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Transcranial electrical stimulation in patients with apallic syndrome

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Muscle responses (MEPs) to transcranial electrical stimulation were studied in 7 patients with apallic syndrome. All the patients showed clinical signs of upper motor neurone impairment in the upper and lower limbs. MEPs were absent or markedly delayed in 4 of the 7 patients. Since patients with apallic syndrome show only minimal voluntary movement, transcranial stimulation is the only way to demonstrate abnormalities of fast corticospinal axons in these patients. Even though these patients often look similar clinically, with tetraplegia and decorticate or decerebrate posture, only some cases showed dysfunction of fast corticospinal neurons.

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The apallic syndrome describes a condition in which patients recover from deep coma, but become awake without being aware (1). Apallic syndrome has been described after traumatic and non-traumatic acute brain damage (2–4) as well as during progressive disorders affecting the cerebral structures (5). Post-mortem examination shows extensive abnormalities of the cortex or its connections, with relative sparing of the brain stem (6).

Apallic patients are usually in a decorticate or decerebrate posture and unable to perform voluntary movements. Because the pyramidal tract can be studied by means of electrical stimulation of the scalp (7, 8), we have used this methodology to ascertain whether the corticospinal tracts are responsible for the motor abnormalities and the abnormal posturing showed by these patients.

Patients and methods

Seven patients (mean age 21.5 ± 14.7 yrs, range 6–42) suffering from apallic syndrome, five post-traumatic (pts 1–5 of Table 2) and two post-anoxic (pts 6–7 of Table 2), were studied and the results were compared to a group of 15 normal age-matched subjects. The study was approved by the local ethical committee. All the patients were chronically bedridden with decorticate or decerebrate posture and no contact with the surroundings. The clinical data of the patients and the CT localisation of the lesions are shown in Table 1. The patients were studied at least 2 months after the brain injury, when the motor deficit was in a subacute or chronic stage. The motor cortex and cervical region were stimulated with an electrical stimulator (Digitimeter model 180) supplying a maximum of 750 volts. The stimulus time constant was 100 μs. The stimulation was performed through surface electrodes placed on the scalp, with the cathode on the vertex and the anode 7 cm down a line between the vertex and the external auditory meatus. The cervical root was stimulated with surface electrodes, with the cathode on T1 (over the spine of the 1° thoracic vertebra) and the anode on C4 (over the spine of the 4° cervical vertebra). Muscle evoked potentials (MEPs) were recorded bipolarly by surface electrodes from biceps and thenar muscles and stored on a Mystro-Medelec EMG recording device. Eight MEPs were obtained from each stimulation site at maximum stimulation intensity. The central conduction time (CCT) was obtained by subtracting the latency of cervical MEPs from the latency of cortical MEPs. The MEPs were considered abnormal when they were absent or the CCT exceeded the normal range.

Results

In all the patients with the apallic syndrome, MEPs evoked by cervical stimulation were normal. Abnormality of cortical MEPs was present in patients 1,

Table 1. Clinical data of patients with apallic syndrome

Patient	Duration (months)	Strength		Tone		Reflexes		Babinski		CCT scan
		right	left	right	left	right	left	right	left	
1	3	n.e.	n.e.	++	++	++	++	–	–	R temp.– occipital hypodensity R int. caps.– thalamus hypodensity R basal ganglia hypodensity
2	3	n.e.	n.e.	++	++	++	++	+	+	R front subdural hematoma R temp., thalamus, hypothalamus hypodensity
3	7	n.e.	n.e.	++	++	++	++	–	+	L temp. hypodensity
4	15	n.e.	n.e.	++	++	++	++	–	–	Cortico-subcortical atrophy
5	8	n.e.	n.e.	++	++	++	++	–	–	R front. subdural hematoma L int.– ext. caps. hypodensity
6	2	n.e.	n.e.	++	++	++	++	–	+	Cortico-subcortical atrophy
7	45	n.e.	n.e.	++	++	++	++	–	–	Cortico-subcortical atrophy

Strength n.e. = not evaluable
Tone –=reduced, +=normal, ++=increased
Reflexes +=normal, ++=increased
Babinski –=absent, +=present

2, 3 and 6 (Table 2) of the seven with apallic syndrome. The abnormalities consisted of the absence of MEPs ("no response" in Table 2) or in the prolongation of the CCT (Fig. 1).

Whereas in Patients 1 and 3 the latency of MEPs was slightly prolonged, in Patients 2 and 6 cortical stimulation either evoked no response or evoked only very late responses.

In Patient 1 CT scan showed ipsilateral lesions, in Patients 2 and 3 a lesion contralateral to the MEP abnormalities and in Patient 6, cortical and subcortical atrophy. All the patients with MEP abnormalities were in a decorticate posture, showed signs of upper motor neurone involvement and had suffered brain injury more recently than in the other patients with normal MEPs.

Cortical stimulation evoked responses with normal CCT in Patients 4, 5 and 7. The CT scan demonstrated diffuse cortical and subcortical atrophy in Patients 4 and 7 and bilateral lesions in Patient 5. These patients were also in a decorticate posture, showed clinical upper motor neurone signs on both sides, and the apallic syndrome lasted longer (longest interval from the brain injury) than in the other patients.

Table 2. Central conduction time (CCT) of patients with apallic syndrome

Patients	Side	CCT	
		Biceps (ms)	Thenar (ms)
1	r	9.7	9.7
	l	4.1	4.0
2	r	4.7	4.6
	l	21.1	61.4
3	r	6.6	9.9
	l	4.6	3.5
4	r	4.6	6.0
	l	5.8	4.8
5	r	5.0	6.0
	l	5.5	5.9
6	r	4.0	5.0
	l	43.5	/
7	r	5.7	5.1
	l	5.7	4.6
Normal subjects			
Mean+SD		4.1+0.7	5.1+0.5
Range		2.9–5.3	4.3–6.1

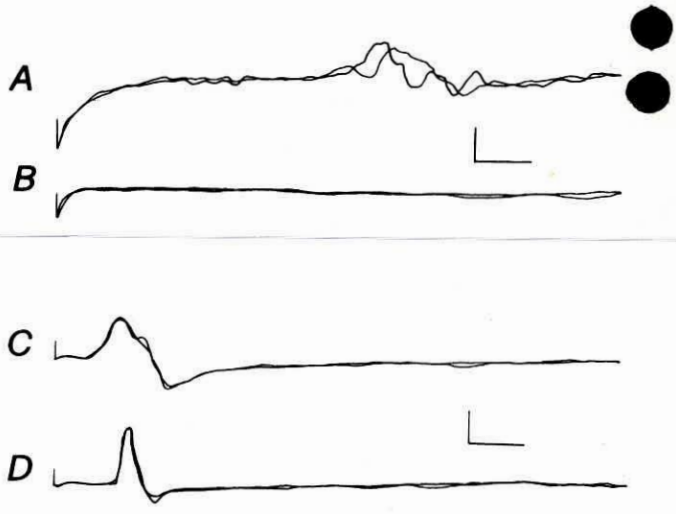

Fig. 1. Cortical and cervical stimulation in Patient 6 with traumatic apallic syndrome. Cortical responses are markedly delayed in biceps (A) and absent in the thenar muscles (B). Cervical MEPs are normal in both biceps and thenar muscles (C, D). Superimposition of two single trials – Vertical calibration 0.5 mV (A, B) and 2 mV (C, D) – Horizontal calibration 10 ms.

Discussion

With the technique of electrical stimulation of the motor cortex described by Merton & Morton (8) it is possible to study the fast conducting axons of the corticospinal tracts (7).

This study demonstrates an abnormal central conduction time in four of seven patients with apallic syndrome; the abnormalities consisted in absence or delay of MEPs. The marked delay seen in 2 of 4 cases suggests that these responses might be mediated by non-pyramidal pathways. Similar changes can also be seen in patients with upper motoneurone syndrome (9, 10).

Because of the multiplicity of CT lesions it was not possible to correlate the MEP abnormalities and the clinical signs: signs of upper motor neurone syndrome were present in all the patients but only some had abnormal MEPs. The abnormalities were more common in patients with shorter duration of the apallic syndrome, even though patients with short or long-lasting apallic syndrome look similar clinically in our population.

Since patients with apallic syndrome make very little voluntary movements, transcranial stimulation may be the only way to evaluate function of fast corticospinal neurons in these patients. Patients with apallic syndrome sometimes have fast corticospinal neurone dysfunction that is often not apparent clinically. Since the interval from brain injury seems to play a role, such a dysfunction could recover with time, although apallic syndrome and decorticate or decerebrate posturing persist.

The finding of normal MEPs in some apallic patients suggests that the abnormal posturing and clinical upper motor neurone signs may depend on the impairment of non-pyramidal or smaller pyramidal fibers.

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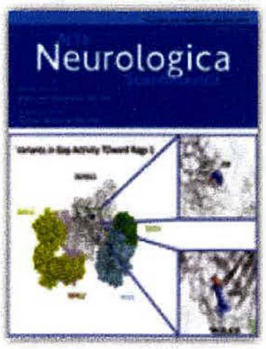

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