

Cortical evoked potentials (N19 & P37) show dramatically increased amplitude and decreased latency over the period of 2 months. The rapid improvement suggests demyelination resulting in abnormal central conduction.

In neurotoxic disease, exposure to different levels of the same substance may result in dramatically different clinical pictures, but a certain toxin associated with structural damage to the nervous system produces a similar pattern of disease, commensurate with the dose and duration of exposure.⁵ Podophyllin is considered to be one of the neurotoxins belonging to this group. From a previous reported case³ and our patient, it appears that podophyllin destroys the sensory system selectively from peripheral nerves to posterior column resulting in deafferentation. It thus appears that podophyllin can provide an experimental model for investigating deafferentation.

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Paroxysmal hemidystonia induced by prolonged exercise and cold

Paroxysmal choreoathetosis is a rare usually familial disorder. Depending upon the age of onset, frequency and duration of the attacks, trigger factors and response to treatment, three subgroups are identified:¹ (1) Paroxysmal dystonic choreoathetosis;² (2) Paroxysmal kinesiogenic choreoathetosis;³ (3) Intermediate type.^{4,5}

In this sporadic case of the intermediate type there were previously unreported features.

This 18 year old cobbler, was seen in February 1988 with a nine month history of paroxysmal involuntary movements affecting the right half of the body. His first attack involved two trigger factors. He was caught in a sudden shower of rain while he was returning home from a nearby village. After running two kilometres in about 10 minutes he started to experience a dragging sensation in the right shoulder which was immediately

followed by strong irregular jerky movements. This spread rapidly to the whole of the right half of the body. He assumed it was the effect of "cold" and tried to "rub it down." He did not experience any pain in the affected part and remained fully conscious during the attack which lasted about 10 minutes. His speech remained normal. The attack faded as he changed into dry clothes. He had no motor or sensory deficits after the attack. Since then, he has experienced similar attacks whenever he runs for more than six to 10 minutes and also when taking cold baths. The frequency of the attacks varied from two to three per month.

A month later, whilst working at the anvil, he experienced an attack of involuntary movements similar to the previous ones. It also started at the right shoulder and spread to involve the right half of the body and lasted about 10 minutes.

He had not previously experienced such attacks with sudden movements, startle or fatigue. They were also not precipitated with cold drinks, tea or coffee. He did not drink alcohol or smoke. There appeared to be no diurnal variation in the attacks. He received no treatment.

He was born of non-consanguineous parents and there was no relevant past or family history.

Detailed and neurological examination was unremarkable. Attempts were made to induce the attacks and the results were video recorded and studied.

Running and treadmill exercising induced the attacks in about eight minutes each. Striking a hammer against a stone, using the right hand, induced an attack in about six minutes. However, similar exercise with the left hand did not provoke an attack. Passive movements, application of a vibrator to the limbs, sudden movements and startle did not induce an attack. A cold water shower (temperature 15°C) and exposure to cold breeze blowing from an air cooler, induced the attacks within three to four minutes. A warm water shower did not induce an attack. Figures 1 and 2 show the patient during attacks induced by treadmill exercising and a cold shower, respectively.

All observed attacks were similar in nature and lasted about 10 minutes each. An attack started with the patient experiencing a dragging sensation in the region of the right shoulder. The involuntary movements, started within five to 10 seconds thereafter. During the period of the attack the arm was generally extended at all the large joints while the leg generally took the posture of flexion at the large joints. While the fist remained partially closed the foot was extended (fig 1). There were repeated brisk irregular jerky and sometimes bizarre movements at the various joints. These produced repeated extension, abduction and lateral rotation of the arm, extension and supination of forearm, extension and lateral deviation of the hand, flexion and medial rotation of the thigh, flexion of the leg and flexion-inversion of the foot. Frequent mild irregular jerking movements were seen affecting the trunk, neck and face on the right side. Each attack usually increased in intensity in the initial two minutes, and after five to six minutes tapered down over another two minutes and stopped. The patient remained fully alert and oriented during the attacks. His speech remained completely normal and he experienced no pain during the attack. There was no post-ictal deficit. The left half of the body remained unaffected.

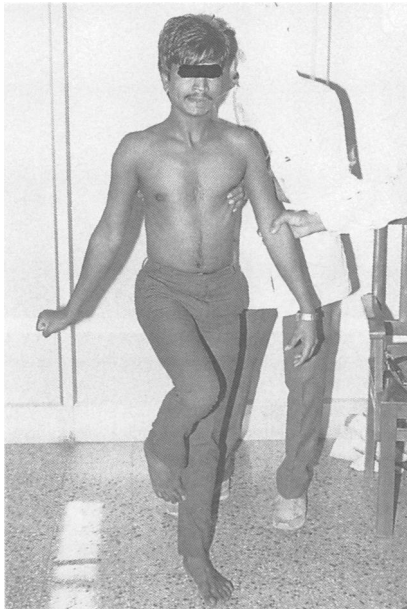


Figure 1 An attack of right hemidystonia induced by treadmill exercising.

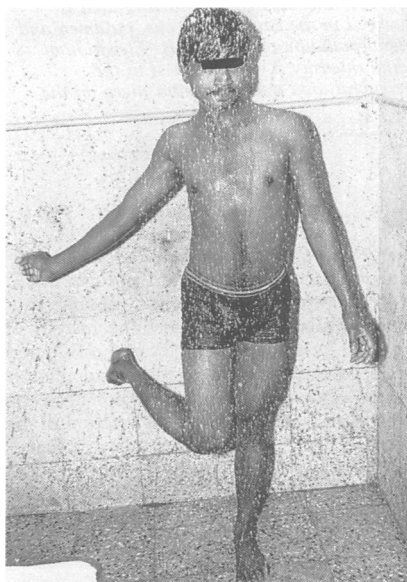


Figure 2 An attack induced by cold water shower.

Haemogram, liver function tests, serum, calcium, phosphorus, copper, caeruloplasmin estimations were normal. Blood sugar levels in the fasting state and during the ictus were normal. Slit lamp examination for K-F ring was negative. Radiological evaluation with CT scan of the head, radiographs of the chest and skull was normal. An interictal EEG whilst awake, showed frontally dominant intermittent rhythmic delta activity. No epileptiform discharges were seen.

Serial trials of phenobarbitone (120 mg/day), phenytoin sodium (300 mg/day) and clonazepam (2 mg/day) for four weeks each did not help. A year later the attacks were unchanged.

Paroxysmal choreoathetosis emerged as a clinical entity in a description of a family by Mount and Reback in 1940.² Clinical features of the three subgroups are summarised in the table. Our case belongs to the inter-

Table Familial paroxysmal choreoathetosis

	Paroxysmal dystonic choreoathetosis	Paroxysmal kinesigenic choreoathetosis	Intermediate type
Inheritance	Autosomal dominant Single sporadic Case	Autosomal dominant Sporadic ? Recessive	Autosomal dominant
Age of onset	Neonatal/infancy	Infancy to 4th decade	2/month; rarely daily
Frequency of attacks	1-2/month to 3/day	1/month to 100/day	5 to 30 minutes
Duration of attacks	2 min to 6 hours	Usually 1 minute; always less than 5 minutes	
Induced by	Alcohol, other drinks, fatigue, rest after physical stress, excitement	Sudden movements, startle	Continuous exertion, passive movements, local vibration
Therapy	Clonazepam	Phenytoin Phenobarbitone	Clonazepam?

Modified from Buruma and Roos¹

mediate type and is the first sporadic case in this group. The history, examination and investigations ruled out underlying conditions, such as, cerebral palsy, multiple sclerosis, Wilson's disease, idiopathic hypoparathyroidism, hypoglycaemia, encephalitis or space occupying lesion which could produce focal dyskinesias.

The well documented families of Lance⁴ and Plant *et al.*⁵ which form the basis of the intermediate type, are characterised by cases whose attacks were induced by prolonged exercise. In our case the attacks were not only induced by prolonged exercise, such as, running, but also by prolonged work with a part of the ipsilateral limb. A similar observation is made in case 1 by Plant *et al.*⁵ in which writing to dictation could induce the attacks of involuntary movements.

As a trigger factor cold temperature has not been reported in the intermediate type. Absence of response to warm water shower indicates that the trigger factor is cold related and not related to the striking force of the water drops to the body surface. Cold breeze also induced the attacks. This is the first case of the intermediate type to show unilaterality the involuntary movements. In the cases of Lance⁴ and Plant *et al.*⁵ the involuntary movements predominantly affected both the lower limbs and only when severe, involved the whole body. The EEG finding of frontally dominant intermittent rhythmic delta activity in the awake state is another new observation although possibly of non-specific signifi-

cance. Previously reported cases had normal EEGs.

The trigger factors and involuntary movements in our case, suggest a functional disturbance of the contralateral basal ganglia or their connections leading to abnormal responsiveness to external stimuli sensed by kinaesthetic and cold receptors.

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An intracerebral abscess following an aneurysm clipping

A sixty one year old right handed woman was admitted as an emergency having sustained a subarachnoid haemorrhage resulting in coma. A CT showed subarachnoid blood in the basal cisterns and a Sylvian fissure. Cerebral pan-angiography showed a bilobular anterior communicating artery aneurysm. On day 17 after the bleed, a right pterional craniotomy was performed and the anterior communicating artery aneurysm was clipped. Her recovery was uneventful except for a urinary tract infection. She was discharged on day 13 after the operation.

Four weeks after the operation she was again admitted with a history of seven days of right frontal headache. There was no neurological deficit and no papilloedema. CT (figure a) suggested a right frontal infarct. A lumbar puncture was normal.

She improved with simple analgesics and was allowed home after four days. Six weeks later she again presented with a two week history of progressive generalised headaches, vomiting and a left sided weakness. Examination revealed a left sided weakness, the presence of primitive frontal reflexes but no papilloedema. She was pyrexial (temperature 37.5°C). Further CT (figure b) showed a cystic enhancing mass in the right frontal area. A right frontal burr-hole was made and the mass aspirated, 40 mls of pus was obtained. Bacteriological studies showed Gram positive cocci and a growth of *Staphylococcus aureus*.

She was initially treated with gentamycin, metronidazole and benzyl penicillin. This was later changed to lincomycin, sodium fusidate and metronidazole. Antibiotics were continued for ten weeks—one week intravenously, nine weeks orally. Her symptoms improved as did the left hemiparesis; the