SOMATOSENSORY CEREBRAL EVOKED POTENTIALS IN SPINAL CORD DISEASES

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Abstract. Somatosensory cortical-evoked potentials (CEP) were investigated in 16 patients with localised myelopathy and in five patients with generalised myelopathy, and then compared with those found in 30 control subjects.

Key words: Cortical-evoked Potentials (CEP); Myelopathies.

Introduction

DAWSON (1947) was the first to pick up specific evoked potentials over the cortex (CEP) of the contralateral hemisphere in response to the stimulation of a peripheral nerve. Today, CEPs have been measured in response to the stimulation of various different peripheral nerves (Allison, 1962; Goff *et al.*, 1962; Debeker *et al.*, 1964; Giblin, 1964; Calmes *et al.*, 1971; Desmedt, 1971; Cracco, 1972; Desmedt *et al.*, 1973 and others).

The first reports on changes in the CEP seen in diseases of the spinal cord, were made by Alajouanine *et al.* (1958), Halliday *et al.* (1963), Giblin (1964), and Bergamini *et al.* (1965). A striking observation was the fact that patients with dissociation sensibility manifested only slight changes in the CEP. These findings were confirmed by later investigations carried out by Desmedt (1971), Nakanishi (1974). Baust *et al.* (1972, 1974) made use of the determination of the CEP following segmental stimulation, to localise the level of pathological processes within the spinal cord. We carried out investigations of 16 patients with localised diseases of the spinal cord, and five patients with generalised spinal cord pathology, and compared the results we obtained with a group of 30 healthy test subjects.

Method

The examination was carried out in a partially darkened room, at a constant temperature of 31°C. Any peripheral nerve damage was excluded by determining the conduction velocities of the motor and sensory nerves.

Stimulation

Stimuli were applied to the tibial nerve at the medial aspect of the ankle joint of both legs. For special questions, segmental stimulation was additionally performed on the trunk.

To carry out stimulation, a bipolar surface electrode (Disa 13 K 62) was employed, the stimulating poles being spaced 25 mm apart. The intensity of the stimulus was 2-3 times greater than the sensory threshold of intensity. The stimuli consisted of square-wave pulses having a duration of 0.2 msec and were applied at a frequency of 1/sec.

Recording

Recording was effected by using two EEG needle electrodes placed in the galea in accordance with the international 10–20 Jasper System (1957), over the sensory 'foot field', the active electrode being located on point Cz and the indifferent electrode on the point Fz. The band width of the amplifier employed was between 2 Hz and 10 kHz. A computer (Type Histomat S manufactured by Intertechnik) having 1024 addresses and an analysis time of 300 msec, was used as the averager. Each stimulation was performed twice, in order to eliminate artefacts.

The cortical-evoked potentials were recorded in such a way that the negative potential peak (N) pointed upwards, while the positive potential peak (P) pointed downwards (Fig. 1).

Following Giblin (1964) the individual potential peaks are identified with N or P and are numbered consecutively. The potential peak P₁—regularly recorded

FIG. 1

Cortical-evoked responses on stimulation of the tibial nerve. Potential peaks identified in accordance with Giblin (1964).

on stimulation of the median nerve—was not taken into account, since it was picked up in only 33 per cent of the healthy control subjects in response to stimulation of the tibial nerve. The latency of the potential peaks N₁ to N₅, and the peak-to-peak amplitudes were measured. The potential peaks N₁ to N₃ were collectively designated as the primary response, the subsequent potentials as secondary response.

Results

Standard figures: Thirty healthy control subjects, aged between 17 and 72 years, were examined. The latencies of the primary response N₁ to N₃ of the CEP following stimulation of the tibial nerve at the ankle were determined accurately in every examination. The potential peaks of the secondary response P₄ to N₅, were frequently not reliably determinable (N₅ only in some 6 per cent) with increasing potential duration, so that potential fluctuations after N₅ were not taken into account. The mean value of the latencies for N₁ is 31.5 msec. The range of scatter of the latencies increases continuously from N₁ onwards (Table 1).

The *amplitudes* of the primary response increase up to N₃, while in the secondary response, the amplitudes decrease. The scatter range is large, so that it was possible to assess the amplitudes only in a bilateral comparison (Table II).

TABLE I

Cortical-evoked potentials in response to stimulation of the tibia nerve. Latency of the potential peaks in 30 healthy test subjects

	N	X (msec)	Scatter (msec)	2-s range (msec)	s _x (msec)
<i>Primary response</i>					
N1	30	31.5	28.2-36.9	27.3-35.7	2.11
P2	30	38.6	36.3-42.3	34.2-48.0	2.18
N2	30	46.9	38.1-56.7	40.2-53.6	3.34
P3	30	57.1	44.7-66.6	47.9-66.4	4.62
N3	30	70.5	60.3-84.3	60.4-60.7	5.06
<i>Secondary response</i>					
P4	30	96.7	73.3-122.1	73.8-119.6	11.44
N4	30	123.9	80.4-161.4	84.0-163.8	19.96
P5	26	145.0	99.9-199.2	88.6-201.5	28.22
N5	20	170.8	117.6-229.9	104.3-237.3	33.24

TABLE II

Cortical-evoked potentials in response to stimulation of the tibial nerve. Amplitude of the potential peaks in 30 healthy test subjects

	N	x (μ V)	Limit values	Scatter (μ V)
<i>Primary response</i>				
N1	30	0.27	0.01-0.74	0.73
P2	30	1.85	0.82-5.29	4.47
N2	30	3.32	1.18-9.17	7.99
P3	30	2.59	0.32-5.91	5.59
N3	30	3.58	0.97-7.98	7.01
<i>Secondary response</i>				
P4	30	3.51	0.87-7.68	6.81
N4	27	2.15	0.68-6.07	5.39
P5	25	1.46	0.00-2.85	2.45
N5	20	1.25	0.44-2.66	2.22

CEP in localised myelopathies

In eight patients with a *complete* paraplegic syndrome, no cortical-evoked potentials were demonstrable on stimulation of the tibial nerve. On the basis of segmental stimulations, it was possible to carry out an accurate localisation of the levels of the pathological processes, and it was frequently seen that stimulation below the clinical 'paraplegic limit' led to an incipient cortical-evoked response (Fig. 2).

Six patients presenting with an *incomplete* paraplegic syndrome due to a vascular lesion of the spinal cord, revealed varying changes in the CEP, depending upon the degree of clinical severity or the damage to the afferent tracts.

Table III. In many cases, the clinical syndrome permits conclusions to be drawn as to the pathological-anatomical pattern of injury. In the case of patients

TABLE III

Symptomatology and measured data of the CEP in six patients with incomplete paraplegia due to circulatory damage to the spinal cord

Patient			Diagnosis	Clinical features						
				Motor power	Pain	Position	Latency N ₁ (ms)	Amplitudes N ₂	N ₃	
K.R.m.	23	J.	Haematomyelia	re.	I	++	—	29·7	0·54	2·65
			C ₂ —C ₈	li.	2	+	—	30·6	2·24	2·08
M.K.m.	22	J.	Brown-Séguar	re.	I	—	—	33·9	4·28	5·23
			C ₆ —C ₇	li.	0	++	—	33·6	2·54	4·27
A.G.w.	64	J.	Myelopathy	re.	I	—	—	29·1	2·16	2·56
			from C ₄	li.	I	+	+	48·5	2·68	0·88
B.R.m.	53	J.	Paraplegia	re.	3	++	—	31·8	0·96	2·26
			incompl. Th ₄	li.	I	++	++	—	—	—
J.E.m.	52	J.	Paraplegia		I	+	+	38·4	2·06	1·34
			incompl. Th ₄							
S.H.m.	46	J.	Caisson disease		2	+	—	31·2	2·35	3·38
			Conus-S							

Motoricity: 0 = none, 3 = severe disorder
 Sensitivity: — = none, ++ = severe disorder
 re. = right, li. = left

FIG. 2

Patient B.R., 26-years-old: Complete paraplegia at the Th₄ level, following compression fracture of the 4th thoracic vertebra. Segmental stimulation revealed that a CEP is obtained from dermatome Th₅.

FIG. 3

Patient M.K., male, 22-years-old: Brown-Séquard syndrome at C_{6/7} with dissociated sensory loss on the left side from dermatome C₆. Reduction in the amplitude of the CEP on stimulation of the left tibial nerve. Latencies normal on each side.

N. tibialis = Tibial nerve; rechts = right; links = left

with dissociated sensory loss, normal CEP latencies were found in each case, whilst, in contrast, the amplitudes of the primary and secondary responses were diminished (Fig. 3).

Similar findings were established in two patients presenting with syringomyelia of the cervical spinal cord, in whom a dissociated sensory loss was seen in both legs (Fig. 4).

FIG. 4

Patient L.J., male, 45-years-old: Syringomyelia in the cervical spinal cord, C₄ to C₈. Dissociated sensory loss below C₈ on both sides. Reduction of the amplitudes, normal latencies of the CEP.

N. tibialis = Tibial nerve; rechts = right; links = left

FIG. 5

Patient B.H., male, 50-years-old, tabes dorsalis. Delay in the latencies, and deformation of the CEP.

N. tibialis li. = Left tibial nerve
N. tibialis re. = Right tibial nerve

TABLE IV
Symptomatology and measured data of the CEP in five patients with diffuse localisation of myelopathy

Patient			Diagnosis	Clinical features					
				Motor power	Pain	Position	Latency N ₁ (ms)	Amplitude N ₂	N ₃
W.A.m.	48	J.	Funicular myelosis B ₁₂ -deficiency	2	++	++	49·5	2·54	2·30
K.A.w.	57	J.	Funicular myelosis B ₁₂ -deficiency	3	++	++	not determinable		
G.F.w.	69	J.	Funicular myelosis lat. B ₁₂ -deficiency	1	+	+	37·8	2·93	3·39
J.H.w.	74	J.	Tabes dorsalis Status after cordotomy	0	++	++	34·5	3·65	4·34
B.H.m.	50	J.	Tabes dorsalis	0	—	++	not determinable		

Motoricity: 0 = none, 3 = severe disorder
Sensitivity: — = none, ++ = severe disorder

CEP in patients with generalised myelopathy

In five patients with generalised pathology of the spinal cord, considerable changes, both in the latencies and in the amplitudes of the CEPs were found (Table IV). Particularly marked were the changes of the CEP in the myelopathies with predominant involvement of the posterior tracts (dorsal columns), as, for example, in patients with tabes dorsalis. The loss of position and vibration sensation, with normal pain and tactile sensation, led to a long delayed latency of the CEP. The potential is deformed, the primary response cannot be accurately delimited (Fig. 5). In the three patients presenting with combined tract pathology, considerable changes in the CEP were also found.

Discussion

The influence of spinal cord pathology on the cortical-evoked potentials (CEPs) by stimulation of the tibial nerve, can best be recognised in the group of patients with a vascular myelopathy.

In common with Alajouanine *et al.* (1958) and Baust *et al.* (1974), we also observed a diminution in the amplitudes of CEP in subjects with dissociated sensory loss. In our patients, the later potential peaks were particularly involved, and this accorded with the results obtained by Tsumoto (1973). The latencies were, by and large, within the normal range. From this, it may be deduced that the sensory stimuli that give rise to the CEP, are conducted largely along the posterior tract (dorsal column). In contrast, the ventrolateral tract appears, in the main, to determine the amplitude and the degree of the secondary response. Grieshop *et al.* (1970) and Blair *et al.* (1975), in patients with chronic pain, were able to produce a change in the CEP after stimulation of the tibial nerve, by direct stimulation of the dorsal columns in the lumbar region. They discovered a reduction in amplitudes, in particular of the late potential peaks. There was a direct correlation between the reduction in amplitude and the amelioration of pain. Like Giblin, Bergamini *et al.*, Namerow *et al.* and Baust *et al.*, we also observed the greatest changes in the CEP in myelopathies where the dorsal columns were severely affected, for example, in funicular myelosis and in tabes dorsalis. In some cases, the deformation of the potential was so marked that the reliable identification of the potential peaks, and therefore a comparison of the latencies with those of the healthy group, was not possible.

In a person with paraplegia where there is loss of all the sensory modalities, there will always be a marked change in the CEP. In those with complete paraplegia, no CEP can be induced by stimulating an area of skin distal to the 'sensitivity boundary'. Our findings with respect to segmental stimulation, confirm the reports of Baust *et al.* (1974), namely, that, by this means, an accurate localisation of the level of the process in the spinal cord can be obtained. In patients with cervical myelopathy, Matsukado *et al.* (1976) described a marked delay in the latencies of the CEP on stimulation of the median nerve. The CEP induced by stimulating the nerves in the arm, however, is, we believe, unsuitable for assessing cervical myelopathy, since, according to Desmedt *et al.* (1974), a radicular disorder can also lead to a delay in the CEP, and thus any conclusions regarding a circumscribed spinal cord lesion is no longer possible. In the case of diffuse diseases of the spinal cord, in particular those caused by lack of vitamin B₁₂, the peripheral, sensory and motor nerve conduction rates must, of course, be determined in order to exclude a neuropathic process as the cause of a change in the CEP. At the same

time, it is essential to exclude a disturbance in the central transmission of the stimulus in the brain-stem and in the cerebrum. The technique proposed by Ertekin (1976), involving the direct pick-up of the evoked potentials by introducing electrodes into the spinal canal, cannot yet be employed for clinical diagnosis.

Summary

Cortical-evoked potentials (CEPs) induced by the stimulation of the tibial nerve, were investigated in 16 patients with localised myelopathy, and in five patients with generalised myelopathy, and compared with the results obtained in 30 healthy test subjects.

The investigations show that the pulses that give rise to the CEP, are conducted mainly by the posterior columns. Damage to the ventrolateral tracts, in contrast, leads only to a reduction in the amplitude of the CEPs.

If account is taken of possible artefacts and misinterpretations, the method described is highly suitable for determining the level of a lesion in complete paraplegia and for obtaining information as to the size and extent of spinal cord damage in myelopathies.

RÉSUMÉ

Cérébraux évoqués potentiels (CEP) par stimulation du nerf tibial ont été étudiés chez 16 patients avec une affection spinale localisée et chez 5 patients avec une myélopathie généralisée. Les résultats ont été comparées avec les résultats obtenues chez 30 sujets normaux. La comparaison des résultats fait voir que les cordons postérieurs sont la voie principale des impulsions évoquant la réponse cérébrale. L'affection du cordon antérieure—latérale cause une diminution des amplitudes du CEP.

En tenant compte des artefacts possibles et des interprétations fautives la méthode décrite est propre pour déterminer le niveau de la lésion spinale chez les paraplégiques et pour éclaircir l'extension des lésions de la moelle chez les myélopathies.

ZUSAMMENFASSUNG

Cortikale evozierte Potentiale (CEP) durch Reizung des N. tibialis wurden bei 16 Patienten mit lokalisierter Myelopathie und bei 5 Patienten mit generalisierter Myelopathie untersucht und mit den Ergebnissen von 30 gesunden Kontrollpersonen verglichen. Die Untersuchungen zeigen, daß die für die Entstehung des CEP verantwortlichen Impulse hauptsächlich über die Hinterstrangsbahnen geleitet werden. Schädigungen der Vorderseitenstrangbahn führen dagegen nur zu einer Amplitudenreduktion des CEP. Bei Berücksichtigung möglicher Artefakte und Fehlbeurteilungen eignet sich die beschriebene Methode gut zur Höhenbestimmung bei kompletter Querschnittslähmung sowie zur Aussage über Umfang und Ausdehnung der Rückenmarksschäden bei Myelopathien.

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