News Letter

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INDIAN SOCIETY OF NEUROANAESTHESIOLOGY AND CRITICAL CARE

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Dept. of Neuroanaesthesiology & Chief, Neurosciences Centre

All India Institute of Medical Sciences, New Delhi-110 029

2nd Conference of
Asian Society for Neuroanesthesia and Critical Care
&
12th Annual Conference of
Indian Society of Neuroanaesthesiology and Critical Care

Date:
February, 25-27, 2011

Venue:
India Habitat Centre, Lodhi Road, New Delhi (India)

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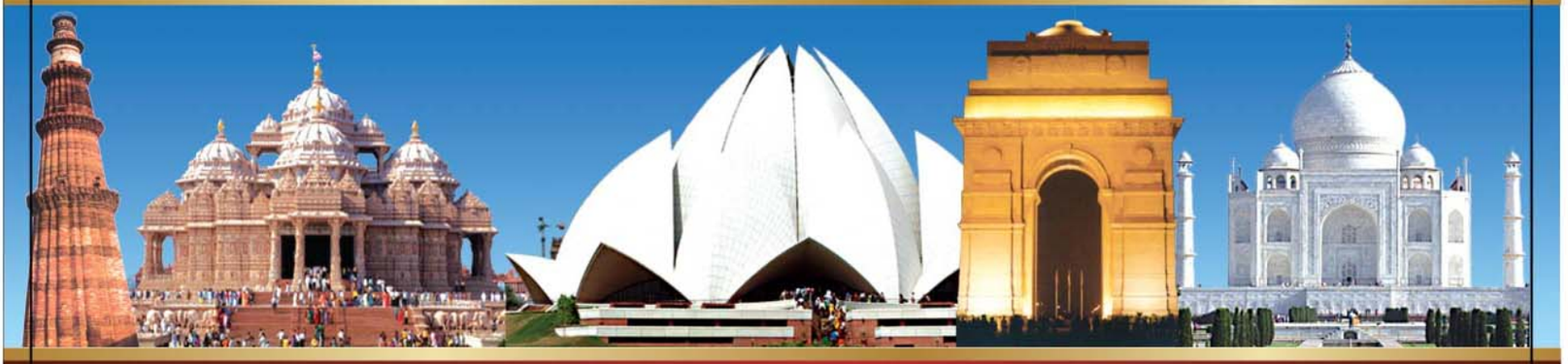

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From the Editor's desk

Time does not wait for anybody. Last year (2010) has already gone and we have welcomed 2011 with pump and show. May I have the honour of wishing all our esteemed members "a very happy, healthy and prosperous new year". New Year always brings new optimism, commitments and priorities not only for individuals but, also for the organizations. Before, we plan our priorities for ISNACC let us introspect our activities in the year gone by.

Year 2010 will be remembered for a long time because of few reasons. The first and the foremost is conferring the Fortes Hospital, Noida to start the "certificate course in Neuroanaethesiology" under the able stewardship of Prof. Mary Abraham. A three member committee consisting of President, Secretary and the Editor of the society visited the hospital to inspect whether the hospital has the requisite facilities and infrastructure to undertake the certificate course. I congratulate Prof. Mary Abraham for creating all the facilities deemed necessary to start the certificate course in Neuroanaesthesiology in Fortes Hospital, Noida under her mentorship.

Secondly, we had a very fruitful meeting with the Executive Director of National Board of Examinations, Professor A.K. Sood pertaining to the DNB fellowship course in Neuroanaesthesiology and Critical Care in India. He sounded very encouraging and advised the ISNACC team members as to how to proceed to achieve this goal. His tips will be of immense help to start the course in various private and government institutions. Work in this direction have already been initiated by the office bearers of ISNACC and we are very sanguine of a positive outcome by the end of this year.

Last bust most outstanding achievement in 2010 for Indian Society of Neuroanaesthesiology and Critical Care was the recognition by the International Society of Neurosciences Anesthesiology and Critical Care (SNACC) in their annual meeting held at San Diego, USA (October 14-15, 2010) for which the founder President, "I" was decorated with the most prestigious "Teachers of the Year 2010" award.

Alongside the achievements the society has witnessed failures in few fronts. Among the failures the superspeciality of Neuroanaesthesiology is yet to receive due recognition from Medical Council of India. Our concerted efforts to seek an appointment with Professor Shiv Sarin, President MCI have so far been unsuccessful. We are trying our best to get an audience with him so that we can apprise him about the benefits of superspeciality course which is already in vogue at AIIMS and Sree Chitra Tirunal Institute for Medical Sciences & Technology. Other major setback is the commitment of our members to submit the scientific manuscripts for publication in ISNACC News letter. Despite, my fervent and umpteen appeals in the past, I did not receive any manuscript for publication in this edition of our Newsletter. Once again, May I request all our esteemed members please think sincerely and submit your research work for publication. In absence of good research work the society cannot dream of having a journal of its own.

I pray the Almighty to shower the society with its choicest blessings in 2011 and make the "2nd ASNACC conference" a memorable event. A successful and glitch free 1st International Neuroanaesthesia conference in India will bring name and fame to ISNACC. May I request all our members to participate in large numbers and make the conference a talking point for years to come. So far we have encouraging response from all the Asiatic countries and I am sure, our members will actively take part in the conference and make the conference a resounding success. Long live ISNACC.

H. H. DASH & P. K. Bithal
AIIMS

Scientific Sessions

DAY 1 (Friday: 25 .02. 2011)

8:30 am	**REGISTRATION**
9:00 - 9:15 am	WELCOME OF DELEGATES & INAUGURATION OF CME
	SESSION I : TOPICS (Talk Time: 25 min) SPEAKERS
9:15 - 9 :45 am	Intraoperative monitoring of motor evoked potentials during neurosurgical procedures ***Dr. Masahiko Kawaguchi***
9:45 - 10:15 am	When will we have general purpose neuroprotection? ***Dr. John Hartung***
10:15 - 10:45 am	Cerebral blood flow measurement techniques & neurosurgery ***Dr. H K Venkatesh***
	DISCUSSION (15 Mins)
10:45 - 11:00 am	**TEA**
11:00 - 11:45 am	**SESSION II : DR. MALTHI ORATION**
	Neuroanesthesia – History and the Future ***Dr. Takefumi Sakabe***
	SESSION III: MRI IN NEUROSURGERY
11:45 - 12:15 pm	An update of safety issues for anaesthesia in MRI ***Dr.Peter Farling***
12:15 - 12:45 pm	Anaesthesia for intraoperative MRI ***Dr. Kyeong Tae Min***
12:45 - 1:15 pm	Neuroanaesthesia considerations for intraoperative MRI: Saudi Arabia experience ***Dr. Mohammad Takrouri***
	DISCUSSION (15 Mins)
1:15 - 2:00 pm	**LUNCH**
2:00 - 2:45 pm	**SESSION IV: WORKSHOP**
	TCD in Neuroanaesthesiology ***Dr. Deepak Sharma***
	SESSION V: NEURO TRAUMA & NEUROCRITICAL CARE
2:45 - 3:15 pm	Protocol based management in neurotrauma ***Dr. Dilip Kulkarni***
3:15 - 3:45 pm	Multimodal cerebral monitoring after severe brain injury ***Dr. Arun Gupta***
3:45 - 4:15 pm	Paediatric neurotrauma ***Dr. Monica Vavilala***
	DISCUSSION (15 Mins)
4:15 - 4:30 pm	**TEA**
	SESSION VI: COMPLICATIONS IN NEUROSURGERY
4.30 - 5.00 pm	Cardiopulmonary complications of brain injury ***Dr. Martin Smith***
5.00 - 5.30 pm	Postoperative blindness following surgery on the spine ***Dr. Smita Sharma***
5.30 - 6.00 pm	Need for intensive care after neurosurgery ***Dr. S. Manikandan***
	DISCUSSION (15 Mins)
6.30 PM Onwards	**INAUGURATION & CULTURAL PROGRAM**
	DINNER AT INDIA HABITAT CENTRE

DAY 2 (Saturday: 26. 02. 2011)

SESSION I: DRUGS IN NEUROANAESTHESIA & NEUROCRITICAL CARE

TOPICS (Talk Time: 25 min) **SPEAKERS**

9.00 - 9.30 am	Nitrous Oxide: Is it appropriate for neurosurgery and neurologically at-risk patients? ***Dr. V K Grover***
9:30 - 10:00 am	The role of beta blockers for neuroprotection ***Dr. Toru Goyagi***
10.00 - 10:30 am	Dexmedetomidine in neurosurgery ***Dr. Pryombodho***
10: 30 -11:00 am	Magnesium Sulphate : Old drug new concepts ***Dr. Sri Rahardjo***
	DISCUSSION (15 Mins)
11:00 – 11.15 am	**TEA**

SESSION: II

11:15 - 12:00 Noon	**PROF. G. R. GODE ORATION**
	Slow but Great Wave of ASNACC ***Dr. Haekyu Kim***

SESSION: III

12.00 - 12.30 pm	Controversies in management of cerebral aneurysms ***Dr. Adrian Gelb***
12.30 - 1.00 pm	Intravenous magnesium sulphate for aneurysmal subarachnoid hemorrhage ***Dr. Matthew Chan***
	DISCUSSION (15 Mins)
1:00 - 2:00 pm	**LUNCH**

SESSION IV : FLUID THERAPY & BLOOD CONSERVATION

2.00 - 2.30 pm	Fluid management in the neurosurgical patient Dr. C. Tommasino
2:30 - 3:00 pm	Does exogenous lactate infusion improve cognitive impairment in post-traumatic brain injury patients? ***Dr. Tatang Bisri***
3.00 - 3.30 pm	Blood conservation during neurosurgery ***Dr. Baoguo Wang***
	DISCUSSION (15 Mins)
3.30 - 4.15 pm	**PANEL DISCUSSION**
	Non technical skills in neuroanaesthesia & neurointensive care ***Dr. R. Mahajan, Dr. A Bhattacharya, Dr. A. Gelb, Dr. Thomas Lew, Dr. A K Singh***
4.15 - 4.30 pm	**TEA**
4:30 - 5:15 pm	WORKSHOP : Airway Management ***Dr. Anil Parakh***
5:30 - 6.30 pm	**GENERAL BODY MEETING**
	DINNER

DAY 3 (Sunday: 27. 02. 2011)

SESSION I:

TOPICS (Talk Time: 25 min) SPEAKERS

9.00 - 9.30 am	Glycaemic control in neurosurgical patient	***Dr. Nidhi Panda***
9:30 - 10:00 am	Anaesthesia for intervention neuroradiological procedures	***Dr. R. C. Rathod***
10:00 - 10:30 am	Anaesthesia for endoscopic neurosurgical procedures	***Dr. Shashi Srivastava***
10:30 - 11:00 am	Postoperative pain management following neurosurgical procedures	***Dr. M. Radhakrishnan***

DISCUSSION (20 Mins)

11:00 – 11:15 am TEA

11:15 - 12:00 Noon PROF. HARIWIR SINGH ORATION

Outcome of neurological patients- A neuroanaesthesiology perspective
Dr. G. S. Umamaheswara Rao

SESSION II: IMPROVING THE SCIENCE OF NEUROANAESTHESIA

12.00 - 12.30 pm	The application of improvement science in Neuroanaesthesia and critical care	***Dr. Thomas Lew***
12:30 - 1:00 pm	Safety in medicine – a paradigm shift?	Dr. Ravi Mahajan

DISCUSSION (10 Mins)

1.00 – 2.00 pm LUNCH

2:00 - 2:45 pm WORKSHOP

Utility of TEE in Neuro-Anaesthesia & Neuro Intensive Care ***Dr. K. Subramaniam***

SESSION IV: ANAESTHETIC CONSIDERATIONS FOR SPECIFIC CONDITIONS

2:45 - 3.15 pm	Anaesthesia for deep brain stimulation	***Dr. Joseph Monteiro***
3:15 - 3:45 pm	Emergence after neurosurgery	***Dr. Hemant Bhagat***
3:45 - 4:15 pm	Anaesthesia for neurosurgery in pregnancy	***Dr. Indranil Ghosh***

DISCUSSION (15 Mins)

4:15 - 4:30 pm VALEDICTORY FUNCTION & TEA

POSTER SESSION: ON ALL THREE DAYS

(SEPARATE SCHEDULE IS PREPARED FOR POSTER SESSION)

COAGULOPATHY IN MODERATE HEAD INJURY. THE ROLE OF EARLY ADMINISTRATION OF LOW MOLECULAR WEIGHT HEPARIN

Pahatouridis D, Alexiou GA, Zigouris A, Mihos E, Drosos D, Voulgaris S.
Department of Neurosurgery, University Hospital of Ioannina, Ioannina, Greece.

Brain Inj. 2010;24:1189-92.

Abstract

Introduction: Abnormalities in blood coagulation are relatively common after traumatic brain injury (TBI). We prospectively studied the safety of the early antithrombotic prophylaxis with low molecular weight heparin.

Methods: We prospectively evaluated 61 patients with moderate TBI. Patients requiring surgical treatment and/or with injuries in other systems were excluded. Coagulation studies included among others prothrombin time (PT), plasma fibrinogen levels and D-dimer levels. Blood samples were collected on admission and 24 h, 48 h, and 72 h later. Prophylaxis was started within 24 hours with tinzaparin.

Results: In 42 of 61 patients a form of disseminated intravascular coagulation (DIC) was detected. The severity of head injury was correlated with the severity of the coagulation disorders. The PT was prolonged in the first two days. Plasma fibrinogen levels dropped initially and increased to above normal values 2-3 days later. D-dimer levels were significantly elevated and in 19 patients remained elevated throughout the study period. Clinical manifestations of DIC were not observed. Conclusions: Patients with moderate TBI are at a serious risk of developing brain intravascular microthrombosis. Our study supports the early use of low molecular weight heparin.

ALVEOLAR RECRUITMENT MANEUVER IN PATIENTS WITH SUBARACHNOID HEMORRHAGE AND ACUTE RESPIRATORY DISTRESS SYNDROME: A COMPARISON OF 2 APPROACHES.

Nemer SN, Caldeira JB, Azeredo LM, Garcia JM, Silva RT, Prado D, Santos RG, Guimarães BS, Ramos RA, Noé RA, Souza PC.
Hospital de Clínicas de Niterói, Rua La Salle 12, Centro, Niterói, Rio de Janeiro 24020-090, Brazil.

J Crit Care. 2010 Jun 18. [Epub ahead of print]

Abstract

Purpose: The purpose of the study was to compare 2 alveolar recruitment maneuvers (ARMs) approaches in patients with subarachnoid hemorrhage (SAH) and acute respiratory distress syndrome (ARDS).

Material and Methods: Sixteen SAH patients with ARDS were randomized in 2 similar groups. One received ARM with continuous positive airway pressure (CPAP) of 35 cm H(2)O for 40 seconds (CPAP recruitment), whereas the other received pressure control ventilation with positive-end expiratory pressure of 15 cm H(2)O and pressure control above positive end-expiratory pressure of 35 cm H(2)O for 2 minutes (pressure control recruitment maneuver [PCRM]). Intracranial pressure (ICP) and cerebral perfusion pressure (CPP) were measured before and after ARM. The ratio of arterial oxygen tension to fraction of inspired oxygen was measured before and 1 hour after the ARM.

Results: After ARM, ICP was higher in CPAP recruitment (20.50 +/- 4.75 vs 13.13 +/- 3.56 mm Hg; P = .003); and CPP was lower in CPAP recruitment (62.38 +/- 9.81 vs 79.60 +/- 6.8 mm Hg; P = .001). One hour after the ARM, the ratio of arterial oxygen tension to fraction of inspired oxygen increased significantly only in PCRM (108.5 to 203.6; P = .0078). CONCLUSION: In SAH patients with ARDS, PCRM did not affect ICP and decreased CPP in safe levels, besides improving oxygenation.

XE-CT AND TRANSCRANIAL DOPPLER IN SYMPTOMATIC VASOSPASM SUBARACHNOID HEMORRHAGE PATIENTS UNDER EUVOLEMIC TREATMENT WITHOUT SEDATION.

Xu Y, Wang Y, Bao Y, Zhang H, Ling F.
Department of Neurosurgery, Capital Medical University, Xuanwu Hospital,
45 Changchun Street, Beijing - 100 053, China.

Neurol India. 2010;58:407-11.

Abstract

Background: Delayed cerebral ischemia from cerebral arterial vasospasm following aneurysmal subarachnoid hemorrhage (aSAH) is associated with significant morbidity and mortality. Early recognition of the cerebral arterial vasospasm and institution of appropriate treatment can reduce the consequences.

Aim: We investigated the association of transcranial Doppler (TCD) and Xe-CT with the characteristics of symptomatic vasospasm secondary to aneurysmal subarachnoid hemorrhage (SAH) in patients who underwent euvolemic treatment without sedation.

Materials and Methods: Data collected prospectively in patients with aSAH admitted to a neurocritical care unit in a regional hospital were retrospectively analyzed. Out of the 98 consecutive patients with aSAH, 30 patients underwent paired Xe-CT (not sedated) and TCD studies. Correlation between cortical cerebral blood flow (CBF) and mean blood flow velocity in middle cerebral artery (MCA) territories was analyzed. The lowest cortical regional CBF and MCA velocity were compared between patients with and without symptomatic vasospasm.

Results: Symptomatic vasospasm occurred in 12 patients. No correlation was found between CBF and mean blood flow velocity of the MCA territory. The differences between MCA velocity and lowest cortical CBF in patients with symptomatic vasospasm were significantly different from patients without symptoms.

Conclusion: TCD does not help to predict regional CBF in the MCA territory in patients with aSAH on euvolemic treatment.

ENDOVASCULAR TREATMENT OF MEDICALLY REFRACTORY CEREBRAL VASOSPASM FOLLOWING ANEURYSMAL SUBARACHNOID HEMORRHAGE.

Jun P, Ko NU, English JD, Dowd CF, Halbach VV, Higashida RT, Lawton MT, Hetts SW.
Departments of Radiology, Neurology and Neurosurgery, University of California, San Francisco, California.

AJNR Am J Neuroradiol. 2010:31:1911-6.

Abstract

Background and Purpose: CV following aneurysmal SAH is a significant cause of morbidity and mortality. We review our experiences using PTA and IA verapamil infusion for treating medically refractory cases.

Materials and Methods: We performed a retrospective review of patients with SAH admitted from July 2003 to January 2008.

Results: Of 546 patients admitted within 72 hours of symptom onset, 231 patients (42%) developed symptomatic CV and 189 patients (35%) required endovascular therapy. A total of 346 endovascular sessions were performed consisting of 1 single angioplasty, 286 IA verapamil infusions, and 59 combined treatments. PTA was performed on 151 vessel segments, and IA verapamil was infused in 720 vessel segments. IA verapamil doses ranged from 2.0 to 30.0 mg per vessel segment and from 3.0 to 55.0 mg per treatment session. Repeat treatments were necessary in 102 patients (54%) for persistent, recurrent, or worsening CV. There were 6 treatment-related complications, of which 2 resulted in clinical worsening. No deaths were attributable to endovascular therapy. At follow-up, 115 patients (61%) had a good outcome and 55 patients (29%) had a poor outcome. Sixteen patients died from causes related to SAH, while 3 died from other medical complications.

Conclusions: Endovascular treatments are an integral part of managing patients with medically refractory CV. In our experience, PTA and IA verapamil are safe, with a low complication rate, but further studies are required to determine appropriate patient selection and treatment efficacy.

THE RELATIONSHIP BETWEEN INFLAMMATORY MARKERS AND POST STROKE COGNITIVE IMPAIRMENT

Rothenburg LS, Herrmann N, Swardfager W, Black SE, Tennen G, Kiss A, Gladstone DJ, Ween J, Snaiderman A, Lanctot KL

J Geriatr Psychiatry Neurol. 2010;23:199-205.

Abstract

Objective: To determine whether there is a relationship between inflammatory markers (serum C-reactive protein (CRP) and cytokines) and post stroke cognitive impairment (PSCI).

Methods: This was a cross-sectional observational study. Patients were recruited from 4 sources: (1) the acute stroke unit of a general hospital, (2) an outpatient stroke prevention clinic, (3) a stroke rehabilitation unit in a specialized geriatric hospital, or (4) a stroke rehabilitation unit of a rehabilitation hospital. Patients meeting National Institute of Neurological and Communicative Disorders and Stroke (NINCDS) and World Health Organization Multinational Monitoring of Trends and Determinants in Cardiovascular Disease (WHO-MONICA) project criteria for stroke were invited to participate in this study within the first 5 to 31 days post stroke. Patients with subarachnoid or intracranial hemorrhage, decreased level of consciousness, severe aphasia or dysarthria, or a significant acute medical, neurological, or psychiatric illness were excluded. Clinical assessments included the Mini-Mental State Examination (MMSE) for cognition, the National Institutes of Health Stroke Scale (NIHSS) for stroke severity, and the Center for Epidemiological Studies-Depression Scale (CES-D) for depressive symptoms. Enzyme-linked immunosorbent assay (ELISA) was used to measure serum concentrations of CRP, interleukin 6 (IL-6), and interferon gamma (IFN-gamma). Results: A total of 48 patients with ischemic stroke (age [mean +/- SD] 71.6 +/- 13.2 years, 54.2% male, MMSE 26.4 +/- 3.8, NIHSS 6.8 +/- 4.0) were recruited within their first month post stroke. Backward stepwise elimination linear regression showed that higher concentrations of serum CRP (betaCRP = -0.46, pCRP = 0.002) predicted lower post stroke global cognition ([MMSE], F1,44 = 11.31, P = .002), with age (P = .92), level of education (P = .22), infarct side (P = 0.49), IL-6 (P = 0.36), and IFN-gamma (P = .57) removed from the final model.

Conclusions: A post stroke inflammatory response may be important in subacute, PSCI.

SPHENOPALATINE GANGLION STIMULATION FOR VASOSPASM AFTER EXPERIMENTAL SUBARACHNOID HEMORRHAGE

Takahashi M, Zhang ZD, Macdonald RL.
Section of Neurosurgery, Department of Surgery, University of Chicago Medical Center and Pritzker School of Medicine, Chicago, Illinois; and.

J Neurosurg. 2010 Jul 2.

Abstract

Object: Sphenopalatine ganglion stimulation activates perivascular vasodilatory nerves in the ipsilateral anterior circle of Willis. This experiment tested whether stimulation of the ganglion could reverse vasospasm and improve cerebral perfusion after subarachnoid hemorrhage (SAH) in monkeys.

Methods: Thirteen cynomolgus monkeys underwent baseline angiography followed by creation of SAH by placement of autologous blood against the right intradural internal carotid artery, the middle cerebral artery (MCA), and the anterior cerebral artery. Seven days later, angiography was repeated, and the right sphenopalatine ganglion was exposed microsurgically. Angiography was repeated 15 minutes after exposure of the ganglion. The ganglion was stimulated electrically 3 times, and angiography was repeated during and 15 and 30 minutes after stimulation. Cerebral blood flow (CBF) was monitored using laser Doppler flowmetry, and intracranial pressure (ICP) was measured throughout. The protocol was repeated again. Evans blue was injected and the animals were killed. The brains were removed for analysis of water and Evans blue content and histology.

Results: Subarachnoid hemorrhage was associated with significant vasospasm of the ipsilateral major cerebral arteries (23% +/- 10% to 39% +/- 4%; $p < 0.05$, paired t-tests). Exposure of the ganglion and sham stimulation had no significant effects on arterial diameters, ICP, or CBF (4 monkeys, ANOVA and paired t-tests). Sphenopalatine ganglion stimulation dilated the ipsilateral extracranial and intracranial internal carotid artery, MCA, and anterior cerebral artery compared with the contralateral arteries (9 monkeys, 7% +/- 9% to 15% +/- 19%; $p < 0.05$, ANOVA). There was a significant increase in ipsilateral CBF. Stimulation had no effect on ICP or brain histology. Brain water content did not increase but Evans blue content was significantly elevated in the MCA territory of the stimulated hemisphere.

Conclusions: Sphenopalatine ganglion stimulation decreased vasospasm and increased CBF after SAH in monkeys. This was associated with opening of the blood-brain barrier.

COMPETITIVE INHIBITION AT THE GLYCINE SITE OF THE N-METHYL-D-ASPARTATE RECEPTOR MEDIATES XENON NEUROPROTECTION AGAINST HYPOXIA-ISCHEMIA

Banks P, Franks NP, Dickinson R.
Biophysics Section, Blackett Laboratory, Department of Anaesthetics, Pain Medicine and Intensive Care, Imperial College London, London SW7 2AZ, United Kingdom.

Anesthesiology. 2010;112:614-22.

Abstract

Background: The general anesthetic gas xenon is neuroprotective and is undergoing clinical trials as a treatment for ischemic brain injury. A small number of molecular targets for xenon have been identified, the N-methyl-D-aspartate (NMDA) receptor, the two-pore-domain potassium channel TREK-1, and the adenosine triphosphate-sensitive potassium channel (KATP). However, which of these targets are relevant to acute xenon neuroprotection is not known. Xenon inhibits NMDA receptors by competing with glycine at the glycine-binding site. We test the hypothesis that inhibition of the NMDA receptor at the glycine site underlies xenon neuroprotection against hypoxia-ischemia.

Methods: We use an in vitro model of hypoxia-ischemia to investigate the mechanism of xenon neuroprotection. Organotypic hippocampal brain slices from mice are subjected to oxygen-glucose deprivation, and injury is quantified by propidium iodide fluorescence.

Results: We show that 50% atm xenon is neuroprotective against hypoxia-ischemia when applied immediately after injury or after a delay of 3 h after injury. To validate our method, we show that neuroprotection by gavestinel is abolished when glycine is added, confirming that NMDA receptor glycine site antagonism underlies gavestinel neuroprotection. We then show that adding glycine abolishes the neuroprotective effect of xenon, consistent with competitive inhibition at the NMDA receptor glycine site mediating xenon neuroprotection.

Conclusions: We show that xenon neuroprotection against hypoxia- ischemia can be reversed by increasing the glycine concentration. This is consistent with competitive inhibition by xenon at the NMDA receptor glycine site, playing a significant role in xenon neuroprotection. This finding may have important implications for xenon's clinical use as an anesthetic and neuroprotectant.

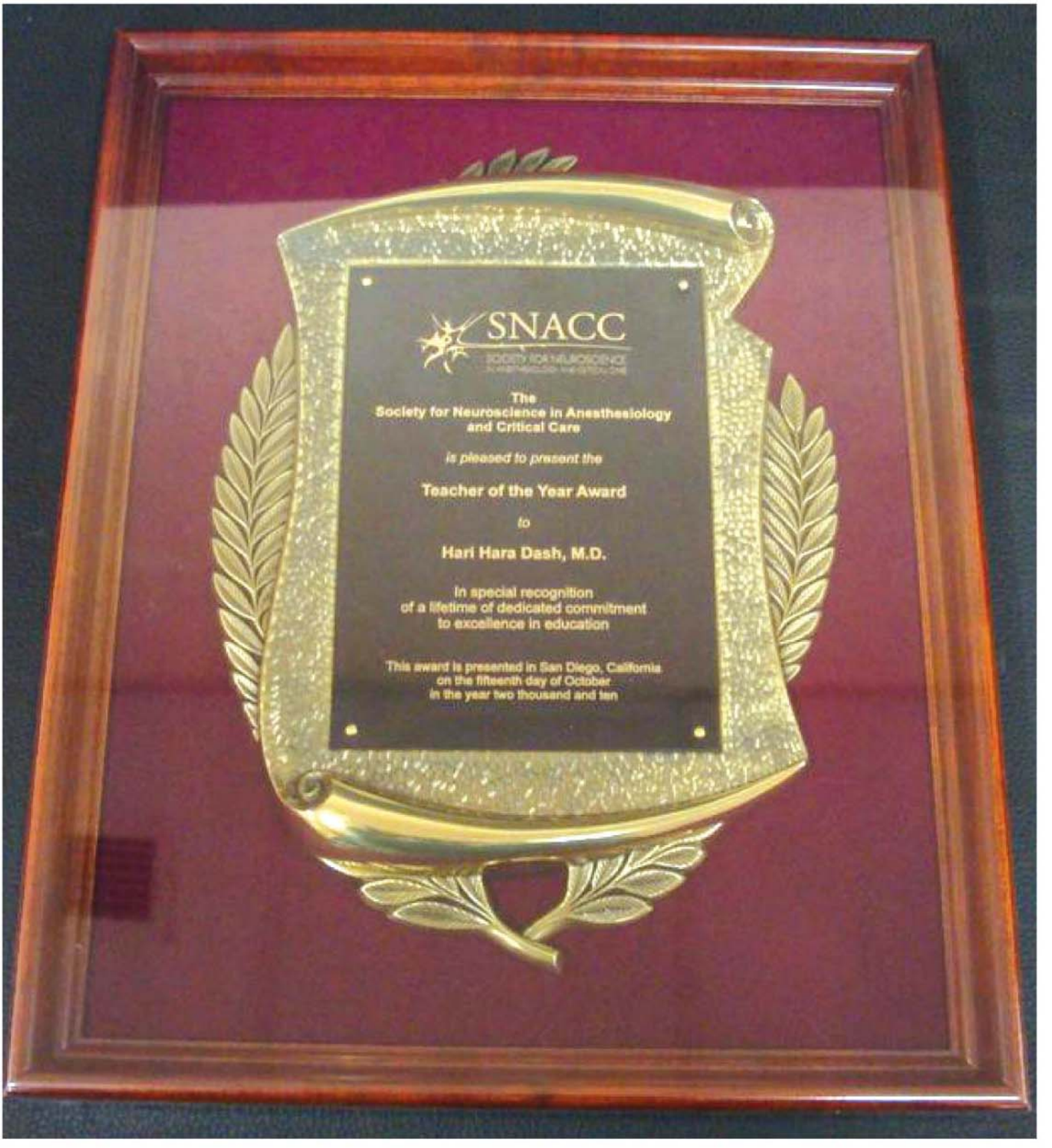
XENON PRECONDITIONING CONFERS NEUROPROTECTION REGARDLESS OF GENDER IN A MOUSE MODEL OF TRANSIENT MIDDLE CEREBRAL ARTERY OCCLUSION

Limatola V, Ward P, Cattano D, Gu J, Giunta F, Maze M, Ma D.
Department of Anaesthetics, Pain Medicine and Intensive Care, Imperial College London, Chelsea and Westminster Hospital, London, UK.

Neuroscience. 2010 Feb 3;165(3):874-81.

Abstract

Xenon preconditioning induces tolerance to the consequences of an injurious stimulus such as cerebral ischaemia. There have been surprisingly few studies investigating gender difference in the efficacy of pharmacological preconditioning, despite the known ability of oestradiol to exert neuroprotectant activity. We explored this paradigm using a mouse model of transient middle cerebral artery occlusion. C57BL/6 mice both male and female received either 2 h of 70% xenon (preconditioning) or 70% nitrogen (control) balanced with oxygen. Twenty-four hours later animals underwent 1 h of middle cerebral artery occlusion and then allowed to recover. After a further 24 h, functional neurological outcome and cerebral infarct size were evaluated. Western blotting was used to detect activity of signalling pathways involving hypoxia-inducible factor (HIF)-1alpha and phospho-Akt for the preconditioning effect. Both xenon preconditioned male and females showed improved functional outcome on focal deficit scales ($P<0.05$). Cerebral infarct volumes were significantly reduced in both xenon treated male and females ($P<0.01$). There was no significant difference between the male and female cohorts. HIF-1alpha and phospho-Akt were quantitatively upregulated in both sexes. Our data suggested that xenon preconditioning improved histological and neurological functional outcome in both gender in a stroke model of mice.



NOTICE

Members / Institutions interested in hosting the 2012 & 2013 Annual Conference of ISNACC may submit their application to Dr. G. Parameshwara, Secretary, ISNACC.

Eligibility Criteria: The member must be a life member of ISNACC for more than one year.

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 2011.

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 2011.

NEWS ITEM

Dr. Nilay Chatterjee passed D.M. Neuroanaesthesia in December 2010 from Sree Chitra Tirunal Institute for Medical Sciences & Technology (SCTIMST)

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