis, and negative serum antibodies and CSF polymerase chain reaction for varicella zoster virus. The co-occurrence of rapid aneurysmal enlargement with non-adherence to antiretroviral therapy suggests an elevated HIV burden may accelerate vasculopathy.

Conclusions:
Rapid enlargement and re-rupture of intracranial aneurysms may be seen in HIV infected patients with an elevated viral burden. Identifying the unique clinical and radiological features of HIV vasculopathy may lead to earlier recognition and novel therapeutic approaches.

Financial Support: None

Poster 75

NEW ONSET REFRACTORY STATUS EPILEPTICUS (NORSE) ASSOCIATED WITH CREUTZFELDT-JAKOB DISEASE

Abhijit Lele, Michael McNearney, Manoj Mittal
University of Kansas Medical Center, Kansas City, KS, United States

Introduction:
Patients with de novo refractory status epilepticus are often referred to as having NORSE. The clinical course is often prolonged (range: 6-68 days), and morbidity and mortality is high (50%). CJD is a rare cause of refractory convulsive and non-convulsive status epilepticus. We describe here a patient with NORSE who had probable sporadic CJD.

Methods:
72 year old, 65 kg, Caucasian female with a past medical history of systemic hypertension, pulmonary hypertension, and prior pulmonary MAI complex presented to the ER with delirium and accelerated hypertension. Initial examination revealed encephalopathy. Patient had 3 generalized tonic clonic seizures, with rapid progression to status epilepticus which was refractory to dilantin (150 mg tid), keppra (1000mg bid), phenobarbitone (60 mg tid), midazolam (6mcg/kg/min), and propofol (75mcg/kg/min) infusion. She was then put in pentobarbital coma (6 m/kg/hr) for 48 hours, and was found to be refractory to withdrawal of pentobarbital. On day 19 of her status epilepticus the patient had an episode of massive pulmonary hemorrhage and went into PEA, from which she couldn't be revived.

Results:
Workup for NORSE including MRI brain, CT chest, abdomen and pelvis, failed to reveal any evidence of stroke, PRESS, neoplasm, meningo-encephalitis. Paraneoplastic antibody panel was negative. Toxicology, metabolic, haematological, vascular, and immunological workup was negative. CSF analysis revealed a WBC of 16, protein 51 and Glucose 68 and protein 14-3-3 was positive.

Conclusions:
To our knowledge this is the first case report of NORSE complicating CJD. NORSE complicating CJD is associated with high mortality. Our case is also unique for its acute onset, absence of myoclonus, and absence of extra pyramidal features commonly seen in CJD. In patients with refractory status epilepticus with no obvious cause, CJD should be considered in the differential diagnoses.

Financial Support: None

Poster 76

ONLY TIME WILL TELL: A CAUTIONARY TALE OF NEURON-SPECIFIC ENOLASE PREDICTING POOR OUTCOME AFTER CARDIAC ARREST

Anna Allred, Wengui Yu, Joanna Rives, Christiana Hall
UT Southwestern Medical Center, Dallas, TX, United States

Introduction:
Predicting recovery after cardiac arrest continues to challenge neurointensivists. Updated 2006 AAN practice parameters add two new evidence-based elements to traditional clinical examination criteria 1) absence of bilateral N20 response on SSEPs and 2) Neuron-specific enolase (NSE) > 33 µg/dl as measured within 3 days. Concurrently moderate hypothermia has emerged as an efficacious therapy, with the possibility of modifying the predictive power of criteria established independent of such intervention.

Methods:
Case: a 71 year old woman undergoing breast lumpectomy was resuscitated following interoperative asystole. ROSC was secured by 5 mins; but she arrived to NSICU comatose with only minimal pupillary and corneal reflexes. Moderate hypothermia with target temperatures of 32-34^{o}C was achieved by 2 hours and maintained for 24 hours before slow re-warming. At 48 hours, N20s were bilaterally present, but NSE was 35 µg/dl. Brain MRI at day 7 was normal. Over 5 weeks she remained comatose with absent motor response requiring aggressive therapy for bouts of refractory non-convulsive status epilepticus. She continued to have intermittent transient myoclonic movements 4 months after cardiac arrest.

Results:
Eye opening without awareness of surroundings began at ICU week 5 with gradual return to consciousness. Subsequently she has made slow steady improvement, conversing appropriately with memory of family names and past experiences. Now 4 months post-arrest on the inpatient rehabilitation unit she moves all extremities with 3-4/5 muscle strength. Her most recent FIM score is 57.

Conclusions:
Prognostication after cardiac arrest remains complex. Application of hypothermia may alter the validity of predictors established previously. Confounders and convergent evidence must be considered over any single data point. As in the past, time remains the final arbiter of certainty.

Financial Support: None

Poster 77

CEREBRAL OXIMETRY VIA NEAR INFRARED SPECTROSCOPY MIRRORS DESATURATIONS OF BRAIN TISSUE PARTIAL PRESSURE OF OXYGEN: A CASE STUDY

Elissa Wible, Carmelo Graffagnino, DaiWai Olson, Michael James
Duke University, Durham, NC, United States

Introduction:
Near infrared spectroscopy is a non-invasive method of monitoring cerebral oxygenation. By employing time and spatial resolution of several light wavelengths, cortical blood flow, volume and oxygenation can be quantified (cerebral oximetry). We present a case utilizing cerebral oximetry in a patient with cerebral vasospasm after subarachnoid hemorrhage (SAH) with concurrent use of brain tissue partial pressure of oxygen (PbtO2) monitoring.

Methods:
A 46 year old woman developed severe diffuse vasospasm following SAH. We monitored intracranial pressure (ICP) as well as tissue oximetry ($PbtO_2$) via Licox (Integra) catheter placed in the distribution of the left MCA. Over a 24-hour period during the third day of vasospasm, CerOx (Ornim) monitoring was applied over the left fronto-temporal area to evaluate the relationship between $PbtO_2$ and non-invasive cerebral oximetry.

Results:
There were 12 episodes of $PbtO_2$ desaturation (<20 mmHg for >5 mins) over the period of dual monitoring. Over 80% of these $PbtO_2$ desaturations were preceded by >20% decline in CerOx values from baseline. There were 22 episodes of cerebral oximetry desaturation (<50% for >10 mins). Less than 50% of CerOx desaturations were temporally related to a decline in PbtO2 to less than 20 mmHg. Hemoglobin was stable at 10 mg/dL and ICP was well controlled (<15 mmHg) during the entire 24 hours.

Conclusions:
In this subject, desaturations of $PbtO_2$ appeared to be related to desaturations by non-invasive cerebral oximetry; the converse was not the case. Perhaps cortical oxygen desaturations (CerOx) occur with increased frequency compared to subcortical oxygen desaturations ($PbtO_2$) in diffuse vasospasm after SAH. CerOx monitoring may provide an enhanced understanding of oxygen delivery and utilization during periods of ongoing cerebral ischemia. Further studies are required to substantiate these findings.

Financial Support: None

Poster 78

ELECTROCONVULSIVE THERAPY FOR REFRACTORY STATUS EPILEPTICUS: A CASE SERIES

Hooman Kamel, Susannah Brock Cornes, Manu Hegde, Stephen Hall, S. Andrew Josephson
University of California, San Francisco, San Francisco, CA, United States

Introduction:
Status epilepticus refractory to conventional anti-epileptic drugs typically carries a poor prognosis, but patients may recover well if seizures can be stopped. Case reports suggest that electroconvulsive therapy (ECT) may stop seizures in patients with refractory status epilepticus, and we sought to examine its effectiveness in a series of patients.

Methods:
Three consecutive patients with refractory status epilepticus at our institution were treated with ECT after other therapies had failed. All 3 patients were women, with age ranging from 26 to 41 years, and none had a significant medical history. Extensive diagnostic testing was unrevealing, and all patients were empirically treated for infectious and autoimmune encephalitis. ECT was begun because of ongoing seizures despite potent combinations of conventional anti-epileptic drugs, multiple trials of complete EEG suppression with anesthetic agents, and trials of more infrequently used therapies such as inhaled anesthetic agents and ketamine.

Results:
ECT stopped seizures in 2 of 3 patients. One patient recovered completely, and in outpatient follow-up had a normal neurological examination and a score of 30 on the Mini Mental State Examination. The second patient was left with mild cognitive impairment and epilepsy, but returned to independent living. In the third patient, seizures continued despite ECT, and care was withdrawn at the family's request. Autopsy revealed evidence of active meningoencephalitis despite treatment with antiviral therapy and high-dose steroids.

Conclusions:
ECT stopped seizures in 2 of 3 patients with refractory status epilepticus. Our results and those of prior case reports suggest that ECT may be an effective therapy for refractory status epilepticus, and warrants further study for this indication.

Financial Support: None

Poster 79

CONIVAPTAN TO INDUCE THERAPEUTIC HYPERNATREMIA IN THE NEUROCRITICAL CARE UNIT

Wendy Wright, Bill Asbury, Susan Samuel, Jane Gilmore, Owen Samuels
Emory University Hospital, Atlanta, GA, United States

Introduction:
Conivaptan, an AVP-receptor antagonist, has been used in neurocritical care patients to treat euvolemic hyponatremia. Therefore, it would stand to reason that the aquaretic effect of conivaptan could also be used to induce a state of therapeutic hypernatremia. Therapeutic hypernatremia is one of the standard modalities for the treatment of cerebral edema.

Methods:
Conivaptan bolus +/- continuous infusion was administered to three patients with cerebral edema in the neurocritical care unit. All patients were initially treated with conventional measures to induce therapeutic hypernatremia, yet were not meeting the desired serum Na goal. Conivaptan was used in these patients to augment the effects of hypertonic saline.

Results:
One patient received a single 20 mg bolus of conivaptan in addition to 23.4% NaCl and his [Na] increased an average of 3 meq/L. One patient received a single 20 mg bolus in addition to 23.4% NaCl + 3% NaCl + NaCl tablets + fludrocortisone and his [Na] increased an average of 6meq/L. A third patient received conivaptan boluses + infusion in addition to 3% NaCl and his [Na] increased an average of 3 meq/L. Fluid balances were not adversely affected in any of these patients.

Conclusions:
Conivaptan added to hypertonic saline therapy appears to be a rational strategy for achieving therapeutic hypernatremia in patients with cerebral edema without adversely affecting fluid balance. Further study is needed to assess the effects of conivaptan on intracranial pressure, cerebral perfusion pressure and intravascular volume.

Financial Support: Dr. Wright has served as a consultant for Astellas Pharma US

Poster 80

DELAYED CEREBRAL ISCHEMIA IN MENINGITIS: CASES AND REVIEW

Valerie Coon, Elaine Skalabrin
University of Utah, Salt Lake City, UT, United States

Introduction:
Delayed cerebral ischemia from vasospasm is an under-recognized, yet potentially treatable cause of morbidity and mortality in meningitis. While cerebral vasospasm has been documented via transcranial Doppler sonography in patients with meningitis, few reports document vasospasm by cerebral angiography in this population.

Methods:
We report two patients who suffered neurological decline resulting from angiographically documented vasospasm during treatment for meningitis.

Results:
The first patient was a 51-year-old woman who developed acute aphasia and hemiplegia during treatment for meningitis. Formal cerebral angiography demonstrated left anterior circulation vasospasm. She was treated with verapamil into the left internal carotid artery and aggressive hypervolemia and hypertension. Within 12 hours, she was neurologically normal. The second patient was a postpartum woman with meningitis who presented with aphasia and hemiplegia. Magnetic resonance imaging showed areas of diffusion restriction consistent with her examination. Although she initially made clinical improvement with antibiotic therapy and was discharged, she re-presented days later with severe left anterior circulation vasospasm and massive left hemisphere stroke and later died. In our cases, as well as those described by the TCD literature, neurological decline and vasospasm occurred within 12 days from the diagnosis of meningitis. This suggests that the “window” for vasospasm secondary to meningitis may be similar to that of vasospasm from SAH.

Conclusions:
The development of focal neurologic symptoms in patients with meningitis should prompt radiographic evaluation for vasospasm. Current treatment algorithms do not include the routine use of cerebrovascular imaging during treatment for meningitis, and thus this potentially treatable complication may be under diagnosed. Prospective studies evaluating cerebrovascular complications in acute meningitis using neuroimaging coupled with directed hypervolemic–hypertensive therapies should be undertaken and may lead to a reduction in the persistently high morbidity and mortality associated with this common disease.

Financial Support: None

Poster 81

OPSOCLONUS-MYOCLONUS ASSOCIATED WITH POSTERIOR REVERSIBLE ENCEPHALOPATHY SYNDROME

Dolores Santamaria, Jamie Strause, Valerie Dechant, William McBride, Monisha Kumar, Rodney Bell, Michael Moussouttas
Thomas Jefferson Medical Center, Philadelphia PA, United States

Introduction:
Opsoclonus-myoclonus syndrome (OMS) is typically associated with a paraneoplastic syndrome or viral encephalitis. Various locations, including the crerebellum, have been proposed as anatomic correlations to this syndrome. OMS as a result of posterior reversible encephalopathy syndrome (PRES) has not been previously described.

Methods:
A 60 yo man with past medical history of poorly control hypertension, hyperlipidemia, and peripheral neuropathy presented with confusion and visual difficulties. Initial exam demonstrated a fever of 101, BP= 170/70 (max 210/110), and lethargy. Laboratory studies revealed acute renal insufficiency (ARI) (Cr=1.6) and rhabdomyolysis (CPK=845). MRI showed faint hyperintensities in the cerebellum and paieto-occipital subcortical areas (image 1). The patient subsequently developed agitation with diffuse multifocal myoclonus and pronounced opsoclonus.

Repeat MRI (image 2) showed extensive hyperintensities in the subcortical hemispheres bilaterally and in the cerebellum, consistent with PRES (image 2). EEG showed diffuse slowing, and LP showed elevated protein (124mg/dl). CSF cultures, VDRL, Lyme antibody, Listeria antibodies, West Nile Virus PCR, HSV PCR, and JC virus PCR were all negative. CT scan with contrast of the chest, abdomen and pelvis, revealed no neoplasms. Results of serum paraneoplastic antibodies are pending. Elevations in SPEP and UPEP were determined to be due to monoclonal gammopathy of unknown significance.

Control of the hypertension resulted in improvement in the patient's menation and resolution of the OMS. Hydration induced clearance of the CPK and resolution of the ARI.

Conclusions:
We present a unique case of PRES presenting as OMS. Involvement of the cerebellum may have been causative in this case. The most likely explanation for the development of PRES was hypertension with ARI. The acute onset, negative viral studies and body CT scan, resolution of symptoms with control of hypertension and reversal of ARI, and the characteristic MRI findings all supported PRES as the cause of OMS.

Financial Support: None

Poster 82

PERIPARTUM BILATERAL SIMULTANEOUS VERTEBRAL ARTERY DISSECTIONS : A DESCRIPTION OF TWO CASES

Eugene Wang, Jeffrey Frank, Fernando Goldenberg, Agnieszka Ardelt, James Brorson, Ritesh Kaushal, Meziane Guerch, Jose Biller
[1]*University of Chicago, Chicago, United States,* [2]*Loyola University, Chicago, United States*

Introduction:
There are few case reports of spontaneous cerebral artery dissections (carotid and vertebral), presenting immediately after childbirth. To our knowledge there have been no reports of patients in the literature that have presented with simultaneous bilateral extra-cranial vertebral artery dissections (BSEVAD) in the early postpartum period.

Methods:
We describe the clinical, imaging, and follow-up details of two patients who developed BSEVAD in the peripartum period to enhance the early recognition of this uncommon but important and potentially disabling complication. Both patients were initially misdiagnosed with post-dural puncture headaches (PDPH).

Results:
Both patients were in their thirties (ages 31 and 38), had epidural anesthesia, and developed their symptoms within days of delivery. Patient #1 developed postural headache within 24 hours of delivery, and Patient #2 developed severe neck pain and bioccipital headache 5 days after delivery. Both received epidural blood patch for presumptive diagnosis of PDPH without any significant relief. Patient #1 developed nausea, vomiting, and ataxia 8 days post-partum with a follow-up MRI revealing acute bilateral cerebellar infarcts and a unilateral pontine infarct. Patient #2 performed unusual physical positions during her pushing phase of labor (drawing provided). Initial neuroimaging with CT, MRI brain in both were reported as normal. However, 4-vessel cerebral angiography in both patients revealed BSEVAD. Both patients did well with medical therapy without sequelae. The risk factors, diagnostic clues, and therapeutic considerations are discussed.

Conclusions:
1. BSEVAD is a rare peripartum complication.
2. BSEVAD presents with clinical features that resemble PDPH but have some distinguishing features to facilitate differentiation.
3. BSEVAD can lead to stroke similar to non peripartum cerebral arterial dissections.
4. Early recognition of this rare complication can potentially lead to protecting patients from devastating posterior circulation strokes.

Financial Support: None

Poster 83

VENTILATORY FAILURE DUE TO POMPE'S DISEASE: A POTENTIALLY TREATABLE ENTITY

Morgan Johnson, Peter Pytel, Helene Rubeiz, Jeffery Frank, Fernando Goldenberg
University of Chicago, Chicago, Il, United States

Introduction:
The late-onset form of Pompe's disease presents generally with limb girdle weakness. Respiratory failure develops later and is the most frequently reported cause of death. We describe a case of late onset Pompe's disease emphasizing the need to incorporate this rare entity into the differential diagnosis of patients with ventilatory failure

Methods:
A 35 year old man presented with progressive weakness and shortness of breath. 4-5 years previously he noticed gradual lower extremity weakness. He began having difficulty breathing while lying flat. He was admitted to the NeuroICU due to progressive respiratory failure. Neuromuscular junction disease, neuropathy and typical myopathies were excluded.

Results:
EMG: Normal nerve conduction studies. Needle exam showed complex repetitive discharges, myotonic discharges and fibrillation potentials consistent with a myopathic process.
Muscle Biopsy: Myopathic changes associated with features of a vacuolar myopathy with abnormal glycogen accumulation and markedly increased acid phosphatase reactivity consistent with acid maltase deficiency.
Dried blood spot serum assay for acid α-glucosidase was undetectable.

Conclusions:
Pompe's disease is a rare condition that is now recognized as a treatable entity. Therefore, it should be included in the differential diagnosis of adult patients with gradually progressive myopathy and respiratory muscle weakness. Its treatment, enzyme replacement with recombinant human alpha-glucosidase (rhGAA), although not yet FDA-approved for patients over 18 years of age, has shown significant clinical benefit when started early in the course of the disease.

Financial Support: None

Poster 84

THE USE OF THERAPEUTIC HYPOTHERMIA FOR REFRACTORY INTRACRANIAL HYPERTENSION FOLLOWING A POOR GRADE SUBARACHNOID HEMORRHAGE

Patty Gessner, Guy Dugan
Alexian Brothers Medical Center, Elk Grove Village, Il, United States

Introduction:
Owing to the success of therapeutic hypothermia (TH) post cardiac arrest, additional indications are now being explored. This case report documents successful application of TH for treatment of refractory intracranial hypertension due to poor grade subarachnoid hemorrhage (SAH) without the need for decompressive hemicraniectomy.

Methods:
A 22-year-old male (DA) became unconscious after complaining of a bad headache. In the ED, DA was listed as unresponsive to all stimuli. CT revealed a Hunt/Hess grade 5 SAH. An aneurismal clipping was performed the following day. On day 4, he experienced severe vasospasm not amendable to angioplasty and refractory to osmotherapy, cerebrospinal fluid drainage, and mild hyperventilation. At one point the intracranial pressures exceeded 100 mmHg and his left pupil became fixed and dilated. A CT showed extensive edema and a worsening midline shift. It was then decided to initiate TH in anticipation for worsening vasospasm.

Results:
Once the target temperature of 33 degrees C was achieved, the ICP stabilized. Attempts to re-warm on days 8 and 10 led to increases in ICP therefore aborted. Finally, on day 13 (TH day 9), DA was re-warmed successfully. CT results that originally showed a large area of edema and midline shift was resolved. DA was extubated day 17, a VP shunt was placed on day 18 and discharged to home after a rehabilitation stay with a good neurological recovery on day 48.

Conclusions:
Considering the mortality of a high grade SAH can exceed 70%, we believe TH contributed significantly to a good neurological outcome. A recent study described the need for a decompressive hemicraniectomy prior to attempting mild hypothermia. Our case report documents successful application without invasive surgery and may be an option for others.

Financial Support: None

Poster 85

PURTSCHER'S RETINOPATHY IN A POSTPARTUM PATIENT WITH HYPERTENSIVE ENCEPHALOPATHY

Dhimant Dani, Thomas Wolfe, Mohammad Taqi, John Lynch, Brian Fitzsimmons, Michel Abraham, Ann Helms, Osama Zaidat, Michel Torbey
Medical College of Wisconsin, Milwaukee, WI, United States

Introduction:
Amaurosis is an uncommon complication of pregnancy encountered by neurologists. Two common causes of blindness in during the peripartum period are: (1) Reversible posterior leukoencephalopathy syndrome (RPLS), and (2) preeclampsia.

Methods:
Case Report:
A 31-year old woman delivered twins at 35-weeks because of severe preeclampsia. In the early postpartum period, she developed altered mental status, and by postpartum day #2, she was responsive only to first name, unable to follow commands, and increasingly combative. Blood pressures were elevated up to 179 mmHg systolic, and 120 mmHg diastolic. She had limited vision by absence of blink response to confrontation bilaterally. Head CT and EEG were unremarkable. Based on findings on cerebral magnetic resonance imaging (MRI), a diagnosis of hypertensive encephalopathy was made. However, the occipital lobes are clearly spared. Her condition substantially improved, and by postpartum day 4 she was able to cooperate with visual acuity and funduscopic examinations. Visual acuity was 20/800 bilaterally. Bilateral fundi showed discrete patches of retinal whitening located between the arterioles and venules, few retinal hemorrhages, and normal optic discs .By discharge, her blood pressure and mental status were at baseline, her vision improved to 20/200, and repeat MRI showed resolution of the earlier findings.

Results:
Cause of blindness in this patient was related to Purtscher's Retinopathy.

Conclusions:
We believe that this is the first documented case of a patient with a hypertensive encephalopathy and Purtscher's retinopathy. This observation indicates that transient visual loss in the setting of elevated systemic blood pressures does not have to be cortical in nature.

Financial Support: None

Poster 86

CONTINUOUS CARDIAC OUTPUT PULMONARY ARTERY CATHETERS CAUSE UNIQUE EEG ARTIFACTS

William A Knight IV, Jordan B Bonomo, David M Ficker, Jay A Johannigman, Lori A Shutter
University of Cincinnati, Cincinnati, OH, United States

Introduction:

Continuous EEG monitoring of neurocritical care patients is becoming more common. Over 20% of critically ill patients with altered sensorium are diagnosed with non-convulsive seizures or status epilepticus. As continuous EEG is increasingly used with critically ill patients, it is important for practitioners to recognize artifacts that may mimic clinically relevant pathologic discharges. We describe a newly discovered artifact from an invasive hemodynamic monitoring device.

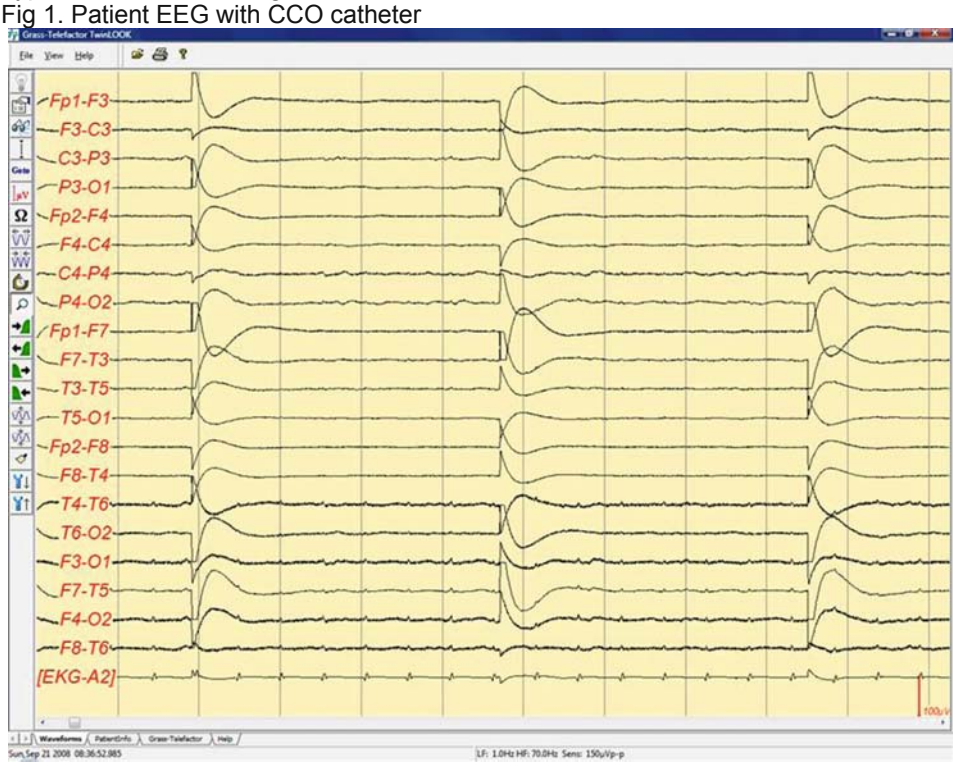
Methods:

Seven patients who had a Ref/Ox (Edwards LifeSciences) continuous cardiac output pulmonary artery catheter (CCO) placed for hemodynamic monitoring and were simultaneously monitored with continuous EEG were retrospectively identified. All patients were cared for in a 20 bed Neuroscience ICU in an urban Level 1 Trauma Center. All EEGs were interpreted by a board certified epileptologist.

Results:

All patients' EEGs demonstrated a distinctive artifact believed to be associated with the CCO catheter. This artifact has not been previously described with other pulmonary artery catheters. The EEG was characterized by an intermittent high amplitude, narrow complex, spike-like artifact followed by a high amplitude slow wave. It is hypothesized that this signal results from current flow to the thermal element of the catheter.

Fig 1. Patient EEG with CCO catheter



Conclusions:

Neurocritical care patients frequently undergo multi-modality monitoring. This newly identified EEG artifact with CCO monitoring has an appearance that may be confused with epileptic spike/wave discharges or burst suppression. The impact of this potential artifact generated by the use of CCO devices requires further characterization. As neurocritical care patients increase in complexity and are subjected to more invasive monitoring, the identification of new EEG artifacts may become more common.

Financial Support: None

Poster 87

FULMINANT GUILLAIN-BARRE SYNDROME MIMICKING CEREBRAL DEATH: CASE REPORTS AND LITERATURE REVIEW

Mohammed Rehman, Ximena Arcila-Londono, Gurjeet Singh, Panayoitis Varelas, Jessy Corry, Jody Wellwood, Tamer Abdelhak
Henry Ford Hospital, Detroit, MI, United States

Introduction:
The diagnosis of Gullian-Barre syndrome (GBS) is based on a combination of clinical and laboratory features. GBS typically presents as a monophasic, subacute, symmetrically, predominantly motor neuropathy. In rare cases, GBS can present with acute quadriparesis and cranial nerve involvement. We report two cases of patients who presented in a state mimicking brain death with complete dysfunction of efferent nerves which turned out to be fulminant GBS.

Methods:
Two cases with rapidly progressive weakness presented to our institution with very rapid deterioration requiring mechanical ventilation. Over a very short course of time, both patients became paralyzed with complete absence of brainstem reflexes. Brainstem function tests were performed as part of full neurological examination which revealed that both patients had non-reactive mydriasis with complete internal and external ophthalmoplegia. Rest of the neurological exam including deep tendon reflexes showed no reponse. Due to lack of identifiable cause of patient's condition, further diagnostic test were carried out.

Results:
Both patient's underwent CSF analysis which revealed evidence of albuminocytological dissociation. A diagnosis of severe Guillain-Barre syndrome with involvement of peripheral and cranial nerves was suspected. Electrophysiological studies were performed that showed this was suggestive of severe, axonal, sensorimotor peripheral polyneuropathy with profuse ongoing denervation in bulbar, cervical and lumbosacral innervated muscles. Extensive laboratory evaluation including GQ1B Antibody were carried out. After prolonged course, both patients made some functional recovery.

Conclusions:
Both these cases proved that in rare cases, GBS can present with signs of coma and absent brainstem reflexes. Brain-death protocols require that before the declaration of brain-death, an etiology needs to identified that could explain the clinical picture and all reversible causes are excluded. These cases illustrate the importance of electrophysiologic, laboratory and imaging studies in patients with suspected brain death where a cause is not clearly determined.

Financial Support: None

Poster 88

REVERSAL OF HYPERACUTE HYPERPERFUSION SYNDROME MIMICKING ACUTE RESTENOSIS AFTER SUCCESSFUL CAROTID STENTING

Julius Gene Latorre, Tarakad Ramachandran, Yahia Lodi
SUNY Upstate Medical University, Syracuse, NY, United States

Introduction:
Hyperperfusion syndrome is a serious complication after carotid revascularization procedure associated with poor outcome, developing between day 1 to 14 days after procedure. In addition to increased cerebral blood flow, clinical manifestation include headache, seizure and intracerebral hemorrhage. Equally rare is stent thrombosis especially in patients who were adequately treated with antiplatelets prior to procedure. Distinction between the two condition requires prompt diagnosis to achieve good outcome.

Methods:
Case report and medical record review

Results:
85M presented with acute L hemiparesis and was treated with IV tPA. NIHSS was 7 on admission and 0 after 24 hours. CT brain did not show any acute infarction. Cerebral angiogram showed 70% R ICA stenosis.The patient underwent R carotid stenting on day 5. Postprocedure MAP was maintained between 80-100 mmHg using oral and IV antihypertensive agents. 12 hours post procedure the patient developed L hemiparesis, dysarthria, right gaze preference and L hemianopia. Blood pressure was augmented to MAP 100-120 mmHg while en route to CT/MRI and angiography for suspected acute stent occlusion. Patient's hemiparesis worsened. CT brain showed unilateral subtle R hemispheric edema but no hemorrhage. MRI showed patchy DWI along R hemisphere with subtle cortical and meningeal enhancement. Emergent angiography showed patent stent. Blood pressure was immediately controlled to MAP 70-90 mmHg and patient improved. Within 48 hours, patient was ambulatory. Repeat CT brain did not show any acute infarction. 6 months after discharge, he was asymptomatic.

Conclusions:
Hyperperfusion syndrome can develop even with relatively controlled blood pressure post-carotid revascularization. Emergent vascular imaging is necessary to differentiate acute stent occlusion from hyperperfusion syndrome so that appropriate measures may be done. When aggressively managed, symptoms associated with hyperperfusion syndrome are fully reversible if not associated with hemorrhage.

Financial Support: None

Poster 89

ACCURATE CORRELATION OF BRAIN AND CORE TEMPERATURES DURING INTRAVASCULAR THERAPEUTIC HYPOTHERMIA

Mohammed Zaman[1], Matthew Smith[1], David Decker[1], Karen Wilson[3], Mark Rumbak[2], Reza Behrouz[1]
[1]University of South Florida College of Medicince - Department of Neurology, Tampa, FL, United States, [2]University of South Florida College of Medicine - Department of Internal Medicine, Tampa, FL, United States, [3]Tampa General Hospital - Neurosciences Intensive Care Unit, Tampa, FL, United States

Introduction:
Fulminant hepatic failure (FHF) or diffuse anoxic injury can lead to the development of cerebral edema and increased intracranial pressure. Hypothermia has been utilized in both clinical scenarios in attempt to prevent the development cerebral edema and manage elevated intracranial pressure. In this case, we sought to determine a correlation between brain and core temperatures ~~in~~ specifically in intravascular therapeutic hypothermia (IHT)

Methods:
This observation was conducted in a 32 year-old woman with grade III hepatic encephalopathy (HE) due to FHF. IHT using CoolGard® Icy catheter (Zoll Medical) was started immediately as the patient progressed to grade III HE. Esophageal and Foley temperature probes were utilized for recording core body temperature. Monitoring of brain temperature and intracranial pressure was conducted via Licox® system (Integra). Goal temperature range for IHT was between 32-34° C. Brain and core temperatures were recorded hourly during IHT which was a period of 87 hours. Data was collected and plotted to show correlation between the three temperatures over time.

Results:
Measurements were obtained over the course of 87 hours to log temperatures. The results showed: Brain temperature: $y=-0.0068x +32.425$. $R^2 =0.1915$. Bladder temperature: $y = -0.0045x +32.311$. $R^2 =0.08689$. Esophageal temperature: $y = -0.0053x +32.322$. The results show a direct linear correlation between brain, esophageal, and bladder temperatures

Conclusions:
Accurate correlation between brain and core temperatures was demonstrated during IHS. Further investigation using larger number of subjects is needed to confirm this.

Financial Support: None

Poster 90

NOVEL INTRACRANIAL BLOOD FLOW PATTERNS IN PATIENTS WITH IMPLANTABLE LONG-TERM VENTRICULAR ASSIST DEVICES

Fernando Goldenberg, Christi Kordeck, Meziane Guerch, Jai Raman, Shahab Akhter, Agnieszka Ardelt, Jeffrey Frank
University of Chicago Medical Center, Chicago, Illinois, United States

Introduction:
The cerebral circulation is normally pulsatile except for short periods of time in patients subjected to extracorporeal circulation, commonly used during cardiac surgery. A new generation of Left Ventricular Assist Devices (LVAD) generates continuous, non-pulsatile blood flow. In patients with implantable continuous flow LVAD (CFLVAD), peripheral arterial pulsatility will exist as long as the native heart is capable of maintaining enough contractility to generate some stroke volume during systole.

Methods:
We explored the intracranial circulation with Transcranial Doppler (TCD) in 8 patients that had a CFLVAD implanted and were neurologically intact. Doppler insonation was performed through the routine temporal and occipital bone windows and proximal intracranial vessels were surveyed. Transthoracic Echocardiogram was performed in all patients to assess the LV function.

Results:
4/8 patients (50%) that had some preservation of the native heart function exhibited an intracranial flow pattern consisting of: high end diastolic flow velocity, very low pulsatility index and sometimes a sinusoidal wave appearance coincident with the native heart's systolic contraction.
4/8 patients (50%) that had extremely poor heart contractility exhibited a distinctive pattern of continuous flow where it was impossible to distinguish between systolic and diastolic flow.

Conclusions:
With the advent of new mechanical cardiac support for patients with end-stage heart failure, new peripheral and cerebral blood flow patterns develop and clinicians need to be aware of these distinctive and novel findings.
This scenario opens an enormous opportunity to understand and better characterize a new physiological situation. It could also limit the usefulness of bedside TCD as a complementary method for the diagnosis of cerebral circulatory arrest given the lack of pulsation, known generator of the isolated systolic spikes or the "To and Fro" pattern considered pathognomonic findings of the absence of intracranial circulation.

Financial Support: None

Poster 91

NEED FOR AN ADVANCED NEUROLOGICAL LIFE SUPPORT COURSE

Edgar Samaniego[1], Gregory Kapinos[2]
[1]*Stanford University, Palo Alto, CA, United States,* [2]*Weill Cornell Medical Center, New York, NY, United States*

Introduction:
The Advanced Cardiovascular Life Support (ACLS) and Advanced Trauma Life Support (ATLS) provider courses are excellent resuscitation tools directed towards respiratory and hemodynamic stabilization, nevertheless, survival rates with good neurological outcome are dismal.

Methods:
The neurological content of ACLS and ATLS training manuals was reviewed. An Advanced Neurological Life Support (ANLS) course is proposed based on the deficiencies of ACLS and ATLS (Table).

Results:
The neurological content of ACLS is 11% and covers only ischemic stroke, with no mention of hemorrhagic stroke or other neurological emergencies. The neurological content of ATLS is 6%, with a brief description of intracranial hemorrhages, increased intracranial pressure and spinal cord injuries management. Both courses overlooked frequent devastating neurological emergencies like status epilepticus, anoxic encephalopathy, acute paralysis and meningitis.

Table. ACLS and ATLS comparison with proposed ANLS.

	ACLS	ATLS	ANLS
Audience	All residents, EMS	Surgery residents and trauma team.	Neurocritical care team, neurology and neurosurgery residents, ER
Primary Survey	Airway , breathing, circulation, defibrillator	Airway , breathing, circulation, disability and exposure	Airway , breathing, circulation, disability and exposure.
Secondary Survey	Airway, breathing, circulation, differential diagnosis	Full history, head-to-toe physical exam, differential diagnosis, chest and plevis X-rays interpreataion. Sonography and deep peritoneal lavage	Airway, breathing and circulation. Brain stem reflexes, GCS and NIHSS.
Semiological approach	Cardio-circulatory versus respiratory	Traumatic assessment, hypovolemia versus neurogenic shock	Anoxic encephalopathy, traumatic brain injury, stroke, status epilepticus, increased intracranial hypertension
Practical Skills	Intubation, CPR, EKG interpretation	Intubation, chest x-ray interpretation, chest tubes, GCS, central line	Lumbar puncture, CT and basic EEG interpretation, cooling
Testing	1 megacode	2 trauma codes	2 neurocodes.

Conclusions:
Many basic concepts of neurological critical care management are missing in advanced resuscitation courses. We advocate the creation of an ANLS provider course to improve neurological outcomes of patients who undergo resuscitation.

Financial Support: None

Poster 92

THE EFFECT OF DECOMPRESSIVE HEMICRANIECTOMY ON BRAIN TEMPERATURE AFTER SEVERE BRAIN INJURY

Kazuma Nakagawa, Diane Morabito, Pratik Patel, Geoffrey Manley, J. Claude Hemphill III
University of California San Francisco, San Francisco, CA, United States

Introduction:
Animal studies have shown that even a temperature elevation of one degree Celsius can worsen neuronal injury after brain ischemia. Since the skull acts as a thermal insulator, we hypothesized that decompressive hemicraniectomy lowers brain temperature by facilitating the heat convection from the brain to its surrounding air.

Methods:
Fifty patients with severe brain injury (TBI=40, ICH=10) requiring continuous brain temperature monitoring (Licox, Integra LifeSciences, Plainsboro, NJ) from January 2007 to March 2009 were retrospectively studied and grouped into "hemicraniectomy" (n=22) or "no hemicraniectomy" group (n=28). The core body (T_{Core}) and brain (T_{Br}) temperature measurements were recorded at 1-min intervals over 12 ± 7 ICU days. As a surrogate marker for the degree of external heat loss from the brain, $\Delta T_{Br\text{-}Core}$ was calculated as the difference between T_{Br} and T_{Core} with each recording. T-tests were used to initially assess the difference between groups. Then, in order to account for clustering of observations in individual patients, generalized estimating equations (GEE) were used to assess association of hemicraniectomy with $\Delta T_{Br\text{-}Core}$, adjusting for core body temperature and diagnosis.

Results:
A total of 316,453 temperature data pairs were collected for analysis (median[IQR] per patient: 5166 [3132 – 9551]) Baseline characteristics were similar in age, sex, diagnosis, GCS, ICU mortality, and ICU LOS between those with and without hemicraniectomy. By univariate t-test, hemicraniectomy patients had a significantly greater $\Delta T_{Br\text{-}Core}$ than those without hemicraniectomy (-1.21±0.88 ^{o}C vs. -0.75±0.86 ^{o}C, p<0.0001). However, after adjusting for intra-individual variability using GEE, only higher core body temperature, but not hemicraniectomy, was associated with difference in $\Delta T_{Br\text{-}Core}$ (p=0.001 for T_{Core}; p=0.53 for hemicraniectomy). This suggests that the $\Delta T_{Br\text{-}Core}$ temperature difference is larger at higher body temperature.

Conclusions:
Substantial variability exists in the brain-to-body temperature gradient across patients and core body temperatures. However, this difference is not due to the presence of a hemicraniectomy.

Financial Support: None

Poster 93

YOUNG PEOPLE'S ATTITUDE TOWARDS STROKE AND DECOMPRESSIVE HEMICRANIECTOMY

Kazuma Nakagawa[1], Matt Bianchi[2], Shawn Nakagawa[3], Farzaneh Sorond[3]
[1]University of California San Francisco, San Francisco, CA, United States, [2]Massachusetts General Hospital, Harvard Medical School, Boston, MA, United States, [3]Brigham and Women's Hospital, Harvard Medical School, Boston, MA, United States

Introduction:
The assumption is often made that young people would want decompressive hemicraniectomy after a large stroke as a life-saving measure. However, this assumption favoring aggressive life-saving treatment, and the perception of quality of life after neurological disability, have not been adequately studied.

Methods:
We conducted a cross-sectional questionnaire-based survey that consisted of demographic information (age, sex, race, marital and family status, religion, income, education level, access to healthcare), and attitude towards neurological disability (based on the highest acceptable modified Rankin scale (mRS) that they would be "willing to live with"). Young adults in the Los Angeles County were surveyed and grouped by whether or not they would want hemicraniectomy after a large stroke despite a high likelihood of disability. Findings from the two groups were compared using Student's t-test and chi-square test. Logistic regression analysis was used to determine the factors predicting willingness to accept decompressive hemicraniectomy.

Results:
In this pilot study, 64 young adults (mean age: 24±6) were surveyed. The highest acceptable mRS (0–5) participants felt "willing to live with" were: 9.4% (0), 28.1% (1), 28.1% (2), 21.9% (3), 9.4% (4), 3.1% (5). Despite a high likelihood of severe disability, 44 of 64 (69%) reported they would undergo hemicraniectomy after a severe stroke. Neither the demographic factors nor the highest acceptable mRS were associated with the willingness to seek aggressive treatment and hemicraniectomy.

Conclusions:
The results from our preliminary study support the commonly held assumption that young adults are generally willing to accept decompressive hemicranietomy as a life-saving measure. However, a substantial subset (~30%) were not willing to accept this aggressive measure, which emphasizes the importance of discussing the individual's previously stated wishes, even in the young population. Further study in larger populations is needed to better characterize the factors impacting young adults' decisions regarding aggressive care.

Financial Support: None

Poster 94

CSF ADRENERGIC PROFILE AMONG PATIENTS WITH NEUROGENIC CARDIOMYOPATHY

Keith Dombrowski, John Khoury, J Ramlall, Milton Maltenfort, E Lae, Matthew DeCaro, Michael Moussouttas
Thomas Jefferson University Hospital, Philadelphia, PA, United States

Introduction:
Neurogenic Cardiomyopathy (NC) is believed to result from sudden excessive adrenergic activity. Currently there is no data on the central adrenergic profile in patients with NC. The purpose of this study is to document the cerebrospinal fluid (CSF) levels of adrenergic compounds and metabolites in patients with SAH, and to compare adrenergic levels between patients with and those without NC.

Methods:
40 consecutive H/H Grade 3-5 patients requiring a ventriculostomy were included in this study. CSF (1ml) was collected from each patient and transthoracic echocardiography (TTE), to assess for the presence of NC, was performed within 72 hours of symptom onset. CSF samples were assayed by HPLC for epinephrine (Epi), norepinephrine (NE), and dihydroxyphenylglycol (DHPG) levels.
Statistical analyses were performed to identify clinical and laboratory predictors of NC. Variables demonstrating a possible association, and variables identified as predictors of NC from the literature, were then entered into a logistic regression model.

Results:
Mean age was 58yo, and 65% were female. 53% were G3 and 47% were G4/5. NC patients tended to be younger (52yo vs 59yo, p=.091) and tended to have a higher H/H score (G4/5) (75% vs 41%, p=.082). CSF levels of Epi and NE were not elevated in patients with NC compared to those without NC (23pg/ml vs 21pg/ml, p=.839 & 112pg/ml vs 238pg/ml, p=.198), but a trend was noted for DHPG levels (519pg/ml vs 776pg/ml, p=.07).
In the multivariate regression model incorporating age, H/H grade and drug use, no correlation was demonstrated for Epi (p=.154), but a trend was noted for NE levels (p=.073), and a significant correlation was seen for DHPG (p=.018).

Conclusions:
Our study supports the theory of a CNS mediated adrenergic mechanism for NC, based on the presence of increased CSF levels of NE and its DHPG metabolite.

Financial Support: None

Poster 95

BIOCHEMICAL ASPIRIN RESISTANCE IS ASSOCIATED WITH RECURRENT ISCHEMIC STROKE LESIONS DURING THE ACUTE PERIOD

Sang-Beom Jeon, Ha-Sup Song, Bum Joon Kim, Hye-Jin Kim, Dong-Wha Kang, Jong S. Kim, Sun U. Kwon
Asan Medical Center, University of Ulsan College of Medicine, Seoul, Korea, Republic of

Introduction:
Recent studies have suggested that recurrent stroke during aspirin treatment might have been caused by biochemical aspirin resistance (BAR). We hypothesized that patients with BAR would develop early recurrent ischemic lesions (ERILs) on diffusion-weighted imaging (DWI) more than those without BAR.

Methods:
We included 120 consecutive patients who: 1) were admitted to our center within 24 hours of stroke onset; 2) had a final diagnosis of acute ischemic stroke, confirmed by DWI, or TIA; 3) underwent follow-up DWI within seven days after initial DWI; 4) received aspirin therapy; and 5) underwent tests for BAR. Aspirin was administered to patients soon after initial imaging study. BAR was measured using the VeryfyNow Rapid Platelet Function Assay-Aspirin (Accumetrics Inc., San Diego, CA). An ARU ≥550 was defined as BAR. ERILs were defined as new lesions on follow-up DWI with decreased apparent diffusion coefficient, which were not detected on initial DWI scans.

Results:
The number of patients with BAR was 16 (13.3%). ERILs were detected in 10 (8.3%) patients. BAR was related to the development of ERILs: among 16 patients with BAR, five (31.3%) patients developed ERILs; and among 104 patients without BAR, five (4.8%) developed ERILs (OR, 5.44; 95% CI, 1.34 – 22.08; $p = 0.028$).

Conclusions:
BAR is associated with the development of ERILs during the first week after development of ischemic stroke. This suggests that increased thrombogenicity is one of important mechanisms of ERILs and that aggressive antiplatelet therapy is warranted during the acute phase.

Financial Support: None

Poster 96

EFFECT OF MANNITOL ON BRAIN TISSUE METABOLISM AND OXYGENATION IN PATIENTS WITH SEVERE BRAIN INJURY AND INTRACRANIAL HYPERTENSION

Raimund Helbok[1], Pedro Kurtz[2], Michael Schmidt[2], Morgan Stuart[3], Luis Fernandez[2], Richi Malhotra[2], Sander Connolly[3], Kiwon Lee[2], Neeraj Badjatia[2], Stephan Mayer[2], Jan Claassen[2]

[1]Medical University Innsbruck, Clinical Department of Neurology, Neurocritical Care Unit, Innsbruck, Austria, [2]Columbia Universtiy Medical Center, Department of Neurology, Division of Neurocritical Care, New York, NY, United States, [3]Columbia Universtiy Medical Center, Department of Neurosurgery, New York, NY, United States

Introduction:
In this study we examined the effects of mannitol 20% on brain metabolism and brain tissue oxygenation (PbtO2) in severely brain-injured patients with intracranial hypertension.

Methods:
Twenty-two episodes of raised intracranial pressure (>20 mm Hg) resistant to standard therapy that required infusions of mannitol were prospectively studied in 13 comatose patients with multimodality monitoring of intracranial pressure (ICP), PbtO2, and microdialysis. We compared mean arterial blood pressure (MAP), ICP, cerebral perfusion pressure (CPP), PbtO2, and brain lactate, pyruvate, and glucose using cerebral microdialysis, for 3 hours preceding and 4 hours after hyperosmolar therapy. Time series data were analyzed using a multivariable general linear model (GLM) utilizing generalized estimating equations (GEE) for model estimation to account for within-subjects and between-subjects variations over time.

Results:
1 g/kg of 20% mannitol solution led to a maximal reduction of ICP at 30 minutes (from 27 ± 13 to 19 ± 16 mm Hg, P <0.001). CPP increased at a peak of 45 minutes (from 73 ± 18 to 85 ± 22 mm Hg, P = 0.002) after mannitol infusion was started, whereas MAP and PbtO2 did not change significantly. Compared to lactate-pyruvate ratio (LPR) at the time of osmotherapy (44 ± 20,) , mannitol resulted in a an 18% decrease over 2 hours (to 35 ± 16, P = 0.002). Brain glucose levels remained unaffected.

Conclusions:
Mannitol effectively reduces ICP and augments CPP, and appeared to benefit oxidative metabolism as measured by the LPR.

Financial Support: None

Poster 97

SYSTEMIC GLUCOSE AND BRAIN ENERGY METABOLISM AFTER SUBARACHNOID HEMORRHAGE: A MICRODIALYSIS STUDY

Raimund Helbok[1], Michael Schmidt[2], Pedro Kurtz[2], Khalid Hanafy[2], Luis Fernandez[2], Morgan Stuart[3], Sander Connolly[3], Kiwon Lee[2], Neeraj Badjatia[2], Stephan Mayer[2], Jan Claassen[2]

[1]Medical University Innsbruck, Department of Neurology, Neurolocritical Care, Innsbruck, Austria, [2]Columbia Univesity Medical Center, Division of Neurocritical Care, Department of Neurology, New York, NY, United States, [3]Columbia University Medical Center, Department of Neurosurgery, New York, NY, United States

Introduction:
Brain energy metabolic crisis (MC) and lactate-pyruvate ratio (LPR) elevations have been linked to poor outcome in comatose patients. We sought to determine if acute reductions in serum glucose are associated with MC and LPR elevations in poor-grade subarachnoid hemorrhage (SAH) patients.

Methods:
Twenty-eight comatose SAH patients that underwent multimodality monitoring with intracranial pressure and microdialysis were studied. MC was defined as lactate/pyruvate ratio (LPR) ≥40 and brain glucose <0.7 mmol/L. Time series data were analyzed using a multivariable general linear model with a logistic link function for dichotomized outcomes.

Results:
Multimodality monitoring included 3178 hours of observation (mean 114 ±65 hours per patient). In exploratory analysis, serum glucose significantly decreased from 8.2 mmol/L (148mg/dL) 2 hours before to 6.9 mmol/L (124 mg/dL) at the onset of MC (P<0.001). Reductions in serum glucose of 25% or more were associated with new onset MC (adjusted odds ratio [OR] 3.6, 95% confidence interval [CI] 2.2–6.0). This association was independent of the absolute serum glucose level. In a second model we chose an elevation of the LPR by 25% or more as the outcome variable. Again, reductions in serum glucose of 25% or more were independently associated with an LRP rise (adjusted OR 1.6, 95% CI 1.1–2.4). All analyses were adjusted for significant covariates including Glasgow Coma Scale and cerebral perfusion pressure.

Conclusions:
Acute reductions in serum glucose, even to levels within the normal range, may trigger brain energy metabolic crisis and LPR elevation in poor-grade SAH patients.

Financial Support: None

Poster 98

HYPONATREMIA AFTER SUBARACHNOID HEMORRHAGE: RISK FACTORS AND IMPACT ON OUTCOME

Luis Fernandez, Raimund Helbok, Pedro Kurtz, Khalid Hanafy, Matthew Vibbert, Rishi Malhotra, Viktor Szeder, Kiwon Lee, Jan Claassen, Neeraj Badjatia, E. Sander Connolly Jr., Stephan A. Mayer
Columbia University Medical Center, New York City, NY, United States

Introduction:
Hyponatremia develops in up to one-third of patients after subarachnoid hemorrhage (SAH), and is usually attributed to cerebral salt wasting or SIADH. Our goal was to identify risk factors for hyponatremia after SAH, and to determine its impact on outcome.

Methods:
We analyzed 1015 consecutive SAH patients enrolled in the Columbia University SAH Outcomes Project between July 1996 and June 2006. Hyponatremia was defined as sodium level ≤130 mEq/l occurring at any point during hospitalization. Multivariate analysis was performed to identify risk factors for hyponatremia. Functional disability was evaluated at discharge and 12 months with the modified Rankin Scale (mRS, score 4-5) and Barthel Index (BI, score <90)

Results:
The frequency of hyponatremia in or cohort was 13% (131/1015). Hyponatremia developed on median post bleed day 6 with most cases occurring between days 2 and 9. Logistic regression adjusted for gender and initial Hunt-Hess grade identified older age (OR 1.02, 95% CI 1.00-1.03, P=0.012), renal failure (defined as any creatinine level >2.5 mg/dl, OR 3.4, 95% CI 1.5-8.1, P=0.004), fever >38.6 °C (OR 1.9, 95% CI 1.2-2.9, P=0.003) and hydrocephalus requiring CSF diversion (OR 1.6, 95% CI 1.0-2.5, P=0.046) as risk factors for hyponatremia. There was no association of hyponatremia with in-hospital delirium, seizures, cerebral edema, or mortality. After adjusting for age, gender and Hunt-Hess grade, hyponatremia was an independent predictor of moderate-to-severe disability at discharge assessed by both the mRS (OR 1.8, 95% CI 1.1-2.8, P=0.02) and BI (OR 1.8, 95% CI 1.1-2.9, P=0.02). This association was no longer present at 3 and 12 months.

Conclusions:
Hyponatremia occurs in 13% of SAH patients, is predicted by older age, fever, renal failure and hydrocephalus, and is associated with reversible functional disability at discharge. Failure to correct hyponatremia my potentially interfere with rehabilitation and recovery after SAH.

Financial Support: None

Poster 99

THE RELATIONSHIP BETWEEN AGE AND CEREBRAL VASOSPASM AFTER ANEURYSMAL SUBARACHNOID HEMORRHAGE

Michael Schimdt, Luis Fernandez, Guillermo Linares, Alex Choi, Sang Bae Ko, Barry Czeisler, E. Sander Connolly, Jan Claassen, Neeraj Badjatia, Stephan Mayer, Kiwon Lee
Columbia University Medical Center, New York City, NY, United States

Introduction:
Previous studies have reported that younger patients have a higher incidence of clinical deterioration from vasospasm after subarachnoid hemorrhage. We sought to determine the relationship between age, with the incidence of vasospasm defined by angiographic, TCD, or clinical criteria.

Methods:
We analyzed 1000 consecutive SAH patients enrolled in the Columbia University SAH Outcomes Project between July 1996 and June 2006. Vasospasm was assessed using angiography and/or a mean flow velocity greater than 120 cm/s in any vessel. Symptomatic vasospasm was defined as clinical deterioration (i.e. a new focal deficit, decrease in level of consciousness, or both) and asymptotic vasospasm included a new infarct on CT that was not visible on the admission or immediate postoperative scan.

Results:
A TCD velocity greater than 120 cm/s was observed in 50% of patients and of the patients that had follow-up angiography performed (48%), 52% of those patients had vessel narrowing consistent with angiographic vasospasm. In contrast symptomatic vasospasm was observed in only 17% of all patients with 11% of patients suffering infarction attributed to vasospasm. In total 21% of patients had either symptomatic vasospasm or asymptomatic infarction from vasospasm. Multivariable logistic regression revealed that after accounting for disease severity (Hunt & Hess), modified Fisher score, gender, and history of smoking, younger age was significantly related to the occurrence of angiographic (OR: 2.3, 95% CI: 1.5-3.4) and TCD>120cm/s (OR: 3.4, 95% CI: 2.4-4.9) spasm, but was not significantly associated with symptomatic vasospasm (p=. 23) or delayed infarction from vasospasm (p=. 51).

Conclusions:
Our data support the findings that younger patients are more likely to experience vasospasm defined by TCD and angiography than older patients, but in our cohort we did not observe a higher incidence of clinical vasospasm or infarction. Aggressive treatment of TCD-based and angiographic vasospasm with intra-arterial vasodilators or balloon angioplasty may mitigate the effect of age.

Financial Support: None

Poster 100

ADMISSION CT FINDINGS PREDICT RISK OF POSTTRAUMATIC VASOSPASM

Krista Keachie[2], Irene M. Hutchins[1], Nancy Rudisill[1], Lori Madden[1], J. Paul Muizelaar[1], Kiarash Shahlaie[3]
[1]University of California, Davis, Sacramento, CA, United States, [2]Gundersen Lutheran Hospital, La Crosse, WI, United States, [3]University of California, San Francisco, San Francisco, CA, United States

Introduction:
Post-traumatic vasospasm (PTV) occurs in 20-50% of patients with severe traumatic brain injury (TBI), and is an independent predictor of neurological outcome. Although PTV incidence has been associated with injury severity, there are conflicting reports regarding patterns of intracranial hemorrhage that may correspond with development of posttraumatic vasospasm. Some authors report that subarachnoid hemorrhage or subdural hematoma is necessary to develop PTV, while others have reported significant PTV in the absence of these lesions.

Methods:
We performed a review of prospectively collected CT scan data from 394 consecutive head injured patients treated at a tertiary Level I Trauma Center. Rotterdam CT score data was reviewed from all patients in the TBI registry, and admission head CT scans from patients with severe TBI (GCS ≤ 8) with (n=8) and without (n=37) clinically significant PTV (csPTV) were re-evaluated by a 'blinded' investigator. CsPTV was defined as demonstrated neurological decline with CT angiographic evidence of arterial vasospasm.

Results:
Rotterdam CT Score significantly correlated with the development of csPTV($p=0.03$). The components of this score were further investigated. We found no correlation between epidural hematoma, subdural hematoma, midline shift, or cisternal compression and the development of csPTV. The presence of intraparenchymal hemorrhage ($p=0.02$) and cisternal subarachnoid hemorrhage ($p=0.035$), however, significantly correlated with risk of csPTV. All cases of csPTV were diffuse in anatomic distribution, and, therefore, did not correlate with side of maximal injury.

Conclusions:
Rotterdam CT score, intraparenchymal and cisternal subarachnoid hemorrhage on admission CT are significantly correlated with the incidence of csPTV. This suggests that risk of cerebral vasospasm following traumatic brain injury is increased not only in subarachnoid hemorrhage, but also intraparenchymal hemorrhage, and that Rotterdam CT score may be a useful metric for assessing risk of csPTV in severe TBI patients.

Financial Support: None

Poster 101

MANAGEMENT OF POST-TRAUMATIC VASOSPASM: PATIENT SELECTION FOR INTRA-ARTERIAL THERAPY

Krista Keachie[2], Irene M. Hutchins[1], Karen Smith[1], Nancy Rudisill[1], Lori Madden[1], J. Paul Muizelaar[1], Kiarash Shahlaie[3]

[1]Univeristy of California, Davis, Sacramento, CA, United States, [2]Gundersen-Lutheran Hospital, La Crosse, WI, United States, [3]University of California, San Francisco, San Francisco, CA, United States

Introduction:

Posttraumatic vasospasm (PTV) is an under-recognized source of secondary injury following TBI that significantly affects clinical outcome. Optimal treatment strategies for PTV are unknown, with published recommendations ranging from clinical observation alone to hyperdynamic therapy, systemic drug therapy, local intra-arterial drug infusion, and transluminal balloon angioplasty (TBA).

Methods:

We reviewed 394 patients from a tertiary Level 1 Trauma Center TBI Registry and identified 10 patients with clinically significant PTV (csPTV), defined as demonstrated neurological decline with CT angiographic evidence of arterial vasospasm. Patient charts were reviewed to characterize the natural history, treatment and efficacy of treatment in csPTV.

Results:

Treatment strategies for patients with csPTV included observation, “triple-H” therapy, oral statins, intra-arterial verapamil infusion and TBA. The decision to pursue intra-arterial therapy was based on severity of spasm and clinical exam. Observation alone was used in patients with mild, diffuse spasm on CTA and rapid clinical improvement (n=2), whereas those with persistent signs of spasm all underwent medical therapy (n=7). Intra-arterial verapamil infusion was used in patients with moderate to severe spasm (n=5). TBA was performed in patients who had severe and diffuse spasm (n=2). In all cases, therapy was effective in reducing (n=4) or reversing (n=5) PTV. Three month functional outcome data revealed no significant differences between patients with and without csPTV.

Conclusions:

Treatment of PTV is effective in reducing or reversing arterial vasospasm. A variety of therapies exist, which should be chosen based on clinical exam and the degree and distribution of spasm. Although it is unknown if treatment improves outcome, our data suggest that patients with csPTV have similar outcomes to those without csPTV when they are adequately treated. A clinical pathway is presented to aid in the screening, diagnosis, and treatment of PTV.

Financial Support: None

Poster 102

NEUROINTENSIVIST PERFORMED SIMULTANEOUS BEDSIDE TRACHEOSTOMY AND GASTROSTOMY IN NEURO-ICU SETTING: RESULTS SINCE PROGRAM INITIATION

Vinay Bangalore, Pretesh Patel, Paul McCarthy, Steve Conrad, L Keith Scott
Louisiana State University Health Sciences Center, Shreveport, LA, United States

Introduction:
Tracheostomy and gastrostomy are common procedures in patients suffering neurologic insults. We report our current data of these procedures performed simultaneously at bedside by a neurointensivist using percutaneous techniques.

Methods:
Database of all tracheotomies, gastrostomies and combined procedures performed by the Neuro-Critical Care team was retrospectively analyzed. Also, satisfaction surveys by nursing and house staff were employed to reassess and refine the service.

All procedures were completed at bedside in the neurointensive care or other intensive care units utilizing two critical care fellows, an intensive care nurse and a respiratory therapist under the direction of the neurocritical care attending.

The team followed each patient daily and reported any complication until discharge. Complications were categorized as major (requiring additional surgical intervention) or minor (no additional surgical intervention).

Results:
- To date the team has performed over 75 combined percutaneous tracheostomy and gastrostomiesin patients with primary neurologic pathology.
- There were two major complications and five minor complications reported.
- The neurologic pathology was mixed as was the,age and weight ranges

Conclusions:
- Combined tracheostomy and PEG tube placement can be performed safely by a neurointensivist
- Complications rates are low and no catastrophic events reported.
- Attendings, house staff and nursing supports the continuation of this programs
- The Neuro-Critical Care service now performs the majority of these procedures in our institution
- Based on the success of the service, non-neurologic related services are consulting the Neuro-Critical Care team to perform these procedures.

Financial Support: None

Poster 103

ANGIOPOIETIN I AND II LEVELS IN THE CSF OF PATIENTS WITH AND WITHOUT TRAUMATIC BRAIN INJURY

Sean Troxclair, Shashikant Patel, Paul McCarthy, Steven Conrad, L Keith Scott
Louisiana Stte University Health Sciences Center, Shreveport, LA, United States

Introduction:
Traumatic brain injury (TBI) is a complex disease state that includes disruption of the blood-brain barrier (BBB) and inflammatory changes. Angiopoietins are a family of growth factors integral in maintaining endothelial integrity and controlling inflammation. Angiopoietin I (Ang1) induces phosphorylation of the Tie2 ligand enhancing endothelial integrity. Angiopoietin II (Ang2) inhibits this action. In animal models, Ang2 is up regulated in TBI while Ang1 appears unchanged. Injury models in other tissues suggest that the ratio of Ang1 to Ang2 may be significant. Little is known about their role in humans with TBI. We collected CSF from patients with TBI (6 patients) and compared it controls (4 patients). Individual levels and ratios were compared.

Methods:
Each non-TBI CSF had < 5 cells/um, negative gram stain and cultures and normal protein and glucose levels. CSF samples were collected from the TBI group within 24 hours of drainage placement.. Ang1 and Ang2 were analyzed using an ELISA method and reported in pg/ml.

Results:
The levels of Ang1 in the control group and TBI group were not significantly different (p value of 0.37). There was significant increase in Ang-2 in the TBI group (p = 0.001). Comparing the ratios of Ang1 and Ang2, Ang1 was 6 times higher (1:6) in the control group than in the in the TBI group (0.95:1).

Conclusions:
This data correlates with animal data that shows an increase in Ang2 after TBI. This data further demonstrates a significant change in the ratio of Ang1 to Ang2 after TBI. What happens over time and how this relates to severity and prognosis is yet to be investigated. Restoring an Ang-1 to Ang-2 ratio to normal may be a therapeutic strategy worthy of investigation.

Financial Support: None

Poster 104

ANTIBIOTIC USE WITH EXTERNAL VENTRICULAR DRAINS AND INTRACRANIAL PRESSURE MONITORING DEVICES: A SURVEY OF CURRENT PRACTICES

Paul McCarthy, Cody Ford, Shashikant Patil, Steve Conrad, L Keith Scott
Louisiana State University HEalth Sciences Center, Shreveport, LA, United States

Introduction:
External ventricular drains (EVD) and intracranial pressure monitoring equipment are used frequently in neuroscience intensive care units. Because of the potential for the development of nosocomial infection prevention is important. Current practice varies from using no antibiotics to continuous antibiotics while devices are in place. There is inadequate foundation to support a particular practice.

Methods:
To define practice patterns, a survey was sent to >8,000 neurosurgeons, critical care, neurocritical care, and infectious diseases specialists. The same survey was also submitted to members of the Neurocritical Care Society but filtered to exclude redundancy. Ten percent of practitioners solicited responded to the survey.

Results:
Eighty seven percent of respondents were from North America, followed by Asia, Europe, and South America. Two-thirds practiced in academic centers and had > 10 years experience. Seventy seven percent of respondents were neurosurgeons, followed by neurocritical care, infectious diseases, and critical care.

Of the respondents:

- 2/3 use antibiotics (ABX) in some capacity.
- For EDV:
  - 48% use ABX for duration device in place.
  - 28% use peri-operative (< 24hours)
  - 24% use no ABX
- For other devices:
  - 38% use ABX for duration device in place.
  - 25% peri-operative ABX
  - 37% use none.
- One third of respondents use antibiotic coated EVD's
- There are differing practices among the specialties surveyed.

Conclusions:

- A majority of the respondents use ABX for the duration the devices are in place.
- There are differences in practice among respondents based on specialty, geography, years of practice and type of practice.
- Eighty percent of respondents think a randomized trial comparing ABX strategies is needed.

Financial Support: None

Poster 105

COAGULATION ABNORMALITIES ASSOCIATED WITH THERAPEUTIC HYPOTHERMIA

Muhammad Taqi, Thomas Wolfe, Michael Abraham, Dhimant Dani, John Lynch, Michel Torbey
Medical College of Wisconsin, Milwaukee/WI, United States

Introduction:

Therapeutic hypothermia (TH) has been increasingly used in the neuroICU. Complications of TH include immunosupression, cardiac arrhythmias, metabolic/electrolyte alteration and coagulopathy. Effects on the coagulation cascade include dysfunction of platelets, thrombocytopenia, and alterations in the coagulation cascade, resulting in mild bleeding diathesis. Most TH trials have reported very low risk of bleeding associated with treatment.

Methods:

Retrospective chart review of prospectively identified patients. Per institutional protocol, patients were cooled to a nadir of 33^0 C. Baseline Prothrombin Time (PT), Partial Thromboplastin Time (PTT) and platelet count were obtained and followed at least daily during TH and rewarming. Data was evaluated for the development of new abnormalities following TH induction.

Results:

Thirty six patients received TH for various clinical indications, including cardiac arrest and intracranial pressure control (related to subarachnoid hemorrhage, intracerebral hemorrhage, ischemic stroke, and traumatic brain injury). Duration of goal temperature maintenance varied from 4 hours to 120 hours. After induction of TH, 6/36 (16.7%) showed abnormal PT, 5/36 (13.9%) had abnormal PTT, and 4/36 (11.1%) patients developed thrombocytopenia (platelet count <150,000/μL). In those developing abnormalities, normalization was not seen for any parameter within 48hrs of rewarming. Overall, 22% of the patients demonstrated some form of new abnormality following TH, none of which had clinically significant bleeding episodes. Overall, 30-day mortality was 72%; no mortality was attributable to TH.

Conclusions:

We concur with the previously reported findings that TH is associated with coagulation abnormalities. A high proportion of patients were found to demonstrate such abnormalities, which persisted following rewarming; the exact clinical significance of these findings is not clear. In addition to this standard laboratory testing, changes in radiographic imaging may serve as a more sensitive adjunctive measure to evaluate the significance of TH related coagulopathy.

Financial Support: None

Poster 106

REVERSAL OF TRANSTENTORIAL HERNIATION WITH HYPERTONIC SALINE IN PATIENTS WITH RENAL FAILURE

Karen Hirsch[1], Todd Spock[1], Robert Stevens[1], Matthew Koenig[2], Romergryko Geocadin[1]
[1]Johns Hopkins University School of Medicine, Baltimore, MD, United States, [2]The Queens Medical Center, Honolulu, HI, United States

Introduction:
Transtentorial herniation (TTH) is a clinical syndrome consisting of pupillary dilatation with loss of pupillary light reflex and decreased level of consciousness in the setting of a large intracranial mass lesion. Reversal of TTH is defined as return of pupillary light reflex with or without immediate improvement in level of consciousness. The role of renal function in the mechanism of hypertonic therapy remains unclear. We evaluated the efficacy and safety of 23.4% saline in TTH in patients with end-stage renal disease (ESRD) on hemodialysis.

Methods:
Patients with clinically defined TTH and ESRD on hemodialysis treated with 23.4% saline (30 to 60 mL) were included in the analysis of a retrospective cohort.

Results:
Of 254 subjects over 7 years, we identified 6 patients with ESRD that had 11 TTH events. Lesions were related to stroke (n=1), intracerebral hemorrhage (n=4), and subdural hemorrhage (n=2). All patients received a 23.4% saline bolus, along with mannitol (91% of events), hypertonic saline maintenance fluids (82%), ventriculostomy (n=1), and hemicraniectomy (n=1). Clinical reversal of TTH occurred in 10/11 events (91%); 2 of 6 patients survived to discharge. In 4 patients, ICP recording of 6 TTH events showed a reduction from ICP of 41$\pm$ 3.8 mmHg (mean $\pm$ SEM) with TTH to 20.8 $\pm$ 3.9 mmHg (p=0.05) one hour after the 23.4% saline bolus. Serum sodium increased from 141.4 mmol/L to 151.1 mmol/L 24 hours after 23.4% saline bolus (p=0.001). No patients were undergoing hemodialysis at the time of the TTH event, and the post-infusion serum creatinine did not change.

Conclusions:
Treatment with 23.4% saline was associated with rapid clinical reversal of TTH and reduction in ICP in this small cohort of patients with ESRD. This finding supports that hypertonic saline may be effective in cases of ESRD.

Financial Support: None

Poster 107

SPONTANEOUS HYPERVENTILATION AND VASOSPASM IN PATIENTS WITH SUBARACHNOID HEMORRHAGE: A RETROSPECTIVE COHORT STUDY

Othman Solaiman, Niall Ferguson, Jeffrey M Singh
University of Toronto, Toronto, Canada

Introduction:
Intubated patients with subarachnoid hemorrhage (SAH) may spontaneously hyperventilate despite minimal ventilatory support. The impact of this is unclear, although hypocapnea may be harmful in traumatic brain injury. We set out to determine the incidence of spontaneous hyperventilation in patients with SAH and its association with clinical outcomes.

Methods:
We identified consecutive, intubated patients with spontaneous SAH from clinical databases (2006-2008). Demographics, clinical and ventilation data (for the first 10 days post-bleed) were collected. Hypocapnea was defined as an arterial pCO2 <= 35 mmHg. Primary outcomes were (1) the presence of symptomatic vasospasm (defined by both angiographic vasospasm and clinical symptoms); (2) death in the intensive care unit. Associations between hypocapnea and outcomes were explored with multivariate analysis.

Results:
We identified 99 patients with SAH and a median duration of ventilation of 4 days [IQR 1-12]. Hypocapnea was observed on at least one day in 91 patients (92%), and 68 patients (69%) had at least 1 pCO2 <30mmHg. All hypocapnea was associated with alkalemia. Ventilatory support was minimal (CPAP or PS 5cm H2O) in 68% of hypocanea measurements. Sedation normalized pCO2 in 63% of cases, and use of neuromuscular blockade was rare. Median duration of hypocapnea (at least one pCO2 <=30mmHg each day) was 3 days [IQR 2-6]. Duration of hypocapnea was associated with increased odds of symptomatic vasospasm (OR 1.24 for each day with hypocapnea; p=0.02) after adjusting for Fisher CT grade. Duration of hypocapea was not associated with ICU mortality after adjustment for APACHE II and WFNS grade (p=0.88).

Conclusions:
The incidence of spontaneous hyperventilation is high in intubated patients with SAH, despite minimal ventilator support. Duration of hypocapnea was independently and statistically significantly associated with symptomatic vasospasm.

Financial Support: None

Poster 108

EVALUATION OF A CHECKLIST TO IMPROVE CONTENT OF FAMILY CONFERENCE DISCUSSIONS IN THE NEUROCRITICAL CARE UNIT

Kristi Tucker[1], Michael Williams[2], Anastasia Shevchenko[3], Wendy Ziai[3]
[1]Wake Forest University Health Sciences, Winston Salem, NC, United States, [2]Sinai Hospital of Baltimore, Baltimore, MD, United States, [3]Johns Hopkins University School of Medicine, Baltimore, MD, United States

Introduction:
Few studies have evaluated physician-family interactions and decision-making in the Neurocritical Care Unit (NCCU). We sought to determine if the ICU team's use of a structured checklist for family conferences (FC) would improve family satisfaction.

Methods:
We conducted a prospective pilot pre- and post-intervention study. We designed an 18-item checklist of key content for FC conducted with the intent of making significant patient management decisions. Phase I was observational, with a nurse covertly documenting the key content covered during the FC. Phase II was interventional. We asked the ICU team to use the checklist during FC to cover all key content. A family member and the ICU team member completed an immediate post-FC written survey, and the FS-ICU 24, a family satisfaction survey, was mailed to the family 3 months after NCCU discharge.

Results:
12 families enrolled (7 Phase I; 5 Phase II), with patient age 57 ± 20 years, APACHE III score 66 ± 30 and NCCU LOS 20 ± 20 days. 8 patients died (4-PI; 4-PII). Median key content covered was 10 items in Phase I and 12 items in Phase II (p=0.5). In Phase II, ICU team member self-report of key content was higher than documented content (18 vs. 12 items; p=0.1). Post-FC survey scores increased from 87.5 (Phase I) to 100 (Phase II) (p=0.4). The FS-ICU 24 decision-making subscale median score was 92.5 in Phase I and 85 in Phase II (p=0.8).

Conclusions:
Use of a FC checklist in the NCCU marginally improved coverage of key elements in family conferences, however post FC family satisfaction was improved. Further evaluation of the influence of checklists on patient outcomes and family satisfaction for family conferences in the NCCU is warranted.

Financial Support: None

Poster 109

A SLOWLY EXPANDING INTRACRANIAL MASS CAUSES A THRESHOLD CHANGE IN SOMATOSENSORY EVOKED POTENTIALS (SEPS)

H. Adrian Püttgen[1], Jai Madhok[3], Xiaofeng Jia[2], Anil Maybhate[2]
[1]Johns Hopkins University Medical Institutions, Baltimore, MD, United States, [2]Johns Hopkins University School of Medicine, Baltimore, MD, United States, [3]Johns Hopkins University, Baltimore, MD, United States

Introduction:
SEP's represent the brain's response to sensory electrical stimulus. Current clinical methods require averaging a large number of SEP waveforms for meaningful prognostication. Automated SEP monitoring could be used as a non-invasive bedside tool for conditions that severely affect somatosensory conduction due to elevating intracranial pressure (ICP) such as cerebral oedema or intracerebral haemorrhage.

Methods:
3 adult Wistar rats were used in this pilot study. To model intracranial hypertension, a latex micro-balloon (300µL maximum volume) was surgically inserted into the epidural space via a burr hole on the left hemisphere (2mm off the sagittal suture). Using a micro-pump, the balloon was slowly inflated with water at 20µL/min for two 5min periods with a 10min pause. SEPs were recorded after electrically stimulating the hind limb at 0.5Hz. ICP was recorded using a transducing catheter inserted in the subdural space over the right hemisphere.

Results:
Balloon inflation was accompanied by a steady increase in the ICP. The increase in ICP beyond a certain level was accompanied by the sudden disappearance of SEP's within a few seconds (2 to 3 sweeps). In our pilot experiments, the peak to peak amplitude of the SEP dropped steeply from about 496$\pm$65µV to 115$\pm$52µV before a complete and sudden disappearance when the balloon volume reached approx. 200$\pm$27µL.

Conclusions:
This pilot study demonstrates the effect of an intracranial mass on the integrity of the somatosensory pathway. The finding of a threshold of lesion magnitude after which further expansion causes a dramatic disappearance of SEP points to the possibility of using continuous SEP for monitoring rapidly evolving mass lesions such as cerebral oedema or intracerebral haemorrhage.

Financial Support: None

Poster 110

PREDICTORS OF INCREASED INTRACRANIAL PRESSURE IN INTRACEREBRAL HEMORRHAGE

Venkatesh Aiyagari, Troy Munson, Anjana Nair, Weihua Gao, Fernando Testai
University of Illinois at Chicago, Chicago, IL, United States

Introduction:
Patients with intracerebral hemorrhage (ICH) may have increased intracranial pressure (ICP) due to hematoma volume, hydrocephalus or cerebral edema. The incidence and predictors of elevated ICP in ICH are not known. Extrapolating from traumatic brain injury guidelines, ICP is commonly monitored in ICH patients with a Glasgow Coma Scale (GCS) score of ≤8.

Methods:
We conducted a retrospective analysis of all patients with a non-traumatic supratentorial ICH admitted to the University of Illinois at Chicago over a 2-year interval (Oct 2006-Oct 2008). We compared subjects with/without an intraventricular catheter (IVC) and in the former group, with/without early elevated ICP (EEICP) (<48 hours). We conducted a univariate analysis of the following variables: demographics, etiology and location of ICH, time from onset to admission and to external ventricular drain (EVD) placement, GCS score on admission and at EVD placement, ICH volume, hydrocephalus score, intraventricular hemorrhage (IVH) score, length of stay and hospital outcome. We then conducted a multivariate logistic regression analysis including variables with a $p<0.25$ in the univariate analysis.

Results:
140 subjects were included. An IVC was placed in 52 (41/55 GCS ≤8, and 11/85 GCS >8). Sixteen patients had EEICP. On univariate analysis, the median GCS was significantly different (4 vs. 7, $p=0.007$) between patients with or without EEICP while demographics, etiology and location, ICH volume, hydrocephalus score and IVH score were not. On multivariate analysis, variables associated with EEICP included the GCS at EVD placement (OR 0.23, 95% CI 0.06-0.86) and ICH volume (OR 1.02, 95% CI 1.00-1.037).

Conclusions:
The GCS score is an independent predictor of EEICP in ICH. The interpretation of these results needs consideration of the fact that the IVC catheter, in addition to monitoring, also treats elevated ICP.

Financial Support: None

Poster 111

THE EPIDEMIOLOGY AND IN-HOSPITAL OUTCOME OF ACUTE KIDNEY INJURY IN THE SETTING OF ACUTE STROKE

Gwendolyn Lynch, J. Javier Provencio, Rebecca Forkapa, Peter Rasmussen
Cleveland Clinic Foundation, Cleveland, Ohio, United States

Introduction:
Acute Kidney Injury (AKI) is associated with increased morbidity and mortality in acutely ill patients. Our understanding of the epidemiology and in-hospital outcome of AKI in the setting of acute stroke is lacking. We assessed the prevalence of AKI at admission and discharge for acute stroke, identified risk factors, and investigated outcomes (hospital discharge disposition(HDD) and length of stay (LOS)).

Methods:
A single-center retrospective analysis of consecutive admissions of patients with acute ischemic and hemorrhagic stroke during calendar years 2007 and 2008. AKI was defined as serum creatinine $\geq$1.5. Serum creatinine on admission and discharge was assessed. Risk factors were identified using the cofactors of age, gender, race, BUN value and history of hypertension, diabetes mellitus, dyslipidemia, coronary artery disease, and stroke. HDD was dichotomized to poor (death, transfer to a skilled nursing facility, and transfer to a ventilatory weaning facility) and good (transfer to an acute rehabilitation center and discharge to home).

Results:
Of the 628 patients with acute ischemic and hemorrhagic stroke 9% of patients had a history of renal disease on admission, and 8% had newly diagnosed renal disease on admission. The remaining results represent analysis of individuals with no prior history of renal disease (569 patients). There was a 24% net increase from admission to discharge in number of patients with AKI. Multivariate analysis demonstrated that men, and patients with history of hypertension were at significant risk for AKI on admission. While men, non-whites, and older age patients showed an increased risk for AKI at discharge. AKI at discharge was associated with a 2.4 relative risk of poor discharge disposition ($p<0.001$). No significant relation to length of stay was identified(AKI 9 days vs. Non-AKI - 8 days).

Conclusions:
Renal failure is prevalent in acute stroke patients at time of admission and is associated with poor outcome at discharge. Consistent implementation of renal protective management, and more broad use of drugs with potential renal protective properties may result in better outcome.

Financial Support: None

Poster 112

INTRACORTICAL EEG FOR THE DETECTION OF VASOSPASM IN PATIENTS WITH POOR-GRADE SUBARACHNOID HEMORRHAGE

R. Morgan Stuart[1], Allen Waziri[2], David Weintraub[1], Michael Schmidt[1], Luis Fernandez[1], Raimund Helbok[1], Pedro Kurtz[1], Kiwon Lee[1], Neeraj Badjatia[1], Ron Emerson[1], Stephan Mayer[1], E. Sander Connolly[1], Lawrence Hirsch[1], Jan Claassen[1]

[1]Columbia College Of Physcians and Surgeons, New York City, NY, United States, [2]University of Colorado Health Sciences Center, Denver, CO, United States

Introduction:

We studied the feasibility of intracortical electroencephalography (ICE) including quantitative EEG (qEEG) analysis for the detection of vasospasm in a series of poor-grade SAH patients.

Methods:

From a consecutive series of SAH patients who underwent ICE placement, the alpha/delta ratio (8-13 Hz/1-4 Hz; ADR) was calculated at twenty second intervals from the ICE and scalp EEG recordings. Percent changes between averaged values over 4-6 hours of the baseline EEG and the EEG prior to angiography were calculated. The entire continuous qEEG recording for each patient was then reviewed to determine optimal automated alarm criteria.

Results:

ICE recordings revealed an improved signal-to-noise ratio when compared to surface EEG recordings. The ADR calculated from the ICE decreased between baseline EEG and follow-up EEG on average by 42% (mean ADR decrease 0.56±0.07 to 0.32±0.03) for those with vasospasm (N=3) compared to 17% (0.62±0.06 to 0.51±0.03) for those without vasospasm (N=2). A sustained decrease in the ADR by at least 25% from baseline for a minimum of 4 hours occurred in patients with vasospasm 1-3 days before angiographic confirmation of vasospasm. This was not seen in patients without angiographic vasospasm.

Conclusions:

EEG recordings from ICE are promising to reliably detect vasospasm in severely brain injured SAH patients. Absence of artifact allows for automated qEEG analysis of ICE recordings.

Financial Support: None

Poster 113

HEAD-OF-BED ELEVATION IN THE NEUROCRITICAL CARE SETTING: RECOGNITION BY HEALTH-CARE PROFESSIONALS

Thiago Coelho, Joana Carvalho, Manuel Cunha e Sa
Hospital Garcia d'Orta, Almada, SET, Portugal

Introduction:
Raising the head-of-bed (HOB) js a very important step in taking care of critically ill patients, particularly in the neurocritical care, as it influences abdominal pressure, decreases incidence of pneumonia associated with aspiration secondary to the decrease in gastroesophageal reflux and reduces intracranial pressure, improving cerebral perfusion pressure. Nevertheless, this relatively simple maneuver is still not widely applied.

Methods:
After explaining the goals of raising the HOB individually, each health-care worker (HCW) attending in our 8-bed neurocritical care service was requested to position the HOB between 30 and 45 degrees. As our beds are able to measure HOB angulation, it was later conferred. After this simple procedure, HCW were again explained and a folder was distributed. It contained a questionnaire and pictures of HOB at 15, 30, 45 and 60 degrees. Later on, they were again requested to position HOB at the adequate position.

Results:
The first poll revealed that 22 out of 23 participating nurses did not or only partially knew the reasons HOB should be positioned between 30 and 45 degrees and almost 70% of the attempts resulted in failures. Among physicians, 87.5% only partially knew the reasons. They could rightly position HOB in 56% of the attempts. After the questionnaire, every and each one of the HCW could name the reasons of HOB positioning and almost 90% of the attempts resulted in right results.

Conclusions:
HCW should be constantly reminded the importance of simple tasks in the care of neurocritical patients. HOB elevation should not be regulated by random trials. Automatic beds are very important devices, particularly when some features are present, such as HOB angulation and, even though it could seem an expensive device, it will finally allow cheaper and less risky expenditures.

Financial Support: None

Poster 114

PRES: IS IT JUST ABOUT THE BLOOD PRESSURE?

Ryan Merrell[1], Jay Mandrekar[1], Jennifer Fugate[1], Alan Yee[1], Osman Kozak[2], Olayemi Durosaro[1], Alejandro Rabinstein[1]

[1]Mayo Clinic, Rochester, MN, United States, [2]University of Minnesota, Minneapolis, MN, United States

Introduction:

Posterior reversible encephalopathy syndrome (PRES) can be caused by hypertensive crisis and is often associated with rapid fluctuations in blood pressure (BP). The role of these BP changes in the pathogenesis of PRES has not been formally studied. We sought to analyze the relationship between blood pressure (BP) fluctuations and the occurrence of PRES.

Methods:

Consecutive hospitalized patients who developed PRES were compared with randomly selected controls matched for age, gender, and history of hypertension. Systolic BP (SBP) and diastolic BP (DBP) were collected every 2 hours for 48 hours before developing PRES symptoms. SBP, DBP, mean arterial pressure (MAP), and pulse pressure (PP) changes over a 48-hour window was summarized for each individual by calculating an M value as described by Service et al (1970). M values were compared using Wilcoxon signed rank test. Tests were two sided and p values less than 0.05 were considered statistically significant.

Results:

We analyzed the BP profiles in 25 cases of PRES and 25 controls. Median age of PRES patients was 54 years (range 31-72). 14 of them (56%) had pre-existing hypertension. Hypertensive encephalopathy was considered the etiology of PRES in 13 patients (52%). At symptomatic onset of PRES, mean SBP was 182 ± 20 mmHg (range 218-145), DBP 95 ± 16 mmHg (range 134-62), MAP 124 ± 15 (range 152-93), and PP 87 ± 18 (range 123-46). While BP was higher in PRES cases, hypertension severity was variable and BP fluctuations were not significantly more common than in controls (p values for SBP, DBP, MAP, PP were 0.38, 0.79, 0.25, 0.73, respectively).

Conclusions:

BP fluctuations do not appear to be more common in hospitalized patients who develop PRES compared with matched controls. Other predisposing factors must therefore contribute to the development of PRES.

Financial Support: None

Poster 115

MONITORING CEREBRAL OXYGEN SATURATION IN ACUTE STROKE PATIENTS

Xiao Androulakis[1], Mukta Panda[2], Kent Hutson[2], Thomas Devlin[1]

[1]NSLIJ-Albert Einstein College of Medicine LJM, Manhasset, NY, United States, [2]University of Tennessee COM at Chattanooga, Chattanooga, TN, United States

Introduction:

Technologies allowing emergent detection of focal cerebral hypoxia would be of great utility in the treatment of ischemic stroke by facilitating diagnosis, tracking reperfusion, and identifying re-thrombosis. Non-invasive brain oxymetry using Near-Infrared Reflectance Spectroscopy (NIRS) technology incorporated into the INVOS device (Somanetics, Troy, MI), provides direct measurement of regional oxyhemoglobin saturation (rSO2) within the cerebral cortex. This study utilized the INVOS device to determine the predictive value of cortical rSO2 monitoring in the assessment of ischemia in patients presenting with large hemispheric strokes.

Methods:

Patients exhibiting acute ischemic strokes involving proximal MCA or ICA occlusions on CT angiography were enrolled prospectively. The INVOS device was applied according to the manufacturer's recommendations. rSO2 data was recorded at 10 s intervals for at least 10 hr in each patient. Concomitant vital signs, HGB, oxygen saturation, PaO2, and PaCO2 were collected. Three out of 5 patients underwent emergent cerebral angiography. A neuroradiologist, blinded to the INVOS results, evaluated all CT, CT perfusion, and cerebral angiography studies.

Results:

CT perfusion imaging confirmed large hemispheric strokes in all patients. Mean time from symptom onset to start of rSO2 monitoring was 7 hours (range =3-12). Data analysis consistently demonstrated mean rO2 saturation levels on the ischemic hemisphere to be either the same or higher than that of the non-ischemic hemisphere. rSO2 levels were independent of BP, HGB, oxygen saturation, PaO2 or PaCO2 levels. Cerebral angiography demonstrated significant collateral flow over the affected hemisphere despite deep large vessel occlusions.

Conclusions:

These findings suggest that NIRS technology has limited utility in the assessment of patients with acute ischemic stroke. Patency of cortical collaterals and increased tissue oxygen extraction during ischemia, among other factors, may offset a decrease of cortical rSO2 within the affected hemisphere.

Financial Support: None

Poster 116

INCIDENCE AND RISK FACTORS FOR VENOUS THROMBO EMBOLISM IN NEURO-CRITICAL CARE PATIENTS

ABHIJIT LELE, ROBERT BUCHMANN, STACY SMITH
UNIVERSITY OF KANSAS MEDICAL CENTER, KANSAS CITY, KS, United States

Introduction:

Venous thromboembolism is a common problem in critically ill patients. Neurosurgical patients even though at higher risk; often do not receive timely pharmacological thromboprophylaxis for fear of bleeding risks. Recent literature points towards the safety and efficacy of early prophylaxis (SCD's + Heparin/Lovenox); however this has not been tested extensively in a randomized controlled trial.

Methods:

A retrospective chart review of patients with a primary diagnosis of subarachnoid hemorrhage (SAH), intracerebral hemorrhage (ICH), and subdural Hematoma (SDH) admitted from January 2008 to June 2009 was conducted for ICD-9 codes of DVT and or PE, and for presence of associated risk factors. All patients received intermittent compression devices (SCD's) on all patients from time of admission to time of discharge, surveillance doppler ultrasound evaluation of both lower extremities once every week, and doppler screening of symptomatic upper extremities.

Results:

Risk Factors for Venous Thromboembolism

Risk Factors		Total (n=18)		SAH (n=6)		ICH (n=7)		SDH (n=5)	
		n	(%)	n	(%)	n	(%)	n	(%)
Age ≥ 60 years		9	50	4	67	2	29	3	60
Gender	Male	11	61	3	50	4	57	4	80
	Female	7	39	3	50	3	43	1	20
BMI ≥ 30		8	44	0	0	6	86	2	40
Smoking		5	28	2	33	1	14	2	40
CAD		3	17	1	17	1	14	1	20
CHF		2	11	1	17	0	0	1	20
Limb Paralysis	≤2 Limbs	10	56	3	50	4	57	3	60
	4 Limbs	4	22	1	17	2	29	1	20
Prior stroke		1	6	1	17	0	0	0	0
Craniotomy		10	56	6	100	1	14	3	60
Location of DVT	Upper	10	56	3	50	3	43	4	80
	Lower	8	44	4	67	2	29	2	40
	Both	2	11	1	17	0	0	1	20
Central Lines		12	67	4	67	6	86	2	40
PICC Line		6	33	3	50	1	14	2	40
PICC Congruent w/ DVT site		6	100	3	100	1	100	2	100
Day of DVT Diagnosis		10.97		11		11.42		10.5	

Overall incidence of DVT was 6.92% (n=260). The incidence of DVT was 8.69% in SAH, 5.42% in ICH, and 8.06% in SDH. The incidence of PE was 0.76%.The presence of intraventricular hemorrhage was seen in 83.3% of patients with SAH who had DVT.

Conclusions:

This study shows almost double the incidence of DVT than reported in the recent literature. PICC line and central lines were associated with higher incidence of DVT.The timing of the diagnosis of DVT falls in a time window where intracranial bleeding risks from anti-coagulation are far less than in the acute stage.

This study will provide us a unique cohort of patients whom we can compare in a prospective manner to patients who will receive subcutaneous heparin along with SCD's in the future, since we are changing our policy to implement heparin thromboprophylaxis.

Financial Support: None

Poster 117

PROGNOSTIC IMPORTANCE OF INTRAVENTRICULAR HEMORRHAGE VOLUME IN PATIENTS WITH RUPTURED CEREBRAL ANEURYSMS

Andreas Kramer[1], Ivan Mikolaenko[2], Bart Nathan[2]
[1]University of Calgary, Calgary, AB, Canada, [2]University of Virginia, Charlottesville, VA, United States

Introduction:
The presence of intraventricular hemorrhage (IVH) is predictive of worse outcomes following aneurysmal subarachnoid hemorrhage (SAH) [1]. However, the amount of IVH can vary considerably. No previous studies have assessed the association between actual hematoma volume (in ml) and subsequent complications or outcomes.

Methods:
We performed a cohort study involving 152 consecutive patients with concomitant SAH and IVH. With investigators blinded to subsequent events, CT scans were graded using two systems. First, to determine the volume of IVH, we used the IVH Score, recently shown to correlate exceptionally well with computerized volumetric assessment [2]. Second, to examine the relative amount of subarachnoid blood, we applied the SAH component of the Hijdra Score [3]. Using logistic regression to adjust for SAH Score and other potential confounders, we assessed the association between IVH volume and poor neurological outcomes (Glasgow Outcome Scale 1-3), as well as symptomatic vasospasm and delayed infarction.

Results:
Compared with patients who had a favourable outcome, those with poor outcomes had significantly larger baseline IVH volumes (mean 3.8 ml vs. 11.8 ml, p=0.001). In the multivariable analysis, IVH volume remained an independent predictor of poor neurological outcome (OR per ml: 1.11, 1.04-1.18, p=0.01). Patients in the highest quartile for IVH volume were far more likely to progress to poor outcomes compared with those in the lowest quartile (OR 4.09, 1.32-12.65, p=0.01). In contrast, IVH volume was not associated with either vasospasm or delayed infarction. Interobserver agreement in the determination of IVH Score was good.

Conclusions:
Volume of IVH is a strong, independent predictor of death and poor neurological recovery, even when one adjusts for the amount of concomitant subarachnoid blood. Future studies should assess whether measures aimed at accelerating the clearance of IVH (e.g. intraventricular thrombolysis) can modify this association.

Financial Support: None

Poster 118

TIGHT GLUCOSE CONTROL, CEREBRAL INFARCTION, AND FUNCTIONAL OUTCOME AFTER SUBARACHNOID HEMORRHAGE

Andrew Naidech, Kimberly Levasseur, Storm Leibling, Rajeev Garg, Michael Shapiro, Michael Ault, Sherif Afifi, Hunt Batjer
Northwestern University, Chcago, IL, United States

Introduction:
While many ICUs have implemented protocols for tight glucose control, there are few data on relative hypoglycemia and neurologic outcomes. We addressed the hypothesis that lower glucose leads to worse neurologic outcomes after subarachnoid hemorrhage (SAH).

Methods:
One hundred seventy-two (172) consecutive patients were treated with a protocol designed to achieve serum glucose 80 – 110 mg/dL. We prospectively ascertained patients on admission and recorded medical history and clinical events. Glucose measurements from the hospital laboratory were electronically retrieved. (A separate analysis of bedside glucose results found similar results.) Cerebral infarction was prospectively documented with neuroimaging. Outcomes were assessed with the modified Rankin Scale (mRS) at 14 days, 28 days and 3 months.

Results:
Worse neurologic injury at admission ($P<0.001$) and a history of diabetes ($P=0.002$) were associated with increased glucose variance. Patients with radiographic cerebral infarction (81 ± 15 vs. 87 ± 16 mg/dL, $P=0.02$), symptomatic vasospasm (78 ± 12 vs. 84 ± 16 mg/dL, $P=0.04$) and angiographic vasospasm (79 ± 14 vs. 86±16 mg/dL, $P=0.01$) had lower nadir glucose, but maximum and mean glucose were not different. Glucose < 80 mg/dL was earlier and more frequent in patients with worse functional outcome ($P<0.001$). Progressive reductions in nadir glucose were associated with increasing functional disability at 3 months ($P=0.001$) after accounting for neurologic grade and mean glucose. Severe hypoglycemia (<40 mg/dL) occurred in one patient.

Conclusions:
In patients with SAH, nadir glucose below the <80 mg/dL is associated with cerebral infarction, vasospasm, and worse functional outcomes in multivariate models. Protocols for target glucose 80 – 110 mg/dL effectively control hyperglycemia, but may place patients with SAH at risk for vasospasm, cerebral infarction and poor outcome even when severe hypoglycemia does not occur.

Financial Support: None

Poster 119

ANTICONVULSANT USE AND OUTCOMES AFTER INTRACEREBRAL HEMORRHAGE

Andrew Naidech, Rajeev Garg, Storm Liebling, Kimberly Levasseur, Micheal Macken, Stephan Schuele, Hunt Batjer
Northwestern University, Chicago, IL, United States

Introduction:
There are few data on the effectiveness and side effects of anti-epileptic drug (AED) therapy after intracerebral hemorrhage (ICH). We tested the hypothesis that AED use is associated with more complications and worse outcome after ICH.

Methods:
We prospectively enrolled 98 patients with ICH and recorded AED use as either prophylactic or therapeutic along with clinical characteristics. AED administration and free phenytoin (PHT) serum levels were retrieved from the electronic medical record. Patients with depressed mental status underwent continuous EEG monitoring. Outcomes were measured with the NIH Stroke Scale and modified Rankin Scale (mRS) at 14 days or discharge, and the mRS at 28 days and 3 months. We constructed logistic regression models for poor outcome at 3 months with a forward-conditional model.

Results:
Seven (7%) patients had a clinical seizure, five on the day of ICH. PHT was associated with more fever (P=0.03), worse NIH Stroke Scale at 14 days (23 [9-42] vs. 11 [4-23], P=0.003) and worse mRS at 14 days, 28 days and 3 months. In a forward-conditional logistic regression model PHT prophylaxis was associated with an increased risk of poor outcome, OR 9.8 (1.4-68.6) P=0.02, entering after admission NIH Stroke Scale and age. Excluding patients with a seizure did not change the results. Levetiracetam was not associated with demographics, seizures, complications, or outcomes.

Conclusions:
PHT was associated with more fever and worse outcomes after ICH.

Financial Support: None

Poster 120

CLINICAL AND RADIOLOGICAL FEATURES OF STATUS EPILEPTICUS IN PRES

Jennifer Fugate, Daniel Claassen, Alejandro Rabinstein
Mayo Clinic, Rochester, MN, United States

Introduction:
Posterior reversible leukoencephalopathy syndrome (PRES) is characterized by seizures, headache, encephalopathy and visual disturbances associated with reversible vasogenic edema on brain imaging. Status epilepticus (SE) has been infrequently described as an initial manifestation of PRES. The clinical and radiological features of patients with PRES and SE have not been well described.

Methods:
Patients with SE were identified from a mostly prospectively collected database of patients (n=113) with PRES. We collected data on general demographics, clinical presentation, history of epilepsy, peak systolic and diastolic blood pressures, and predisposing conditions. Brain MRIs were analyzed independently by two neuroradiologists for lesion location and distribution, severity, presence of hemorrhage and presence of restricted diffusion.

Results:
Of 113 patients with PRES, 20 (18%) presented with SE. Only 2 had a prior history of epilepsy. Mean peak SBP was 199 mm Hg (160-268) and mean DBP was 109 mm Hg (60-144). Etiologies of PRES included hypertension (n=13), cytotoxic medications (n=3), sepsis (n-2) and other (n=2). Renal failure was present in 11 (55%) cases and 5 (25%) had pre-existing chronic kidney disease. Twelve patients (60%) had a history of autoimmunity. Among 19 patients with brain MRI, 8 (42%) demonstrated mild edema and 11 (58%) had moderate-severe edema. The cortex was involved in only 2 patients (11%). Almost all had edema in the parietal-occipital region (n=18, 95%). When compared with the rest of our PRES cohort, we did not identify any significant clinical or radiological predictors of SE.

Conclusions:
SE is not an infrequent presentation of PRES. Its occurrence is not correlated with the severity of radiologic edema and the great majority of patients actually lack cortical involvement. Recognition that PRES may present with SE is important because, besides anticonvulsants, appropriate treatment requires identifying and treating the underlying cause of PRES.

Financial Support: None

Poster 121

THE EFFECTS OF FLUID BALANCE AND VASOPRESSOR USE IN PATIENTS WITH SEVERE TRAUMATIC BRAIN INJURY

Jeff Fletcher MD, Karen Bergman RN
Bronson Methodist Hospital / Michigan State University, Kalamazoo, MI, United States

Introduction:

Hypervolemia is known to lead to peripheral and pulmonary edema however the effect on intracranial pressure (ICP) following traumatic brain injury (TBI) is unclear. There is no direct evidence in humans linking hypervolemia independently to elevated ICP. Compelling evidence suggests that fluid restriction should be avoided and limited evidence suggest significant hypervolemia may be associated with worse outcome following TBI [1]. The use of fluids and vasopressors to elevate cerebral perfusion pressure (CPP) >70mmHg has been to shown to increase the risk of pulmonary complications (but not clearly effect ICP) following TBI and is not recommended by guidelines [2,3,4]. Despite this, some ancillary monitoring protocols recommend elevating CPP to treat episodes of cerebral hypoxia.

Methods:

Retrospective observational cohort study of severe TBI patients admitted over a 2-year period to a Neuro-Trauma unit. Data extracted: Characteristics; fluid balance; development of refractory intracranial hypertension (RIH); pulmonary complications; use of vasopressors; ancillary monitoring. Patients with unsurvivable injuries, early withdrawal of care or the development of refractory intracranial hypertension (RIH) within 24 hours were excluded.

Results:

Forty-one patients with mean age 35.2; 73% male; 73% automobile accidents; 61% polytrauma; average best GCS of 6.4 (a subgroup presented with higher GCS with declined secondary to neurological injury). RIH was associated with lower fluid balance but not hypervolemia (overall Q1= 50%, IQ = 21%, Q4 =27%). An early low fluid balance and hypervolemia both are associated with more pulmonary complications. The use of vasopressors, and to a lesser extent Licox monitoring is associated with a higher incidence of pulmonary complications and possibly RIH. [Tables 1-4]

Table 1: Day 1

Characteristic	Q1	IQ	Q4
Fluid Balance: mean (range) in Liters	0.4 (-1.5 - 0.9)	2.3 (1 - 5.6)	10.5 (6.1 – 20.5)
Average best GCS (prior to any neurological decline)	7	6.2	5.9
Use of vasopressors for >48 hrs AND >12 hrs for CPP >70mmhg	40% (4/10)	25% (5/20)	60% (6/10)
Licox Monitoring	40% (4/10)	20% (4/20)	45% (5/11)
RIH	40% (4/10)	20% (4/20)	45% (5/11)
Pulmonary complications	70% (7/10)	40% (8/20)	73% (8/11)

Table 2: Day 3

Characteristic	Q1	IQ	Q4
Fluid Balance: mean (range) in Liters	1.6 (-0.7 – 2.7)	6.3 (3.1 – 9.5)	17.3 (9.7 – 29.1)
Average best GCS (prior to any neurological decline)	5.3	6.7	6.4
Use of vasopressors for >48 hrs AND >12 hrs for CPP >70mmhg	40% (4/10)	25% (5/20)	55% (6/11)
Licox Monitoring	30% (3/10)	25% (5/20)	45% (5/11)
RIH	40% (4/10)	25% (5/20)	36% (4/11)
Pulmonary complications	30% (3/10)	55% (11/20)	82% (9/11)

Table 3: Day 7

Characteristic	Q1	IQ	Q4
Fluid Balance: mean (range) in Liters	1 (- 1.7 – 3.4)	11 (4.2 – 17.4)	21.2 (17.7-25.7)
Average best GCS (prior to any neurological decline)	7.7	6.4	6.3
Use of vasopressors for >48 hrs AND >12 hrs for CPP >70mmhg	22% (2/9)	20% (5/20)	70% (7/10)
Licox Monitoring	44% (4/9)	10% (2/20)	60% (6/10)
RIH	56% (5/9)	20% (4/20)	30% (3/10)
Pulmonary complications	33% (3/9)	50 % (10/20)	90% (9/10)

Q1 = first quartile, IQ = interquartile, Q4 = fourth quartile; RIH = failure of first tier therapy by brain trauma foundation; Pulmonary complications = ARDS or pulmonary edema with P/F ratio <300; Use of Vasopressors = for ≥ 48 hours ***and*** > 12 hours to maintain CPP > 70mmhg

Table 4: Association of vasopressors and ancillary monitoring on RIH and Pulmonary complications (cohort of 41 patients)

Characteristic	Vasopressors use (37%)	No vasopressor use (63%)	Licox (PbTiO2) 32% (13/41)	No Licox
RIH	53% (8/13)	19% (5/26)	38% (5/13)	25% (7/28)
Pulmonary Complications	80% (12/15)	38% (10/26)	77% (10/13)	46% (13/28)

Conclusions:

Following severe TBI hypovolemia should be avoided as it's associated with increased ICP and pulmonary complications [1]. Extreme hypervolemia should be avoided, if possible, to minimize pulmonary complications. Ancillary monitoring protocols should be used with caution, as the components that may improve outcome versus those that may harm are incompletely defined. Without correction for patient demographics, severity of illness, and head CT findings further conclusions cannot be made.

References:

1. Clifton GL, Miller ER, Choi SC. Fluid thresholds and outcome from severe brain injury. Crit Care Med 2002;30(4):739-45.
2. Contant CF, Valadka AB, Gopinath SP, Hannay HJ, Robertson CS. Adult respiratory distress syndrome: a complication of induced hypertension after severe head injury. J Neurosurg 2001;95(4):560-8.
3. Robertson CS, Valadka AB, Hannay HJ, Contant CF, Gopinath SP, Cormio M, Uzura M, Grossman RG.Prevention of secondary ischemic insults after severe head injury.Crit Care Med 1999;27(10):2086-95.
4. Braintraumafoundation.org

Financial Support: None

Poster 122

PRESENCE AND SEVERITY OF ICU-ACQUIRED PARESIS AT TIME OF AWAKENING ARE ASSOCIATED WITH INCREASED ICU AND HOSPITAL MORTALITY

Tarek Sharshar[1], Sylvie Bastuji-Garin[3], Marie-Christine Durand[2], Isabelle Malissin[5], Pablo Rodriguez[4], Charles Cerf[5], Hervé Outin[7], Bernard De Jonghe[7]

[1]Department of Intensive Care Medicine, Raymond Poincaré Hospital; University Versailles Saint-Quentin en Yvelines, Faculty of Medicine, Garches, France, [2]Department of Physiology, Raymond Poincaré Hospital; University Versailles Saint-Quentin en Yvelines, Faculty of Medicine, Garches, France, [3]Department of Public Health, AP-HP, Albert Chenevier-Henri Mondor, Créteil, France; University Paris 12, Faculty of Medicine, Créteil, France, [4]Department of Medical Intensive Care Medicine , AP-HP, Albert Chenevier-Henri Mondor, Créteil, France; University Paris 12, Faculty of Medicine, Créteil, France, [5]Department of Surgical Intensive Care Medicine, AP-HP, Albert Chenevier-Henri Mondor, Créteil, France; University Paris 12, Faculty of Medicine, Créteil, France, [6]Departments of Anesthesiology and Critical Care Medicine; Neurology; and Neurosurgery, Johns Hopkins University School of Medicine, Baltimore, United States, [7]Department of Intensive Care Medicine, Poissy-Saint-Germain en Laye Hospital, Poissy, France

Introduction:
To assess whether the presence and severity of ICU-acquired paresis are associated with ICU and in-hospital mortality

Methods:
This is a prospective observational study in two medical, one surgical, and one medicosurgical intensive care units in two university hospitals and one university affiliated hospital.with. A total of 115 consecutive patients were enrolled after >7 days of mechanical ventilation The Medical Research Council (MRC) score (from 0 to 60) was used to evaluate upper and lower limb strength at time of awakening, identified as the ability to follow 5 commands. ICU-acquired paresis was defined as a MRC score <48. Patients were followed until hospital discharge. The primary endpoint was hospital mortality.

Results:
At awakening, median MRC score was 41 (IQR, 21-52) and 75 (65%) patients had ICU-acquired paresis. Hospital non-survivors had a significantly lower MRC score at awakening (21 [11-43] versus 41 [28-53], p=0.008) and a significantly higher rate of ICUAP (85.1% versus 58.4%, p=0.02) compared to survivors. After multivariate risk adjustment, ICU-acquired paresis was independently associated with higher hospital and ICU mortality (OR for hospital mortality 2.02, 95% CI 1.03-8.03, p=0.048). Each MRC point decrease was associated with a significantly higher hospital mortality (OR: 1.03; 95%CI: [1.01-1.05], p = 0.033).

Conclusions:
Both the presence and severity of ICU-acquired paresis at the time of awakening are associated with increased ICU and hospital mortality; the mechanisms underlying this association need further study.

Financial Support: None

Poster 123

NOVEL INFLAMMATORY BIOMARKERS IN PATIENTS WITH SUBARACHNOID HEMORRHAGE ARE ELEVATED IN PATIENTS WITHOUT VASOSPASM

Paul Nyquist[1], Wendy Ziai[1], Rafael Tamargo[1], Hung Hui Wang[2], Christopher Devor[3], Anthony Suffredini[1]
[1]Johns hopkins School of Medicine, Baltimore, United States, [2]Critical care medicine department National Institute of Health, Bethesda, United States, [3]Laboratory of Pathology, National Cancer Institute, National Institutes of Health,, Bethesda, United States

Introduction:
Vasospasm is an inflammatory condition. Changes in the blood proteome may reflect changes in inflammatory components that enhance or suppress the development of vasospasm after subarachnoid hemorrhage (SAH).

Methods:
28 patients with SAH had blood sampled within three days of the onset of SAH (D-0) and 8 ± 2.7 days after onset (D-8). These patients were divided into two groups. Those with TCD evidence of vasospasm (n = 14, identified by a Lindegard ratio of greater than 3 on day 8) and those without TCD evidence of vasospasm (n = 14). Pooled sera from each group at D-0 and D-8 was screened using an antibody–based array containing 507 elements (RayBiotech). Proteins that were differentially expressed were then validated in individual patient samples using standard ELISA or reverse-phase protein arrays. Differences between vasospasm and non-vasospasm groups at D-0 and D-8 were then determined.

Results:
By day 8, in patients without vasospasm, serum amyloid A protein (1.2u vs 0.9u, p=0.001) and apolipoprotein E (1.4u vs 0.9, p=0.006) were increased. C-reactive protein was higher in patients with vasopasm compared to the non-vasospasm group at D-8 (85u vs. 82u, p<0.001). There was a trend toward higher serum amyloid protein and serum apolipoprotein E levels in those patients without vasospasm versus those with vasospasm (1.2u vs 1.0u p=0.06) and (1.4u vs 1.0u p=0.065) respectively.

Conclusions:
In patients with lower TCD velocities not meeting TCD criteria for vasospasm there were elevations of serum amyloid protein and apolipoprotein E on day 8. These proteins may help to elucidate biological processes that suppress vessel constriction in patients with SAH. Further, they may serve as biomarkers helping to identify patients with vasospasm.

Financial Support: None

Poster 124

PLASMA CORTISOL LEVELS IN GUILLAIN-BARRE-SYNDROME

JONATHAN STRAUSS[1], JEROME ABOAB[1], MARTIN ROTTMAN[2], RAPHAEL PORCHER[3], LEON IKKA[1], ANDREA POLITO[1], MARIE CHRISTINE DURAND[4], DAVID ORLIKOWSKI[1], CHRISTAN DEVAUX[1], FREDERIQUE LOFASO[4], DJILLALI ANNANE[1], JEAN LOUIS GAILLARD[2], TAREK SHARSHAR[1]

[1]MEDICAL INTENSIVE CARE UNIT, RAYMOND POINCARE TEACHING HOSPITA, GARCHES, FRANCE, [2]MICROBIOLOGY DEPARTMENT, RAYMOND POINCARE TEACHING HOSPITAL, GARCHES, FRANCE, [3]BIOSTATISTICS AND MEDICAL COMPUTER SCIENCE DEPARTMENT, SAINT-LOUIS TEACHING HOSPITAL, PARIS, FRANCE, [4]FUNCTIONAL TESTING DEPARTMENT, RAYMOND POINCARE TEACHING HOSPITAL, GARCHES, FRANCE

Introduction:

Invasive mechanical ventilation is required in one third of patients with Guillain-Barré syndrome (GBS). There are few early indicators of subsequent progression to respiratory failure. Adrenal function has rarely been studied in patients with GBS. We assessed the relationship between plasma cortisol level and GBS related complications, notably respiratory failure.

Methods:

Plasma cortisol levels were measured before (T0) and 60 minutes (T60) after corticotrophin test in 93 GBS patients at admission, 16 (17%) of which were ventilated within 24 hours from admission, 17 (18%)ventilated after the 24th hour and 60 (65%) never ventilated.

Results:

Mean plasma cortisol levels at T0 and T60 were 22.9±11.3 ng/ml and 45.4±16.1 ng/ml. Mean plasma cortisol level at T0 was significantly higher in the 17 (18%) patients who developed respiratory failure at least 24 hours later (28.5±12.1 vs 20.4±9.6 ng/ml; p = 0.003) and dysautonomia (33.1±14.3 vs 21.4±10.2 ng/ml, p = 0.003). When adjusting on only validated clinical predictors (i.e. delay between onset and admission < 7 days, inability to lift head and vital capacity < 60%), plasma cortisol level at T0 was the only independent risk factor for respiratory failure (OR: 2.45 per 10 ng/ml [1.23 to 4.88], p = 0.01). Fifty-nine patients underwent electrophysiological testing. When adjusting on validated electrophysiological model (i.e. peroneal p/d CMAP ratio and vital capacity), plasma cortisol level at T0 remained an independent predictor (OR: 2.50 per 10 ng/ml [1.14 to 5.51], p = 0.02).

Conclusions:

Measurement of baseline plasma cortisol levels can be helpful for early detection of GBS patients at risk for respiratory failure at least 24 hours later.

Financial Support: None

Poster 125

VENTRICULOSTOMY AND CSF DRAINAGE IN SAH

Anthony M. Amato[1], Nina Pluskowski[1], Meg Zomorodi[2], Gavin Britz[1], Michael L. James[1], Ali Zomorodi[1], Carmelo Graffagnino[1], DaiWai M. Olson[1]

[1]Duke University, Durham, NC, United States, [2]University of North Carolina, Chapel Hill, NC, United States

Introduction:

The volume of subarachnoid hemorrhage (including intraventricular blood) following aneurysmal rupture is associated with the development of vasospasm. Intraventricular catheters (IVC) facilitate cerebral spinal fluid (CSF) drainage and may reduce the incidence or severity of vasospasm but little evidence exists from which clinicians may determine the best practice. The purpose of this study was to provide the foundation for designing a trial that will explore how different methods of CSF drainage may impact outcomes in these patients.

Methods:

This observational pilot study enrolled 34 adult SAH patients. Data was collected through chart abstraction. Attending neurosurgeons determined whether each patient's IVC was primarily left open to drain CSF resulting in intermittent ICP monitoring (drain-first group), versus an IVC that was primarily set to monitor ICP resulting in intermittent CSF drainage at a set pressure threshold (monitor-first group).

Results:

Subjects were primarily female (68%), mean 54 years old. Subjects in the drain-first group (N=22) and the monitor-first group (N=12) had similar Hunt/Hess (p=0.43) and Fisher scores (p=0.69). Although there are no statistically significant differences between groups, this pilot study was not designed to test a hypothesized difference. The monitor-first group had lower mean CSF output (115 vs 132 ml/day), lower rates of vasospasm (42% vs 68%), lower incidence of complication (25% vs 64%), shorter length of stay (18 vs 21 days), and lower modified Rankin scores at discharge (3.6 vs 3.8).

Conclusions:

This observational study suggests that the method of IVC management may impact clinical outcomes. Although the monitor-first group method appears to be favourable, it is difficult to attribute differences in a non-randomized trial. A larger randomized controlled clinical trial is now in progress.

Financial Support: None

Poster 126

OSMOREGULATION OF VASOPRESSIN SECRETION IS ALTERED IN THE POST-ACUTE PHASE OF SEPTIC SHOCK

Shidasp Siami[1], Juliette Bailly-Salin[2], Andrea Polito[2], Raphael Porcher[3], Anne Blanchard[4], Philippe Haymann[5], Kathleen Laborde[6], Virginie Maxime[2], Catherine Boucly[7], Djillali Annane[2], Tarek Sharshar[2]
[1]Department of Intensive Care Medicine, Etampes, France, [2]General Intensive Care Medicine – Assistance Publique Hôpitaux de Paris, Raymond Poincaré Hospital, University of Versailles Saint-Quentin en Yvelines, Garches, France, [3]Biostatistics and Medical Computer Science Department, Saint-Louis Teaching Hospital, Paris, France, [4]Clinical Investigation Centre - Assistance Publique Hôpitaux de Paris, European Georges Pompidou Teaching Hospital, University René Descates and INSERM CIC9201, Paris, France, [5]Department of Functional Investigations - Assistance Publique Hôpitaux de Paris, Tenon Teaching Hospital, University of Pierre et Marie Curie-Paris 6 and INSERM U702, Paris, France, [6]Department of Department of Physiology - Assistance Publique Hôpitaux de Paris, Necker Enfants-Malades Hospital, University René Descartes, Paris, France, [7]Department of Biochemistry – Assistance Publique Hôpitaux de Paris, Raymond Poincaré Hospital, University of Versailles Saint-Quentin en Yvelines, Garches, France

Introduction:
Septic shock, a life-threatening disease, can be associated with a relative vasopressin (AVP) deficiency that occurs about three days after its onset. It may contribute to persistent hypotension, as vasopressin controls volemia and induces arterial vasoconstriction. It might be related to an alteration of baroreflex and osmosreceptors mediated secretion. These mechanisms have never been assessed in patients with septic shock. For safety reason, we opted to only assess the change in plasma AVP level during an osmotic challenge

Methods:
If normonatremic and at more than 72 hours from onset of septic shock, patients underwent an osmotic challenge, consisting on infusing 500 mL of hypertonic glucose (with 24 g of NaCl) over 120 minutes. Plasma AVP levels were measured 15 mn before the test and then four times every 30 minutes. A slope of the relation between AVP and sodium plasma levels less than < 0.5 pg/mEq defined non-responders.

Results:
33 patients were included. Seventeen (52%) patients were considered non-responders. During osmotic challenge, Variations throughout the test in plasma sodium levels, blood pressure and central venous pressure were comparable between the two groups. AVP increased from 4,8[3,3-6,4] to 14,4[11,2-23,3] pg/ml in responders but only from 2,8[2,3-4] to 4[3,1-5,3] pg/ml in non-responders (p<0,0001). AVP at baseline was higher in responders than responders 4,8 versus 2,8 pg/ml (p=0,031). Responders had a more severe hematosis alteration. Non-responders had more frequently been referred from ward, undergone surgery and they had more frequently a bacteraemia. There was no difference in terms of critical illness severity, hemodynamic alteration, hydroelectrolytic disturbances, treatment and outcome.

Conclusions:
Osmoregulation is dramatically altered in half of patients with prolonged septic shock. Pneumonia might have up-regulated in responders while prolonged sickness down regulated the osmoreceptors in non-responders.

Financial Support: None

Poster 127

NON-INVASIVE IMAGING HAS LOW DIAGNOSTIC YIELD IN ANGIOGRAM-NEGATIVE SUBARACHNOID HEMORRHAGE

Jay Joshi, Vivien Lee, Shyam Prabhakaran, Sayona John
Rush University Medical Center, Chicago, IL, United States

Introduction:
Subarachnoid hemorrhage (SAH) patients whose initial angiogram does not locate a bleeding source are often classified as having perimesencephalic hemorrhages. However, many patients do not fit into this benign picture and are non-perimesencephalic, angiogram-negative SAH (NPAN-SAH). Though the conventional angiogram remains the gold standard for diagnosis, multiple non-invasive imaging tests, beyond a second angiogram, are often performed in the acute evaluation of NPAN-SAH.

Methods:
With IRB approval, we retrospectively reviewed 226 non-traumatic SAH patients admitted to our institution from January 1, 2007 to June 1, 2009. Hunt-Hess and Fisher scores, in-hospital complications, and imaging data were abstracted from medical charts.

Results:
Of the 226 SAH patients, 45 (19.9%) had no aneurysm on initial angiography. Of these angiogram-negative SAH patients, 12 (26.7%) were perimesencephalic SAH and 33 (73.3%) were NPAN-SAH. There were 4 deaths (8.9%, 95% CI 3.0-21.3), all in NPAN-SAH cohort with 2 (4.4%) deaths due to re-bleeding. Forty-one (91.1%) had repeat angiography within two weeks and 3 (6.7%) had a third angiogram at long-term follow-up. All 33 (100%) NPAN-SAH had a repeat angiography. Forty-one (91.1%) had at least 1 CTA of the head, 16 (35.6%) 2 CTAs, and 5 (11.1%) had > 2 CTAs. Forty-two (93.3%) had MRI and MRA of brain and cervical spine, 20 (44.4%) had additional thoracic MRI, and 18 (40%) had additional MRI of the entire spine. An average of 3.2 non-invasive studies was performed on each patient. In these 142 additional non-invasive studies, there was no additional diagnostic yield in finding the source of SAH.

Conclusions:
Non-perimesencephalic angiogram-negative SAH has a worse prognosis compared to perimesencephalic SAH. Additional non-invasive neuroimaging provided no diagnostic yield in either patient population.

Financial Support: None

Poster 128

TRANSFER TIMES AND OUTCOMES FOR PATIENTS WITH ACUTE SUBDURAL OR EXTRADURAL HAEMATOMA

Julian Barnbrook, Marcus Hickson, Chris Taylor
National Hospital for Neurology and Neurosurgery, London, United Kingdom

Introduction:
Guidelines suggest an ideal time from injury to surgical decompression of less than four hours in patients with acute traumatic subdural or extradural haematoma. Previous audits at our centre showed this standard was not consistently achieved. We looked for a relationship between the length of this time interval and adverse neurological outcome at six months.

Methods:
We retrospectively reviewed all patients with acute traumatic subdural (ASDH) or extradural (AEDH) haematoma transferred to our neurosurgical centre over a three year period (December 2005-November 2008) for emergency surgical decompression. We identified the time elapsed from presentation at the Emergency Department to commencement of surgical decompression. We then assessed neurological function at six months post surgery using a Glasgow Outcome Score.

Results:
We were able to include 59 patients in our study (35 ASDH, 24 AEDH).
The mean time from presentation to surgery was 7:52 hours. At six months 52.5% of patients had a good neurological outcome (GOS 1-3), 47.5% had a poor outcome (GOS 4-5).
Of those presenting with GCS <9, 33% had a good outcome compared to 65% of those with an admission GCS of 9 or above.

Conclusions:
Achieving definitive surgery within four hours of presentation, let alone injury remains elusive. We were unable to associate prolonged length of transfer time with worse neurological outcome at six months. Our study was retrospective and the numbers were small.

Our unit accepts a significant number of patients from outside its normal referral area, meaning there may already be a significant delay in many cases.

In most cases there was no single identifiable reason for delay and a few cases showed that transfer could be achieved very rapidly.

Financial Support: None

Poster 129

CORRELATION BETWEEN CSF ADRENERGIC LEVELS AND MCA VASOSPASM ON TRANSCRANIAL ULTRASOUND

John Khoury, Keith Dombrowski, Jerry James, Anna Gutgarts, Carissa Pineda, Rodney Bell, Michael Moussouttas
Thomas Jefferson University, Philadelphia, PA, United States

Introduction:
Activation of the sympathetic autonomic nervous system may possibly contribute to the development of vasospasm (VS) following subarachnoid hemorrhage (SAH). The purpose of this study is to correlate cerebrospinal fluid (CSF) levels of adrenergic compounds and metabolites to the occurrence of middle cerebral artery (MCA) spasm as determined by transcranial ultrasound (TCUS).

Methods:
SAH patients who had Hunt-Hess Grades 3-5, a ventriculostomy, and TCUS performed for at least 7 days were included in this study. CSF 1ml was collected from each patient during the first 72 hours and assayed by HPLC for levels of epinephrine (Epi), norepinephrine (NE), and dihydroxyphenylglycol (DHPG). MCA VS was defined as a mean velocity (MV) > 120cms/s with a MCA/ICA ratio of >3 at any time. Analyses were calculated on a per-case and per-vessel basis.

Results:
Of the initial 40 patients included, 15 were excluded due to incomplete data as a result of early mortality or absent bone windows. From the remaining 25 patients, 3 had only ipsilateral bone windows, resulting in a total of 47 vessels amenable to insonation. Mean age was 58yo, and 65% were female. CT scores (Frontera et al.) were 1=3%, 2=24%, 3=5%, & 4=68%.

On a per-case basis, patients with MCA VS were younger (49yo vs 64yo, p=.003), but no correlation was observed between MCA VS and adrenergic levels. On a per-vessel basis, HH grade tended to correlate with MCA VS (p=.084), but again no association was observed between MCA VS and adrenergic levels.

Conclusions:
No connection was seen between CSF adrenergic levels and MCA VS. Our study is limited by small numbers, but our findings are consistent with the available literature whereby the association between the sympathetic nervous system and VS remains uncertain.

Financial Support: None

Poster 130

ANALYSIS OF CLINICAL STUDIES INVESTIGATING DEXMEDETOMIDINE IN THE NEUROSCIENCE SETTING 1997-2009

Marek Mirski

[1]Hospital of the University of Pennsylvania, Philadelphia, PA., United States, [2]Johns Hopkins Medical Institutions, Baltimore, MD., United States

Introduction:

Dexmedetomidine is an alpha-2 adrenoreceptor agonist with sedative, analgesic and anxiolytic properties approved for the intubated adult patient in the ICU setting. It possesses well described attributes for the neurological population; a rapid ability to sedate and awaken the patient allowing continuing neurologic assessment and no relevant respiratory depression. Properties including neuroprotection, cardioprotection and renoprotection have been proposed and investigated in various settings. Demonstrated clinical benefits in the ICU neuroscience setting are just emerging. This synopsis reviews the literature in regards to clinical studies conducted to evaluate dexmedetomidine in the neurosciences. Characteristics of the studies were categorized by study design, setting, patient population, and comparisons to other agents.

Methods:

Human clinical studies were identified through a search of PUBMED from 1997-2009. Key words include dexmedetomidine, NICU, neurocritical care and CEA. Study designs include randomized, observational, retrospective and case series.

Results:

Twenty-seven studies were included in the final analysis. The majority are case-studies or anecdotal and the literature consists of mostly surgical patients vs. the ICU population. The leading hypothesis is that dexmedetomidine is safe and efficacious in the neurosurgical population and may provide neuroprotection. Consistent findings are the attenuation of hemodynamic and endocrine response, smoother extubation and facilitation of neurological assessment.

Conclusions:

Dexmedetomidine has gained popularity in applications beyond its labeled indication and dosage, in various ICU's, and in special populations. The literature points to gained acceptance and favorable conditions for sedation without toxicity on CNS parameters and a rapidity of onset and offset. There is brevity of literature which demonstrates positive outcomes in the neuroscience setting and the primary data does not represent level 1 or 2 evidence. More studies should be done to validate this drug for common use as there appears to be great advantages in the neuroscience population.

Financial Support: None

Poster 131

ANEURYSM SIZE IS NOT RELATED TO HEMORRHAGE VOLUME FOLLOWING ANEURYSMAL SUBARACHNOID HEMORRHAGE.

Ahmed Hassan, Sayona John, Shyam Prabhakaran, Vivien Lee, Michael Chen, Yousef Mohammad, Richard Temes
Rush University Medical Center- Department of Neurological Sciences, Section of Neurocritical Care, Chicago, IL, United States

Introduction:

The Hijdra Scale was developed to quantify the volume of blood following aneurysmal subarachnoid hemorrhage (aSAH). We investigated the relationship of hemorrhage quantity utilizing the Hijdra scale and aneurysm size among patients with aSAH.

Methods:

We prospectively followed up a cohort of SAH patients annually to document outcome events after obtaining informed consent. We abstracted demographic, clinical, and past medical history data by chart review on a subset of patients with documented aSAH after excluding those with cryptogenic, traumatic, and non-aneurysmal SAH. The primary outcome of interest, hemorrhage quantity, was analyzed as both an ordinal measure (small, moderate, large) using tertiles and a dichotomous measure using the median. Proportional odds logistical models for ordinal response measures and simple logistical regression for dichotomous responses were constructed to investigate the relationship between hemorrhage quantity and aneurysm size.

Results:

From 03/2008 to 05/2009 a total of 73 patients were enrolled in the Rush University SAH database. Of these, we identified 53 patients with documented aSAH; 75% were female; 44% white, 37% black, 17% Hispanic; and the mean age was 60 ±13 years. The mean ruptured aneurysm size was 5.7 mm and the median Hijdra score was 18 (range 0-28). We found no relationship between ordinal (p=0.49) and dichotomous (p=0.42) hemorrhage quantity and aneurysm size. No relationships were found between hemorrhage quantity and age, sex, race, APACHE II score, and history of anti-platelet use. There was a trend for significance among patients with a past medical history of hypertension and having large hemorrhage quantity (OR 3.03, 0.96-9.52; p=0.06).

Conclusions:

We found no relationship between aneurysm size and quantity of hemorrhage among patients with aSAH. Future studies should focus on clinical variables such as hypertension and their role in hemorrhage quantity.

Financial Support: None

Poster 132

INTRACEREBRAL HEMORRHAGE CARRIES A HIGHER LIKELIHOOD OF EARLY MORTALITY COMPARED TO OTHER STROKE SUBTYPES

Ahmed Hassan, Ivan Rocha Ferreira da Silva, Bichun Ouyang, Richard Temes, Vivien Lee, Shyam Prabhakaran, Sayona John

Rush University Medical Center- Department of Neurological Sciences, Section of Neurocritical Care, Chicago, IL, United States

Introduction:

Stroke is the third leading cause of death in the United States. Among the stroke subtypes intracerebral hemorrhage (ICH), subarachnoid hemorrhage (SAH), and ischemic stroke (IS), ICH and SAH are associated with the highest mortality, followed by IS. Most deaths due to stroke occur within the first 30 days, though it is unclear if any specific stroke subtype carries a significantly higher risk of early mortality (within the first 48 hours from presentation) when compared to the other subtypes.

Methods:

With IRB approval, we retrospectively reviewed 80 stroke patients transferred to our institution between November 2007 and April 2009 who died during hospitalization. We collected data including primary diagnosis, confirmed by CT or MRI, and time from presentation to our institution to death from any cause.

Results:

Among the 80 in-hospital stroke deaths, ICH was the diagnosis in 49 (61%), SAH in 18 (23%), and IS in 13 (16%). Thirty-three of 49 (67%) ICH deaths occurred within 48 hours of transfer. Of the remaining stroke subtypes, 8/18 (44%) SAH deaths and 5/13 IS deaths occurred within 48 hours. Among in-hospital stroke deaths, the odds of early death (within 48 hours) are estimated to be 2.86 times (95%CI: 1.13-7.24, p=0.025) higher in ICH compared with the other stroke subtypes (SAH, IS).

Conclusions:

Amongst in-hospital stroke deaths, ICH was the stroke subtype associated with the highest likelihood of early mortality. This may indicate the severity of the disease process and a lack of effective early therapeutic measures available for ICH.

Financial Support: None

Poster 133

ENDOTHELIAL NITRIC OXIDE SYNTHASE POLYMORPHISM (ENOS -786T>C) AND POOR FUNCTIONAL OUTCOME AFTER ANEURYSMAL SUBARACHNOID HEMORRHAGE (SAH)

Nerissa Ko[1], Helen Kim[1], Randall Higashida[1], Michael Lawton[1], Jonathan Zaroff[2]
[1]University of California, San Francisco, San Francisco, CA, United States, [2]Kaiser Permanente, San Francisco, CA, United States

Introduction:
Cerebral vasospasm is a common complication of SAH and remains a major cause of death and disability after aneurysm rupture. The eNOS promoter (-786T>C) CC genotype has been associated with a three-fold increased risk of angiographic cerebral vasospasm, however, its effect on adverse neurologic outcomes after SAH has yet to be determined. We hypothesize that eNOS genotype would predict worse outcome, likely through its effect on risk of vasospasm.

Methods:
Subjects included 284 patients with confirmed aneurysmal SAH enrolled in a longitudinal cohort study. We analyzed data from 90 subjects for whom we had genotype information as well as 6-month follow-up assessment. Patients who died prior to follow-up were excluded. Univariate analyses used Chi-square, Wilcoxon ranksum or Students t-test for the individual predictor variables. Modified Rankin Scale score (mRS) was our primary outcome. Logistic regression analysis for poor outcomes (mRS >2) included genotype and adjusted for age, sex, race/ethnicity, and Hunt-Hess grade.

Results:
Of the 284 subjects, 18% were dependent or severely disabled (mRS > 2) at 6 months. Older age, higher Hunt Hess grade and presence of vasospasm were associated with poor outcomes. The eNOS (-786T>C) CC genotype was associated with mRS>2 with an adjusted OR of 1.67 (95%CI 0.54-5.18).

Conclusions:
Our results support a trend between eNOS (-786T>C) CC genotype and 6-month poor functional outcome (mRS>2). Although these results are not as robust as the association with angiographic cerebral vasospasm, it demonstrates the ability to integrate genetic information with clinical outcomes. Limitations are primarily the small sample size and ability to adequately adjust for all clinical factors that could influence outcome. Whether the effect on outcome of eNOS genotype is related to cerebral vasospasm risk will require further study.

Financial Support: None

Poster 134

STAY COOL WHILE YOU'RE AHEAD: PROPHYLACTIC HYPOTHERMIA IN PATIENTS WITH ANEURYSMAL SUBARACHNOID HEMORRHAGE

Shirley Chen, Nobl Barazangi, Jeffrey Thomas, David Tong, Jack Rose, Evelyn Taverna, Ann Bedenk, Jackie Que-Huong Phan
California Pacific Medical Center, San Francisco, CA, United States

Introduction:
Therapeutic hypothermia has been utilized in various brain injury models, including aneurysmal subarachnoid hemorrhage (SAH). Hypothermia has been used to treat refractory cerebral edema or severe vasospasm in this setting. However, there is very little data on hypothermia as a prophylactic measure before potential complications of SAH have occurred. We evaluated the safety and feasibility of prophylactic hypothermia in patients with aneurysmal SAH.

Methods:
We conducted a retrospective chart review of patients admitted with aneurysmal SAH at a tertiary stroke center from July 1, 2007 to June 30, 2009, who were also treated with induced hypothermia. Only patients who had hypothermia initiated prior to symptomatic vasospasm onset were included.

Results:
A total of 10 out of 103 patients were treated prophylactically with mild hypothermia (32-34 degrees Celsius). Three patients presented with Hunt and Hess Grade I-III, and seven patients with Grade IV-V SAH. Average time at initiation of hypothermia was on SAH day 1 (range day 0-3). Six (60%) patients underwent treatment of aneurysm by endovascular coiling or surgical clipping within 72 hours of symptom onset. The average duration of hypothermia was 10 days (range 1-27 days). Nine (90%) patients developed evidence of vasospasm on computed tomography angiography or transcranial doppler. Five (50%) patients survived to discharge. Causes of death included irreversible global hypoxia from cardiac arrest (2), severe refractory cerebral vasospasm (1), and malignant MCA infarction (2). All four patients with Grade V SAH died. When these patients were excluded, of the remaining 6 patients, 5 (83%) survived to discharge, and 1 (17%) died.

Conclusions:
Prophylactic hypothermia may be effective and safe in selected patients with aneurysmal SAH. Additional studies are needed to further define timing and parameters for therapeutic hypothermia in this setting.

Financial Support: None

Poster 135

NEUROPROTECTIVE EFFECT OF MELATONIN AND PINOLINE IN FOCAL CEREBRAL ISCHEMIA

Santiago Ortega Gutierrez[1], Pankhil Shah[2], Jane Madden[2], Joaquin Garcia Garcia[3], Michel T Torbey[1]
[1]Columbia University Presbiterian, New York, United States, [2]Medical College of Wisconsin, Milwaukee, United States, [3]Universidad de Zaragoza, Zaragoza, Spain

Introduction:
Melatonin and Pinoline are indolamines which have shown an antioxidative direct and indirect protection effect in vitro and in vivo models.

Methods:
Fourteen 8- week-old male, C57B16 mice underwent reversible middle cerebral artery occlusion ischemia (1.5 hours) followed by 22 hr of reperfusion. The animals received pinoline (15 mg/kg i.p.; n=4), melatonin (30 mg/kg i.p.; n=5) or vehicle (n = 5) at ischemia, immediately upon reperfusion, and at 3 and 4.5 hr post-ischemia. Another three animals in each group received the same doses but were sacrificed at 4.5 hours and used for protein oxidation quantification by Western blot. Rectal temperature, surgical time, time to ischemia and time to reperfusion were recorded continuously. Initial neurological damage by modified stroke score was grossly assessed at ischemia, reperfusion, and at 24 hr. Infarction volume was quantified using 2, 3, 5-triphenyltetrazolium chloride (TTC) staining, digital photography, and imaging analysis software. Means (± SD) were calculated and compared using Student's t-test or ANOVA. $P \leq 0.05$ was set as statistically significant.

Results:
Total hemispheric infarction volume was reduced in the pinoline and melatonin-treated mice compared with the non-treated group ($19 \pm 7\%$ vs. $36 \pm 13\%$; $p < 0.05$) and ($34 \pm 8\%$ vs. $53 \pm 7\%$; $p < 0.01$) respectively. Pinoline score was 1.40 vs. 1.80 in the control group at 24h. No statistical difference was observed in the melatonin group. Optical net intensity ratio was statistically significance at cortical level on the 40KD band in the melatonin and 50 KD on the pinoline groups.

Conclusions:
Pinoline and melatonin treatment appeared to confer neuroprotection on a cerebral ischemia in vivo model. Although its anti-ischemic mechanism needs to be elucidated, both molecules are potent free radical scavenging properties may offer a potential therapy.

Financial Support: None

Poster 136
OUTCOMES FROM THE USE OF PENTOBARBITAL: A RETROSPECTIVE ANALYSIS
Eric Adelman, Teresa Jacobs, Devin Brown
University of Michigan, Ann, Arbor, MI, United States

Introduction:
Many authors suggest using pentobarbital when elevated intracranial pressure or seizures are refractory to other agents. Due to the lack of outcome data after the use of this agent, we investigated the outcomes of patients treated with pentobarbital over the past five years.

Methods:
Patients were identified using a pharmacy database that tracks inpatient medication dispensing at our tertiary referral center. All patients, older than 18, cared for in adult ICUs, who received pentobarbital between 2004 and 2008 were included. Inpatient mortality was compared between patients older and younger than 40 as well as those who required vasopressors using Fisher's exact test.

Results:
Twenty-two patients received pentobarbital. The mean patient age was 43 (SD=16). Just over half (55%) were men. Pentobarbital was used in 64% of the patients to treat intracranial hypertension; the remainder were treated for refractory status epilepticus. The most frequent underlying disorders were toxic-metabolic disease processes.

Fourteen patients (64%) died in the hospital. Care was withdrawn in 55%. Of the 8 patients (36%) who were alive at the time of discharge: 3 (14%) were discharged to acute rehabilitation, 2 (9%) to an extended care facility, 1 (5%) to sub-acute rehabilitation, 1 (5%) to hospice, and 1 (5%) to home.

Hypotension, renal failure, and pneumonia were common (40-86%) in patient receiving pentobarbital. There was no significant association between inpatient mortality and reason for pentobarbital use. Age and need for vasopressors were not significantly associated with in hospital mortality.

Conclusions:
Pentobarbital use was associated with significant morbidity and mortality (greater than 60%), but 18% of patients were discharged home or to acute rehabilitation facilities. Further study is needed to better clarify the risks and benefits of pentobarbital to treat refractory intracranial hypertension and status epilepticus.

Financial Support: None

Poster 137

THE IMPACT OF WEEKEND ADMISSION ON IN-HOSPITAL MORTALITY IN SUBARACHNOID HEMORRHAGE

Manisha Gupte, Jay Joshi, Sayona John, Shyam Prabhakaran, Vivien Lee
Rush University Medical Center, Chicago, IL, United States

Introduction:
The "weekend effect" phenomenon suggests that admission day of the week is an independent predictor of mortality. We evaluated the effect of weekend admission on SAH in-hospital mortality at a single academic center.

Methods:
With IRB approval, we retrospectively reviewed 226 consecutive SAH patients admitted to our institution from August 1, 2006 to June 1, 2009. Weekend was defined as Saturday or Sunday. Data was collected on day of the week admission, in-hospital mortality, aneurysm type and treatment. CT images were reviewed by the study neurologist and scored for Fisher grade.

Results:
Of 226 SAH patients admitted to our institution, 152 (67%) were female. The mean age was 57.5 years (range, 21 to 90). CT brain Fisher score was as follows: Fisher 3 (72%), Fisher 2 (14%) and Fisher 4 (10%). The cerebral aneurysm distribution was ACOM (24%), PCOM (13%), MCA (9%), multiple (6%), and angiogram negative (18%). Surgical clipping was performed in 52 (23%) and endovascular treatment was performed in 99 (44%). The overall SAH in-hospital mortality rate was 23%. Weekday admission accounted for 73%, and weekend admission occurred in 26%. Age, Fisher grade, and treatment modality were not significantly different between weekday versus weekend admission. The mean time from admission to treatment of aneurysm was 1.04 days and did not differ significantly by weekend versus weekday admission (p 0.8). Mortality rate was 25% for SAH patients admitted on a weekend versus 22% for SAH patients admitted on a weekday (p 0.6).

Conclusions:
The weekend effect does not appear to be a significant factor in mortality outcomes of SAH patients. The time to definitive aneurysm treatment does not appear to be impacted by weekend admission.

Financial Support: None

Poster 138

ICH SCORE MORTALITY IS MAGNIFIED IN WARFARIN INTRACEREBRAL HEMORRHAGE

David McCollum, William Freeman
Mayo Clinic, Jacksonville, FL/Southeast, United States

Introduction:
The ICH score is a simple clinical-radiographic scale in patients with intracerebral hemorrhage (ICH) that helps estimate 30-day mortality. We hypothesize that the ICH score can be applied to patients with warfarin ICH (W-ICH) to help estimate thirty-day mortality.

Methods:
A retrospective review was conducted using a consecutive series of patients with acute W-ICH presenting from December 2002 until February 2009. Acute warfarin ICH was defined as patients presenting with acute stroke symptoms and verification of a spontaneous ICH confirmed by non-contrast head CT in the setting of warfarin use and anticoagulation (INR >1.3). Exclusion criteria included traumatic ICH and patients with insufficient data for ICH score calculation.

Results:
Twenty-eight patients with acute W-ICH were identified. Four patients were excluded because they were lost to follow-up after discharge. The ICH score was then calculated for each W-ICH patient [W-ICH score] and they were divided into groups based on their W-ICH score [W-ICH = 0 (n=5), W-ICH = 1 (n=6), W-ICH = 2 (n=6), W-ICH = 3 (n=5), W-ICH = 4 (n=2), W-ICH = 5 (n=0); N = 24 total]. A general increase in 30-day mortality was observed with increasing W-ICH score with the exception of W-ICH score of two [W-ICH = 0 (20% mortality), W-ICH =1 (50% mortality), W-ICH = 2 (16.67% mortality), W-ICH = 3 (100% mortality), W-ICH = 4 (100% mortality)]. All patients had their warfarin coagulopathy reversed as per institutional policy depending on admission INR and comorbidities.

Conclusion:
W-ICH patients have an increasing trend toward 30-day mortality with increasing ICH score similar to patients with ICH not taking warfarin. However, 30-day mortality was higher at lower relative ICH scores, suggesting that warfarin anticoagulation (INR 1.4 or greater) increases mortality independent of other clinical and radiographic variables.

Financial Support: None

Poster 139
ANEMIA IN CEREBROVASCULAR DISEASE
Archana Hinduja, Gwendolyn Lynch
Cleveland Clinic Foundation, Cleveland, OH, United States

Introduction:
Anemia is a highly prevalent condition among hospitalized patients. We hypothesize that patients with acute cerebrovascular disease and anemia on admission have poor prognosis in terms of death, length of stay and disposition.

Methods:
A retrospective analysis of patients admitted to our institution with acute stroke (ischemic, hemorrhagic, subarachnoid hemorrhage) between October 2007 and March 2008 was performed. They were dichotomized based on hematocrit levels of < 36, >/= 36 for women and <39 and >/=39 for men using the WHO definition of anemia. Covariates used include diagnosis, demographic information and past medical history. The best admission hematocrit cutoff points for distinguishing between those with increased risk of death, disposition to SNF (skilled nursing facility), increased LOS (length of stay) were identified.

Results:
Of the 201 patients, 106 (52.7%) were female with a mean age of 61.4 years. Of these 54 patients (26.9%) were anemic. Ten patients died and nine were dispositioned to SNF. While the relationship between disposition and anemic status was not significant (p=0.14), there was evidence that those who died were more likely to be anemic (p=0.025). LOS did not differ statistically between anemic and those without anemia.
None of the variables were statistically significant on univariable analysis for mortality. The admission hematocrit cutoff points for distinguishing risk of death, discharge disposition to SNF, and increased length of stay are 38.4 (95% CI - 29.4, 49.5) p=0.044C, 32.1 (28.9, 48.1) p=0.14C, 50.1 (30.8, 50.1) p=0.028W, respectively.

Conclusions:
Anemia on admission did not predict death, disposition to SNF or LOS, but there was a tendency that patients who died were more likely to be anemic. The admission hematocrit cutoff point for distinguishing risk of death, disposition to SNF was slightly lower and increased LOS was higher than the WHO definition of anemia.

Financial Support: None

Poster 140

INTRACRANIAL HEMORRHAGE FOLLOWING NEUROENDOVASCULAR PROCEDURES WITH ABCIXIMAB IS ASSOCIATED WITH SIGNIFICANT MORTALITY

Ryan D. Walsh[1], Maria I. Aguilar[2], Giuseppe Lanzino[3], Ricardo A. Hanel[1], Brian W. Chong[2], W. David Freeman[1]
[1]Mayo Clinic Jacksonville, Jacksonville, FL, United States, [2]Mayo Clinic Scottsdale, Scottsdale, AZ, United States, [3]Mayo Clinic Rochester, Rochester, MN, United States

Introduction:
Abciximab, a glycoprotein IIb/IIIa receptor inhibitor (GPIIb/IIIa), is used during neuroendovascular procedures both to prevent and treat ischemic sequelae. Experience with abciximab in this setting is limited and major bleeding complications, including fatal intracranial hemorrhage (ICH), are of particular concern. We report our multicenter experience with ICH following administration of abciximab during neuroendovascular procedures.

Methods:
We identified neuroendovascular procedures in which abciximab was used at three academic institutions from November 2000 through April 2009. Cases of periprocedural ICH were identified and pertinent demographic, historical, procedural, laboratory, and radiographic data were collected. Clinical outcome was measured by the Glasgow Outcome Scale (GOS) either at death or discharge.

Results:
Abciximab was used in 51 neuroendovascular procedures; 9 ICH cases (17.6%) were identified. Procedures performed and indications for abciximab use varied. Route of abciximab administration included IV bolus only (n=4), IA bolus and IV infusion (n=3), IV bolus and IV infusion (n=1), and IV infusion without preceding bolus (n=1). All patients but one received periprocedural antiplatelet, anticoagulant, or thrombolytic agents. All ICH were detected within 7 hours of abciximab administration, (except Patient 4; 40 hours); 5 were detected within 3 hours. ICH patterns varied and included subarachnoid hemorrhage (SAH) with intraventricular hemorrhage (IVH) (n=5); intraparenchymal hemorrhage (IPH) with IVH (n=2); SAH, IVH, and IPH (n=1); and a combination of SAH, IVH, IPH, and subdural hemorrhage (n=1). Four patients died following ICH (i.e. GOS score of 0); GOS scores at discharge for the remaining cases were 3 (n=3), and 5 (n=2).

Conclusions:
ICH was common (17.6%) after neuroendovascular procedures using abciximab and was associated with a 44% mortality. Future management strategies should focus on earlier recognition of GPIIb/IIIa-related-ICH; development of direct GPIIb/IIIa antidotes; comparisons with shorter-half-life GPIIb/IIIa drugs; and identification of optimal abciximab dose and route.

Financial Support: None

Poster 141

INITIAL EXPERIENCE AND FEASIBILITY OF A NON-INVASIVE BRAIN OXYGEN MONITOR IN NEUROCRITICAL CARE

Vineeta Singh[1], Diane Morabito[1], Nicolas Phan[1], Shirley Stiver[1], Guy Rosenthal[2]
[1]University of California, San Francisco, San Francisco, CA, United States, [2]Hadassah-Hebrew University Hospital, Jerusalem, Israel

Introduction:
CerOx 3110 is a novel noninvasive brain and tissue oxygen saturation monitor based on NIRS and ultrasound technology. The purpose of this prospective observational study of patients with both traumatic and non-traumatic brain injuries is to determine if the CerOx 3110 correlates with existing measures of cerebral oxygen metabolism which are currently used as part of regular care in the management of patients with severe brain injury.

Methods:
We enrolled patients with severe brain injury (TBI = 6, ICH = 4) who had at least one invasive cerebral oxygen monitor in addition to an intra-cranial pressure monitor. CerOx 3110 adhesive patches were placed bilaterally over the frontal regions of the scalp and optical probes were attached to the patch clips. Monitoring with CerOx 3110 continued for up to 7 days. High density physiological data, e.g., MAP, brain tissue oxygen, jugular venous saturation, ICP, were collected at Q 1 minute intervals into our Neurocritical Care database. Physiological data were then merged with CerOx 3110 measurements.

Results:
Ten patients requiring invasive neuromonitoring were enrolled during this 3-month study period. The duration of non-invasive recording was 1-10 days (mean= 4 days) with maximum length of uninterrupted recording being 72-hours. CerOx 3110 measurements ranged from 24-84.5% (mean = 57%) on the left and 30-77% (mean = 56.2%) on the right. In this group of patients, the brain tissue oxygen tension ranged from 7.4-98.2 mm Hg, the jugular venous saturation was 36.5-98% and the cerebral blood flow varied from 0.6-122 ml/100gm/min.

Conclusions:
Continuous monitoring with CerOx 3110 is safe and feasible in neurocritical care setting. It has the potential of providing information about cerebral metabolism needed for close monitoring and management of patients with severe brain injury

Financial Support: None

Poster 142
HYPERCHOLESTEROLEMIA AND ELEVATED BLOOD PRESSURE INCREASES THE RISK OF DVT IN NON-AMBULATORY ICH PATIENTS
Ifeanyi Iwuchukwu, Brittany Mcmurren, Martin Gizzi
NJ Neuroscience institute, Edison, NJ, United States

Introduction:
Deep vein thrombosis (DVT) is a common complication of intracerebral hemorrhage (ICH) and has been associated with immobility in the lower extremities. [1] Atherosclerotic risk factors (hypertension, diabetes mellitus (DM) and hypercholesterolemia) are associated with arterial thrombosis and have been postulated play a role in venous thrombosis. [1]

We hypothesized that a history of atherosclerotic risk factors increases the risk of DVT in ICH patients.

Methods:
Retrospective analysis of patients diagnosed with spontaneous ICH at our institution between January 2005 and December 2007 was performed. Demographics, history of hypertension, DM or hypercholesterolemia; systolic blood pressure at presentation; presence of immobility or hemiparesis and diagnosis of DVT were collected. Logistic regression analysis was used to predict the risk of DVT.

Results:
62 of 114 patients with spontaneous ICH were immobile and were selected for analysis. All patients had sequential compression devices applied on admission. The overall incidence of DVT diagnosed by lower extremity Doppler was 29% and pulmonary embolism was 3.2%. Mean time to diagnosis of DVT was 16.9 (SD 12.2) days. After stepwise logistic regression analyses, significant predictors of DVT in immobilized ICH patients were, history of hypercholesterolemia (OR 3.57 p=0.0092) and SBP on admission >180 (OR 5.14 p=0.0442).

Conclusions:
Immobilized ICH patients with a history of hypercholesterolemia were three times more likely to develop DVT. A SBP >180 on admission was five times more likely to predict DVT. Thus atherosclerotic risk factors may play a role in the pathophysiology of DVT in immobilized ICH patients, suggesting a possible etiopathologic link between arterial and venous thrombosis.

Landi G, D'Angelo A, Bocardi E et al. Venous thrombosis in acute stroke. Prognostic importance of hypercoaguability. Arch Neurol 1992; 49:279

Financial Support: None

Poster 143

ROLE OF EMERGENT CAROTID STENTING IN REVASCULARIZATION OF THE MIDDLE CEREBRAL ARTERY AND ANTERIOR CEREBRAL ARTERY IN PATIENTS WITH ACUTE ISCHEMIC STROKE

John Cullen, Veena Yashaswi, Amar Swrankar, Ravi Patel, Adham Kamel, Ziad El-Zammar, Julius Gene Latorre, Eric Deshaies, Tara Ramachandran, Yahia Lodi
Upstate Medical University, Syracuse, NY, United States

Introduction:
Acute ischemic stroke due to the occlusion of the internal carotid artery (ICA) is associated with malignant stroke and poor outcome. Without revascularization of ICA perfusion to the middle cerebral artery (MCA) and anterior cerebral artery (ACA) is not possible. Objective: Objective of our study is to evaluate the technical feasibility of emergent carotid artery revascularization using stent and to evaluate the impact of stenting in distal cerebral perfusion.

Methods:
From an established stroke database consecutive patients with acute ischemic stroke who underwent emergent carotid stenting and thrombolysis/clot retrieval of the MCA and ACA from July 2007 to December 2008 were enrolled. Patients' demographics including presenting National Institute of Health Stroke Scale (NIHSS), degree of revascularization, hemorrhagic conversion and 30 days outcome data using Glasgow Outcome Scale (GOS) were collected.

Results:
Successful ICA stenting was possible in 20/21 (95%) patients. The average age of patients was 62 years (ranges 40-93) and average NIHSS was 18 (ranges 9-29). Carotid stenting facilitated successful revascularization of the MCA and ACA using TPA and MERCI clot retriever device in 18 patients (85.7%), 14 (67%) of which has achieved complete recanalization in the MCA and ACA. In complete recanalization group a 4 point or higher NIHSS improvement was observed in 11/14 (78.5%) patients. Symptomatic intracranial hemorrhage was observed in 9.5% patients. Seven of 14 patients who achieved complete recanalization had a good outcome. Five of 7 patients who did not achieve complete recanalazation of the MCA and ACA died and had NIHSS ≥ 20. NIHSS ≥ 20 was associated with incomplete recanalization of the MCA and ACA with poor outcome.

Conclusions:
Emergent carotid revascularization is not only technically feasible in patients with acute ischemic stroke due to the carotid occlusion, but it also facilitates successful renalization of the MCA and ACA. Further study is necessary.

Financial Support: None

Poster 144

STENT-ASSISTED COILING OF THE RUPTURED INTRACRANIAL WIDE NECK ANEURYSM

Yahia Lodi[1], Ravi Patel[1], Amar Swrankar[1], Eric Deshaies[1], John Whapham[3], Ali Malek[4], Gene Latorre[1], John Collen[1], Tara Ramachandran[1], Richard Fessler[2]

[1]Upstate Medical University, Syracuse, NY, United States, [2]Wayne State Medical University, Detroit, MI, United States, [3]Loyola University, Chicago, IL, United States, [4]St. Marys Hospital, Tempa Bay, FL, United States

Introduction:

Stent-assisted coiling of wide neck intracranial aneurysm requires therapeutic dose of antiplatelets to prevent stent thrombosis. Stent-assisted coiling of the ruptured intracranial aneurysms also requires a loading of both loading dose of aspirin and plavix. Objective: To report any potential complication associated with the use of both aspirin and plavix in stent-assisted coiling of ruptured wide neck intracranial aneurysm.

Methods:

Consecutive patients who underwent stent-assisted coiling for ruptured wide neck intracranial aneurysm were enrolled from 2005 to 2009. Patient's demographics including the Hunt & Hess grade, Fished scale, use of ventriculostomy catheter, location and size of aneurysm were collected. Any complication such rupture of aneurysm, ventriculostomy associated hemorrhage or systemic bleeding was recorded. Additionally a 30 days outcome measurement was obtained using Glasgow Outcome Scale (GOS).

Results:

21 patients with mean age of 50 ± 13 underwent stent-assisted coiling. A loading dose of plavix (300 mg to 375 mg) and aspirin 325 mg were given prior to stent placement. 14 patients received ventriculostomy catheter, 11 cases before and 3 cases after the procedure. There was no intraoperative ruptured of aneurysm or hemorrhage related to ventriculostomy or systemic hemorrhagic event. There were two episodes of stent thrombosis; one was an asymptomatic which developed during stent-assisted coiling procedure and resolved spontaneously, the other was symptomatic required intra-arterial administration of thrombolytic. There was no mortality and good outcome was observed in 85% of patient.

Conclusions:

Stent-assisted coiling of the ruptured wide neck intracranial aneurysm using therapeutic dose of aspirin and plavix is not associated with increased bleeding complication such as rupture of aneurysm or intracranial hemorrhage related to ventriculostomy. However, the thromboembolic events remain the main challenge in stent-assisted coiling of ruptured intracranial wide neck aneurysm. Therefore, antiplatelets should not be withheld prior to a stent-assisted coiling of ruptured wide neck aneurysm.

Financial Support: None

Poster 145

EFFECT OF MILD HYPOTHERMIA ON BRAIN TISSUE OXYGENATION IN PATIENTS WITH SEVERE TRAUMATIC BRAIN INJURY

Takashi Tokutomi, Tomoya Miyagi, Yasuharu Takeuchi
Kurume Universuty School of Medicine, Kurume, Japan

Introduction:
We have used mild therapeutic hypothermia in patients with severe traumatic brain injury. In this study we investigated the effects of hypothermia on brain tissue oxygenation.

Methods:
Brain tissue oxygen tension ($PbtO_2$) in addition to intracranial pressure (ICP), cerebral perfusion pressure (CPP), and jugular venous saturation (SjO_2) were monitored in 12 consecutive patients with a Glasgow Coma Scale score of 4 to 7 (ages 16 to 64 years). Patients were cooled to a target temperature of 35.0 °C. Patients with good recovery and moderate disability on the Glasgow Outcome Scale were regarded as having favorable outcomes.

Results:
Seven patients had favorable outcomes and five had unfavorable outcomes. In nine patients, the temperature was reduced below 35 °C, and in four, below 34 °C. ICP decreased with decreasing temperature to 35 °C (16.9±9.1 mmHg in 35-36 °C vs. 20.2±7.3 mm Hg in 37-38 °C, $p<0.001$), but a further reduction of the temperature did not lower the ICP. $PbtO_2$ decreased significantly below 35 °C (25.4±12.3 mm Hg below 35 °C vs. 31.6±11.2 mm Hg above 35 °C, $p<0.001$). The mean value of $PbtO_2$ was significantly lower in the unfavorable outcome group (25.0±9.7 mm Hg in unfavorable vs. 34.7±10.4 mm Hg in favorable outcome group, $p<0.001$)

Conclusions:
These results suggest that decreasing the temperature to 35 °C can reduce intracranial hypertension while hypothermia below 35 °C may impair brain tissue oxygenation.

Financial Support: None

Poster 146

A STRATEGY FOR MANAGING VENOUS THROMBO-EMBOLIC DISEASE IN THE NEUROCRITICAL CARE UNIT (NCCU)

Bryan Gaspard, Hartmut Uschmann, Dale Hoekema, Kinjal Desai
Univ of Mississippi Medical Center, Jackson, MS, United States

Introduction:
NCCU patients may have multiple risk factors for venous thrombo-embolic disease including a hypercoaguable state, prolonged bed rest and immobility. Many patients cannot tolerate aggressive anticoagulation due to the risk of intracerebral bleeding. A strategy to avoid fatal pulmonary emboli (PE) and diagnose and treat deep venous thrombosis (DVT) early is needed. We hypothesize, surveillance lower extremity venous Doppler exams every 5-7 days, along with lower extremity sequential compression devices (SCD's), compression stockings (CS's), early anticoagulation escalated step wise, and inferior vena cava filters (IVCF's) when full anticoagulation is not possible, will prevent fatal PE.

Methods:
A retrospective review of a six-month period in a University NCCU was performed where patients were treated according to the above hypothesis. Anticoagulation was usually started with heparin 5000 units sq q12 hr within the first 48 hours and increased to 5000 units sq q 8 hr after 48 hours. Anticoagulation was increased in many cases to enoxaparin 30 mg sq q 12 hr after another 48 hours.

Results:
Of the 542 patients who received care during the six-month period, 41 patients (7.5%) were diagnosed with lower extremity DVT that were asymptomatic in 90% of the cases. IVCF's were placed in 27 patients (65% of those with DVT). Two patients were diagnosed with pulmonary emboli (.4%). There were no fatal pulmonary emboli. There were no significant bleeding complications or IVCF complications.

Conclusions:
Surveillance lower extremity venous Dopplers every 5-7 days, SCD's, CS's, and escalating doses of anticoagulation as is tolerated and safe lower the risk of DVT in this high risk population and identify early asymptomatic DVT. Fatal PE can be prevented with IVCF placement and more aggressive anticoagulation as permitted by the diminishing risk of bleeding as time passes from the acute injury.

Financial Support: None

Poster 147

HEPARIN INDUCED THROMBOCYTOPENIA IN ANEURYSMAL SUBARACHNOID HEMORRHAGE

Valerie Dechant, Mohamed Baio, William McBride, Carissa Pineda, David Brock, Rodney Bell, Robert Rosenwasser, Michael Moussouttas, Monisha Kumar
Thomas Jefferson University Hospital, Philadelphia, PA, United States

Introduction:
Heparin induced thrombocytopenia (HIT) is a common yet under-recognized condition in the neuro ICU. It is caused by an autoimmune reaction to heparin-platelet factor 4 (PF4) complexes which causes activation of platelets and leads to thrombosis. Patients with aneurysmal subarachnoid hemorrhage treated by endovascular means are exposed to large doses of unfractionated heparin and therefore may be at high risk for HIT.

Methods:
The medical records of 117 consecutive patients with aneurysmal SAH were reviewed. Diagnosis of HIT was made by clinical determination.

Results:
Twenty-one patients (18%) met the diagnosis for HIT. Mean platelet (PC) nadir was 91,047 ± 27.3 in the HIT patients and 187.2 ± 55.07 in the non-HIT patients. HIT patients were more likely to be female, (86% vs. 59%, $p<0.023$) had more high grade (4 or 5) Hunt Hess scores, (38% vs. 14%, $p<0.010$) and had more aneurysms arising from the anterior circulation (100% vs. 69%, $p<0.016$). The groups did not significantly differ in age, aneurysm size, or Fisher score. Seventy-one percent HIT patients and 63% of non-HIT patients had endovascular coiling. HIT patients were more likely to have a new hypodensity on CT (71% vs. 21%, $p<0.0001$) and more like to die during their hospitalization (28% vs. 3.2 %, $p<0.001$). HIT patients who survived to discharge were more likely to have moderate to severe disability on the modified rankin scale than non-HIT patients (75% vs. 47%, $p<0.07$). PF4-platelet antibody ELISA and Serotonin Release Assay were sent in 14 of the HIT patients and were positive in 3 cases.

Conclusions:
Clinically diagnosed HIT is common in the SAH population. Patients with HIT are at higher risk for cerebral infarction, in-hospital mortality and disability. A high suspicion for HIT is appropriate in patients with aneurysmal SAH treated by endovascular means.

Financial Support: None

Poster 148

JUMPSTARTING MILD HYPOTHERMIA IN THE NEURO POPULATION: HITTING THE TARGET OF 33°C

Mary Kay Bader[1], Robert McCary[2], Joy Toyama[3], Anthony Kim[1], Robert Jackson[1], Ched Nwagwu[1], Farzad Massoudi[1], Sylvain Palmer[1]

[1]Mission Hospital, Mission Viejo CA, United States, [2]UCLA School of Nursing, Los Angeles CA, United States, [3]UCLA, Los Angeles CA, United States

Introduction:

Mild Hypothermia (32-34°C)[1]has been investigated in a variety of neurologic diseases and disorders. Since the 1990s research has shown that hypothermia provides vital neuroprotection after sustaining brain/spine injury from a trauma, stroke, or cardiac arrest. Hypothermia reduces increased ICP and improves neurologic outcomes.[1-4] Translation of the research to clinical practice poses many challenges such as determination of the most effective method of cooling, maintaining hypothermia, and slowly re-warming back to normothermia.

Methods:

A neuro hypothermia protocol was instituted in March 2008. 20 patients underwent mild hypothermia using a hydrogel-pad cooling system. This retrospective study analyzed the data related to induction start times and associated variables (BMI and BSA) and sought to determine whether any correlation existed between the variables and degree/hour induction to goal temperature - 33 + 0.2°C. Additionally, data was collected related to hours at 33°C and assessment of device control of ascent rate to 37°C.

Results:

Using the Pearson correlation coefficient and the Bonferroni standard correction method, 20 patient charts were reviewed and data assessed to determine the statistical relevance of several variables: gender- males, age 43, BMI 23.5, BSA 1.866, induction start temperature 36.3, target temperature 33.1, hour to target temperature 2.5, and temperature descent of 1.4 degrees/hour. It was determined that there was a significant statistical association between temperature changes (degrees/hour) and BMI/BSA values. The P-values for the BMI was determined to be .0096 and the BSA .00235. Target temperatures were maintained at 33°C with minimal variances. The ascent to 37°C was controlled at 0.05 °C/hour for the brain injured patients and 0.25°C/hour for the spinal cord injured patients.

Conclusions:

The final analysis of the data revealed that an individual's BMI and BSA does directly affect both the induction of hypothermia and the controlled re-warming back to the targeted normothermic goal.

References:

1. Tang X, Liu L, and Yenari L. Combination therapy with hypothermia for treatment of cerebral ischemia. J of Neurotrauma 26: 325-331, 2009.
2. Jiang J. Clinical Study of mild hypothermia treatment for severe traumatic brain injury. J of Neurotrauma 26, 339-406, 2009.
3. Hayashi N. 2009. Management of pitfalls for the successful clinical use of hypothermia treatment, J of Neurotrauma 26, 445-453, 2009.
4. Clifton G, Drever P, Valadka A, et al. Multicenter trial of early hypothermia in severe brain injury. J of Neurotrauma 26, 393-397, 2009.
5. Brain Trauma Foundation. Guidelines for the Management of severe traumatic brain injury. 3rd Edition. MR Bullock and JT Povlishock (Eds). Journal of Neurotrauma. May 1, 2007, 24(supplement 1): i-vi. doi:10.1089/neu.2007.9999.

Financial Support: None

Poster 149

LUNG INJURY, PNEUMONIA, AND FEVER DRIVE DURATION OF MECHANICAL VENTILATION AFTER CEREBRAL HEMORRHAGE

Storm Liebling, Isis Duran, Andrew Naidech
[1]Northwestern University, Chicago, Illinois, United States, [2]Northwestern Memorial Hospital, Chicago, Illinois, United States

Introduction:
Mechanical ventilation is associated with worse outcome after intracerebral (ICH) and subarachnoid hemorrhage (SAH). We sought to examine the predictors of duration of mechanical ventilation.

Methods:
We prospectively identified patients with spontaneous ICH and SAH who required invasive mechanical ventilation. Ventilator settings and measurements were recorded daily from the initiation of ventilation. Complications were prospectively recorded. Data are presented as mean +/- SD or N(%) when appropriate. Variables for multiple linear regression were chosen with a stepwise algorithm (in order of decreasing significance).

Results:
Of the 62 patients in this study, 37 (59.7%) were female, 35 (56.5%) were Caucasian, and the mean age was 61.2 +/- 15.4 years. Patients spent an average of 9.3 +/- 6.3 days ventilated. An average of 5.8 +/- 3.9 days were spent on Pressure Support Ventilation, 2.7 +/- 2.3 days on SIMV mode, and 1.6 +/- 3.1 days on AC mode. The maximum plateau pressure was 21.1 +/- 5.6 cm H20. Forty-nine (79.0%) patients had no pulmonary edema, three (4.8%) patients had pulmonary edema but no lung injury, four (6.5%) patients had ALI, while six (9.7%) patients had ARDS. In multiple linear regression, number of days febrile (3.0 per quartile, 95%CI 1.7-4.3 days), highest plateau pressure (0.4 cm H2O per day, 95%CI 0.1-0.6) and pneumonia (3.6 days, 95%CI 0.3-7.0 days) were associated with increased duration of mechanical ventilation ($P<0.04$ for all). Other variables did not enter the model.

Conclusions:
Duration of fever, maximum plateau pressure, and pneumonia are associated with increased duration of mechanical ventilation for patients with ICH and SAH. Minimizing fever, avoiding barotrauma and preventing pneumonia are attractive targets to decrease the need for mechanical ventilation after cerebral hemorrhage.

Financial Support: None

Poster 150

USING CONTINUOUS EEG (CEEG) TO PREDICT AND MONITOR ANTERIOR REGION VASOSPASM IN PATIENTS WITH SUBARACHNOID HEMORRHAGE (SAH)

Rahul Rathakrishnan, Jean Gotman, Francois Dubeau, Mark Angle
Montreal Neurological Institute and Hospital, Montreal, Canada

Introduction:
Permanent ischemic damage from anterior vasospasm causes significant morbidity. Previous studies reported the utility of CEEG in detecting vasospasm in patients with poor clinical grades of SAH. We aimed to ascertain if CEEG can aid prediction of onset of clinical vasospasm and monitor treatment response in unselected SAH patients.

Methods:
Patients with aneurysmal SAH of all clinical grades were prospectively studied. Regional anterior alpha power was quantitatively analysed. We assessed alpha power and variability using the product of standard deviation and mean power over a 6-hour duration, repeated along a window sliding by 30 minutes and graphically displayed. An independent clinician predicted the status of patients as improvement, deterioration or no change from the previous day. This was first done using only clinical data. CEEG trends prior to that day were then presented and another prediction made. Results were compared with the true clinical states that were determined independently. Clinical evolution in patients who were treated for vasospasm was correlated with daily mean alpha power.

Results:
8 patients were included (Hunt-Hess 1-5). 4 received intravenous milrinone for radiologically confirmed clinical vasospasm. 42 daily predictions were made. The sensitivity of predicting deterioration rose from 30% to 70% (specificity 75 and 72% respectively) and that of an improvement, from 9% to 45.4% (specificity 64% and 68%) when CEEG data was included. Three patients developed recurrences of vasospasm and underwent dose adjustments of milrinone. Reduction in mean daily alpha power was detected between 24 and 96 hours prior to clinical deterioration.

Conclusions:
CEEG can enhance the prediction of clinical vasospasm irrespective of the grade of SAH. It is a useful tool to monitor the response to treatment of vasospasm, aiding management decisions prior to clinical manifestations.

Financial Support: None

Poster 151

MANAGEMENT OF SUBARACHNOID HEMORRHAGE AND INTRACEREBRAL HEMATOMA: CLIPPING AND CLOT EVACUATION VERSUS COIL EMBOLIZATION FOLLOWED BY CLOT EVACUATION

Kenneth De Los Reyes, Arjun Gowda, Errol Gordon, Aman Patel, Joshua Bederson, Jennifer Frontera
Mount Sinai School of Medicine, New York, NY, United States

Introduction:
Aneurysmal Subarachnoid hemorrhage (SAH) with associated intracerebral hemorrhage is often treated with concomitant surgical clipping and clot evacuation. Aneurysm coiling followed by clot evacuation may be an alternative treatment.

Methods:
A retrospective review was conducted of SAH patients between 7/200-3/2009. Inclusion criteria were: aneurysmal SAH, ICH>30ml or with midline shift>5mm, and candidacy for surgical clot evacuation. The Mann Whitney-U nonparametric test or Fisher exact test was used to compare demographic and radiographic criteria, time to aneurysm protection, 3-month functional outcome (categorized as dead or GOS 1-3), length of stay (LOS), and treatment complications of patients who underwent coiling followed by clot evacuation (group A) versus clipping with evacuation (group B).

Results:
Of 265 SAH patients, 10 underwent coiling followed by clot evacuation and 8 underwent clip with clot evacuation. Compared to Group B, Group A had a lower GCS score (median 5.5 versus 7.5), higher ICH score (median 3 versus 2), worse modified Fisher score (median 4 versus 3) and higher rate of herniation at admission (50% versus 25%). The median time to aneurysm protection was significantly shorter in Group A (299 versus 885 minutes, $P<0.001$). Comparing group A to B, the rates of death (30% versus 25%), poor outcome (70% versus 50%), median ICU LOS (20 versus 22 days), median hospital LOS (27 versus 29 days) and total median direct costs ($64,537 versus $61,243) were similar (all P=NS). Complications of treatment occurred in 1 patient in group A and 0 in group B (P=1.00) and residual aneurysm occurred in 1 patient in each group (P=1.00). Rebleed occurred in 1 patient in group A and 0 in group B (P=1.00).

Conclusions:
Coiling followed by clot evacuation is associated with a faster time to aneurysm protection and similar outcome, LOS, and cost as clipping and evacuation. This may be a viable alternative treatment strategy.

Financial Support: None

Poster 152

NEUROANATOMIC BASIS FOR POSTURING REFLEXES IN ACUTE HEAD TRAUMA: RE-EVALUATING THE EVIDENCE

Hans Puttgen, Robert Kowalski, David Efron, Romergryko Geocadin
Johns Hopkins University, Baltimore, MD, United States

Introduction:
The neurologic mechanism leading to unresponsiveness after acute traumatic brain injury is not well understood. Posturing reflex examination in evaluating comatose patients is ubiquitous. The reliability of this practice has not been systematically evaluated.

Methods:
From the trauma service registry at a Level 1 Trauma Center, all admissions between from 7/1/1999 to 6/30/2007 where the patient had a Head component of the Abbreviated Injury Score >1 were identified. From this group of 3,478 patients, the records of the 193 patients with a Glasgow Coma Scale (GCS) ≤8 on presentation and a brain CT scan performed in the ED were evaluated. CT scans were scored for injury by location and the motor component of the GCS (GCS_M) was noted from ED documentation.

Results:
The study population was young (mean age 38.4) and predominantly male (84%). The GCS_M was ≤2 (indicating extensor posturing or no response) for 127 patients and >2 (indicating flexor response or better) for 66 patients. On univariate analysis, intra-axial injury above the thalamus did not correlate with the GCS_M (42 with GCS_M≤2, 18 with GCS_M>2, p=0.545). A second analysis of intra-axial injury above the midbrain again showed no reliable correlation with GCS_M (54 with GCS_M≤2, 25 with GCS_M>2, p=0.079). Patients with extensor or worse exams were less likely to have a Glasgow Outcome Score >3 (41 (32%) with GCS_M≤2 , 33 (50%) with GCS_M>2, p=0.018).

Conclusions:
GCS_M responses of flexor or worse did not reliably correlate with injuries at the level of the thalamus or below. However, lower GCS_M was still associated with poor GOS. This study points to a need for reinvestigation into the neuroanaotmic basis for posturing and unresponsiveness to enhance the understanding and improve acute management of these patients.

Financial Support: None

Poster 153

IMPLEMENTATION OF CEREBRAL MICRODIALYSIS IN A COMMUNITY-BASED HOSPITAL SETTING—A FOUR YEAR EXPERIENCE

Jeff Chen, Sandy Cecil, Patrick Chen, Susan Rowland, Sarah Callaway, David Adler
Legacy Emanuel Medical Center, Portland, Oregon, United States

Introduction:
Since the CMA 600 cerebral microdialysis analyzer received FDA approval for clinical use in 2005 in the United States, cerebral microdialysis has gained increasing acceptance as an adjunct in the multimodality monitoring of the brain after traumatic brain injury, subarachnoid hemorrhage, and stroke. We describe a single institutional four year experience with CMA 600 and recent ISCUS^{flex}.

Methods:
The CMA 600 and ISCUS^{flex} analyzers, CMA 107 pump, and CMA 70 microdialysis catheters were obtained from CMA Microdialysis (Solna, Sweden). Perfusion fluid CNS (artificial CSF) was perfused at 0.3 ul/min, and samples collected hourly. Lactate, pyruvate, glucose, glycerol, and glutamate levels were entered into the ICU pilot program along with neurophysiologic parameters to analyze relationships/trends. All cerebral microdialysis catheters were implanted by board certified attending neurosurgeons at a single community-based hospital. Catheters were implanted directly into the brain via a 1mm diameter corticectomy at the time of craniotomy. Catheters were placed via twist drill hole/bolt when craniotomies were not performed.

Results:
From July 2005 to July 2009, 154 cerebral microdialysis catheters were implanted in 114 multi-modality monitored patients with 130 being placed via open craniotomy, and 24 via twist drill hole/bolt technology. The age range of patients: 5 months-90 years with a M:F ratio of 2:1. Cranial pathologies included: subdural hematoma 15, epidural hematoma 3, intracerebral hemorrhage 30, subarachnoid hemorrhage 16, mixed injury 34, other 16. No infections were associated with the microdialysis catheters.

Conclusions:
1. Cerebral microdialysis was readily implemented in a community-based hospital.
2. Specialized nursing training/education is vital.
3. The Lactate/Pyruvate ratio demonstrated the expected correlations with ↑ICP and ↓CPP.
4. Cerebral microdialysis allowed optimization of cerebral glucose.
5. Cerebral microdialysis provides additional information to guide therapy to optimize neurologic outcome.

Financial Support: None

Poster 154

EXPERT VS. NON-EXPERT USE OF QUANTITATIVE EEG TOOLS FOR SEIZURE IDENTIFICATION IN THE ICU

Cecil Hahn, Craig Stewart, Vera Nenadovic, Ayako Ochi, Hiroshi Otsubo, Jamie Hutchison
The Hospital for Sick Children, Toronto, Ontario, Canada

Introduction:
Although continuous EEG monitoring is a valuable tool for the monitoring of brain function in the ICU, EEG interpretation is customarily performed by experts who are not continually present at the bedside of critically ill patients. Prompt recognition of EEG seizures by bedside caregivers may be facilitated by displaying quantitative EEG trends at the bedside. Our aim was to compare the utility of one such trending tool, Color Density Spectral Array (CDSA), for seizure identification in the hands of EEG experts versus bedside ICU nurses.

Methods:
27 continuous EEGs performed in a pediatric ICU were transformed into 8-channel CDSA displays. Three neurophysiologists, 3 EEG technologists and 2 bedside nurses underwent training on the use of CDSA for seizure identification. Participants were then presented with CDSA displays of all EEG recordings and asked to mark events that they suspected to be seizures. Their performance was then compared to seizures identified by a board-certified electroencephalographer on the raw 13-21 channel EEG.

Results:
The 27 EEG recordings contained 553 discrete seizures over 487 hours. Sensitivity for seizure identification and false-positives are reported below. Values are median (range).

	Sensitivity (%)	False-positive rate (# / hour)
ICU nurses (n=2)	89.0 (88.2–89.7)	0.32 (0.29–0.35)
Neurophysiologists (n=3)	83.3 (73.3–86.7)	0.06 (0.00-0.13)
EEG technologists (n=3)	73.3 (66.7–76.7)	0.00 (0.00-0.14)

Using CDSA, ICU nurses were the most sensitive at identifying seizures, however they also demonstrated the highest false-positive rate. Neurophysiologists and EEG technologists demonstrated slightly lower sensitivity, and much lower false-positive rates. However, on individual EEG recordings performance varied greatly, with group median sensitivities ranging from at 0% to 100%.

Conclusions:
Neurophysiologists, EEG technologists and bedside nurses demonstrated comparable performance in seizure identification using CDSA. The observed differences in sensitivity and false-positive rates between different groups of reviewers are smaller than the variability in their performance on individual EEG recordings.

Financial Support: None

Poster 155

THROMBOELASTOGRAPHY AS AN ADJUNCT TOOL TO ASSESS COAGULATION DISTURBANCES IN ISOLATED TRAUMATIC BRAIN INJURY PATIENTS

Asaph Nini[1], David Livingstone[1], Elena Mishuk[1], Alexander Kogan[1], Eran Segal[2]
[1]Haim Sheba Medical Center, Tel Hashomer, Israel, [2]Assuta Medical Center, Tel Aviv, Israel

Introduction:
Coagulation disorders are common after traumatic Brain Injury (TBI), and may contribute to morbidity and mortality (1). These disorders are complex and dynamic over time, making clinical evaluation of coagulation status of the patients difficult (2). Thromboelastography (TEG) has been suggested as a tool for rapid assessment of such states. TEG is a test of clot formation and lysis, providing a holistic assessment of clot formation time and strength. It is an easy to perform, point of care test that enables clinicians to differentiate hypo or hypercoagulability, and the factors contributing to each, and evolution over time.

The aim of this work is to show the contribution of TEG to the evaluation of coagulopathies in patients with isolated TBI.

Methods:
We have retrospectively inspected TEG records and routine coagulation studies from patients with isolated TBI, and checked for signs of either bleeding tendency or signs of hypercoaguilation

Results:
Ten patients with isolated traumatic brain injury were evaluated using TEG. Reasons for tests included workup of suspected bleeding, assessment of hypercoagulable states, or planned invasive procedures. Three of these patients showed increased LY30, indicating thrombolysis, and two patient showed prolongation of the R value, indicating prolonged clotting time. Two patients showed increased Maximal Amplitude (MA), indicating a hypercoagulable state.

Conclusions:
Thromboelastography is a useful adjunct tool in the assessment of coagulation status in isolated TBI patients, and may help in clinical decision making in such patients. Further work, relating thromboelastography results, prognosis and management are warranted.

Financial Support: None

Poster 156

DIFFERENCES IN RESTING ENERGY EXPENDITURE OF CRITICALLY ILL NEUROLOGY PATIENTS ADMITTED TO A NEUROSCIENCE INTENSIVE CARE UNIT

Christine Hartney, Kathryn Keim, Diane Sowa, Richard Temes
Rush University Medical Center, Chicago, IL, United States

Introduction:
The objective of this study was to compare differences in resting energy expenditure (REE) results of critically ill neurology patients based on gender, body mass index (BMI) class and race.

Methods:
This study was a retrospective chart review of patients admitted to the Neurosciences Intensive Care Unit at an urban medical center who were started on enteral nutrition support. The research methods received approval from the Institutional Review Board for Human Studies.

Results:
Forty-six patients (25 male) with average age of 56.3 $\pm$ 2.3 years (mean $\pm$ standard deviation) and BMI= 28.2 $\pm$ 1.2 kg/m^2 met inclusion criteria and had at least one metabolic cart measurement with a Medgraphics CPX Ultima Metabolic Cart (Medical Graphics Corporation, Minneapolis, MN). Results of the male mean REE was 2213 $\pm$ 734 kcal and female mean REE was 1992.2 $\pm$ 597 kcal. There were no significant differences in measured REE based on gender (p=0.289), BMI class (p=0.783), and race (p=0.406).

Conclusions:
The differences between gender, BMI class and race may not have been detected as a limitation of the sample size. Research is needed to further explore the relationship among gender, BMI class and race and use of established predictive equations for the critically ill neurology patient.

Financial Support: None

Poster 157

NEUROSCIENCE ICU PROVIDER ATTITUDES REGARDING PALLIATIVE CARE CONSULT SERVICES

Karen Warford, Angel Sostre, Maria Walker, Owen Samuels, Wendy Wright
Emory University Hospital, Atlanta, GA, United States

Introduction:
Many critically ill neurologic and neurosurgical patients undergo a significant change in functional status or require end-of-life care. Therefore, palliative care is an integral part of care provided by neurocritical care physicians and midlevel providers. At times, the needs of the patients and families can overwhelm these clinicians, whose focus is often on curative measure, so there may be a benefit to integrating a formal palliative care consultation service into the neuroICU.

Methods:
An anonymous survey was conducted among the four neuro ICU physicians and nine nurse practitioners regarding the integration of a palliative care consult service into the neurocritical care service. The survey consisted of seven "yes" or "no" questions and a write-in section for comments.

Results:
All 13 providers thought that it was helpful to have the palliative care consult team in the ICU, and that they provided added support not just for families, but to the physicians and nurse practitioners. 1/13 respondents stated that palliative care was only appropriate for families that wanted to decelerate care. Only 4/13 responded that they were aware of the existence of formal criteria that were designed to trigger the consultation of the palliative care team. Feelings were mixed regarding nurse-driven consults, with only 4 respondents feeling that this was appropriate.

Conclusions:
In general, the formal palliative care consult service was felt to be a welcome addition. Clearly, the existence of formal criteria to trigger a palliative care consult had not been emphasized enough to physician and midlevel providers, and the concept of nurse-driven consults was not accepted by the majority of number of providers. Additional comments obtained will be used to improve the process by which palliative care services are obtained.

Financial Support: None

Poster 158

THE MINIMAL IMPORTANT DIFFERENCE IN TRAUMATIC BRAIN INJURY RESEARCH: RESULTS OF A NEUROCRITICAL CARE SOCIETY SURVEY

Jordan Bonomo[1], Lori Shutter[1], Jonas Ellenberg[2], Wei Yang[2], Peter Le Roux[2]
[1]*University of Cincinnati, Cincinnati, Ohio, United States,* [2]*University of Pennsylvania, Philadelphia, PA, United States*

Introduction:
Most clinical trials in traumatic brain injury (TBI) have failed to demonstrate a therapeutic benefit. One factor implicated in these failures is an inadequate estimate of the smallest clinically meaningful beneficial effect — the Minimal Important Difference (MID). In this study we surveyed the Neurocritical Care Society (NCS) membership to determine an MID for TBI clinical trials.

Methods:
A survey approved by the NCS Research Committee was developed to assess the MID that would lead physicians to recommend a new therapy for TBI patients. The survey was distributed online to all NCS members with a 1-week response period.

Results:
There were 125 responses (15.4%) from 814 NCS members. Respondents included neuro-intensivists (61.2%), neurologists (29.3%), and neurosurgeons (13.8%); 75% were in academic practice on average 10.8 years. Two-thirds (62.6%) cared for 4 to 14 severe TBI patients monthly and 43.3% had participated in TBI clinical trials. One third believed that 50% of patients would consent to minimal risk trials. The preferred primary outcome measures were mortality, Glasgow Outcome Score (GOS), and GOS extended, while the SF-36, neuropsychological measures and sliding dichotomy were the least preferred. The preferred secondary outcome measures were intracranial pressure (ICP) control, therapeutic intensity level (for ICP) and repeat imaging. Organ dysfunction scores and biomarkers were least preferred. A reduction in unfavorable outcome of 10% (IQR 5-10%) was reported as the MID needed to introduce a new therapy. MID rather than “number needed to treat” was the preferred method to describe trial efficacy.

Conclusions:
In this NCS survey, the preferred primary outcome measure for TBI trials was mortality or GOS. A 10% reduction in unfavorable outcome is considered the MID. This information can be used to help define sample size for future TBI clinical trials.

Financial Support: None

Poster 159

QUALITY AND SENSITIVITY COMPARISON OF ELECTROENCEPHALOGRAPHY SIGNALS OBTAINED WITH TEMPLATE VS. TECHNICIAN APPLIED ELECTROENCEPHALOGRAPHY ELECTRODES

Bradley Kolls
Duke University Medical Center, Durham, NC, United States

Introduction:
Compare the quality and sensitivity of electroencephalography signals (EEG) obtained with a disposable template system to EEG obtained by certified EEG technicians.

Methods:
Prospectively acquired EEG data were obtained in 8 hour blocks (matched pairs) from leads placed by a certified EEG technician vs. those placed using a disposable template system (BraiNet®). Quality measures included start and end recording of impedance, elapsed time from physician's order to first recorded EEG, and a blinded subjective evaluation of data quality. All segments of data were de-identified and will be read by a blinded reviewer highly experienced in EEG interpretation.

Results:
Analysis from the first 13 subjects of a 100 subject trial is presented here. Average impedances in the BraiNet® group were within recommended guidelines, but were slightly higher than technician applied leads. Groups had similar impedance variance, lead failure rates, and maximal difference in impedance at the beginning and at the end of the 8 hour blocks. The difference in mean time to first EEG for the BraiNet® group (98 mins) vs. technician applied leads (323 mins) was statistically significant ($p<0.003$). Evaluation of sensitivity is pending collection of the remaining data sets.

Conclusions:
Preliminary analysis indicates the use of a disposable template system that allows a non-technician healthcare provider to place EEG leads is feasible and safe. No significant differences in EEG quality during 8 hours of recording were found, and use of BrainNet® leads was associated with a significant reduction in the time from order to the first page of EEG data. This preliminary assessment does not allow for conclusions about the overall quality and sensitivity of disposable template leads; the complete set of EEG segments needs to be collected and undergo blinded review.

Financial Support: None

Poster 160

ADRENERGIC MARKERS OF CLINICAL AND OUTCOME MEASURES IN SUBARACHNOID HEMORRHAGE

Scott Pello, Keith Dombroski, William McBride, Valerie Dechant, Monisha Kumar, Rodney Bell, Michael Moussouttas
Thomas Jefferson University Hospital, Philadelphia, PA, United States

Introduction:
Outcome following aneurysmal subarachnoid hemorrhage (SAH) is related to various demographic and clinical factors. Biomarkers are an increasingly employed means for determining outcome in neurologically injured patients. The purpose of this study is to correlate cerebrospinal fluid (CSF) adrenergic compound and metabolite levels to clinical and outcome measures.

Methods:
40 consecutive SAH patients with ventriculostomy had CSF collected 1ml within 3d of onset. CSF was assayed for epinephrine (Epi), norepinephrine (NE), and dihydroxyphenylglycol (DHPG) by HPLC. Levels were compared to various demographnic, clinical, and radiological measures, and to mortality at 30 days.

Results:
Mean age was 58yo and 65% were female. HH grade was 3 in 53%, 4 in 42%, and 5 in 5%. No correlation was found for age, but women had greater DHPG levels (658pg/dl vs 408pg/dl, p=.022). Dichotomized HH score demonstrated greater Epi levels in G4/5 patients compared to G3 patients (32pg/dl vs 14pg/dl, p=.033).

Patients who died had also greater Epi levels (38pg/dl vs 16pg/dl, p=.039) yet lower DHPG levels (421pg/dl vs 635pg/dl, p=.064), but regression analysis incorporating HH grade eliminated these associations.

Conclusions:
In SAH, women demonstrate greater elevations in the NE metabolite DHPG, and greater elevations in Epi are present in HH grade 4/5 patients. Patients who die have greater CSF Epi levels which appears related to the severity of the disease.

Financial Support: None

Poster 161

QUID PRO QUO: THE ROLE OF "BASIC SCIENCE" AND ITS PRACTITIONERS IN NEUROCRITICAL CARE RESEARCH

Gail Pyne-Geithman, Opeolu Adeoye, Jordan Bonomo, Carolyn Koenig, Jed Hartings, Lori Shutter
University of Cincinnati, Cincinnati, OH 45267, United States

Introduction:
As in clinical neurocritical care (NCC) practice, effective NCC basic science research requires organized interdisciplinary collaboration. The purpose of this abstract is to share our experience in building a basic science collaborative to facilitate the efforts of others and foster discussion regarding engagement of basic scientists in the Neurocritical Care Society (NCS) and NCC research efforts.

Methods:
Our institution is active in interdisciplinary NCC clinical practice and fellowship training, in addition to conducting various clinical trials relevant to NCC. Collaborating with the clinicians is a core of basic scientists who are working to integrate their funded research into the fabric of care in the Neurosciences ICU. The composition of the team is truly interdisciplinary, spanning multiple clinical and basic science departments and colleges within our institution.

Results:
Frequent meetings among NCC physicians, surgeons, research nurses in the Division of Clinical trials and basic scientists have resulted in fruitful collaborations in teaching, research and funding. Clinical responsibilities of fellows and residents limits time for bench research, so joining an existing project allows time and resources to be used productively. NCC fellows are teamed with a basic science mentor, and these collaborations often continue beyond the tenure of the fellowship. The basic scientists benefit, as current basic research needs to have translational potential to the clinical setting. Basic scientists attend clinical rounds, reinforcing the benefits of truly translational research.

Conclusions:
Gaining a reputation for quality research that enables consistent funding and earns respect from the NCC community requires engagement and input of basic scientists. Individual institutions can solicit interested basic scientists to join in their research planning and execution and augment the training of residents and fellows, thus preparing the next generation of research-trained clinicians.

Financial Support: None

Poster 162

EFFECTS OF TIGHT GLUCOSE CONTROL ON INFECTION RATES ASSOCIATED WITH EXTERNAL VENTRICULAR DRAINS (EVD) IN NEURO INTENSIVE CARE UNIT

Sophie Samuel, David Romerill
The Methodist Hospital, Houston, Texas, United States

Introduction:
The purpose of this study was to evaluate the association between tight glucose control and the incidence of ventriculitis in neuro intensive care unit with EVDs

Methods:
The hospital's computer system was used to identify patients admitted between January 1, 2006 and December 31, 2008 to the neuro ICU with documented EVD placement. Patients' 18 years of age or older and deemed to require insulin therapy by the admitting physician were included in the study. We excluded patients if they had EVD placement or documented CSF infections before admission to the unit, were treated with antibiotics a week prior to admission, and length of ICU hospitalization less than seven days. The primary outcome measure was EVD related infection. The secondary outcome measures were in hospital and ICU length of stay and in hospital death

Results:
The association between glucose control and positive CSF cultures was described using the morning blood glucose for seven consecutive days stratified based on the number of blood glucose readings that fell between 80-120 mg/dl. The binary logistic regression model showed that patients with a higher percent of readings in tight blood glucose range were more likely to have CNS infection (odds ratio 1.05; $p < .01$). The secondary outcomes could not be measured because we did not have enough readings to stratify our data into categories

Conclusions:
Contrary to our hypothesis, the results from our study suggest a possible association between tight blood glucose and an increase in EVD related infections. At this time we are unable to make recommendations based on these results given the inherent limitations of our study ; i.e., small sample size, retrospective design, single centered and single morning blood glucose reading to assess glucose control

Financial Support: None

Neurocritical Care Society
7th Annual Meeting
WHAT'S THE BIG IDEA? POSTER PRESENTATIONS

Authors will be standing by their posters at the hours indicated below:
*Posters 170-*185: Friday, November 13, 2009 from 5:30 – 7:00 p.m.*
**Poster 182: Thursday, November 12, 2009 from 5:30 – 7:00 p.m.*

Poster #	Title	Presenting Author
170	A Multicenter Randomized Controlled Trial Comparing Restrictive and Liberal Transfusion Strategies in Subarachnoid Hemorrhage (Running Title: Transfusion in Subarachnoid Hemorrhage (TISH) Trial)	Peter Le Roux, MD
171	Accuracy of Multidetector Computed Tomographic Angiography and Cerebral Perfusion in Diagnosis of Brain Death	Marcelo Costilla, MD
172	Hypothermia for Refractory Status Epilepticus	Jesse Corry, MD
173	A Prospective Trial of Elective Extubation in Brain Injured Patients Meeting Ventilatory Extubation Criteria	Edward Manno, MD
174	Prognostic Value of Brain Diffusion-Weighted Imaging after Cardiac Arrest	Christine Wijman, MD, PhD
175	Decompressive Craniotomy for Hypertensive Capsulo-Ganglionic Hemorrhage	Salah Keyrouz, MD
176	Hypertensive Hypervolemic Vasodilatory (HHVD) Therapy with Intravenous Nicardipine for the Treatment of Cerebral Vasopsasm	Benjamin Emanuel, DO
177	Weaning from Mechanical Ventilation in the Neurocritical Care Unit	Gene Sung, MD, MPH
178	Detection and Treatment of Cerebral Vasospasm in Bacterial Meningitis to Improve Survival	Valerie Coon, MD
179	Indomethacin in the Treatment of Intracranial Hypertension	Marcelo Costilla, MD
180	Aggressive Hyperglycemia Management after Acute Brain Injury	Julius Gene Latorre, MD, MPH
181	AFTERSHOC (Anti-Fibrinolytic Therapy in the Emergency Room for Subarachnoid Hemorrhage OutCome)	Brad Zacharia, MD
182	Long-term Recovery of Devastating Neurologic Diagnoses	Eli Feen, MD
183	Incremental Multimodal Neuroprotective Therapies—A Proposed Solution to Neuroprotective Nihilism in Clinical Trials	W. Andrew Kofke, MD, MBA
184	Continuous Veno-venous Hemofiltration For The Management Of Refractory Intracranial Hypertension	Jeff Fletcher, MD
185	Prediction of Coma Outcome by MRI: The Need for a North American Arm to an European Study	Louis Puybasset, MD, PhD

WHAT'S THE BIG IDEA? Poster 170
A MULTICENTER RANDOMIZED CONTROLLED TRIAL COMPARING RESTRICTIVE AND LIBERAL TRANSFUSION STRATEGIES IN SUBARACHNOID HEMORRHAGE

RUNNING TITLE:
TRANSFUSION IN SUBARACHNOID HEMORRHAGE (TISH) TRIAL

Peter le Roux[1], Jonas Ellenberg[1], Stephan Mayer[2], Joanne Festa[2], Andrew Naidech[3], Andreas Kramer[4], Loch MacDonald[5]
[1]University of Pennsylvania, Philadelphia, PA, United States, [2]Columbia University, New York , NY, United States, [3]Northwestern University, Chicago IL, United States, [4]Calgary University, Calgary AL, Canada, [5]University of Toronto, Toronto ON, Canada

Background:
Following aneurismal subarachnoid hemorrhage (SAH) half the patients' die and only one third of survivors make a full recovery. The optimal hemoglobin (Hgb) after SAH, however, is uncertain. Higher-goal Hgb and more red blood cell transfusion (RBCT) lead to worse outcome in general critical care. Clinical series suggest that RBCT may increase vasospasm risk and exacerbate outcome after SAH. However other studies suggest that a higher Hgb may be associated with better outcome and less cerebral infarction after SAH.

Hypothesis:
We now will examine the hypothesis that patient outcome after SAH is better when an Hgb level of 8.5g/dl rather than an Hgb of 11g/dl triggers transfusion.

Study design:
We propose a multi-center, prospective, phase III randomized, clinical trial involving adults admitted to University based NICUs within 48 hours of SAH..

Subjects:
Eligible SAH patients will be randomly assigned to one of two treatment groups, 1) restrictive (Hgb of 8.5g/dl) or 2) liberal (Hgb of 11.5g/dl) transfusion triggers stratified by center and SAH severity.

Main outcome measure:
The primary objective is to determine if SAH subjects who have a restrictive transfusion trigger during the first 14 days of care, are more likely to have a favorable 6-month outcome than subjects transfused with a liberal trigger. The trial is designed to detect an overall absolute difference of 10% in the proportion of favorable outcomes (Glasgow Outcome Score of Good or Moderate Disability).

Secondary outcome measures:
Secondary objectives include: 1) Determine if a restrictive policy is associated with less vasospasm. 2) Examine the relationship between Hgb and 6 month outcome and cerebral infarction. The number of randomized subjects is expected to be 758.

Power analysis:
The number of randomized subjects is expected to be 758.

Current status:
To date 25 centers have agreed to participate. This group will receive administrative, statistical and data coordinating support from the University of Pennsylvania Center for Clinical Epidemiology and Biostatistics. The proposal is under review at the NIH.

Financial Support: None

WHAT'S THE BIG IDEA? Poster 171

ACCURACY OF MULTIDETECTOR COMPUTED TOMOGRAPHIC ANGIOGRAPHY (CTA) AND CEREBRAL PERFUSION (CTP) IN DIAGNOSIS OF BRAIN DEATH

Alicia Saenz Valiente[1], Marcelo Costilla[1], Fernando Abramzon[1], Sonia Lesyk[1], Walter Videtta[1], Gustavo Domeniconi[2]
[1]Hospital de Trauma y Emergencias "Federico Abete", Pablo Nogues - Pcia de Buenos Aires, Argentina,
[2]Sanatorio de la Trinidad, San Isidro - Pcia de Buenos Aires, Argentina

Background:
Brain death (BD) is diagnosed clinically by documentation of coma, absence of brainstem reflexes, and apnea unresponsive to hypercarbia. In Argentina (like other countries) other confirmatory tests are required as a part of the diagnostic criteria. The utility of CTA with cerebral perfusion was reported by Qureshi in 2000 and then evaluated by Combes et al and they found 30% false negative rate for the test; moreover, Greer et al reported one case of false positive. In Spain, Otero has reported a sensitivity of 100% in a series of 6 patients who had CTA and CT perfusion. Accuracy of CTA and CTP must be assessed.

Hypothesis:
We propose that CTA and CTP is a reliable confirmatory test for BD, with particular interest in cases where barbiturates or other CNS depressant drugs difficult to diagnose clinically or by electrophysiological studies.

Study design:
Prospective multicenter study to determine the accuracy diagnostic of brain death with CTA & CTP in patients with suspicion of BD according clinical criteria (CC) defined by neurological criteria, apnea test; compared with electrophysiological methods and TCD evaluation.

Subjects:
All adults of at least 18 years of age who meet the CC of BD.

Setting:
Intensive care Unit, Emergency Department, Neurocritical Care Unit, Stroke Unit in Hospitals with availability of TCD, EEG and Multi-row CT 24 hours.

Intervention:
- In patients with CC of BD, we will be performing CTP and then CTA.
- All the case will be made an EEG and TCD evaluation

Main outcome measure:
- Evidence of cerebral circulatory arrest.
- Absence of cerebral perfusion
- Sensitivity and Specificity, PPV and NPV for CTA & CTP compared with CC, EEG & TCD.

Secondary outcome measures:
- Accuracy of CTA & CTP in patients with recent utilisation of CNS depressor or NM blockers to confirm BD before EEG and TCD.
- Complications rate associated with the use of contrast.
- Renal failure post contrast use.

Power analysis:
We need at least 100 patients to to achieve a sample size that allows the analysis of sensitivity, specificity and construction of ROC.

Current status:
Not Started.

Funding is needed to support the project.

Financial Support: None

WHAT'S THE BIG IDEA? Poster 172
HYPOTHERMIA FOR REFRACTORY STATUS EPILEPTICUS
Jesse Corry[1], Michael Diringer[2], Tamer Abdelhak[1], Rajat Dhar[2], Panayiotis Varelas[1]
[1]Henry Ford Hospital, Detroit, MI, United States, [2]Washington University, St Louis, MO, United States

Background:
Status epilepticus (SE) affects 150,000 Americans yearly. 30-50% of cases fail initial therapy. Refractory cases requiring midazolam, pentobarbital, or propofol fail in 8-21% of patients. Outcome is independent of the anaesthetic agent or extent of EEG suppression. Hypothermia (HT) in rodent models of SE abates epileptic discharges and neuronal death. Case reports demonstrate HT as an effective adjunctive or primary treatment for refractory SE (RSE).

Hypothesis:
HT effectively treats RSE.

Study design:
Multicenter, randomized, non-blinded phase IIa trial of HT in RSE evaluating target temperature and duration.

Subjects:
Inclusion criteria: RSE patients failing initial benzodiazepines and phenytoin treatment, $\geq$18yo, and $\geq$35^{o}C upon admission. Exclusion criteria: immunosuppression, active infected, unstable cardiac rhythm, coagulopathy, SE secondary to cardiac arrest/anoxia, active CHF, pregnancy, in-place IVC filter, or DNR/DNI status.

Setting:
ICU

Intervention:
EEG monitored patients with RSE will be randomized. Controls will be managed at the intensivist's discretion. Remaining patients will be varied by HT duration (12hr v 24 hr) and target temperature (32-33^{o}C v 31-32^{o}C). Initially, seizure will be treated with midazolam while endovascular cooling catheters are placed, and a cold saline bolus is given. At goal temperature, midazolam will be weaned to off or the lowest dose necessary for absence of seizure activity.

Main outcome measure:
Anaesthetic medication requirements and seizure burden (i.e. seizure frequency, duration, and number), from achieving goal temperature until ICU discharge, as compared with control.

Secondary outcome measures:
Total IV anaesthetic
ICU duration
Vasopressor/inotrope requirements
Modified Rankin score at discharge
Infectious, device, and coagulopathic complications

Power analysis:
$\geq$ 225 patients ($\geq$ 45 each arm) would be 80% powered to detect a 15% difference in HT vs control (alpha=0.05), and a 20% difference of each arm vs control (alpha=0.0125), in the primary outcome measures of anaesthetic and seizure burden.

Current status:
A phase I study is underway at Washington University and Henry Ford Hospital. This study remains unfunded.

Financial Support: None

WHAT'S THE BIG IDEA? Poster 173
A PROSPECTIVE TRIAL OF ELECTIVE EXTUBATION IN BRAIN INJURED PATIENTS MEETING VENTILATORY EXTUBATION CRITERIA.
Edward Manno
Lerner College of Medicine Cleveland Clinic Foundation, Cleveland, OH, United States

Background:
Traditional dogma has mandated that brain injured patients with a Glasgow coma score of < 8 need to remain intubated for airway protection. Recent prospective studies have suggested that in brain injured patients with intact airway reflexes, prolonged intubation leads to an increase in nosocomial pneumonias and worse outcomes. A recent randomized trial suggested randomization of brain injured patients into early and delayed extubation is safe and feasible.

Hypothesis:
Brain injured patients with a GCS <8 and intact airway reflexes will not have worse outcomes if extubated early compared to a similar extubation group with delayed extubation until their GCS becomes >8.

Study design:
Multi-center non blinded randomized phase 2 trial.

Subjects:
A non inferiority trial of immediate versus delayed extubation accounting for a 0.5 change in modified Rankin Scores would require 110 patients in each treatment arm for 80% power.

Setting:
ICU

Intervention:
Immediate extubation in stable brain injured patients with intact airway reflexes as evaluated by an airway care score.

Main outcome measure:
Hospital discharge modified Rankin Score.

Secondary outcome measures:
Hospital and ICU length of stay, nosocomial pneumonias, reintubations.

Power analysis:
All intubated adult patients with severe brain injury defined as a GCS< 8 are potentially eligible. Exclusion criteria includes: Patients < 18, lack of surrogate informed consent, intubation for therapeutic interventions, anticipated medical or neurological worsening, intubation for airway edema, or prolonged intubation > 2 weeks.

Current status:
A feasibility trial has been completed and published. The study is in search of centers and funding.

Financial Support: None

WHAT'S THE BIG IDEA? Poster 174

PROGNOSTIC VALUE OF BRAIN DIFFUSION-WEIGHTED IMAGING AFTER CARDIAC ARREST

Christine Wijman[1], Stephan Mayer[2]
[1]*Stanford University, Palo Alto, CA, United States,* [2]*Columbia University, New York, NY, United States*

Background:
Quantitative diffusion-weighted imaging MRI (DWI) in comatose post-cardiac arrest survivors holds promise as a prognostic tool. Between 1 and 5 days more than 10% of brain volume with an ADC value < 650x10-6mm2/sec identifies poor outcome patients with 100% specificity and 81% sensitivity (Ann Neurol 2009;65:394–402). This threshold needs validation in an external dataset.

Hypothesis:
We hypothesize that the capacity for recovery of consciousness in comatose cardiac arrest survivors can be predicted with quantitative DWI.

Study design:
Multicenter observational study of DWI MRIs in comatose cardiac arrest survivors obtained between 1 and 5 days after the arrest.

Patient data will be recorded using a web-based data entry form including baseline characteristics, neurological examinations, results of neurophysiological testing, cause of death, and 30-day outcome.

Patients may be entered retrospectively and prospectively.

Brain DWI scans will be blindly analyzed centrally and an outcome measure (survival versus death or vegetative at 30 days) will be assigned.

Subjects:
Patients who remain comatose after cardiac arrest and who have undergone DWI between 1 and 5 days after the arrest.

Setting:
Multiple centers

Main outcome measure:
The specificity of the predefined DWI threshold (ADC of 650x10-6 mm2/sec) for prediction of poor outcome (defined as death or failure to recover consciousness at 30 days).

Secondary outcome measures:
The sensitivity of the predefined DWI threshold in comparison with the 72-hour neurological examination and, if available, SSEPs and peak serum levels of neuron specific enolase.

Power analysis:
Assuming a 37% survival rate, and 100% specificity of DWI for poor outcome, 250 patients are needed to achieve a 0% false positive rate for poor outcome with a 95% confidence interval of 0 to 5%.

Current status:
Centers are invited to participate. Several investigators have expressed interest.

Financial Support: None

WHAT'S THE BIG IDEA? Poster 175
DECOMPRESSIVE CRANIOTOMY FOR HYPERTENSIVE CAPSULO-GANGLIONIC HEMORRHAGE
Salah Keyrouz
UAMS, Little Rock, AR, United States

Background:
Outcome of patients with ICH is dismal. The majority of patients succumb in the first 48 hours to the effect of the hemorrhage causing tissue shifts and herniation. Furthermore, many are comatose, ventilator-dependent because of alteration of consciousness brought upon by pressure on midline diencephalic structures. Both early hemostatic therapy and surgical evacuation failed to improve outcome

Hypothesis:
Simple yet large decompression with durotomy in patients with large, unilateral hypertensive capsulo-ganglionic ICH preceded by administration of rFVIIa for stabilization of clot and thereby prevention of hematoma growth upon decompression should result in less hematoma expansion (safe), reduce pressure on midline structure, and improve mentation and overall outcome

Study design:
Randomized, controlled but not blinded, feasibility and safety multi-center trial

Subjects:
Patients with spontaneous, hypertensive large (≥25 mL) ICH located in the putamen and internal capsule with evidence of mass effect on midline structure (≥5mm septum pellucidum shift), who are not moribund (decerebrate posturing, absent pupillary light and oculocephalic reflexes), and who present within 8 hours from onset of bleed are eligible to be enrolled. Patients should have no contraindication to receiving rFVIIa.

Setting:
Patients are screened upon arrival to the ER or NICU if transferred directly there

Intervention:
80mcg/Kg rFVIIa is given prior to patients undergoing a large, fronto-temporo-parietal decompressive craniotomy with durotomy without clot evacuation. Further care like blood pressure control and osmotic therapy will not be standardized.

Main outcome measure:
Hematoma expansion, improvement in midline shift by ≥50%

Secondary outcome measures:
Deterioration by ≥ 4 points on NIHSS within first 24 hours following decompression, good outcome at 3 months (mRS ≤3), ICU and hospital LOS, days on mechanical ventilation

Power analysis:
With a beta level of 0.9 and alpha 0.02, an assumed hematoma expansion rate of 26% in the non intervention group (FAST trial placebo group hematoma growth rate) and 15% in the intervention group, 152 patients are needed to be randomized

Current status:
Not initiated yet

Financial Support: None

WHAT'S THE BIG IDEA? Poster 176
HYPERTENSIVE HYPERVOLEMIC VASODILATORY (HHVD) THERAPY WITH INTRAVENOUS NICARDIPINE FOR THE TREATMENT OF CEREBRAL VASOSPASM

Benjamin Emanuel, Monica Sapo, David McArthur, Paul Vespa
University of California, Los Angeles, Los Angeles, CA, United States

Background:
Cerebral vasospasm (CV) after aneurysmal subarachnoid hemorrhage (aSAH) remains a significant cause of morbidity. Intravenous nicardipine has been previously studied clinically as a neuroprotectant, and shown to decrease the incidence of angiographic and symptomatic vasospasm in aSAH, and has the potential to avoid rescue intra-arterial rescue therapy and the resultant complications.

Hypothesis:
Hypertensive Hypervolemic Vasodilatory HHVD for CV will result in a reduction in duration CV, fewer delayed ischemic neurologic deficits (DINDS) and better functional outcome.

Study design:
Randomized placebo controlled trial of HHVD versus HH therapy.

Subjects:
Patients with aSAH, ages 18-80, without a history of coronary artery disease (CAD), ischemic cardiomyopathy, neurodegenerative disorder, or chronic kidney disease not on hemodialysis.

Setting:
Multicenter

Intervention:
HHVD therapy using norepinephrine and continuous nicardipine infusion at 2mg/hr initiated at the onset of cerebral vasospasm, for a duration of 7 days.

Main outcome measure:
1. Incidence of delayed ischemic neurologic deficits (DINDS) by computed tomography or MRI.
2. Modifed Rankin Outcome at 3 months.

Secondary outcome measures:
1. Duration of vasospasm
2. Incidence of myocardial infarction or troponin leak
3. Incidence of rescue intra-arterial therapy for vasospasm

Power analysis:
1. For comparing the incidence of rescue therapy is estimated at 17.
2. For comparing the incidence of troponin leak is estimated at 38.
3. For comparing the DINDS differences is estimated at 2,472.

Current status:
Preliminary single center data and protocol.

Financial Support: None

WHAT'S THE BIG IDEA? Poster 177
WEANING FROM MECHANICAL VENTILATION IN THE NEUROCRITICAL CARE UNIT
Gene Sung
University of Southern California, Los Angeles, CA, United States

Background:
There have been several Randomised Controlled Clinical Trials of weaning from mechanical ventilation which has shown quicker weaning and shorter ventilation time in abrupt discontinuation of mechanical support as opposed to gradual step-wise withdrawal. However, there have been no substantial trials of ventilatory weaning in acute brain injury patients or those with neuromuscular diseases.

Hypothesis:
The neurocritically ill patient on mechanical ventilation will require slower step-wise weaning from mechanical ventilation.

Study design:
Multicenter, randomized, non-blinded phase 2 trial feasibility and safety trial.

Subjects:
All patients in the neurocritical care unit expected to be on mechanical ventilation for more than 5 days. Routine scheduled post-operative patients and patients transferred from outside hospitals already on mechanical ventilation for more than 24 hours will be excluded.

Setting:
Neurocritical Care Unit.

Intervention:
Patients will be randomized to slow step-wise SIMV wean versus pressure support wean on CPAP.

Main outcome measure:
Length of time on mechanical ventialtion

Secondary outcome measures:
Pneumonia, urgent re-intubation, hypoxia

Power analysis:
From previous studies in the medical intensice care units, 400 patients, half with acute brain injury, half with neuromuscular diseases.

Current status:
Idea stage

Financial Support: None

WHAT'S THE BIG IDEA? Poster 178
DETECTION AND TREATMENT OF CEREBRAL VASOSPASM IN BACTERIAL MENINGITIS TO IMPROVE SURVIVAL
Valerie Coon, Jennifer Majersik, Elaine Skalabrin
University of Utah, Salt Lake City, UT, United States

Background:
Despite the use of appropriate antimicrobial therapies, the morbidity and mortality associated with bacterial meningitis remains high. Cerebrovascular complications from meningitis, including vasospasm, have been shown to contribute to this poor outcome. Several series have reported TCD velocity elevation correlates with clinical decline and occurs in up to 51% of patients with bacterial meningitis. To date no systematic large trial has been completed to detect or treat this complication.

Hypothesis:
1. Phase II: Clinically significant vasospasm in bacterial meningitis results in higher mortality compared with those with normal TCD velocities.
2. Phase III Goal directed therapy: Triple H, intra-arterial verapamil and angioplasty will increase survival in patients with bacterial meningitis at high risk for vasospasm.

Study design:
1. Multicenter, Prospective, Nonrandomized PhaseII feasibility study assessing mortality and defining the characteristics of the target (high risk for vasospasm) population.
2. Multicenter Prospective Randomized PhaseIII efficacy.

Subjects:
Patients admitted to the ICU with the diagnosis of bacterial meningitis.

Setting:
ICU

Intervention:
PhaseII: Bacterial meningitis enrolled within 24 hours of diagnosis. Subjects receive baseline CTA of head/neck, daily TCDs, angiography when mean velocities >180, daily NIHSS, mortality rate at one month, and MRS at 3 months. Significant vasospasm will be defined as angiographic vasospasm with corresponding increased NIHSS of at least 2 points.

PhaseIII: Target population from Phase II. All subjects will undergo testing and data collection as outlined in phase II. Subjects randomized to aggressive vasospasm treatment vs. standard-care. In the treatment group, vasospasm will be treated with goal-directed therapy.

Main outcome measure:
One month mortality.

Secondary outcome measures:
Independent predictors of the presence of vasospasm.
Rankin score at 3 months.

Power analysis:
Phase II and III: Inference of portions (alpha0.05, beta0.8, delta0.15, n=220subjects)

Current status:
Big idea.

Financial Support: None

WHAT'S THE BIG IDEA? Poster 179
INDOMETHACIN IN THE TREATMENT OF INTRACRANIAL HYPERTENSION
Walter Videtta, Marcelo Costilla, Gustavo Domeniconi
[1]Hospital de Emergencias y Trauma "Federico Abete", Pablo Nogues - Pcia Buenos Aires, Argentina, [2]Sanatorio de la Trinidad, San Isidro - Pcia de Buenos Aires, Argentina, [3]Hospital "Alejandro Posadas", Haedo - Pcia de Buenos Aires, Argentina

Background:
Intracranial hypertension (IH) is the most powerful predictor of poor outcome in severe TBI. Indomethacyn (IM) is a COX inhibitor with a potent vasoconstrictive effect in cerebral arterioles that has been used in TBI, AVM's and intracranial neoplasm. There is a little of evidence that supports its utility in the treatment of IH with special emphasis in type A waves in patients with an impaired cerebral vasoreactivity, improving both cerebral perfusion and response to other second-tier therapeutic tools. However, large, prospective, randomised and controlled studies have not yet been performed to confirm its benefit in patients with TBI

Hypothesis:
The IM could be effective to treat refractory IH in severe TBI with impaired cerebral autoregulation and poor response to other therapeutic strategies. IM can improve indices of cerebral haemodinamics and cerebral oxygenation decreasing neuronal ischemic damage.
This therapeutic approach can reduce TIL to control intracranial pressure, also the length-of-stay in ICU can be reduced too. IM can improve long term functional outcome in severe TBI.

Study design:
Multicenter, randomized controlled trial to evaluate the efficacy and safety of IM in patients with severe TBI that presents intracranial hypertension which have reached a therapeutic intensity level that includes second-tier therapies (ie,deccompresive craniectomy, controlled hypothermia, etc)

Subjects:
All patients (older than 18 years old) that presents with Glasgow coma Scale 8 or <, with ICP above 20 torr and with any evidence of hyperaemia (TCD, SJVO2, AVDO2 etc) or increased CBV (CT perfusion, etc) despite standard therapy (includes mechanical ventilation, evacuation of intracranial mass, profound sedation, osmotherapy, etc).

Setting:
Intensive care unit or neurocritical care unit or neurotrauma unit at a hospital that has multimodal monitoring modality (ICP, CPP, ETCO2 or PCO2, SJVO2 or PTiO2, TCD etc) and Neuroimaging with evaluation of CBV (CT perfusion, etc)

Intervention:
Indomethacyn 0.6 – 0.8 mg/kg at loading dose, followed by continous infusion 0.3 – 0.5 mg/kg/hr or placebo in patients who develop high ICP despite standard therapy for ICP control.

Main outcome measure:
- ICP control (reduction of ICP below 20 torr or 15 torr in DC), normalization of cerebral oxygenation (AVDO2, SJVO2 or PTiO2).
- Improvement of cerebral perfusion measured by cuantitative or cualitative methods
- Neurological outcome (eGOS) at discharge, 30, 90, 180 and 365 days.

Secondary outcome measures:
- LOS in ICU
- Overall mortality at month 1, 6 and 12
- QoL at month 1, 6 and 12
- Evidence of long-term ischemic damage

Power analysis:
We need a sample of at least 100 patients to find statistically significative difference between intervention and placebo group.

Current status:
Not started.
Financial supports is needed.

Financial Support: None

WHAT'S THE BIG IDEA? Poster 180
AGGRESSIVE HYPERGLYCEMIA MANAGEMENT AFTER ACUTE BRAIN INJURY
Julius Gene Latorre, Yahia Lodi, Tarakad Ramachandran
SUNY Upstate Medical University, Syracuse, NY, United States

Background:
Hyperglycemia is very common in acute brain injury (ABI) from ischemic stroke, hemorrhage or trauma and is associated with poor outcome. Tight glucose control is effective in improving outcome in medical/surgical ICU but its role in ABI is uncertain. Studies investigating brain metabolism using microdialysis showed increased brain metabolic crisis with tight glycemic control. Currently the optimal glucose control for patients with acute brain injury is unclear.

Hypothesis:
Aggressive hyperglycemia management will result in improved outcome in ABI compared with standard glucose management.

Study design:
Multicenter, randomized, single-blinded phase II feasibility and safety trial.

Subjects:
Inclusion criteria: Patients admitted to ICU for management of ABI with high likelihood of requiring at least 72 hours of intensive care.

Exclusion criteria include:
- Absence of health care proxy to sign consent.
- Patient with do not resuscitate and do not intubate orders on admission
- History of allergy or known contraindication to insulin
- Moderate to severe baseline disability (pre-ABI modified Rankin Scale 3 or greater)
- Severe terminal concurrent medical illness with expected survival of less than three months.

Setting:
Neurocritical care unit.

Intervention:
Treatment arm will receive continuous insulin infusion targeting blood sugar level 100-140mg/dL.
Control group will receive subcutaneous insulin injection and/or insulin infusion targeting blood sugar level 180-200 mg/dL.

Main outcome measure:
Modified rankin scale at 6 months

Secondary outcome measures:
Rate of medical complications, including infection, new neurologic abnormality, hypoglycaemia and in-hospital mortality.

Length of ICU, hospital stay.

Power analysis:
Based on our retrospective study with expected good clinical outcome of 50% and 25% relative difference in outcome between groups, the study will need 150 patients in each arm with 80% power and 5% two-sided alpha level.

Current status:
Protocol complete. For submission for funding.

Financial Support: None

WHAT'S THE BIG IDEA? Poster 181
AFTERSHOC (ANTI-FIBRINOLYTIC THERAPY IN THE EMERGENCY ROOM FOR SUBARACHNOID HEMORRHAGE OUTCOME)

Brad Zacharia[1], Andrew Ducruet[1], Zachary Hickman[1], Paul Gigante[1], Christopher Kellner[1], Raqeeb Haque[1], Bartosz Grobelny[1], Jan Claassen[2], Kiwon Lee[2], Neeraj Badjatia[2], Stephan Mayer[2], E. Sander Connolly, Jr[1]
[1]Columbia University Department of Neurological Surgery, New York, NY, United States, [2]Columbia University Department of Neurology, New York, NY, United States

Background:
Rebleeding on the first day following aSAH is as high as 9–17%, and approximately half of these occur within 6hrs of onset. Unanticipated delays in aSAH diagnosis and result in failure to secure aneurysms during the period of maximal rebleeding. A novel approach of acute antifibrinolysis (<72h duration) has demonstrated safety, however, there have been no trials powered to demonstrate a difference in long-term outcome. Therefore, a clinical trial evaluating the impact of ε-aminocaproic acid (EACA) on outcome following aSAH is warranted.

Hypothesis:
Acute treatment with intravenous EACA will improve twelve-month outcome in patients with aSAH.

Study design:
Neurological Emergency Treatment Trials (NETT)-based multi-center, randomized, double-blind, placebo-controlled phase-III trial.

Subjects:
All adult aSAH patients presenting to NETT facilities will be screened for enrollment. Patients must receive study drug within 12h of aSAH onset. Those with aneurysm-negative SAH, anticipated treatment within 4h, or recent thromboembolic disorder will be excluded.

Setting:
Subjects will be enrolled and treatment initiated in the emergency department and continued during transfer and the referral-center intensive care unit.

Intervention:
Patients will receive an intravenous placebo or EACA. A 4g loading dose, will be followed by infusion of 1g/hr, to a maximum 72h.

Main outcome measure:
Favorable 12-month modified Rankin Score(1-3)

Secondary outcome measures:
The Barthel and Lawton scales(disability scales), SIP(quality-of-life scale), and a psychometric battery(cognitive/intellectual domains) will be assessed as secondary outcomes. Known sequelae of aSAH and anti-fibrinolytic therapy including rebleeding, vasospasm, hydrocephalus, and thrombotic complications will be tracked.

Power analysis:
Based on a χ^2 analysis with $\alpha=0.05$ and a power of 80%, 900 subjects will be randomized. This calculation is based on conservative estimates from past studies that demonstrate 9% increase in favorable outcome for patients receiving acute EACA.

Current status:
Based on a χ^2 analysis with $\alpha=0.05$ and a power of 80%, 900 subjects will be randomized. This calculation is based on conservative estimates from past studies that demonstrate 9% increase in favorable outcome for patients receiving acute EACA.

Financial Support: None

WHAT'S THE BIG IDEA? Poster 182
LONG-TERM RECOVERY OF DEVASTATING NEUROLOGIC DIAGNOSES
Eli Feen
Saint Louis University School of Medicine Department of Neurology & Psychiatry, St. Louis Missouri, United States

Background:
The outcome of devastating neurologic disease like massive ischemic stroke, intracranial hemorrhage, status epilepticus, and subarachnoid hemorrhage is presumed to be poor. Mortality in studies may be influenced by premature withdrawal of care, and not by natural history or chronic complications.

Hypothesis:
If these patients are given maximal supportive care chronically, their outcome will be better than expected based upon commonly accepted morbidity/mortality.

Study design:
Randomized, controlled non-blinded clinical trial.

Subjects:
Patients with devastating neurologic conditions as listed above who require mechanical ventilation (MV) and feeding tube placement (TF).

Setting:
Neurocritical care unit of major tertiary care centers as part of a multi-center trial.

Intervention:
Families are offered usual standard of care—either withdrawal of care or full supportive care. For those not certain about which course to take, enrolment is offered. The trial would necessarily require initial full supportive care such as tracheostomy and feeding tube placement. The patients are randomized to one of two treatment regimens: 1) aggressive, long-term supportive care involving treatment of intercurrent medical complications and full resuscitation; 2) basic supportive care including MV and TF but not involving these aggressive measures.

Main outcome measure:
Modified Rankin scale at one, two, and five years.

Secondary outcome measures:
Continued need for MV/TF, Barthel index, correlation with initial hospital care in a specialized neurocritical care unit.

Power analysis:
Assuming a 50% event rate (severe disability or mortality) in the control group and expecting a 20% relative risk reduction (about 10% absolute risk reduction), the estimated sample size with an 80% power and an alpha of 0.05 is 400 patients total (200 in each arm).

Current status:
Proposal status. How to structure randomization in concert with ethical principles and which supportive measures can be ethically restricted must be determined.

Financial Support: None

WHAT'S THE BIG IDEA? Poster 183
INCREMENTAL MULTIMODAL NEUROPROTECTIVE THERAPIES—A PROPOSED SOLUTION TO NEUROPROTECTIVE NIHILISM IN CLINICAL TRIALS

W Andrew Kofke
University of Pennsylvania, Philadelphia, PA, United States

Background:
Regarding clinical trials in neuroprotection, Donnan [1] suggests: "We have reached a stage at which research in this area should stop altogether or radical new approaches adopted." This abstract suggests a radical new approach.

An injured biological system is characterized by severity of a pathophysiologically complex injury, S. A regression equation with a large number of disparate and interacting nonlinear heterogenous pathophysiologic factors, F*i*, and weighting factors, W*i*, with a post injury time component, *t,* describes S: $S=\sum W_{it}F^{e}_{i}$.

Additionally, there are also associated nonlinear hospital system factors, H*i*, probably accounting for positive preclinical and single center studies, followed by multicenter failure. The severity regression equation can now be described as $S=\sum W_{it}F^{e}_{i} + \sum W_{it}H^{e}_{i}$

This leads to the notion that the current widely accepted methods of evaluating single facet therapy to attenuate such multifaceted complex problems is generally a fruitless waste of public resources which has produced innovation paralysis.

Preclinical studies have demonstrated the potential for dramatic breakthrough level neuroprotection with a multifaceted approach but a rational systematic method for introduction of multifaceted therapeutic bundles is needed.

Hypothesis:
Multifaceted neuroprotective bundles can be used to demonstrate robust neuroprotection. Can the Plan-Do-Study-Act (PDSA) QI method be used to incrementally add and evaluate individual facets of neuroprotective therapeutic bundles?

Study design:
Single Center PDSA therapeutic bundle development followed by multicenter randomized trial of a therapeutic bundle.

Subjects:
Patients with acute TBI or brain ischemia syndromes

Setting:
ED, OR, and ICU

Intervention:
Multi mechanism multifaceted therapy incrementally and sequentially implemented during active post insult secondary pathophysiologic processes.

Main outcome measure:
Surrogates for functional outcome with sequentially added facets in a therapeutic bundle

Secondary outcome measures:
Ongoing evaluation of functional neurologic outcome.

Power analysis:
None yet

Current status:
Idea phase in effort to prevent continued negative single facet studies.

Financial Support: None

WHAT'S THE BIG IDEA? Poster 184
CONTINUOUS VENO-VENOUS HEMOFILTRATION FOR THE MANAGEMENT OF REFRACTORY INTRACRANIAL HYPERTENSION

Jeff Fletcher MD, Karen Bergman RN, Glenn Carlson MSN, CCRN
Bronson Methodist Hospital / Michigan State Unviversity, Kalamazoo, MI, United States

Background: Refractory intracranial hypertension (RIH) is associated with death or poor neurological outcome in 80-85% of patients and clinical equipoise often exists among management [1]. For patients with cerebral edema or intracranial hypertension who require renal replacement therapy, continuous (CRRT) modes are preferred due to limited data showing improved intracranial stability over traditional intermittent hemodialysis (IHD). This is attributed to better cardiovascular stability, less rapid fluid shifts, bicarbonate and osmolality changes as well as more biocompatible, and highly permeable membranes [2]. Anecdotal reports have suggested improvement in intracranial pressure (ICP) [3 and verbal] during CRRT and we have observed this in 3 patients (Table 1, Figure 1-3).

Patient	Age / sex	Diagnosis / (GCS)	Indication	ICP management	Mode / CBF / TEF rate / Anticoagulant / Vascular access / filter changes during therapy	Discharge / LOS
1	28 / M	Severe TBI (6) Polytrauma MOF ARDS	RIH / Hypervolemia	EVD, sedation, Osmotic therapy, hypothermia (33deg), 23.4% saline, pharmacologic coma	CVVH / 80-180cc/min /20-25cc/kg/hr / Heparin / Femoral vascular catheter / 1	LTCF / 39
2	30 / M	Severe TBI (3) Polytrauma MSOF ARDS	Hypervolemia (RIH present)	Sedation, osmotic therapy, paralytics, mild hypothemia	SCUF / 150cc.min / 150-400cc/hour / Heparin / Femoral vascular catheter / 0	LTCF / 34
3	51 / F	SAH - (treated with coil embolization)	RIH	EVD, sedation, Osmotic therapy, hypothermia (33deg), 23.4% saline, pentobarbital coma	CVVH / 150cc/min / 20cc/kg/hr / Heparin / Femoral vascular catheter / 0	Death / 4

GCS = Glasgow Coma Scale Score; RIH = Refractory intracranial hypertension defined as failure of first tier therapy as defined but the brain trauma foundation; BFR = blood flow rate; TEF = total effluent flow rate; CVVH = hemofiltration; SCUF = slow continuous ultrafiltration; CVVHDF = Hemodialfiltration; IHD = intermittent hemodialysis; ICP = intracranial pressure management ***Prior to*** CRRT; EVD = external ventricular drain; MSOF = multi-system organ failure; acute respiratory distress syndrome; LOS = length of stay; LTCF= long term care facility; TBI = traumatic brain in jury; SAH = subarachnoid hemorrhage

Patient	12 hours prior (mmHg) / P value	4 hours after (mmHg)	12 hours after (mmHg)	Change 1 hour (mmHg)	Change Volume 1 hr (L)	Change volume 12 hr (L)	% FB 12 hr
1	35.5	24.3	21.3	-9	-0.4	- 4.1	-16%
2	23.4	11.5	13.8	-13	-0.16	-2.56	-6%
3	36.7	32.5	29.8	-17	-0.02	-0.62	-41%
All	31.9	22.8	21.6	-13	-0.19	-2.43	-8%

L = liters

Hypothesis: CRRT reduces ICP in patients with RIH

Study design: Prospective observational cohort study to determine safety and efficacy

Subjects: RIH without other indications for CRRT. RIH definition: ICP $\geq$ 25mmHg for 30 minutes (or ICP $\geq$ 15mmHg for 15 minutes in patients with large craniectomy) despite first tier therapy

Setting: Intensive care unit

Intervention: Continuous veno-venous hemofiltration via femoral access with total effluent rate of 35ml kg^{-1} h^{-1}, blood flow rate between 100-300ml/min, high permeability glycerine free polyethersulfone membrane, filter change every 24 hours, pre-filter replacement fluids: prismasate BGK 4/0/1.2 (osmo 296) and sodium-citrate anticoagulant. Fluid management per attending physician. Minimum duration of 36 hours with termination after 12-24 hours of ICP control or at 96 hours.

Main outcome measure: Change in ICP at 1 hour after initiation

Secondary outcome measures: Change in ICP at hours 4 and every 12 hours thereafter; Neurological outcome (Glasgow Outcome Scale score dichotomized unfavorable (1,2,3) / favorable (4,5)); cytokine removal and complications.

Power analysis: A sample of size 24 will be obtained to attain a power of 0.8 at 5% level of significance

Current status: Idea phase

References:

1) Cooper DJ, Rosenfeld JV, Murray L, Wolfe R, Ponsford J, Davies A, D'Urso P, Pellegrino V, Malham G, Kossmann T. Early decompressive craniectomy for patients with severe traumatic brain injury and refractory intracranial hypertension--a pilot randomized trial. J Crit Care. 2008 Sep;23(3):387-93.
2) Davenport A. Renal replacement therapy for the patient with acute traumatic brain injury and severe acute kidney injury. Contrib Nephrol 2007;156:333-9.
3) Fletcher JJ, Bergman K, Feucht EC, Blostein P. Continuous Renal Replacement Therapy for Refractory Intracranial Hypertension. Neurocrit Care 2009 Mar 7. (online first)

Financial Support: None

WHAT'S THE BIG IDEA? Poster 185

PREDICTION OF COMA OUTCOME BY MRI: THE NEED FOR A NORTH AMERICAN ARM TO AN EUROPEAN STUDY

Louis Puybasset[1], Damien Galanaud[2], Stephan Mayer[3]
[1]NeuroICU, Pitie-Salpetriere Hospital, Paris, France, [2]Neuroradiology, Pitie-Salpetriere Hospital, Paris, France, [3]Columbia University, New York, United States

Background:
Given its multiple ethical, medical and economical impacts, efforts should be made to accurately predict coma outcome at one year.

Hypothesis:
Coma outcome can be accurately predicted by combining clinical and quantitative MRI data : DTI (fractional anisotropy) and MRS (NAA/Cr) in specific brain areas*
** Tollard E, Crit Care Med: 2009,37:1448-55*

Study design:
Multicentric, prospective cohort with consecutive inclusions.

Subjects:
Patients with persistent disorders of consciousness, defined as the absence of response to simple orders, ≥ 7 days after the event unexplained by sedation. Signed informed consent. Inclusion of TBI and non TBI comatose patients (ischemia, SAH, hematoma and cerebral anoxia).

Setting:
NeuroICU. MRI under mechanical ventilation. Inclusions during 3 years. Follow up at 6 months and one year by phone interview.

Intervention:
Electronic CRF. Multimodal MRI with MRS (pons and CSI) and DTI under mechanical ventilation.
10 controls per center.

Main outcome measure:
Predictability of dichotomized GOS at year using a composite index combining clinical data and quantified indicators from MRS (NAA/Cr in specific brain regions) and DTI.

Secondary outcome measures:
Design of specific algorithms according to the etiology of coma.

Power analysis:
With 220 subjects, 79% of power to detect a variable with an OR by standard deviation of 1.55 (= 5 %, bilateral test, proportion of patients with poor outcome = 33%, NQuery Advisor® 4.0). 10% of lost to follow-up within 1 year and 5% of drop-out. Total TBI to be included = 250. Same reasoning for non TBI patients.

Current status:
Founded in France 400 000 €. 10 European actively including patients following a similar protocol. 281 patients already included (TBI 154 patients, anoxia 40, intracerebral hematoma 27, SAH 51, arterial ischemia 9). Mortality rate at one year 26 %.

Financial Support: None