

**Table Eight patients with arsenic neuropathy with decreased peripheral nerve functions**

Patient no.	Age, y/sex/arsenic intake, mg	Abnormalities in VPT*			Sural nerve AP, $\mu$ V			Sural nerve SCV, m/s		
		1 mo	3 mo	12 mo	1 mo	3 mo	12 mo	1 mo	3 mo	12 mo
Patients without symptoms										
1	50/M/56	3/4	3/4	1/4	8.1	10.0	6.7†	50	42	51
2	56/F/71	4/4	1/4	1/4	10.0	15.9	8.7	49	50	55
Patients with sensory symptoms										
3	16/F/52	1/4	0/4	0/4	29.5	33.0	41.8	54	53	55
4	17/M/54	1/4	0/4	NE	4.4†	13.0	NE	46	48	NE
5	50/M/68	4/4	NE	NE	6.2†	NE	NE	48	NE	NE
6	44/F/52	2/4	1/4	1/4	13.5	16.3	9.9	52	48	52
7	47/F/95	4/4	4/4	4/4	22.5	15.4	14.9	51	44	45
8	48/M/95	4/4	4/4	3/4	5.8†	4.8†	5.7†	42	43	46

\* Expressed the ratio of abnormalities of VPT (2 SD above the normal range) to four examinations.

†Indicates 2 SD below the normal range.

VPT = vibration perception threshold; AP = action potential; SCV = sensory nerve conduction velocity; NE = not examined.

to impaired energy metabolism.<sup>4</sup> It has been suggested that arsenic interferes with cellular energy metabolism of the neurons, eventually leading to axonal degeneration.<sup>5,6</sup>

Although motor nerve involvement is usual in arsenic neuropathy and tetraplegia develops in severe cases,<sup>5</sup> none of our patients developed motor weakness. Motor nerve conduction velocities and compound muscle action potentials were normal. Only F-responses were absent in two patients, which may reflect subclinical motor nerve involvement. Other authors have also reported the absence of F-response in arsenic neuropathy.<sup>6,7</sup>

Arsenic has an effect on various components of the nervous system. CNS manifestations, including drowsiness and confusion, develop rapidly after intoxication. Paranoid delusions and hallucinations have also been reported.<sup>2,5</sup> The involvement of the cranial nerves is rare, but has been reported.<sup>5</sup> Autonomic nervous system involvement has not yet been reported even in severe motor involvement.<sup>2</sup> In this study, the results of functional tests of the autonomic nervous system were normal in every patient. This selectivity in neurotoxicity of arsenic is of interest and warrants further investigation.

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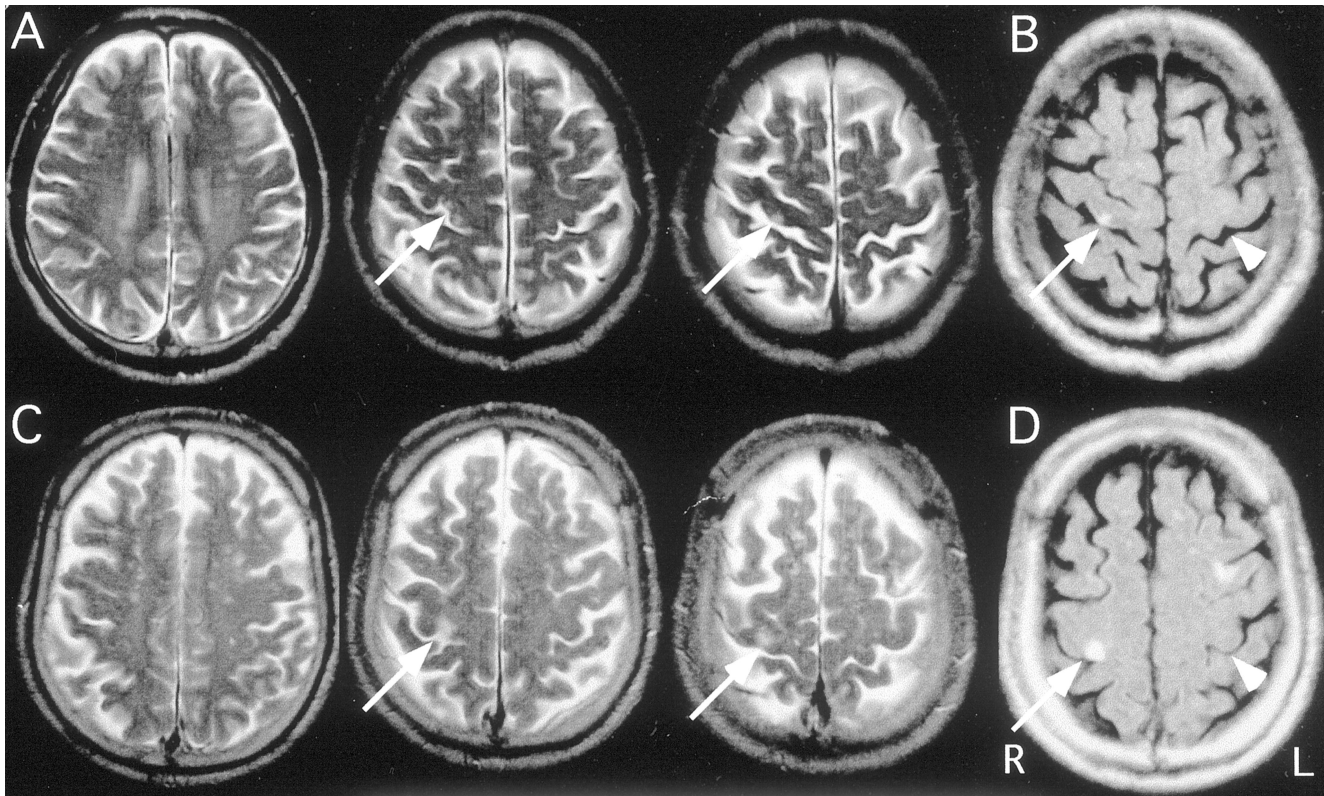
## Monoataxia of upper extremity in motor cortical infarction

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Recent studies using functional imaging have demonstrated that the anatomic location of the primary motor hand area is topographically localized in a specific segment of the precentral gyrus. MRI reveals that such cortical area protrudes posterolaterally from the precentral gyrus to the central sulcus, thus forming a characteristic knoblike shape similar to that of an inverted omega or epsilon in the axial plane.<sup>1</sup> Lesions in the precentral knob cause isolated motor weakness or clumsiness of the corresponding hand.<sup>2</sup> Recently, we encountered two patients who presented with ataxia after a cerebral infarction that selectively involved the motor cortex located at the medial part of the precentral knob.

*Case reports. Patient 1.* A 69-year-old right-handed-man with a history of hypertension and diabetes mellitus was admitted because of sudden difficulties in voluntary movement in his upper left limb. On neurologic examination, he was alert and oriented and there was no sensory extinction. Cranial nerves were all intact. There was no weakness in his limbs. Pain, light touch, position, and vibration sensations were normal. However, he could not maintain his outstretched left arm at a stable position, and irregular persistent oscillations appeared. During performance of finger-to-nose testing, the oscillations of his left arm and shoulder worsened markedly. The ataxia was confined to the proximal part of his upper left extremity, and was not influenced by the presence or absence of visual information. Finger movements were normal. The neurologic examination was otherwise normal. These symptoms gradually improved, and the patient fully recovered within 2 weeks. MRI revealed a small cortical infarction located at the medial part of the precentral knob of the right side (figure, A). There were no identifiable lesions in the corona radiata, internal capsule, cerebral peduncle and pontine base, and the cerebellum. Median nerve somatosensory evoked potentials were bilaterally normal.

*Patient 2.* A 67-year-old right-handed-man with a history of hypertension was admitted because of sudden difficulty in executing voluntary movements of his upper left extremity. On neurologic examination, he was alert and well oriented. Cranial nerves were all intact. There was no motor weakness or sensory disturbance in his limbs. However, when he stretched his arms in front, he could not maintain his left arm at a stable position. During performance of the finger-to-nose testing, severe oscillations of his left arm and shoulder appeared. However, there was no loss of



**Figure.** MRI scans of the brain of Patient 1 (A, B) and Patient 2 (C, D). T2-weighted images (A, C) and fluid-attenuated inversion recovery images (B, D) show that the motor cortical infarctions located in the medial part of the precentral knob (arrows). Arrowheads (B, D) indicate the precentral knob of the contralateral side.

dexterity in the fingers or hand. This symptom gradually improved and disappeared completely within 2 weeks. The brain MRI revealed a recent small cortical infarction located at the medial part of the precentral knob of the right side (see the figure, B).

**Discussion.** Clinical and neuroradiologic characteristics of these two patients were essentially similar. The common manifestation in both patients was unilateral monoataxia of the proximal part of their upper left extremities that worsened when performing voluntary actions. There was no associated motor weakness, sensory disturbance, or loss of finger dexterity. Their MRI studies clearly revealed a small cortical infarction involving the medial part of the precentral knob. Therefore, it is most likely that these small discrete cortical infarctions involving the medial part of the precentral knob were responsible for the motor disturbances in our patients.

It has been reported that not only weakness or clumsiness of the hand but also other symptoms such as tremor or asterixis occasionally appear after a small motor cortical infarction.<sup>3,4</sup> However, the symptom presented by our patients was ataxia in the proximal upper limb; there has been no similar report. Although the exact reason that our patients exhibited ataxia is unclear, one possible speculation is that the lesion may have interrupted the sensory-motor integrity, resulting in unbalanced motor outputs. It has been shown that areas 3a and 4 are closely related in the primary motor cortex,<sup>5</sup> and sensory-motor integration in the motor cortex is essentially important for motor control of the limb.<sup>6</sup> Alternatively, the frontopontocerebellar pathway may have been selectively involved at the cortical level. Another notable finding in the current patients was that the ataxia appeared preferentially in the proximal muscles of the upper extremity, suggesting that the motor cortical territories for the execution of movements of the proximal upper extremity may be located in the medial portion of the precentral knob. Although further studies are required to confirm our speculations, it is clinically important to note that a cortical infarction in the medial precentral knob may cause an isolated monoataxia in the upper extremity.

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## Leptomeningeal metastasis from an intracranial epidermoid cyst

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Epidermoid tumors are benign and constitute 1% of all intracranial tumors. Squamous cell carcinoma (SCC) arising in an epidermoid is rare, and leptomeningeal metastasis (LM) from malignant degeneration of an epidermoid is even less common; we have identified only three such patients in the literature.<sup>1-3</sup>

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