



sodium valproate, clonazepam and zopiclone. The patient had also undergone two courses of electroconvulsive therapy in the past, as well as various psychotherapies and occupational therapy. None of these was particularly successful.

At the PAH a complete physical work-up showed no evidence of lesions or gross abnormalities; blood pressure, blood chemistry, inter-ictal and ictal scalp EEG studies, and spiral computed tomography (CT) were normal. The patient's Mini Mental State Examination score was 29 out of 30, and her Dissociative Experiences Scale score 54. The psychiatric *Diagnostic and Statistical Manual IV (DSM-IV)* diagnosis was: Axis I — conversion disorder and major depressive disorder (recurrent, severe); and Axis IV — marital conflict. An inter-ictal hexamethylpropyleneamine oxime (^{99m}Tc -HMPAO) perfusion study of the brain was done, and areas of mildly reduced perfusion were registered in the right infero-frontal and medio-temporal regions. Since calcium ions play a pivotal role in neuronal function, and disruption of calcium ion homeostasis results in neuronal dysfunction, the patient was started on 40 mg verapamil per day, increased to 80 mg after 2 weeks. The results were dramatic, with no attacks during the following 6 weeks of medication.¹⁻⁴

An on-therapy follow-up SPECT (single photon-emission computed tomography) perfusion study showed some improvement in perfusion in the infero-frontal region.

Two superfamilies of voltage-gated Ca^{2+} channels have been distinguished, the one high-voltage-activated, and the other low-voltage-activated. In the nervous system voltage-gated channels are key regulators of neuronal excitability and neurosecretions. Derangement of the channel system may lead to excess influx of calcium ions, and excess calcium ions are neurotoxic.^{5,6}

Conversion disorder may present at any age, and is more common in women than men (6:1). Symptoms are caused by previous severe stress, emotional conflict, or an associated psychiatric disorder. Many studies have confirmed a high incidence of depressive disorders in patients with conversion disorder.¹

The input of EEG, CT and other screening modalities in the diagnosis of conversion disorder is nonspecific or unclear. Treatment includes behavioural therapy, clinical hypnosis, group psychotherapy, anticonvulsants, antidepressants, neuroleptics and anxiolytic agents. Organic calcium channel blockers are not included in the psychopharmacotherapies usually employed.

Symptomatology of the attacks, the brain lesion identified by radioscinigraphy and the positive response to an organic calcium blocker in this patient suggest epileptic seizures, while the clinical history, the results of the physical examination, EEG, spiral CT and the psychiatric assessment favour a diagnosis of conversion disorder. The patient's excellent

CONVERSION DISORDER AND CALCIUM HOMEOSTASIS

To the Editor. A 45-year-old right-handed, married woman with three grown-up children was referred to the Department of Psychiatry at the Pretoria Academic Hospital (PAH). Early history included a traumatic childhood. Her psychiatric history included 8 years of recurrent major depressive disorder. She was referred because of attacks of absence over the past year — in her own words 'I simply cut out'. These attacks were characterised by staring into space, coarse jerky muscle movements in her right upper limb and sometimes her right lower limb, but without localising neurological signs. The attacks lasted 5–45 minutes and had increased in frequency. Clinical examination revealed no physical disorder. Blood chemistry as well as repeated electroencephalogram (EEG) studies were normal. Previous medication included fluoxetine, venlafaxine, risperidone, lithium carbonate, lamotrigine,



therapeutic response to an organic calcium channel blocker suggests that disruption of neuronal Ca^{2+} homeostasis is involved in the attacks. This poses some interesting questions. For example, have systematic radiosciintigraphic studies been done on conversion syndrome patients and in patients who have received electroconvulsive therapy to rule out therapeutic-elicited brain damage? Possible causes underlying periodic disruption of calcium homeostasis included *inter alia* primary channelopathy and channel dysfunction secondary to intermittent focal vasospasm.⁷⁻¹⁰

Edna K Venter
Greta Lange
Christa Krüger
Bernard J Meyer

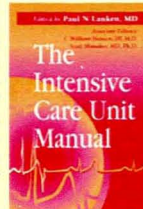
*Departments of Nuclear Medicine and Psychiatry
 Pretoria Academic Hospital*

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