ligated to the pMalc vector. Plasmids were transformed into Escherichia coli DH5. Production of the recombinant fusion protein was induced with isopropyl-β-D-thiogalactopyranoside. The DH5 cells were sonicated and centrifuged, after which the supernatant was applied to an amyllose resin column and eluted with maltose to separate the Yo protein fused to the maltose binding protein (figure). For ELISA, the r-Yo was bound to immunoplates (0-5 μg/well). Non-specific binding was blocked with 1% bovine serum albumin (TBS) containing 0-5% skimmed milk. The patient’s serum then was diluted 1:5000 with the blocking solution and immunostained with avidin-biotin-peroxidase. Absorbance at 495 nm was measured with a spectrophotometer. The normal and disease control samples consisted of serum samples taken from 10 healthy young adults, from 10 patients with spinocerebellar degeneration (mean age 56 (SD 11)), and from seven patients with gynaecological cancer without neurological symptoms. The mean ELISA absorbance of serum samples from the young adult controls was 0-080 (SD 0-023) and that from the disease controls, 0-082 (SD 0-046). The absorbance for serum from the patient with PCD before treatment was 1-200. After three cycles of plasmapheresis, it rose to 1-764. On the basis of her clinical picture and the presence of the characteristic anti-Yo antibody, we did a test laparotomy with her informed consent because the conventional survey had found no tumour. A fallopian tube adenocarcinoma 1-5 cm in diameter was identified and successfully resected. Cancer chemotherapy was started 18 days after operation (cisplatin, adriamycin, cyclophosphamide, and 5-fluorouracil). Two more chemotherapy cycles were given at monthly intervals. The antibody titre of serum taken serially at various times during treatment and during the three year follow up period showed a gradual reduction to 0-401. Patients with PCD who have anti-Yo antibodies have been found to have mostly breast or gynaecological cancers. More than half of these patients with PCD showed neurological symptoms only for several months, no underlying cancer being detected despite extensive surveys for malignancy. The detection of antineuronal antibodies, therefore, is important for the early detection of the underlying cancer. These autoantibodies can be detected from the staining distribution obtained by immunohistochemical means as well as by their molecular sizes on immunoblots. But, as there are many molecules of similar size, we constructed r-Yo to use as the ELISA antigen to group patients with PCD with an autoantibody that recognises the same molecule.

In ELISA, our patient’s serum and CSF both had a high titre for the anti-Yo antibody. This titre increased two weeks after plasmapheresis, indicative of rebound overproduction of the antibody. After tumour resection and subsequent anticancer chemotherapy, the patient’s anti-Yo antibody titre gradually decreased. Her neurological symptoms showed mild improvement, suggesting that early tumour resection ameliorates neurological symptoms. Increases in antibody titre indicate the immunological state of the host, indirect evidence of tumour antigen stimulation. Therefore it is very important to follow up changes in specific antibodies in patients with PCD.

**Letters to the Editor**

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**Almost continuous epileptic activity apparent in the right frontal-frontal sagittal region during drowsiness.**

**Painful paroxysmal dystonia associated with focal epileptic activity**

Painful paroxysmal dystonia, a rare movement disorder, is characterised by recurrent episodes of brief, intensely painful unilateral abnormal posturing of one or two limbs unassociated with clonic movements or impairment of consciousness. The mechanism remains obscure, and most cases with identifiable causes have subcortical lesions, particularly in the brain stem, as in multiple sclerosis. Our patient had typical painful paroxysmal dystonia associated with cortical epileptic activity in the EEG.

A 72 year old woman was admitted for the evaluation of recurrent painful dystonic posturing of her left extremities. These attacks were characterised by sudden unprovoked torsion of the head forward, adduction of the left arm at the shoulder with flexion at the elbow and at the wrist and extension of the fingers, extension of the left knee, and plantar flexion of the left ankle. She had a 18 month history of progressive bilateral leg weakness. Her medical history included hypertension, coronary artery disease, and herpes zoster. She had undergone a cholecystectomy for cholelithiasis.

On admission she was conscious and orientated. She complained of severe pain and weakness of both lower extremities. She had a 18 month history of progressive bilateral leg weakness. Her medical history included hypertension, coronary artery disease, and herpes zoster. She had undergone a cholecystectomy for cholelithiasis.

On examination, her vital signs were normal. She was afebrile and her blood pressure was 120/80 mm Hg. She had a pulse of 70 beats per minute, and her respiratory rate was 18 breaths per minute. Her oxygen saturation was 98% on room air.

She was alert and oriented to person, place, and time. She had a normal mental status examination and no evidence of cranial nerve abnormalities. Her visual acuity was 20/20 in both eyes, and her visual fields were full.

Her motor examination was notable for decreased strength in the left lower extremity. She had a positive Babinski sign on the left side. She had a 18 month history of progressive bilateral leg weakness. Her medical history included hypertension, coronary artery disease, and herpes zoster. She had undergone a cholecystectomy for cholelithiasis.

Her sensory examination was normal. She had a 18 month history of progressive bilateral leg weakness. Her medical history included hypertension, coronary artery disease, and herpes zoster. She had undergone a cholecystectomy for cholelithiasis.

Her cognitive examination was normal. She had a 18 month history of progressive bilateral leg weakness. Her medical history included hypertension, coronary artery disease, and herpes zoster. She had undergone a cholecystectomy for cholelithiasis.

Her speech was clear and fluent. She had a 18 month history of progressive bilateral leg weakness. Her medical history included hypertension, coronary artery disease, and herpes zoster. She had undergone a cholecystectomy for cholelithiasis.

Her gait was normal. She had a 18 month history of progressive bilateral leg weakness. Her medical history included hypertension, coronary artery disease, and herpes zoster. She had undergone a cholecystectomy for cholelithiasis.

Her cranial nerves were intact. She had a 18 month history of progressive bilateral leg weakness. Her medical history included hypertension, coronary artery disease, and herpes zoster. She had undergone a cholecystectomy for cholelithiasis.

Her fundi were normal. She had a 18 month history of progressive bilateral leg weakness. Her medical history included hypertension, coronary artery disease, and herpes zoster. She had undergone a cholecystectomy for cholelithiasis.

Her deep tendon reflexes were normal. She had a 18 month history of progressive bilateral leg weakness. Her medical history included hypertension, coronary artery disease, and herpes zoster. She had undergone a cholecystectomy for cholelithiasis.

Her plantar responses were normal. She had a 18 month history of progressive bilateral leg weakness. Her medical history included hypertension, coronary artery disease, and herpes zoster. She had undergone a cholecystectomy for cholelithiasis.

Her abdominal examination was normal. She had a 18 month history of progressive bilateral leg weakness. Her medical history included hypertension, coronary artery disease, and herpes zoster. She had undergone a cholecystectomy for cholelithiasis.

Her cardiovascular examination was normal. She had a 18 month history of progressive bilateral leg weakness. Her medical history included hypertension, coronary artery disease, and herpes zoster. She had undergone a cholecystectomy for cholelithiasis.

Her respiratory examination was normal. She had a 18 month history of progressive bilateral leg weakness. Her medical history included hypertension, coronary artery disease, and herpes zoster. She had undergone a cholecystectomy for cholelithiasis.

Her gastrointestinal examination was normal. She had a 18 month history of progressive bilateral leg weakness. Her medical history included hypertension, coronary artery disease, and herpes zoster. She had undergone a cholecystectomy for cholelithiasis.

Her neurologic examination revealed right-sided hyperreflexia with positive Babinski sign. She had a 18 month history of progressive bilateral leg weakness. Her medical history included hypertension, coronary artery disease, and herpes zoster. She had undergone a cholecystectomy for cholelithiasis.

She was started on valproic acid and clonazepam. She had a 18 month history of progressive bilateral leg weakness. Her medical history included hypertension, coronary artery disease, and herpes zoster. She had undergone a cholecystectomy for cholelithiasis.

Her pain improved with treatment and she was discharged to home with a plan for follow-up in the outpatient clinic.

**References**


**Almost continuous epileptic activity apparent in the right frontal-frontal sagittal region during drowsiness.**
had posturing. There were 10 with a frequency clonic "painful spasm" of she after sleep. The frontal-frontal epileptic activity was apparent in the right frontal-frontal epileptiform focus in the EEG and the disappearance of both after carbamazepine treatment, suggest a causal relation between the electrophysiological phenomena and the benzodiazepine responsiveness of the patient's toric epilepsy. They may represent a distinct type of painful paroxysmal dystonia of cortical epileptic origin.

The patient had typical unprovoked painful paroxysmal dystonia associated with focal cortical epileptic activity in the contralateral fronto-frontal sagittal region. This approximates to the posterior and supplementary motor region that, when activated, may result in postural changes. Although we have not obtained an ictal recording, the association of the clinical events with the focal epileptic focus in the EEG and the disappearance of both after carbamazepine treatment, suggest a causal relation between the electrophysiological phenomena and the benzodiazepine responsiveness of the patient's toric epilepsy.

Mild depressive symptoms had been present since the age of 60, being treated with fluoxetine and benzodiazepines intermittently. There was no family history of epilepsy or other neurologi- cal diseases.

Her interictal general and neurological examination was unremarkable. Blood counts and serum biochemistry were normal. The routine EEG recording was normal, after admission the awake EEG disclosed no abnormalities; during drowsiness and sleep an almost continuous focal epileptic activity was apparent in the right frontal-frontal sagittal region (figure). Comparative study with video and MRI contrast and MRI of the brain were performed and showed mild symmetric brain atrophy without any evidence of focal structural abnormality. The EEG and MRI suggested a demyelinating disease. Treatment with carbamazepine (200 mg three times daily) led to dramatic cessation of attacks and disappearance of the epileptic activity on the EEG.

Paroxysmal dystonia was previously described by different terms, such as painful tonic seizures and painful tonic spasm.1 It represents one of the distinct paroxysmal features of multiple sclerosis.1 Ephaptic activation of axons within demyelinated plaques or other structural lesion may be responsible for both motor and sensory components of the paroxysmal dystonia.1 Bennett2 attributed painful paroxysmal dystonia to the heterogeneous group of paroxysmal dykinesias, stressing their non-epileptic character and emphasising their importance. They represent a differential diagnosis of epilepsy. The most characteristic features of these fits are their painful nature and typical pattern of unilateral limb posturing.1,2 Both ictal and interictal EEGs were reported to be normal or showed non-specific changes1 supporting the non-epileptic origin of painful paroxysmal dystonia. By contrast, dystonic posturing may occur in epilepsy, although it is usually not painful.1

Paroxysmal pain associated with motor phenomena may also be a rare manifestation of epilepsy.3,4 It has been suggested that epileptic pain originates from the contralateral Rolandic area or superior and medial parts of the parietal lobe.5 The motor component of painful epileptic seizures was reported to be produced by unilateral clonic or tonic-clonic convulsions with or without march, tonic deviation of the head and eyes, bilateral clonic movements of the extremities, generalised tonic-clonic convulsions, transient motor weakness without tonic-clonic static posturing, and "stiffening" of the arm or leg.5 Painful epileptic seizures with a characteristic pattern of unilateral posturing of the extremities meeting the criteria of painful paroxysmal dystonia to our knowledge have apparently never been described.

Our patient had typical unprovoked painful paroxysmal dystonia associated with focal cortical epileptic activity in the contralateral fronto-frontal sagittal region. This approximates to the posterior and supplementary motor region that, when activated, may result in postural changes. Although we have not obtained an ictal recording, the association of the clinical events with the focal epileptic focus in the EEG and the disappearance of both after carbamazepine treatment suggest a causal relation between the electrophysiological phenomena and the benzodiazepine responsiveness of the patient's toric epilepsy. They may represent a distinct type of painful paroxysmal dystonia of cortical epileptic origin.

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Treatment of spasmodic torticollis with intramuscular phenol injection

Phenol is a caustic agent that produces tissue destruction and has been used to weaken muscle in patients with spasticity by either nerve block, motor point block, or intramuscular neurolysis.3,4 Local muscle pain and tenderness but no systemic or long term side effects have been reported after such use.1,2 Phenol is the most intramuscular phenol in the treatment of spasmodic torticollis. I have used this agent in patients with spasmodic torticollis who have not responded well to intramuscular injections of botulinum toxin type A (BTX).

Both patients gave informed consent to participate in the trial with the approval of the Duke University Institutional Review Board. The muscles responsible for abnormal head movements were determined by clinical examination and by EMG recordings made with a concentric EMG needle in the sternomastoid, splenius capitus, or other neck muscles involved in head turning. Phenol solution (1% weight/volume USP phenol crystals in sterile aqueous solution) was injected into the involved muscles with a recording monopolar injection electrode to determine that injections were made in muscle active in the abnormal movement. Injections were made at two to six sites in each muscle, in sites where a motor unit action potentials with sharply rising compo-

ments were recorded, indicating that the needle tip was close to muscle fibres that were activated during the abnormal move-