Comparison of stroke survivors with and without emotionalism, assessed in hospital 1 month after stroke

	No emotionalism (n=45) Mean (SD)	*Emotionalism (n=19) Mean (SD)*
GHQ- 12*	3.2 (2.4)	5.3 (3.5)
Recovery locus of control scale	33.2 (3.3)	34.2 (3.7)
Impact of events scale intrusion subscale**	2.9 (4.6)	9.2 (6.6)
Impact of events scale avoidance subscale*	4.7 (4.6)	9.9 (6.1)
MASS Fighting spirit subscale	49.1 (4.0)	48.8 (5.2)
MASS Anxious preoccupation subscale**	22.2 (2.8)	25.2 (4.0)
MASS Fatalism subscale*	20.0 (1.9)	21.3 (2.2)
MASS Avoidance subscale	1.7 (0.8)	1.9 (0.8)
MASS Helplessness/hopelessness subscale**	10.9 (2.5)	14.1 (3.5)

MASS = Mental adjustment to stroke scale.
*p<0.05; **p<0.01, *t* tests.

(*F*=15.33, p<0.001), and avoidance (*F*=11.84, p=0.001); the mental adjustment to stroke scale subscales helplessness/hopelessness (*F*=11.7 1, p=0.00 1) and anxious preoccupation (*F*=8.05, p=0.006). The associations with fatalism (*F*=14.79, p=0.052) and avoidance (*F*=0.06, p=0.80) on the mental adjustment to stroke scale were no longer significant after adjustment for GHQ-12 score.

This study confirms earlier work by showing that stroke survivors with emotionalism have more other mood symptoms (here rated by the GHQ-12) than do those without emotionalism. It goes further however, in showing that they also have intrusive thoughts about their stroke, of a sort similar to those reported by people with post-traumatic stress disorder. This unpleasant remembering is probably responsible for their higher ratings on anxious preoccupation. It is compatible with our finding in a previous study[2] that irritability is associated with emotionalism, as irritability is a common response to threatening intrusive memories of the sort encountered in post-traumatic stress disorder. It may not be that emotionalism is a direct manifestation of post-traumatic stress disorder, although that condition has been described after stroke,[6] but the analogy raises the possibility that an abnormality in processing emotionally important stimuli may be one of the causes of emotionalism. If correct it suggests possible treatment strategies along the lines of those used in post-traumatic stress disorder.

A corollary is our finding of increased feelings of helplessness and hopelessness, coupled with avoidance—at least as a cognitive coping strategy reported on one of our measures. Avoidant coping may perpetuate the symptom of emotionalism, by preventing habituation to the social stimuli which provoke it. Alternatively it may lead to a reduction in social support, exacerbating coexistent mood disturbance. Thus, it may be that avoidant coping is not an integral part of emotionalism, but rather that it is an important maintaining factor.

We predicted that patients with emotionalism would have more "external" scores on the locus of control measure, reflecting their sense of lack of personal control over crying. They did not, perhaps because the emotional expression, although not apparently controllable by internal resources, is none the less perceived as having psychological meaning, so that responsibility for it it cannot readily be devolved to others.

Our study used a relatively weak between-groups design, the number of patients was not large, and we cannot be sure that all confounders were dealt with. None the less, our results suggest that future research into emotionalism could profitably concentrate not just on seeking its biological correlates, but should also explore the psychological factors which might contribute to its cause or continuation.

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Paraneoplastic stiff limb syndrome

Stiff man syndrome (SMS) is a rare, severe progressive motor disorder characterised by painful spasms, symmetric axial muscle rigidity, and uncontrollable contractions leading to distorted posturing. The disorder has been associated with the autoantigens, glutamic acid decarboxylase (GAD), and amphiphysin, which are cytoplasmic proteins in neurons of the CNS. A large series of patients with SMS found that most have autoantibodies against GAD,[1] whereas amphiphysin is presumably the predominant autoantigen in paraneoplastic SMS.[2] Recently, Brown *et al*[3] presented four patients with a stiff leg syndrome marked by progressive rigidity and spasms of the lower extremities. This group of patients tested negative for anti-GAD antibody by immunoprecipitation and demonstrated distinct electrophysiological features. By contrast, another report described two patients with stiff leg syndrome who tested positive for anti-GAD antibody.[4] Finally, in presenting a group of 13 patients, Barker *et al*[5] proposed that the nomenclature "stiff limb syndrome" refers to the focal form of SMS when one or more distal limbs are involved; two of their patients were also anti-GAD antibody positive, but none were tested for antibodies to amphiphysin or identified as having an underlying neoplasm. We present a patient clinically consistent with the stiff limb syndrome who was found to have autoantibody to GAD and breast cancer.

A 68 year old woman presented with a 1 month history of painful spasms in her legs. Cramps were associated with tactile stimuli and emotional upset. Within weeks, inversion began at the left and then right ankle, making ambulation difficult. Her medical history was significant for Graves' disease treated with thyroidectomy and radiation therapy, and hyperlipidaemia. She was a chronic smoker. General examination was noteworthy for lymphadenopathy in the right axilla. Her mental status was worse during periods of lower extremity spasms, during which she became anxious, diaphoretic, and tachycardic. Cranial nerve and motor evaluations were unremarkable, but assessment of the left leg, due to painful spasms elicited by light touch, was difficult. Inversion and plantarflexion were essentially fixed at the left ankle but could be overcome on the right. Deep tendon reflexes were 3+ in the upper and lower extermities, with sustained clonus at the right ankle. Sensory examination, with the exception of hyperaesthesia in the distal lower extremities, and coordination testing were grossly normal. No hyperlordosis or myoclonus was noted. Gait was limited due to ankle posturing.

The laboratory evaluation was noteworthy for a CSF with increased IgG indices (2.5, 3.4; normal, 0.2–0.8) and oligoclonal bands (5, 5) but no pleocytosis. Serological testing for anti-Hu, anti-Yo, and anti-Ri antibodies was unremarkable, and the haemoglobin A1C was 6.6 (5.6–7.7)%. Skin biopsy at three sites on the patient's leg showed diminished epidermal nerve fibre density and terminal axonal swelling distally, consistent with a small fibre sensory neuropathy.[6] The patient would not tolerate EMG. Magnetic resonance images of the brain and the entire spinal cord were normal. Fine needle aspiration of a soft tissue right axillary mass showed metastatic adenocarcinoma. On an open surgical procedure, infiltrating duct carcinoma of the breast was identified. Anti-GAD autoantibodies were positive by immunocytochemical assay and immunoprecipitation, but antibodies to amphiphysin were not detected by immunocytochemistry, immunoprecipitation, or western blotting (Dr P De Camilli, Yale University).

Ongoing therapy with clonazepam and a trial of oral dexamethasone did not improve the lower extremity symptoms. The patient's ankle posturing continued a slow progression to marked inversion, with spontaneous extension of hallucis longus. The patient died 18 months after symptom onset. Gross necropsy attributed the cause of death to aspiration pneumonia. Neuropathological evaluation showed a grossly normal brain and spinal cord. Microscopically, the lumbar cord had mild reactive gliosis in the anterior horns but no evidence of inflammation. Sections of the frontal cortex, pons, and medulla showed mild diffuse reactive astrocytosis.

Stiff man syndrome is increasingly recognised as a heterogeneous disorder.[7] Other case reports have documented patients with "focal" disease involving either lower[3–5] or upper extremity posturing,[8] which contrast

with the "diffuse" axial and subsequent proximal muscle distribution of the classic disorder.[9] Our patient differs from those reported with stiff leg syndrome in that an occult malignancy was present. Unfortunately, we were unable to obtain electrophysiological studies for comparison. The search for a paraneoplastic process was based on the findings of axillary lymphadenopathy and an abnormal CSF. Our patient is only the second reported patient with paraneoplastic SMS associated with anti-GAD antibody; the other had upper limb rigidity in the setting of breast cancer and additionally mounted an immune response to amphiphysin.[8]

Paraneoplastic processes can affect any component of the nervous system and, occasionally, multiple levels, as in the syndrome of sensory neuronopathy-encephalomyelitis. Our patient's findings were not entirely consistent with criteria for classic SMS[9] in that an apparent encephalopathy and a small fibre neuropathy were identified—for example, her dysautonomia (tachycardia and relative hypertension) during spasms may have been a manifestation of involvement of small fibres. The role of autoantibodies in the pathogenesis of SMS and cancer is unclear.[2 7 8] Via its probable function in endocytosis,[10] amphiphysin has been postulated to play a part in the regulation of growth factor internalisation; however, the absence of an autoimmune response to this autoantigen in our patient suggests that other mechanisms of oncogenesis in SMS exist. Given anecdotal evidence of improvement in paraneoplastic SMS after treating the underlying malignancy,[8] we suggest that all patients with SMS, diffuse or focal, be screened for occult cancer.

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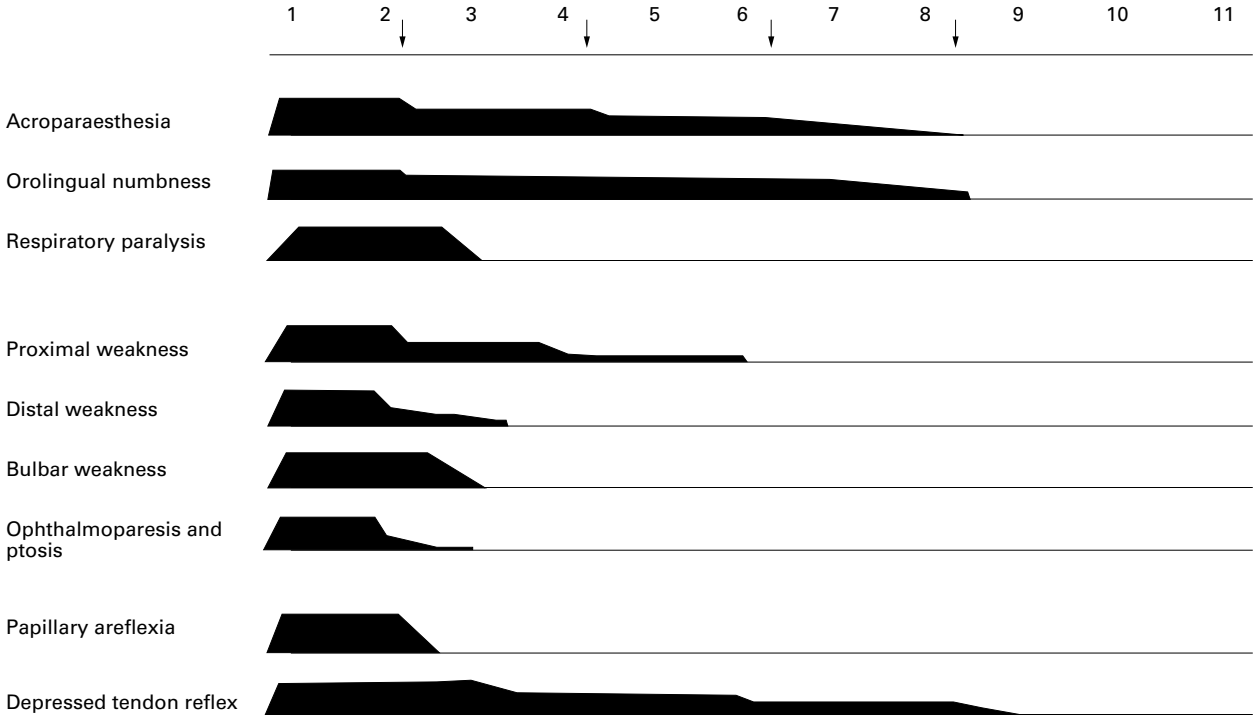
Tetrodotoxin intoxication in a uraemic patient

Tetrodotoxin intoxication results from ingesting puffer fish or other animals containing the toxin. Clinical presentation is mainly acute motor weakness and respiratory paralysis. Death is common in the worst affected victims. Although the severity of the symptoms generally depends on the amount of toxin ingested, it may be influenced by the victim's medical condition, as described in this report. The patient was a 52 year old uraemic woman. The uraemia was of undefined aetiology. Over the past 3 years she has received regular haemodialysis. One day both she and her husband, a healthy 55 year old man, ate a fish soup. About 4 hours after the meal she developed a headache and a lingual and circumoral tingling sensation and numbness at the distal parts of all four limbs. She was dizzy and unsteady, had difficulty in swallowing, and became very weak. She was taken to the emergency service and was placed on machine assisted ventilation as respiratory distress and cyanosis developed. Her husband remained asymptomatic throughout this time.

The patient's condition kept on deteriorating, developing eventually into a comatous-like state with no spontaneous or reflexive eye opening or limb movement within 30 minutes of intubation. On neurological examination, the pupillary light reflex was absent and oculocephalic manoeuvre elicited no ocular movements. All four limbs were areflexic and Babinski's signs were absent. Brain CT and laboratory studies of arterial blood gas (under assisted ventilation), electrolytes, liver function, blood glucose, and CSF study were unremarkable. An examination of renal function indicated chronic renal insufficiency with mild azotaemia (urea nitrogen 70 mg/dl, creatinine 9.1 mg/dl). An EEG, recorded 18 hours after the onset of symptoms when the neurological condition was unchanged, showed posterior dominant alpha waves intermixing with trains of short duration, diffuse theta waves. When brief noxious stimuli were applied to the sternum, they were replaced transiently by beta activities. The findings suggested that the profound neurological dysfunction might be peripheral in origin. The patient was given a course of haemodialysis according to the set schedule for uraemia at 21 hours after onset of the symptoms. Her condition improved dramati-



Changes in the symptoms of poisoning in relation to each course of haemodialysis. Scales in the vertical axis represent the arbitrary measurements of severity of each symptom; the numbers indicating day(s) after onset; ↓= haemodialysis).