President's Message

Dear ASN members,

I thank you for electing me to the prestigious post of President of the Association of Srilankan Neurologists for the year 2009.

Fulfilling the duties that go with this office is no mean task and I hope that with your support we will be able to have a successful programme this year. Neurology has become a very dynamic specialty because of major advances at molecular level. A few years ago, we did not think that Parkinson's disease could have a genetic aetiology. Medications have been developed for epilepsy, Parkinson's disease, multiple sclerosis based on the new knowledge at molecular and physiologic level. Stroke treatment is being revolutionized world-wide with the evidence that show the benefit of thrombolytic therapy and stroke units. Available choice in antiplatelet drugs are increasing and attempt to find more effective treatment for stroke prevention is continuing. Surgical interventions for Parkinson's disease, aneurysms and AVMs have advanced with more effective and safer methods being developed. Carotid endarterectomy and stenting in carotid stenosis and clot retrieval in acute strokes are trying to find their own niche. The best way to treat intracerebral haemorrhage is still not sorted but the current on going trials will settle this issue soon. Still there are many neurological conditions with a hopeless prognosis such as MND, MS, and ataxias. Stem cells may be the answer, and that is the probable future. Therefore continuing medical education and professional development become an integral part of any medical specialty and as in the past years, this year too this will be the main focus. We will continue with the monthly ASN Forum and bimonthly council and general meetings. Case presentations from your own practice would be welcome at the general meeting as this would be an interesting way to learn. Attempts to provide better services to stroke patients should be another main objective of ours while improving the standards of Neurology units in the hospitals. Neurology research at a local level is something that we need to pay special attention and the ASN will encourage and support such efforts. We need to make a mark at international level with good quality research in our country. The ASN annual conference is scheduled to be held on 21 and 22 of November and I request your help in organizing this event. I thank you again for placing your trust in me to head the ASN this year and wish all members the very best.

Professor Saman Gunatilake

ASN EVENTS FOR THE YEAR

- 3RD ANNUAL SCIENTIFIC SESSIONS—NOVEMBER 2009
- NEUROLOGY FORUM—MONTHLY MEETING
- COUNCIL MEETINGS AND CME (CONTINUUM) - BI-MONTHLY
Disorders of Orthostatic Tolerance

Orthostatic intolerance is a well recognized and relatively common clinical problem. Orthostatic intolerance is defined as the development upon standing of symptoms of cerebral hypoperfusion (e.g., lightheadedness, weakness, blurred vision) associated with those of sympathetic activation (such as tachycardia, nausea, tremulousness).

There are three well recognized disorders categorized under the group of orthostatic intolerance:
1. Orthostatic Hypotension
2. Postural Tachycardia Syndrome
3. Vasovagal/Neurally mediated Syncope

Physiology of Blood Pressure Maintenance
In our body the Heart act as the main pump organ which pump blood to the tissues through a resistance side (arteries), and it flows back to the heart from the capacitance side (veins & volume). This process is coordinated by a neural mechanism where the coordinating signals starts in right atrial pressure receptors and affereents go through the vagus nerve to the integrating nuclei in brain stem, i.e. Nucleus Tractus Solitarius Catecholaminergic Brainstem Nuclei. The efferents go via sympathetic nerves to vessels and heart.

Orthostatic Hypotension
Orthostatic hypotension is defined as a drop in systolic pressure of 20 mmHg or a drop in diastolic pressure of 10 mmHg within 3 minutes of standing or in 3 minutes of a tilt study on a table that is at least at a 60 degree angle, accompanied by symptoms of cerebral hypoperfusion.

Clinical features
Cerebral Symptoms of Orthostatic Hypotension
Dizziness, Presyncope and Syncope
Visual disturbance—scotoma, graying out, colour defects
Loss of consciousness
Lightheadedness
Fatigue
Cognitive difficulties (fuzzy head)
Focal neurologic signs

Other Symptoms of Orthostatic Hypotension
Muscle hypo perfusion
Coat hanger ache
Lower backache
Cardiac hypo perfusion
Angina pectoris
Orthostatic dyspnoea inadequate perfusion of ventilated apices
Renal hypo perfusion
Spinal cord hypo perfusion
Weakness (leg buckling)
Falls

Etiology of Orthostatic hypotension
The basic pathogenetic mechanism is degeneration of ANS which can affect
1. Central pathways in brain-multiple system atrophy, parkinson's disease, dementia with Lewy bodies
2. Peripheral autonomic nerves-small fiber neuropathy (DM, Amyloidosis), HSAN 3 4, porphyria, HIV
3. Both Pure autonomic failure

Hallmark of both central and peripheral causes of orthostatic hypotension is failure to increase noradrenaline levels on standing. In patients with pure autonomic failure and small fiber peripheral neuropathy have low resting Nor adrenalin levels due to degeneration of postganglionic neurons. In contrast the patients with Multi system atrophy have normal supine noradrenaline levels. But both groups have failure to increase noradrenaline levels on standing.

Investigations
Autonomic testing
1. Response to deep breathing (sinus arrhythmia) mainly tests cardiac parasympathetic function. It is abnormal early in diabetes.

Cardiac Response to Deep Breathing

Cardiac Response to the Valsalva Maneuver
2. Valsalva maneuver test the cardiac sympathetic, parasympathetic and vasomotor sympathetic function
(continued on page 3)
3. Tilt table test: Differentiates venomotor from arteriomotor sympathetic
   
   **Indications of Tilt table testing**
   
   Recurrent syncope
   Single episode of syncope accompanied by injury or occurring in a high risk setting
   Evaluation of unexplained recurrent falls
   Evaluation of primary or secondary autonomic failure
   Relative contraindications
   Syncope with severe left ventricular outflow obstruction
   Syncope in the presence of critical mitral stenosis
   Syncope in the setting of known critical proximal coronary artery stenosis
   Syncope in conjunction with known critical cerebrovascular stenoses

   **Tilt Table: Orthostatic Hypotension**

   ![Image](attachment://image.png)

   After head tilt the blood pressure steadily drops but due to autonomic failure there is no compensatory increase in HR.

4. Thermoregulatory sweat test Apply a quinsarin dye to the whole body (except face) and ask the patient to wait in hot environment for 1 hour. The amount of sweating can be judged by the degree of colour change from brown to purple. Therefore depending on the areas involved we can decide on central, peripheral or total autonomic dysfunction.

---

**Thermoregulatory Sweat Test**

![Image](attachment://image.png)

**Treatment**

**Physical Conditioning exercises**

- Water drinking (16 oz in am; Shannon, 2002)
- Water jogging
- Self-tilt exercises (Ector, 1998)

**Self-tilt:**

- Stand with back against the wall

---

Dr Darshana Wijegoonesignhe
Consultant Neurologist
Neurology Update—Antiplatelet Therapy for Stroke

Antithrombotic Treatment Associated With Cerebral Microbleeds

**Background** - Cerebral microbleeds are hemosiderin deposits in the brain that are indicative of microangiopathy. Microbleeds in strictly lobar brain locations have been related to cerebrovascular angiopathy, a bleeding-prone disease state.

**Objective** - To investigate the relation between antithrombotic drug use and the presence of cerebral microbleeds, especially those in strictly lobar locations.

**Design** - A population-based, cross-sectional analysis that used magnetic resonance imaging (MRI) to assess the presence and location of microbleeds. Complete information on outpatient use of platelet aggregation inhibitors and anticoagulant drugs before MRI was obtained from automated pharmacy records.

**Setting** - The Rotterdam Scan Study, a population-based imaging study in a general elderly community in the Netherlands.

**Participants** - A population-based sample of 1062 persons from a longitudinal cohort, 60 years and older, free of dementia, who underwent MRI examinations between August 15, 2005, and November 22, 2006.

**Main Outcome Measures** - Presence of cerebral microbleeds on MRI.

**Results** - Compared with nonusers of antithrombotic drugs, cerebral microbleeds were more prevalent among users of platelet aggregation inhibitors (adjusted odds ratio [OR], 1.71; 95% confidence interval [CI], 1.21-2.41). We did not find a significant association for anticoagulant drugs and microbleed prevalence (OR, 1.49; 95% CI, 0.82-2.71). Strictly lobar microbleeds were more prevalent among aspirin users (adjusted OR compared with nonusers, 2.70; 95% CI, 1.45-5.04) than among persons using carbasalate calcium (adjusted OR, 1.16; 95% CI, 0.66-2.02). This difference was even more pronounced when comparing persons who had used similar dosages of both drugs.

**Conclusions** - This cross-sectional study shows that use of platelet aggregation inhibitors is related to the presence of cerebral microbleeds. Furthermore, aspirin and carbasalate calcium use may differently relate to the presence of strictly lobar microbleeds.

(Published at www.nejm.org March 31, 2009)

Effect of Enteric Coating on Antiplatelet Activity of Low-Dose Aspirin in Healthy Volunteers

**Background and Purpose** - Aspirin resistance may be relatively common and associated with adverse outcome. Meta-analysis has clearly shown that 75 mg plain aspirin is the lowest effective dose; however, it is not known whether the recent increased use of enteric-coated aspirin could account for aspirin resistance. This study was designed to determine whether enteric-coated aspirin is as effective as plain aspirin in healthy volunteers.

**Methods** - Seventy-one healthy volunteers were enrolled in 3 separate bioequivalence studies. Using a crossover design, each volunteer took 2 different aspirin preparations. Five aspirin preparations were evaluated, 3 different enteric-coated 75-mg aspirins, dispersible aspirin 75 mg and aspirin (25-mg standard release aspirin plus 200-mg modified-release dipyridamole given twice daily). Serum thromboxane (TX) B2 levels and arachidonic acid–induced platelet aggregation were measured before and after 14 days of treatment.

**Results** - All other aspirin preparations tested were inferior to dispersible aspirin (P < 0.001) in their effect on serum TXB2 level. Treatment failure (95% inhibition serum TXB2 formation) occurred in 14 subjects, none of whom were taking dispersible aspirin. Mean weight for those demonstrating treatment failure was greater than those with complete TXB2 (>95%) inhibition (P < 0.001). Using logistic regression analysis an 80-kg subject had a 20% probability of treatment failure. Aspirin was the most potent preparation in terms of inhibition of platelet aggregation.

**Conclusions** - Equivalent doses of the enteric-coated aspirin were not as effective as plain aspirin. Lower bioavailability of these preparations and poor absorption from the higher pH environment of the small intestine may result in inadequate platelet inhibition, particularly in heavier subjects.

Compiled by:

Dr Harsha Gunasekara
Consultant Neurologist
Neurology Update—Movement Disorders

Heavy Drinking Doubles Risk for Essential Tremor Later in Life

BACKGROUND: Recent postmortem studies have demonstrated pathological changes, including Purkinje cell loss, model adjusting for cigarette pack-years, depressive symptoms and community, the baseline number of drink-years was marginally associated with in the cerebellum in essential tremor (ET). Toxic exposures that compromise cerebellar tissue could lower the threshold for developing ET. Ethanol is a well-established cerebellar toxin, resulting in Purkinje cell loss.

OBJECTIVE: To test whether higher baseline ethanol consumption is a risk factor for the subsequent development of incident ET. METHODS: Lifetime ethanol consumption was assessed at baseline (1994-1995) in a prospective, population-based study in central Spain of 3285 elderly participants, 76 of whom developed incident ET by follow-up (1997-1998).

RESULTS: In a Cox proportional hazards a higher risk of incident ET (relative risk, RR = 1.003, p = 0.059). In an adjusted Cox model, the highest baseline drink-year quartile doubled the risk of incident ET (RR = 2.25, p = 0.018), while other quartiles were associated with more modest elevations in risk (RR: 3rd quartile) = 1.82 (p = 0.10), RR (2nd quartile) = 1.75 (p = 0.10), RR (1st quartile) = 1.43 (p = 0.34) vs non-drinkers (RR = 1.00). With each higher drink-year quartile, the risk of incident ET increased an average of 23% (p = 0.01, test for trend).

CONCLUSIONS: Higher levels of chronic ethanol consumption increased the risk of developing ET. Ethanol is often used for symptomatic relief; studies should explore whether higher consumption levels are a continued source of underlying cerebellar neurotoxicity in patients who already manifest this disease.

(J Neurol Neurosurg Psychiatry. 2009;80:423-425.)

Deep Brain Stimulation for Dystonia Effective Long Term

Background Pallidal deep brain stimulation (DBS) is the best therapeutic option for patients with disabling primary generalized dystonia (PGD) that is refractory to medications. However, little is known about its long-term effects.

Objective To describe long-term clinical outcomes in patients with PGD who underwent pallidal DBS.

Patients Thirty consecutive patients with at least 2 years follow-up after pallidal DBS for intractable PGD.

Interventions Pallidal DBS and annual follow-up examinations up to 8 years after DBS implantation.

Main Outcome Measures Clinical outcome as measured by changes in the Burke-Fahn-Marsden dystonia scale, incidence and prevalence of adverse events, total electrical energy delivered, and implantable pulse generator longevity.

Results Twenty-three patients were followed for 3 years, 13 for 4 years, 9 for 5 years, 5 for 6 years, 5 for 7 years, and 1 for 8 years after DBS. Overall improvement at 1 year was maintained in all at successive yearly examinations. There were no intraoperative complications; hardware-related adverse events were infrequent. Rare stimulation-related adverse events primarily affected speech. Implantable pulse generators were replaced every 24 months on average in patients who received initial stimulation at 120-Hz frequency. No battery was replaced, for up to 48 months, in 20 patients initially stimulated using 60-Hz. Clinical outcome did not depend on high energies of stimulation.

Conclusions Pallidal DBS is a safe and effective treatment for PGD, with improvement sustained for up to 8 years in 1 patient. Low energies of stimulation, although they did not affect clinical outcome, were associated with longer battery life.

(Arch Neurol. 2009;66:502-508.)

Compiled by,
Dr Nilupul Perera
Consultant Neurologist
Q1. The following are the Spinal MRI images of a 28 year old female presenting with paresthesiae of both lower limbs starting 8 days back but had improved over this period. She describes a similar episode 2 years ago. Neurological examination was normal and her sensory symptoms were limited to the feet alone at examination. A tell-tale tuft of hair over the lumbar spine suggested a spina bifida occulta. What is/are the likely diagnosis/diagnoses?

Q2. These are the MRI Brain images from a 65 year old man with a history of epilepsy who was found collapsed at home. He was drowsy and confused since admission, but slow improvement of symptoms were noted. The EEG showed diffuse theta wave activity. What is the likely diagnosis?
In Parkinsonism

**Tidomet Forte**
Carbidopa 25mg + Levodopa 250mg Tablet

Restores normal function rapidly

In schizophrenia

**Arip MT**
Aripiprazole 10/15 mg Melt in mouth tab.

The first dopamine system stabilizer

**Valparin CHRONO**
Sodium valproate 200/333mg + Valproic acid 87/145mg tablet

Complete seizure control, Proven safety

**Deplatt**
Clopidogrel 75 mg tablet

The dependable antiplatelet agent