

'Pure' motor hemiplegia

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SYNOPSIS Attenuation of cerebral evoked responses after stimulation of the median nerve in the hemiplegic limbs suggested that an apparently pure motor hemiplegia in some patients may not have pure involvement of the corticospinal system. Frontoparietal metastasis, infarction in basis pontis and medullary pyramid, and occlusion of internal carotid artery in the neck resulted in pure motor hemiplegia in some individuals.

Fisher and Curry (1965) defined the entity of pure motor hemiplegia as a recent paralysis, complete or incomplete, of the face, arm, and leg on one side and unaccompanied by any impairment of functions of language, sensory system, visual fields, cerebellar pathways, and cranial nerves. In a review of 50 patients, which included nine cases that came to necropsy, they concluded that pure motor hemiplegia resulted from a small infarction (less than 35 mm × 8 mm × 25 mm) in the internal capsule or the basis pontis, probably due to thrombosis of the small penetrating vessels. They further stated that other diagnoses and lesions at other sites need not be strongly considered and angiography was not indicated. The purpose of this presentation is threefold: (1) to demonstrate by recording somatosensory cerebral evoked responses that in some patients an apparently pure motor hemiplegia was not so pure, as there was electrophysiological evidence of involvement of the lemniscal pathways; (2) to document lesions at other sites in pure motor hemiplegia; and (3) to demonstrate the necessity for considering diagnosis other than infarction in 'pure' motor hemiplegia.

METHODS

CLINICAL MATERIAL There were 15 patients who were admitted to the Neurology Service of the Veterans Administration Hospital, Hines, Illinois,

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with a clinical diagnosis of pure motor hemiplegia. The patients' ages ranged from 44 to 81 years. There were 14 men and one woman (case 8). The Table summarizes the pertinent data. The duration of the hemiplegia varied from two days to one month. Blood pressure was normal in eight patients and in the remaining individuals the blood pressure was high, and they all had a history of hypertension and had been on antihypertensive medication. Routine electroencephalography was normal in all, except for slight diminution of the background activity contralateral to the hemiplegic side in case 6. Brain scan was normal in nine patients and was not done in three patients. The scan showed increased uptake of isotope in three other patients. Cerebral angiograms (cases 4-8 and 11) demonstrated occlusion of the internal carotid artery in the neck in cases 4 and 6 contralateral to the hemiplegic limbs. One patient (case 4) had an endarterectomy of the occluded carotid artery. One patient (case 8) had a bronchogenic carcinoma for which she received radiation treatment. A cerebral angiogram demonstrated evidence of a possible metastatic lesion in the left frontoparietal area. She died nine months after she presented with pure motor hemiplegia. Postmortem examination confirmed the presence of a metastatic tumour in the left frontoparietal region. An infarct in the right side of the basis pontis was demonstrated at necropsy in case 10. In case 15 (details will be published separately) postmortem examination revealed a medullary pyramidal infarct.

SOMATOSENSORY CEREBRAL EVOKED RESPONSE STUDY We recorded somatosensory cerebral evoked responses simultaneously by contralateral and ipsilateral scalp electrodes after stimulation of the

TABLE
CLINICAL AND LABORATORY DATA ON 15 PATIENTS WITH PURE MOTOR HEMIPLEGIA

Case no.	Age (yr)	Side affected	Tone	Duration of hemiplegia	BP (mm Hg)	EEG	Brain scan	Cerebral angiogram	SER
1	54	R	I	1 w	130/110	N	N	No	N
2	51	R	D	2 d	176/106	N	N	No	A
3	46	R	I	1 w	220/120	N	No	No	N
4	64	R	I	3 d	160/100	N	Uptake L parietal	Occlusion L carotid in neck	A
5	60	L	I	1 w	142/80	N	N	Normal R brachial	A
6	52	R	I	5 d	190/110	Mildly abnormal	Uptake L fronto-parietal	Occlusion L int. carotid in neck	A
7	52	R	I	2 d	130/90	N	N	Normal L carotid	N
8	54	R	I	3 w	140/90	N	Uptake L parietal	Abnormal L carotid	A
9	63	L	I	1 w	140/90	N	N	No	N
10	76	L	N	2 w	140/70	N	N	No	N
11	53	L	I	1 m	180/120	N	N	Normal R brachial	A
12	78	R	D	10 d	150/110	N	N	No	A
13	81	R	I	3 w	120/70	N	N	No	N
14	55	L	I	10 d	130/70	N	N	No	N
15	77	L	D	10 d	140/80	N	N	No	N

R: right, L: left, SER: somatosensory cerebral evoked responses.
I: increased, D: decreased.
N: normal, A: abnormal.

median nerves on both sides separately in cases 1-14. The median nerve was stimulated at the wrist at the rate of 1/s by means of a Grass Stimulator S-88. The cathode was placed proximally at the stimulus site. We used a pulse duration of 0.1 ms and stimulus intensity just above the threshold to produce a small twitching movement of the thumb. The evoked potentials were recorded bilaterally from the central electrodes (C3 and C4 of the International Nomenclature) with ipsilateral ear as the reference electrode. The responses were averaged for a total of 200 and 400 stimuli. In each trial, the input from the recording electrode was led into a Tektronix differential amplifier 3A72 and displayed on the oscilloscope and the output was summated by a computer of average transients (CAT-400 C, Technical Instruments, Inc.). The stimulator triggered the computer. The first 125, 250, and 500 ms after the stimuli were analysed separately on both sides. We made control recordings without nerve stimulation in the same experimental situation. The patients lay in a comfortable position on a couch in a relaxed state. Throughout the experiment, the EEG was monitored on a Grass 8-channel machine. In all patients, we routinely determined the sensory nerve conduction time after stimulating the sensory fibres of the median nerve in the index finger and recording from the nerve at the wrist bilaterally.

RESULTS

The contralateral and ipsilateral cerebral evoked

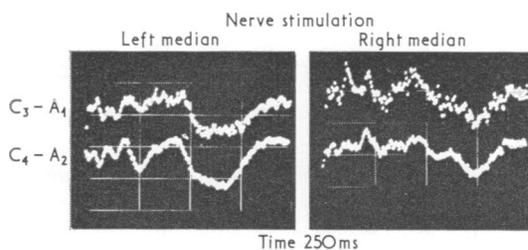


FIG. 1 Case 7. Normal somatosensory cerebral evoked responses.

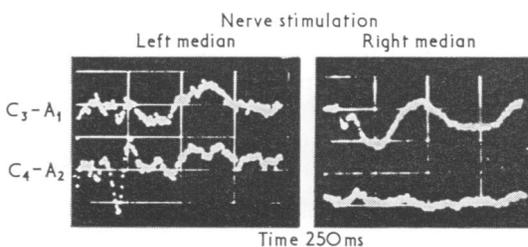


FIG. 2 Case 6. Attenuation of cerebral evoked potentials bilaterally after stimulation of the right median nerve (hemiplegic side). Normal responses after stimulation of the left median nerve.

responses after median nerve stimulation separately on both sides were normal in seven patients. Figure 1 (case 7) shows such a normal response. In the remaining seven individuals, the evoked responses demonstrated moderate attenuation of all components of the evoked potentials in the contralateral hemisphere and slight attenuation of the ipsilateral evoked responses on stimulation of the median nerve on the affected side. The median nerve sensory conduction time was normal in all patients. Figure 2 (case 6)

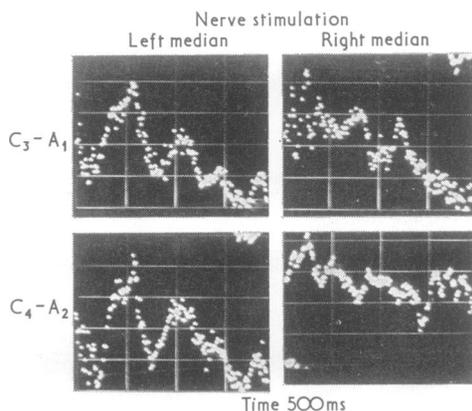


FIG. 3 Case 8. Contralateral and ipsilateral attenuation of cerebral evoked potentials after stimulation of the median nerve on the affected right side. Cerebral responses are normal after stimulation of the left (unaffected side) median nerve.

shows attenuation of the components of bilateral evoked potentials during the first 250 ms after stimulation of the right median nerve (hemiplegic side). Stimulation of the median nerve in the unaffected left side shows normal responses. Figure 3 demonstrates attenuation from another patient (case 8) contralaterally and ipsilaterally after stimulation of the median nerve in the affected right side, but the responses are normal bilaterally after stimulation of the left median nerve (unaffected side).

DISCUSSION

There is little doubt that in many patients pure motor hemiplegia is due to pure corticospinal system dysfunction as suggested by Fisher and

Curry (1965). However, in some patients, pure motor hemiplegia is not so pure, as is clearly shown by our electrophysiological demonstration of involvement of the lemniscal pathways. It is generally accepted that the somatosensory cerebral evoked responses depend upon the integrity of the pathways transmitting proprioception (Williamson *et al.*, 1970). Therefore, normal somatosensory cerebral evoked responses in cases 1, 3, 7, 9, 10, 13, and 14 with clinical features of pure motor hemiplegia indicated pure dysfunction of the corticospinal system. However, in cases 2, 4-6, 8, 11, and 12 evoked responses were abnormal. These latter observations in conjunction with normal sensory conduction velocity of the median nerves suggested subclinical involvement of the lemniscal pathways. This discrepancy between the clinical and the electrophysiological findings could be due to the fact that mild degree of affection of the proprioceptive pathways may escape detection even on very meticulous clinical examination.

In cases 4 and 6, occlusion of the internal carotid artery in the neck was demonstrated angiographically and verified at operation in case 4. Fisher and Curry (1965) mentioned one case, not clinically examined by them, where postmortem examination showed a capsular infarct and an 'old carotid occlusion'. They concluded that the two conditions may not have been related. The same argument may be applied to our patients (cases 4 and 6). However, Aleksic and George (1973) reported two cases of pure motor hemiplegia in whom angiograms revealed occlusion of the internal carotid artery in the neck. They suggested that in some cases, extracranial occlusive vascular disease may play a role in the pathogenesis of pure motor hemiplegia.

In our case 8, a pure motor hemiplegia associated with a metastatic cerebral lesion demonstrated the necessity for considering a diagnosis other than infarct in some cases of pure motor hemiplegia. Weintraub and Glaser (1970) described a case of pure motor hemiplegia due to a nocardial abscess in the motor cortex. Igapashi *et al.* (1972) described a case of pure motor hemiplegia resulting from ischaemia or oedema in the motor cortex after repeat craniotomy for postoperative bleeding.

The postmortem demonstration of infarct in the basis pontis in case 10 supported the sugges-

tion of Fisher and Curry (1965) regarding the sites of involvement in pure motor hemiplegia. They further stated that a medullary pyramidal infarct was unlikely to produce a pure motor hemiplegia, but it was possible for a critically placed lacunar infarct in one pyramid to produce pure motor hemiplegia. Our case 15 conclusively demonstrated that it was indeed possible for a pyramidal infarct to produce pure motor hemiplegia.

On the basis of our observations, we would like to suggest that in cases of pure motor hemiplegia, recording of somatosensory cerebral evoked responses is desirable and if these results are abnormal then angiograms should be performed to demonstrate possible lesions at sites

other than the internal capsule or basis pontis, or to establish a diagnosis other than infarction.

REFERENCES

- Aleksic, S. N., and George, A. E. (1973). Pure motor hemiplegia with occlusion of the extracranial carotid artery. *Journal of the Neurological Sciences*, **19**, 331-339.
- Fisher, C. M., and Curry, H. B. (1965). Pure motor hemiplegia of vascular origin. *Archives of Neurology*, **13**, 30-44.
- Igapashi, S., Mori, K., and Ishijima, Y. (1972). Pure motor hemiplegia after craniotomy for postoperative bleeding. *Archiv für Japanische Chirurgie*, **41**, 32-37.
- Weintraub, M. I., and Glaser, G. H. (1970). Nocardial brain abscess and pure motor hemiplegia. *New York State Journal of Medicine*, **70**, 2717-2721.
- Williamson, P. D., Goff, W. R., and Allison, T. (1970). Somato-sensory evoked responses in patients with unilateral cerebral lesions. *Electroencephalography and Clinical Neurophysiology*, **28**, 566-575.