Letter to Editor

Usefulness of head up tilt test in the diagnosis of neurocardiogenic syncope

Sir,

I read with interest the article published by Udani V et al., which is a retrospective and part prospective study, evaluating the usefulness of head up tilt test in the diagnosis of neurocardiogenic syncope. The authors have made a good attempt to analyze the significance of HUTT in ruling out other perturbing causes of syncope in children and adolescents such as seizure disorders and vertigo. But there are many aspects in this study which are unclear. The study being partly prospective, investigators had an opportunity to include a control group. Without an appropriate control group, it is certainly inappropriate to attribute any significance to the positive test obtained in 16 children. It is not clear why the investigators choose to define a positive test as > 30% drop in blood pressure or heart rate and neither is it mentioned whether they were considering systolic or diastolic blood pressure. Moreover, measurement of blood pressure with a standard sphygmomanometer can produce considerable interpersonal subjective variation in the BP values. As many as 11 children showed a positive test on provocation with nitroglycerin. I would like to know the dose of the S/L nitroglycerin used and whether it is adjusted according to the body weight in this age group. Higher doses of nitroglycerin cause further venous pooling and may decrease arteriolar resistance as well, decreasing systolic and diastolic blood pressure and cardiac output and resulting in pallor, weakness and dizziness. This can lead to more number of false positive tests if the dose is not appropriately adjusted. Considering these ambiguities, this study does not provide any definitive evidence of usefulness of HUTT in the diagnosis of neurocardiogenic syncope.

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Nimodipine in severe head injury

Sir,

I read with interest the recent article by Pillai SV et al. Based on an excellent randomized controlled trial, they conclude that nimodipine does not improve the outcome in patients with severe diffuse head injury. However, I would like to make certain comments.

1. **Patient selection:** This trial included patients on the basis of Glasgow Coma Scale (8 or less) and CT scan findings (diffuse head injury with absence of any operable mass lesion). As nimodipine is proposed to act by ameliorating traumatic vasospasm, it would be prudent to perform Trans Cranial Doppler (TCD) studies at admission to identify the subset of patients with evidence of vasospasm. In a recent study, early treatment with nimodipine was found to improve outcome in a group of patients with concussion who had cerebral spasm confirmed by TCD.2

2. **Dose & mode of administration of nimodipine:** Patients in this trial received only one-third of the standard dose (30 mg Q6H as compared to the standard 60 mg Q4H). Could the lack of effect be partly related to inadequate dose? Moreover, after emptying the contents of nimodipine capsule directly into Ryle’s tube, flushing of the tube with 30 ml 0.9% saline is recommended for facilitating delivery of the entire dose. In addition, contents of capsule should not be admixed with any other solution prior to administration due to the risk of decomposition. Failure to observe these precautions could result in incomplete drug delivery.

3. Finally, as Pillai SV et al point out, this study does not have enough power to address the efficacy of nimodipine in traumatic subarachnoid hemorrhage (tSAH). In a recent systematic review of all published RCTs on this subject, nimodipine showed a beneficial effect in the subgroup of brain injury patients with tSAH.4

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References


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References

Role of nimodipine in severe diffuse head injury

Sir,

I read with interest the article published by Pillai S et al., which is a double blind placebo-controlled trial, evaluating the role of nimodipine in severe diffuse head injury. Although pathological increases in intracellular calcium have been implicated as the major final common pathway leading to neuronal death, the mechanism of increase in intracellular calcium in neurons is largely due to excitotoxicity, following hypoxic-ischemic injury or head trauma. After a severe diffuse head injury, the diffuse neural injury that results is caused primarily by the presence of excess glutamate, due to its action on N-methyl-D-aspartate (NMDA) receptors. The activated NMDA receptor-channels allow an influx of Ca^{2+}, which in excess can activate a variety of potentially destructive processes. Nimodipine, because of its high lipid solubility, was developed as an agent to relax cerebral vasculature, and is effective in inhibiting cerebral vasospasm, but does not have any action on NMDA receptors. Hence nimodipine was found to be effective in conditions causing cerebral vasospasm, such as in severe head injury with contusions and intracranial hematomas, where in, its ability to inhibit vasospasm has a significant beneficial effect in reducing neural damage. In this study by Pillai S et al., the patients included showed radiological evidence of only diffuse head injury without any operable mass lesion like intracerebral hematoma or contusion more than 1 cm in diameter, or extradural and acute subdural hematomas more than 1 cm in maximum thickness. It would have been interesting if cerebral vasospasm was demonstrated in these cases with the help of transcranial doppler. This would have provided a better insight into the role of nimodipine in severe diffuse head injury. In such cases with diffuse head injury, excitotoxic injury by glutamate is more likely to be the major cause of neural injury, compared to cerebral vasospasm, as substantiated in this study with no significant improvement in outcome in patients treated with nimodipine, compared to the placebo group. In these patients with diffuse head injury, agents which block NMDA receptors, such as Mg^{2+}, may have a beneficial effect.

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Ventriculo-peritoneal shunt infection by mycobacterium fortuitum in an adult

Sir,

A 60-year-old male patient with unconsciousness after an assault was operated for decompression of fracture, and right-sided ventriculo-peritoneal (VP) shunt was placed for hydrocephalus. He developed fever after a few days. The above procedures were done at another institution. The patient came to us after one-and-a-half months with fever and pneumonia. The pneumonia was treated with intravenous Amoxycillin and Clavulanic acid combination. But the fever persisted in spite of clearing the consolidation in the lungs as evidenced by the X-ray reports. On exploration, there was an abscess in the neck, which was drained. Pus was sent for culture and sensitivity and VP shunt was removed. The patient developed Cerebrospinal fluid (CSF) rhinorrhea 3 days following shunt removal for which bi-frontal craniotomy and fascia lata dura-plasty was done. After 17 days of shunt removal, the abdominal wound was found to be tender, indurated and hence it was debrided. This was followed by left-sided VP shunt insertion another 10 days later.

Provisional diagnosis was pneumonia and infected VP shunt. Investigations done showed: Hemoglobin-12.2 gm%, CBC-9820 (Neutrophils-76%, lymphocytes-24%). Cerebrospinal fluid (CSF) showed proteins-56 gm% and White blood cells (WBC) count of 20 (30% polymorphs, 65% lymphocytes).

Pus sent for culture and sensitivity yielded no growth. But Zielh Neelsen’s stain for pus samples showed acid fast bacilli

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