News Letter

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INDIAN SOCIETY OF NEUROANAESTHESIOLOGY AND CRITICAL CARE

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Presidential Address

Marching forward with ISNACC…….

Dear Friends,

A decade of existence of Indian Society of Neuroanaesthesiology and Critical Care (ISNACC) has made its impact. Founded at the initiative and under the leadership of Dr. H.H. Dash, it has created awareness among the anesthesiologists of the need for specialised training to manage neurological patients. This is evidenced by the number of anaesthesiologists seeking short term and long term training courses in advanced centers and the inclination of many of the young anaesthesiologists to commit themselves to full time neuroanaesthesia practice. The decade also saw establishment of training programmes both formal and informal in many centers. At least two institutions at the moment are offering D.M. Course in Neuroanaesthesia and many other centers are offering postdoctoral fellowship programs of one year. The ultimate beneficiaries of all these programs are of course, our patients. Most of the specialty hospitals are looking for anaesthesiologists with experience in neuroanaesthesia.

What is the role of the society now? The top priority, in my opinion, is to strive towards uniformly good standards of clinical care all over the country. The first step to achieve this is to understand the current scenario and then plan out a course of action. A national level collaborative effort is necessary to understand the nature of clinical care at different levels. Then, the society may sit to formulate guidelines that are appropriate to our socioeconomic background. It may not be difficult to achieve a reasonable standard.

Research in neuroanaesthesia also has taken a turn for the better. The number of papers in the international journals from India has increased, though they still constitute a minority. But the direction is definitely positive. At the same time, it is important to note that a lot needs to be done on this front. With the vast amount of clinical material that we have, creating large databases and analysing the outcomes should not be a major problem; it requires a little commitment from all the members and that is the least that any member can do to lay the foundation for a glorious future of Neuroanaesthesiology in India. The ISNACC website is almost getting completed. I expect this to play a major role in exchange of ideas and information. Individual institutions with good infrastructure must also enter the arena of experimental investigation and compete with our colleagues in the rest of the world.

The annual conferences of ISNACC are becoming increasingly popular among the anesthesiologists. With good scientific content, these meetings have become a source of continuing education for those seeking latest knowledge in neuroanaesthesia. I invite all of you to participate in large numbers in the forthcoming meeting at Hyderabad on 8-10th Feb 2007.

Long live ISNACC !!!

(Dr. G.S. Umamaheswara Rao)

President, ISNACC

Professor of Neuroanaesthesia
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From The Literature
Efficacy and Safety of Epoetin Alfa in Critically Ill patients

Howard L. Corwin, M.D., Andrew Gettinger, M.D., Timothy C. Fabian, M.D., et al; for the EPO Critical Care Trials Group.

NEJM 357 : 965 - 976, Sept. 6, 2007

Background: Anemia, which is common in the critically ill, is often treated with red-cell transfusions, which are associated with poor clinical outcomes. We hypothesized that therapy with recombinant human erythropoietin (epoetin alfa) might reduce the need for red-cell transfusions.

Methods: In this prospective, randomized, placebo-controlled trial, we enroll 1460 medical, surgical, or trauma patients between 48 and 96 hours after admission to the intensive care unit. Epoetin alfa (40,000 U) or placebo was administered weekly, for a maximum of 3 weeks; patients were followed for 140 days. The primary end point was the percentage of patients who received a red-cell transfusion. Secondary end points were the number of red-cell units transfused, mortality, and the change in hemoglobin concentration from baseline.

Results: As compared with the use of placebo, epoetin alfa therapy did not result in a decrease in either the number of patients who received a red-cell transfusion (relative risk for the epoetin alfa group vs. the placebo group, 0.95; 95% confidence interval [CI], 0.85 to 1.06) or the mean (+SD) number of red-cell units transfused)4.5+4.6 units in the epoetin alfa group and 4.3+4.8 units in the placebo group, P=0.42). However, the hemoglobin concentration at day 29 increased more in the epoetin alfa group than in the placebo group (1.6+2.0 g per deciliter vs. 1.2+1.8 g per deciliter, P<0.001). Mortality tended to be lower at day 29 among patients receiving epoetin alfa (adjusted hazard ratio, 0.79; 95% CI, 0.56 to 1.10); this effect was also seen in pre-specified analyses in those with a diagnosis of trauma (adjusted hazard ratio, 0.37; 95% CI, 0.19 to 0.72). A similar pattern was seen at day 140 (adjusted hazard ratio, 0.86; 95% CI, 0.65 to 1.13), particularly in those with trauma (adjusted hazard ratio, 0.40; 95% CI, 0.23 to 0.69). As compared with placebo, epoetin alfa was associated with a significant increase in the incidence of thrombotic events (hazard ratio, 1.41; 95% CI, 1.06 to 1.86).

Conclusions: The use of epoetin alfa does not reduce the incidence of red-cell transfusion among critically ill patients, but it may reduce mortality in patients with trauma. Treatment with epoetin alfa is associated with an increase in the incidence of thrombotic events.

NXY-059 for the treatment of Acute Ischemic Stroke

Ashfaq Shuaib, M.D., Kennedy R. Lees, M.D., Patrick Lyden, M.D., et al; for the SAINT II Trial Investigators

NEJM 357 : 562 - 571, Aug. 9, 2007

Background: The free-radical-trapping agent NXY-059 showed promise as a neuroprotectant in the Stroke-Acute Ischemic NXY Treatment I (SAINT I) trial, reducing disability when given to patients who had acute ischemic stroke. We sought confirmation of efficacy in a second, larger trial.

Methods: We enrolled 3306 patients with acute ischemic stroke in a randomized, double-blind trial to receive a 72-hour infusion of intravenous NXY-059 or placebo within 6 hours after the onset of stroke symptoms. Our primary end point was the distribution of disability scores on the modified Rankin scale at 90 days. We examined scores on neurologic and activities-of-daily-living scales as secondary end points. We also tested the hypothesis that NXY-059 would reduce alteplase-related intracranial hemorrhage.

Results: The efficacy analysis was based on 3195 patients. Prognostic factors were well balanced between the treatment groups. Mortality was equal in the two groups, and adverse-event rates were similar. The distribution of scores on the modified Rankin scale did not differ between the group treated with NXY-059 (1588 patients) and the placebo group (1607 patients; P=0.33 by the Cochran-Mantel-Haenszel test; odds ratio for limiting disability, 0.94; 95% confidence interval [CI], 0.83 to 1.06). Analysis of categorized scores on the modified Rankin scale confirmed the lack of benefit: the odds ratio for trichotomization into modified Rankin scale scores of 0 to 1 versus 2 to 3 versus 4 to 6 was

0.92 (95% CI, 0.80 to 1.06). There was no evidence of efficacy for any of the secondary end points. Among patients treated with alteplase, there was no difference between the NXY-059 group and the placebo group in the frequency of symptomatic or asymptomatic hemorrhage.

Conclusions: NXY-059 is ineffective for the treatment of acute ischemic stroke within 6 hours after the onset of symptoms.

Saline or Albumin for Fluid Resuscitation in Patients with Traumatic Brain Injury

The SAFE Study Investigators

NEJM 357 : 874 - 884, Aug. 30, 2007

Background: The Saline versus Albumin Fluid Evaluation study suggested that patients with traumatic brain injury resuscitated with albumin had a higher mortality rate than those resuscitated with saline. We conducted a post hoc follow-up study of patients with traumatic brain injury who were enrolled in the study.

Methods: For patients with traumatic brain injury (i.e., a history of trauma, evidence of head trauma on a computed tomographic [CT] scan, and a score of ≤13 on the Glasgow Coma Scale [GCS], we recorded baseline characteristics from case-report forms, clinical records, and CT scans and determined vital status and functional neurologic outcomes 24 months after randomization.

Results: We followed 460 patients, of whom 231 (50.2%) received albumin and 229 (49.8%) received saline. The subgroup of patients with GCS scores of 3 to 8 were classified as having severe brain injury (160 [69.3%] in the albumin group and 158 [69.0%] in the saline group). Demographic characteristics and indexes of severity of brain injury were similar at baseline. At 24 months, 71 of 214 patients in the albumin group (33.2%) had died, as compared with 42 of 206 in the saline group (20.4%) (relative risk, 1.63; 95% confidence interval (CI), 1.17 to 2.26; P=0.003). Among patients with severe brain injury, 61 of 146 patients in the albumin group (41.8%) died, as compared with 32 of 144 in the saline group (22.2%) (relative risk, 1.88; 95% CI, 1.31 to 2.70; P<0.001); among patients with GCS scores of 9 to 12, death occurred in 8 of 50 patients in the albumin group (16.0%) and 8 of 37 in the saline group (21.6%) (relative risk, 0.74; 95% CI, 0.31 to 1.79; P=0.50).

Conclusions: In this post hoc study of critically ill patients with traumatic brain injury, fluid resuscitation with albumin was associated with higher mortality rates than was resuscitation with saline.

Effect of nitric oxide on oxygenation and mortality in acute lung injury: systematic review and meta-analysis

Neill K.J. Adhikari, Karen E A Burns, Jan O Friedrich, et al;

BMJ 334 : 779 - 782, April 14, 2007

Objective: To review the literature on the use of inhaled nitric oxide to treat acute lung injury/acute respiratory distress syndrome (ALI/ARDS) and to summarise the effects of nitric oxide, compared with placebo or usual care without nitric oxide, in adults and children with ALI or ARDS.

Design: Systematic review and meta-analysis.

Data sources: Medline, CINAHL, Embase, and CENTRAL (to October 2006), proceedings from four conferences, and additional information from authors of 10 trials.

Review methods: Two reviewers independently selected parallel group randomized controlled trials comparing nitric oxide with control and extracted data related to study methods, clinical and physiological outcomes, and adverse events.

Main outcome measures: mortality, duration of ventilation, oxygenation, pulmonary arterial pressure, adverse events.

Results: 12 trials randomly assigning 1237 patients met inclusion criteria. Overall methodological quality was good. Using random effects models, we found no significant effect of nitric oxide on hospital mortality (risk ratio 1.10, 95% confidence interval 0.94 to 1.30), duration of ventilation, or ventilator-free days. On day one of treatment, nitric oxide increased the ratio of partial pressure of oxygen to fraction of inspired oxygen (PaO_2/FiO_2 ration) (13%, 4% to 23%) and decreased the oxygenation index (14%, 2% to 25%). Some evidence suggested that improvements in oxygenation persisted until day four. There was no effect on mean pulmonary arterial pressure. Patients receiving nitric oxide had an increased risk of developing renal dysfunction (1.50, 1.11 to 2.02).

Conclusions: Nitric oxide is associated with limited improvement in oxygenation in patients with ALI or ARDS but confers no mortality benefit and may cause harm. We do not recommend its routine use in these severely ill patients.

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ACUTE INTRAOPERATIVE BRAIN BULGE

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The widespread use of microsurgical technique in neurosurgery has been made possible through advanced anaesthetic technique and better intracranial pressure control.

"TIGHT BRAIN" or 'TENSE BRAIN' is the most commonly uttered word in the operation theater by a neurosurgeon once the bone flap is raised. This is also known as "Acute Brain Bulge". Management of this complication is a matter of great concern for the anaesthetist. Unless it is relieved, the neurosurgeon cannot operate. If the brain remains "Tense", there is every likelihood of retractor anaemia or other neurosurgical complications. Prior to management of this we must have little knowledge of why this occurs and how best it can be managed?

Pathophysiology:

Increase in ICP occurs whenever the compensation fails which is known as Monro Kellie Doctrine. The cranial vault is a rigid structure with fixed total volume consisting of Brain(80%), Blood(12%) and CSF(8%). An increase in volume of any one compartment must be offset by an equivalent decrease in another to prevent a rise in ICP.

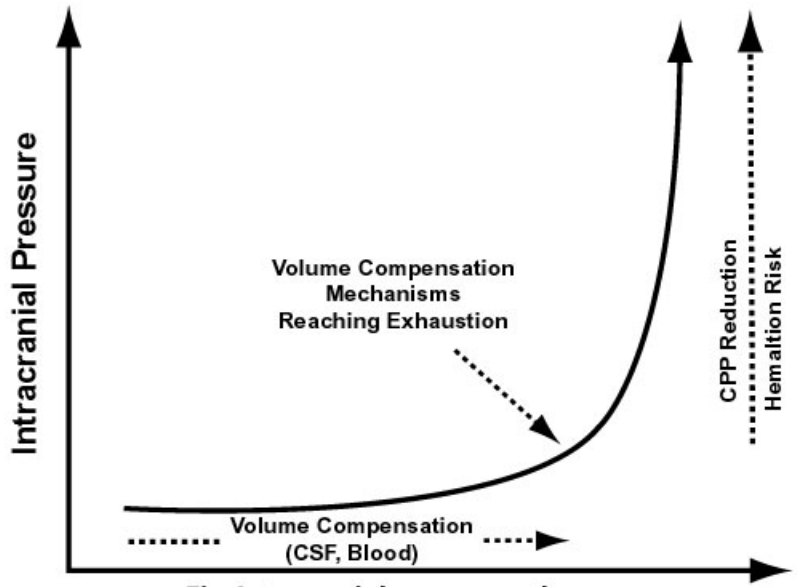


Fig: Intracranial pressure-volume curve

The main goal of neuroanaesthesia is to avoid intracranial compartment volume increase especially cerebral blood volume.

Pathogenesis of intraoperative brain bulge:

The mechanism of development of intraoperative brain swelling depends on the disease condition.

Brain bulge in head injured patients:

During the evacuation of an acute traumatic subdural haematoma, development of contralateral epidural haematoma and consequent brain bulging has become a more frequent event. It has been called a delayed haematoma, an alternative haematoma, or, in conjunction with the initial haematoma, a bilateral haematoma. It manifests with tense dura mater during craniotomy or if the dura has been opened, severe brain protrusion through the craniotomy would occur. It is defined as a haematoma, not present in the initial CT scan, that develops on the contralateral side during craniotomy for an acute extra axial haematoma. This brain displacement doesn't represent the cerebral oedema or hyperemia often associated with traumatic brain injury. Rather it is a displacement of the mass effect in response to the mass effect of the developing contralateral epidural haematoma. This shift is exaggerated because the normal resistance offered by the bone and dura mater are not present intraoperatively after the brain has been exposed. The clinical characteristic of the cases of intraoperative contralateral epidural haematoma form a distinct syndrome. The degree of head injury is usually severe. An extra axial haematoma is present on the intial CT scan, it can be epidural, subdural or mixed epidural and subdural. The patient often have a clinical or radiologic evidence of a skull fracture at the side of contralateral haematoma. Outcome is generally poor. A mechanism that has been hypothesized for the

development of intraoperative contralateral haematoma is simply a loss of the temponade effect of the ipsilateral haematoma after it has been evacuated by craniotomy. With relief of the temponade, the previously injured vessels on the contralateral side bleed with rapid formation of a large haematoma.

Brain bulge during tumour surgery:

There are fundamental differences in the pathological mechanism, neuroradiological findings and outcome between intraoperative brain bulge occurring in post traumatic and elective neurosurgical patients. Brain bulge during craniotomy for intracranial tumour is usually because of the intracranial hypertension by the tumour mass itself. Bleeding inside the tumour cavity can also give rise to severe brain bulging. Subarachnoid and intraventricular haemorrhage were found on post operative CT scan of patients who had intraoperative brain protrusion during surgery of intracranial mass lesion[1]. The proposed mechanism of brain bulge is the combination of actue intracranial hypertension caused by subarachnoid and intraventricular haemorrhage and consequent hyperemia 1. Brain swelling may be because of the sustained brain retraction which is known to result in disturbance of brain perfusion with concomitant electrical dysfunction and loss of autoregulation[2]. Massive intraoperative brain swelling is more common with large vascular tumours which may be related to long operative retraction. Patients with glioblastoma mutiforme and metastasis are more prone to develop intraoperative brain swelling[3]. Vascular tumours like angioblastic meningioma or hemangiopericytoma are also more prone for massive bleeding and intraoperative brain bulge. Whenever such massive bleeding is anticipated preoperative embolization of the tumour vessel should be considred.

Brain bulge during aneurysm clipping:

In patients undergoing craniotomy to clip a cerebral aneurysm following subarachnoiod haemorrhag acute brain swelling may follow rupture of the aneurysm1. Acute intracranial hypertension caused by subarachnoid and intraventricular haemorrhage together with hyperemia has been implicated as the proposed mechanism of brain herniation in these patients[3]. Intraoperative measurement of cerebral blood flow using intravenous ^{133}xenon has demonstrated that cerebral blood flow increases by 46% without increase of mean blood pressure during rupture of aneurysm[4]. It is postulated that excitation of the medullary and pontine nuclei, perhaps due to brainstem circulatory disturbances caused by subarachnoid and intraventricular haemorrhage, results in neurogenic vasodilatation and hyperemia.

Normal perfusion pressure breakthrough syndrome:

The highflow low resistance shunt that is associated with arteriovenous malformations can lead to chronic hypoperfusion of the surrounding brain tissue. The abrupt removal of the shunt from the circulation, by resection of the arteriovenous malformation induces cerebral hyperemia, oedema and haemorrhage into the surrounding brain tissue leading to malignant brain swelling[5]. Infants with vein of Galen malformations are particularly at high risk for these complications.

Awake craniotomy:

During awake craniotomy respiratory obstruction or hypoventilation from excessive sedation can give rise to carbon dioxide retention and subsequent brain swelling. Nausea and vomiting occurs frequently during stripping of the dura or temporal lobe or manipulation of the meningeal vessel which can also lead to severe brain swelling.

Contributing factors for development of intraoperative brain swelling:

A number of contributing factors during anesthesia may initiate the process of intraoperative brain bulge. It is imperative on the part of anesthesiologist to identify these and institute remedial measures.

Venous obstruction:

Decreased venous return leading to cerebral congestion can also give rise to severe brain swelling in the intraoperative period. Obstruction to flow from abnormal head position is one of the most common causes of brain swelling. This is especially true for the patient whose head is supported with a horse shoe rather than pins. The head is often moved gradually so as to occlude venous return. Rigid head fixation can also inadvertently place the head and neck in physiologically extreme positions, causing jugular venous obstruction.

Inadvertent venous occlusion intracranially by the surgeon as a cause of cerebral venous obstruction is always a possibility. Bilateral pressure over the neck veins to stop air entrainment during venous air embolism invariably result in venous congestion and brain swelling. A tight tie around the neck can cause venous obstruction severe enough to cause massive brain swelling. Increase in the intrathoracic pressure from pneumothorax or bronchospasm can also

give rise to severe cerebral venous obstruction and subsequent brain swelling. Small amount of PEEP doesn't cause this problem. A PEEP of 10 cm H2O can be given without significant increase in intracranial pressure.

Why it occurs? :

Neurosurgical Conditions	Midline shifts Gross hydrocephalus Very large tumours Extensive oedema Counter coup Injuries
Patient's Conditions	Increased airway pressures Venous obstruction
Anaesthetic Considerations	Light plane of anaesthesia Inadequate analgesia Inadequate relaxation
Positioning	Improper positioning

How to manage It? :

Proper Positioning	Head up with 15-30 degree head elevation Avoid compression of neck veins No abdominal compression
Adequate anaesthesia and analgesia	Haemodynamics BIS Monitoring
Hyperventilation	Et CO2 28 mmHg
Hyperosmolar Diuretics	Mannitol(0.5-1.0 g/kg)
Loop Diuretics	Frusemide (1.0 mg/kg)
Lignocaine I/V	(1.5 mg/kg) as effective as 3 mg/kg Thiopentone
Metabolic Suppression	Reduce brain bulk and cerebral Metabolic activity
Hypothermia	Mild hypothermia is a safe and effective Method

If everything fails then decompresive craniectomy is undertaken.

Management of High ICP ('Tight Brain") During Anaesthesia

An intraoperative rise in ICP will impede surgery because if the brain is bulky it is more difficult to retract. Increased force will be needed on the brain retractors producing local damage. If during neurosurgery the ICP is noted to be high the likely cause must be identified and corrected. There are also specific methods for lowering ICP.

Checklist for Causes of High ICP During Surgery

- Position

  Head up[6]
  Clear cerebral venous drainage
  No abdominal compression

- Depth of anaesthesia, analgesia and muscle relaxantion
- Avoid inhalational agents
- Airway pressures
- CVP - 5-7, maximum 10-11
- Hyperventilation[7]

- No hypoxia
- Adequate ventilation — $PaCO_2$ low
  - Long expiratory pause
  - No PEEP

Treatment of High ICP:

Hyperosmolar Diuretics

Mannitol, which is perhaps the agent most frequently used, has many systemic and cerebral effects. Given in doses of 0.5-1.0 g/kg, it raises serum osmotic pressure so that water is drawn into the vascular system from the tissues. The increased oncotic pressure draws water from the brain and reduces brain bulk. As the action develops, the circulating blood volume rises and the haematocrit falls. The blood volume remains elevated for 15-30 min and during this time, the blood pressure and CVP may also be elevated. The diuresis limits the rise in blood volume. The decreased haematocrit allows a greater CBF and in patients with intact autoregulation, cerebral vasoconstriction occurs, keeping oxygen supply in balance with demand. The result is that the CBV is reduced and therefore so is the ICP. If autoregulation is impaired then the increased CBF persists, though ICP still falls, though to a small extent.

Loop Diuretics

Frusemide 1.0 mg/kg produces a fall in ICP similar to that produced by 1 g/kg mannitol. It acts by inhibition of sodium and chloride reabsorption in the ascending limb of the loop of Henle and has a separate action in reducing CSF production by suppressing sodium transport. It lowers ICP by mobilizing normal brain extracellular fluid and erebral oedema. The diuresis reduces blood volume and therefore the low cerebral venous pressure allows resorption of CSF. Frusemide appears not to affect the volume/pressure response, as does mannitol.

Steroids

They are particularly effective in patients with focal lesions and are ineffective when there is widespread brain injury. They have little place in the control of intraoperative high ICP but may be given intraoperatively to reduce postoperative swelling. They reduce the extrachoroidal production of CSF.

Hyperventilation

If high ICP occurs, then it is important to check the $PaCO_2$ with an arterial sample to avoid any inadvertent hypercapnia.

Metabolic Suppression

Shapiro described the use of barbiturates intravenously during periods of intraoperative high ICP. The aim is to reduce cerebral metabolic activity, while reducing brain bulk by producing a cerebral vasoconstriction[8]. Propofol and etomidate have also been used to lower ICP and have the advantage over the barbiturates that they are metabolized quickly. Lignocaine (1.5 mg/kg) may also be used to lower ICP, especially in the patient with cardiovascular instability. 1.5 mg/kg lignocaine is said to be as effective as 3 mg/kg thiopentone in lowering ICP[9].

Hypothermia:

Therapeutic moderate hypothermia (32° - 34° C) is safe and has sustained favorable effects on acute derangements of cerebral physiology and metabolism caused by severe closed head injury. Mild hypothermia is a safe and effective method to control traumatic intracranial hypertension and to improve mortality and morbidity rates.

Decompressive Craniectomy:

Surgical therapies such as decompressive craniectomy or temporal lobe resection may be required to save a patient's life or one may decompressed the bulge area of the brain. Outcome may be improved if craniectomy is performed before ICP>40 mmHg for prolonged periods.

The End of the Anaesthetic

Leech et al; measured ICP at the end of surgery, before and after the reversal of the muscle relaxants and after removal of the tracheal tube. After surgery but before reversal of the relaxants, the mean ICP was 11 mmHg but after reversal, with the patient breathing spontaneously, the mean ICP had risen to 21 mmHg. Just before return to the ward, the ICP was still high at 19 mmHg[10]. These are surprisingly high values considering that craniotomy had been performed and CSF drainage had taken place.

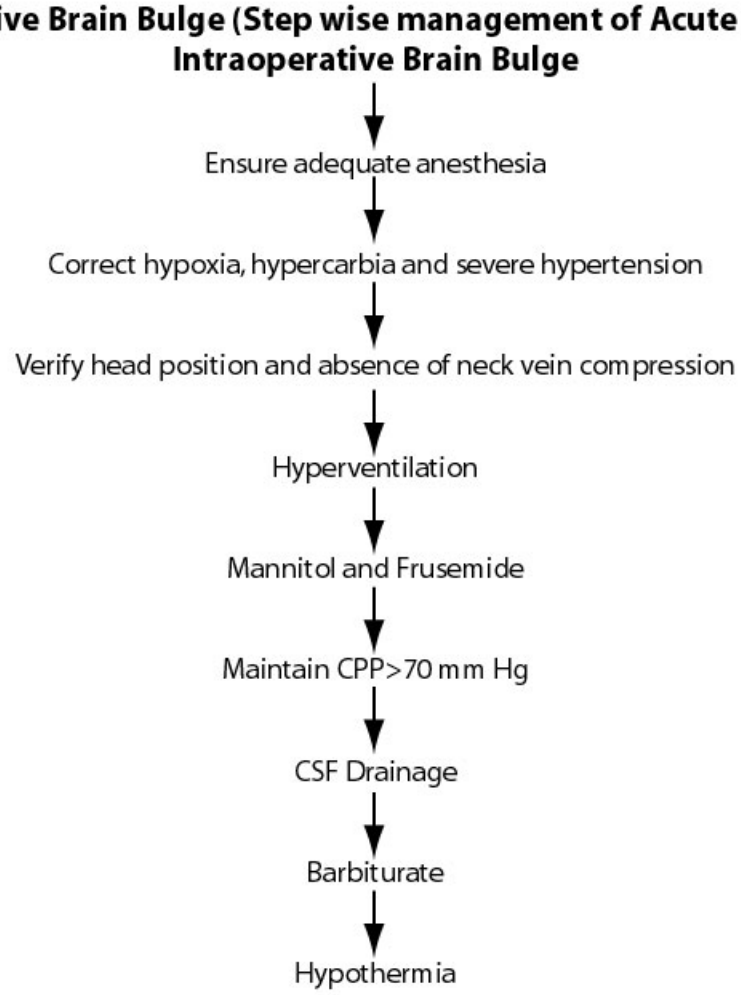
Management in specific situations:

When the brain protrudes from the craniotomy wound for an acute traumatic extra axial haematoma, cerebral oedema, cerebral hyperemia should be considered in the differential diagnosis and treatment should be directed to treat hyperemia and oedema. Hyperventilation should be instituted and mannitol should be given intravenously. If hyperventilation and mannitol fails then intravenous barbiturates are often effective in reducing brain bulge. When these measures fails, the likely cause of brain bulge is displacement by an intraoperative contralateral epidural haematoma. If these measures fails wound should rapidly be closed after achieving haemostasis. It may not be possible to replace the bone flap if the protrusion is severe. Immediate CT scan of the head or diagnostic burr holes are indicated. When a contralateral fracture is identified on a preoperative CT scan or skull X-ray films the first burrhole should be drilled over the fracture site other wise burrhole should be drilled at the standard site starting at the temporal fossa. Contralateral haematoma is a treatable condition and that should rapidly be evacuated to save the life of the patient. This problem should always be anticipated when there is clinical or radiographic evidence of fracture of skull on the contralateral side and patient should be positioned at the time of initial craniotomy in such a way that both the sides of the head are exposed to surgery. In the face of an uncontrollable brain bulge during surgery an exploration of contra lateral side can be done without delay.

Supportive measure:

When brain swelling is associated with or develop as a result of ischemia, measures should be taken to resuscitate the brain. This requires maintenance of adequate cerebral blood flow and brain oxygenation. In acute focal brain ischemai, cerebrovascular autoregulation to the undamaged brain may be intact. Elevation of cerebral perfusion pressure improves oxygen delivery to the ischemic penumbra. However, an inappropriately elevated cerebral perfusion pressure can result in increase in brain volume secondary to increase in cerebral blood volume or may cause disruption of the blood brain barrier and aggravate oedema formation.

Intraoperative Brain Bulge (Step wise management of Acute Brain Bulge)



Prevention:

Anticipation of intraoperative brain swelling is the first step in prevention. Patients with respiratory compromise such as those with bronchial asthma, chronic bronchitis or aspiration pneumonitis need special attention. Embolization of large vascular tumours should be considered before surgery. Patients with peritumoral oedema seen on CT scan or MRI should be given several days of steroid prior to surgery and mannitol infusion during craniotomy. Adequate control of blood pressure and prophylactic use of barbiturate comma can be used after resection of arteriovenous malformation to prevent development of normal perfusion pressure breakthrough syndrome. When brain swelling occurs during an otherwise uncomplicated surgery all possible anaesthetic causes, carbon dioxide retention or venous obstruction should be looked for and corrected.

Conclusion:

Intraoperative brain bulge have multiple aetiologies like formation of intracranial haematoma, ischemia or hyperemia but preventable causes like hypercarbia and venous obstruction also contribute significantly in its development. In most instances the neuroanaesthesiologist holds the key to intracranial pressure and prevent this complication. Rapid reversal of brain swelling is also possible with prompt step wise measures.

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IX Annual Conference of Indian society Neuroanaesthesiology & Critical Care

ISNACC-2008
8th - 10th February 2008
VENUE: Auditorium

Gandhi Medical College Musheerabad. (Secunderabad)

Organised By : Department of Anaesthesiology & Intensive Care

NIZAM'S INSTITUTE OF MEDICAL SCIENCES, PUNJAGUTTA, HYDERABAD-500 082. A. P. INDIA

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	1st Jun - 30th Nov 2007	After 30th Nov 2007&Spot
Life Members	Rs. 2,500	Rs. 3000
Accompanying Person	Rs.1,500	Rs.1,500
Non-Members and SAARC Countries	Rs. 3,000	Rs. 3,500
PG & Nurses	Rs. 1,500	Rs. 2000
Workshops	Rs. 200	Rs. 250
Foreign Delegates	$ 100	$ 150

Please send the DD infavour of **"ISNACC-2008"** payable at Hyderabad. Certificate and Registration form to be attested by Head of Department for PG & Nurses.

Last Date for Abstract Submission - 30th November 2007.

I wish all our readers and members of our society a very happy and prosperous New Year

Dr. H. H. DASH
Editor - in - Chief

TRAVEL GRANT

ISNACC will award Travel Grant to suitable candidates to either visit one of the premier Neuroanaesthesiology centres in India or to present one or more free papers in the ISNACC annual conference. A fixed sum of Rs. 10,000/- each will be awarded to 2 candidates who must fulfill the following criteria:

- Should be a life member of ISNACC.
- If the grant is for attending the annual conference, he or she must present a free paper as first author
- Should provide a certificate attesting that he or she is a Junior Resident or Senior Resident.

Application along with documents supporting your candidature should reach the ISNACC Secretariat by 31 December 2008.

RESEARCH GRANT

ISNACC will award one research grant to a suitable candidate to carry out clinical research in the field of Neuroanaesthesia and critical care in India. A fixed sum of Rs. 10,000/- will be awarded to one candidate who must fulfill the following criteria:

- Candidate must be a life member of ISNACC.
- Working certificate in Dept. of Neuroanaesthesia has to be submitted from the HOD.
- Ethics committee's approval is mandatory.
- Information pertaining to any other financial assistance for the project from other sources must be provided.
- Four copies of the research project, in the proper format should be submitted to the secretariat on or before 31st December 2008.

NEWS ITEM

1. Dr M.P. Pandia, Assistant Professor, Dr Hemant Bhagat (Post doctoral) and Dr Virender Jain (DM Student) from the Dept. of Neuroanaesthesia, AIIMS were awarded Rs. 10,000/- each to attend the Annual Conference of American Society of Anaesthesiology 2007, and SNACC - 2007 held at San Francisco.
2. Dr Virender Jain's paper has been awarded as one of the 10 Best Papers Award by the SNACC.
3. Dr. P. Smita passed her DM (Neuroanesthesia) in December 2007, at Sree Chitra, Trivandrum

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IX Annual Conference of The Indian Society of Neuroanaesthesiology & Critical Care

(ISNACC 2008)

February 8th - 10th , 2008

Venue : Auditorium

Gandhi Medical College
Musheerabad

Organized by :

Department of Anaesthesiology & Intensive Care
Nizam's Institute of Medical Sciences
Hyderabad - 500 082- INDIA.

SCIENTIFIC PROGRAMME

Day I (CME) (8th Feb)

8.00 -8.45 AM	**Registration**	
8.45 - 9.15 AM	Formal Inauguration	
9.30-11.00	**Session I - Chairpersons**	**Speaker**
	R.C.Rathod and Dr.K.J.Choudhury	
	Education in Neuroanaesthesia	H.H.Dash
	Cerebral blood flow :- Relevance to Neuroanaesthesia	G.S.Umamaheswara Rao
	Choice of Anaesthetic Drugs in Neuroanaesthesia -	Adrian .W.Gelb .
11.00-11.15	**TEA**	
11.15 - 12.00	**Oration I Prof .Malathi Devi Oration**	
	Chairpersons	**Speaker**
	G.S.Umamaheswara Rao and G..Parameswara	
	Evidence Based Medicine in Neuroanaesthesia Practice	Christian Werner
12.00 -1.00	**Session II - Chairpersons**	**Speaker**
	Anil Parakh and M.Chandrashekar	
	Pathophysiology of Brain injury and Neuronal cell death.	Piyush Patel
	Recent trends in Neuro-protective Methods.	Yoram Shapira
1.00-2.00	**LUNCH (Poster Presentations- judges: Christian Werner, Radhakishen Rao & Piyush Patel.)**	
2.00 - 5.30 PM	Registered delegates to proceed to WORKSHOPS.	
	1) Pre hospital care in Trauma patients. (EMRI) 2) Interventional pain management for low back-pain. (Wockhardt Hospital) (Details are given in the last page)	
2.00 - 3.00	**Session III - Chairpersons**	**Speaker**
	G.Surender Reddy and Venkatesh H.K.	
	Interventional Neurophysiological Monitoring	Matthew Chan
	Neurochemical methods of Monitoring Transmitter dynamics in Brain.	Martin Smith

3.00 -4.00	**Session IV - Chairpersons**	**Speaker**
	J.M Reddy and P.Surender	
	Current concepts in Management of ICP	Bibhukalyani Das
	Anaesthetic management of Epilepsy Surgery	Mary Abraham
4.00-4.15	**TEA**	
4.15 - 5.15	**Session IV Chairpersons**	**Speaker**
	Dr Kanchan Jagger and Dr.Sai Satyanarayana	
	Anaesthetic considerations in craniofacial surgery.	Ram Kumar
	Intra-operative Fluid Management in Neuroanaestheisa	Pramod.K.Bithal
7.00	**Inauguration ' Dinner**	

Day II (9th Feb)

9.00 AM-11.00 AM		
	Session I - Chairpersons	**Speaker**
	Amna Goswami and L.D.Mishra	
	Current status of hyperventilation including unresolved questions .	Adrian W.Gelb
	Recent advances in care of Stroke Patients.	Martin Smith
	Brain death and Care of Organ Donor.	S.Manimala Rao,
	Outcome Predictors in Neuro-ICU.	Jyotsna Wig
11.00-11.15	**TEA**	
11.15-12.00-	**Oration II Prof.G.R.Gode Oration**	
	Chairpersons	**Speaker**
	H.H.Dash and Prabhavathi	
	Current Trends in Paediatric Neuroanaesthesia.	Kanchan Jagger
12.00- 12.45	**Oration III Prof.Hari Wir Singh Oration**	
	Chairpersons	**Speaker**
	Jyotsna wig and R.Gopinath	
	Changing role of Neuroanaesthesiologist with emerging trends in neurosurgery	K.J.Choudhury
12.45 -1.45	**LUNCH (Poster Presentations- judges: Christian Werner, Radhakishan Rao & Piyush Patel.)**	
1.45 - 3.15	**Session II - Chairpersons**	**Speaker**
	Bibhukalyani Das and A.K..Purohit,	
	Vasospasm and it's management in SAH	J.N.Monterio
	Protective strategies in spinalcord injury.	Kristin Engelhard
	Anaesthetic management of spinalcord injured patient.	Col.TVSP.Murthy
3.15 - 4.00	**Session III - Chairpersons**	**Speaker**
	Aruna Subhash and Kavita Sandhu	
	How I Do It ?	
	Resuscitation of a Patient undergoing surgery in Prone position.	Venkatesh.H.K

	Pregnant Patient in 2nd trimester for Craniotomy.	Shashi srivastava
	MAC for DBS	Rajashree .C.Deopujari
4.00-4.15 -	**TEA**	
4.15-5.45PM	**Session IV - PANEL DISCUSSION On TRAUMATIC BRAIN INJURY**	
	Moderator: Umamaheswara Rao	
	Panelists:	
	Grace Korula Anil Parakh,	
	Pramod.K.Bithal V.J.Ramesh	
6.00 PM	**GBM ---------- BANQUET**	

Day III (10th Feb)

9.00 AM-10.00 AM		
	Session I - Chairpersons	**Speaker**
	Annapurna Rout and Manas Panigrahi	
	Anaesthetic considerations in Neuro-endocrine Disorders.	R.C.Rathod
	Postoperative pain management in Neurosurgical patients.	G.Parameswara
10.00 AM -11.00 AM		
	Session II - Chairpersons	**Speaker**
	Pragati Ganjoo and Dilipkumar Kulkarni	
	Recent radiological tools in neuroscience research.	Rammurti
	ThromboProphylaxis for Neurosurgical patients.	Deep Raj singh
11.00-11.15	**TEA**	
11.15- 12.45		
	Session III - Chairpersons	**Speaker**
	TVSPMurthy and M.Krishna Prasad	
	Information management system in Neuro-OT.	Mahesh
	Role of TCD in Neuroanaesthesia Practice	Deepak Sharma
Pros & Cons		
	Unstable Cervical spine - Intubation with Conventional vs Fiberoptic	Kavita Sandhu &
	Intubation In MP(Mallampati) Grade I and II.	Hemanshu Prabhakar
12.45 -1.30		
	Session IV Open forum - Moderator	**Speaker**
	Marry Abraham	
	Critical Incident monitoring in neuroanaestheisa.	L.D.Mishra
	Ethical and Legal considerations in neurosurgical patients.	S.C.Parakh
1.30 - 2.30	**LUNCH**	
2.30 - 3.30	**Valedictory Function**	

WORKSHOPS & DETAILS :
of IX ISNACC Conference Hyderabad (Feb. 8 - 10, 2008)

1) PRE HOSPITAL CARE IN TRAUMA PATIENTS.

i) Transformation of Emergency Management in India

(EMRI Presentation & EMRI Tour) EMRI 1.30 minutes

ii) Pre hospital care in Head injury and Spinal injury .

(Hands on workshop on dummies for stabilization, resuscitation etc., and The EMRI experience - 1.30 minutes - in batches)

iii) Open discussion -15 Minutes.

Dr Ramana Rao, Dr Sanjay Thakur and other Senior Specialists in field of Neuro Surgery.

EMERGENCY MANAGEMENT & RESEARCH INSTITUE. Medchal Road - Hydearbad

2) INTERVENTIONAL PAIN MANAGEMENT FOR LOW BACK-PAIN.

i) Epidural Injections (Interlaminar, Transforaminal, & caudal)

ii) Radiofrequency Denervation

iii) Sacroiliac joint Injections

iv) Epiduroscopy.

Dr.Muralidhar Joshi - Wockhardt Hospital. Hyderabad

1st Asian Society of Neuroanaesthesia & Critical Care Conference

Nov. 28th - Dec. 1st, 2008
Beijing, China

Dear Prof. H.H.Dash:

I am Dr. Jin, one of the secretaries of the first congress of Asian Society for Neuroanesthsia and Critical Care. I am so honored to report some news of the meeting to you.

After consulting the date with the other countries, we decided the meeting to be held from Nov 28th to Dec 1st at Beijing . We applied a website: www.asnacc.com, where you can get some information of the meeting. The website is under construction now and we will put more information on it. We have planed to inform you after the website is completed so please forgive us for the delay. We sincerely invite the doctors in your country to attend the meeting and we will be very grateful if you could inform them of the meeting.

We look forward to getting more news from you and meeting you next year.

Best regards,

Dr. Hailong Jin

Contact: csna2007@yahoo.co.cn