NGF concentrations in human cerebral arteries

Artery	Number	NGF (ng/g) Mean (SEM)
Anterior cerebral (AC)	7	0.82 (0.19)
Internal carotid (IC)	7	0.78 (0.16)
Posterior cerebral (PC)	7	2.02 (0.53)
Superior cerebellar (SC)	7	2.14 (0.45)
Posterior communicating	6	1.87 (0.58)

P values were determined by student's *t* test.

AC *v* SC *P* < 0.02; IC *v* PC *P* < 0.04; IC *v* SC *P* < 0.015.

Mean NGF concentration *v* age of subject *r* = -0.8, *P* < 0.034.

ache, as in temporal arteritis, and also reduce NGF synthesis, which is increased by inflammation.¹ Modulation of NGF activity may thus provide a new approach to prevent and treat vascular headaches.

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Triphasic waves in serotonin syndrome

The serotonin syndrome was first described in 1960 in depressed patients with delirium due to monoamine oxidase inhibitors and L-tryptophan administration.¹ Symptoms of the serotonin syndrome include mental status changes, behavioural changes, myoclonus, rigidity, hyperreflexia, and autonomic instability with low grade fevers, diarrhoea, headache, tachycardia, and pupillary dilatation.² The serotonin syndrome has been noted to occur with several serotonimetic agents, particularly when multiple agents are used.³

Psychiatry and pharmacology literature has described the serotonin syndrome for several years. As the use of serotonin reuptake inhibitors has increased, cases have begun to appear in the neurology literature—often associated with combination regimens that include serotonin reuptake inhibitors and dopaminergic agents. These cases have been attributed to the serotonimetic effects of dopamine and its agonists. I describe a patient admitted for acute confusion who met criteria for the serotonin syndrome, responded well to supportive care, and whose EEG showed prominent triphasic wave activity.

A 76 year old man had a history of Parkinson's disease, recurrent depression,

chronic constipation, and non-insulin dependent diabetes mellitus. He had right sided tremors, bradykinesia, hypophonia, and significant gait instability with occasional visual hallucinations. Due to his depression and concerns regarding the use of tricyclic antidepressants in a patient already at risk for autonomic dysfunction, he was started on 50 mg sertraline at bedtime. He initially responded well, experiencing no notable side effects. About three days before admission, amantidine was added to his drug regimen which already included sertraline and Sinemet. The patient was brought to the emergency department by his wife due to increasing confusion, diarrhoea, and frequent falls that had begun a day earlier.

On examination, the patient had a low grade fever, extreme rigidity in all limbs, agitation, confusion, and ongoing visual hallucinations. Over the next four hours he developed multifocal and startle myoclonus. He had not received any neuroleptic or antibiotic drugs in more than six months.

Electrolytes, creatine kinase, liver functions, a complete blood count, and ammonia concentration were all normal. Blood cultures and urinalysis were also unremarkable. A 16 channel EEG was obtained and showed pronounced triphasic wave activity and diffuse slowing. Supportive care with intravenous fluids and acetaminophen were initiated and all outpatient medications were stopped. Within 24 hours the patient's myoclonus began to subside and in 48 hours he had returned to baseline without any sequelae. He continues to do well on Sinemet alone for his Parkinson's disease.

Case reports of the serotonin syndrome have noted EEG abnormalities—delta range activity, slow waves, spike and waves, and polyspike and waves—but triphasic waves have not previously been reported.^{4,5} The diagnosis of the serotonin syndrome in the Parkinson's disease population is a difficult one as many of the features of the serotonin syndrome are present in Parkinson's disease alone. A high level of suspicion for the serotonin syndrome in patients with Parkinson's disease taking serotonin reuptake inhibitors is necessary to make the diagnosis. Electroencephalography may play an important part in the diagnosis of the serotonin syndrome, particularly in the setting of other concurrent neurological disease.

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Pseudoseizures or non-epileptic seizures (NES); 15 synonyms

Medical jargon is often confusing, particularly when the condition described falls within the domain of two medical specialties. This confusion reaches its zenith with those seizure disorders that do not have an epileptic aetiology. There are at least 15 synonyms for a condition that occurs in 10% to 26% of adults investigated for refractory seizures. This causes confusion for patients, doctors, and researchers. The adoption of a common term must be the rational way forward, but which one to choose?

The label pseudoseizures is the most commonly used. Its great weakness is that it is not acceptable to patients as the label implies that the seizures are not real. The reality of the 'fit' is seldom an issue. The label pseudoepileptic seizures is both less well known and pejorative. Labels that are offensive to patients are counterproductive and best avoided.

The aetiology of this disorder is currently a matter for speculation. Terms that imply a psychological causation are misleading. Psychogenic seizures, hysterical seizures, psychogenic attacks, and hysterical attacks are all inappropriate for this reason.

A good descriptive label is non-epileptic attacks but this is seldom used. Non-epileptic attack disorder (NEAD) is rarely used and is complicated. Functional seizures, hysteroepilepsy, pseudoepilepsy, hysterical epilepsy, pseudoepileptic attacks, and psychoseizures are the least commonly used terms. These labels should all be abandoned.

This leaves the term *non-epileptic seizures* (NES) as the favoured candidate; it is non-judgmental, often used, acceptable to patients, and best describes the problem without implying causation.

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Multiple sclerosis: longitudinal measurement of interleukin-1 receptor antagonist

Inflammatory activity in multiple sclerosis is regulated by a network of proinflammatory and antiinflammatory cytokines. Identifying downregulatory cytokines opens new potential therapeutic options in multiple sclerosis.^{1,2} The interleukin-1 receptor antagonist (IL-1ra) is the only known naturally occurring specific antagonistic cytokine; IL-1ra competes with IL-1 for receptor binding and lacks agonist activity. IL-1ra has been implicated in the pathogenesis of stroke and several inflammatory diseases.³ Human IL-1ra is available as a recombinant protein; the first controlled study using IL-1ra for therapy (in