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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References


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. **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.

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References