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Motor Evoked Responses Recorded Epidurally in a Patient with Guillain-Barré Syndrome

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Summary. The case of a 75-year-old man with Guillain-Barré syndrome is presented. By means of transcranial electrical stimulation and epidural recording at the spinal level L2-3, distinct potentials with a latency of 21ms were obtained when the patient was tetraplegic. At the same time electromyographic responses of the thenar and anterior tibial muscles were absent following both transcranial and peripheral nerve stimulation. The patient recovered partially within 4 weeks. It is concluded that epidurally recorded motor evoked responses allow electrophysiological assessment of the descending pathways even in severe cases of Guillain-Barré syndrome and might contribute to a more accurate prediction of outcome.

Key words: Motor evoked potential – Guillain-Barré syndrome

Introduction

Transcranial electrical cortex stimulation is capable of eliciting motor evoked potentials (MEP), which can be recorded from the extremity muscles [7] and the epidural space along the spinal cord and cauda equina [2]. We used this technique in a patient who was tetraplegic from a Guillain-Barré syndrome for electrophysiological assessment of the descending pathways. The presence of responses recorded epidurally at the spinal level L2-3 combined with the absence of electromyographic (EMG) responses to transcranial and peripheral nerve stimulation led us to report our findings.

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Case Report

This 75-year-old male patient was reoperated on for right-sided coxarthrosis with implantation of an endoprothesis on 28 July 1987. Although the postoperative course was initially uneventful, he developed respiratory insufficiency and had to be reintubated and artificially ventilated on 5 August 1987. Neurological examinations revealed progressive tetraparesis and finally tetraplegia with absent tendon reflexes and additional involvement of the lower cranial nerves, yet he remained conscious. Neuroradiological investigation, including cranial CT and craniospinal MRI, showed no abnormalities, nor did vertebral angiography. CSF was inconspicuous except for an elevated protein level of 63mg%. Other laboratory tests including

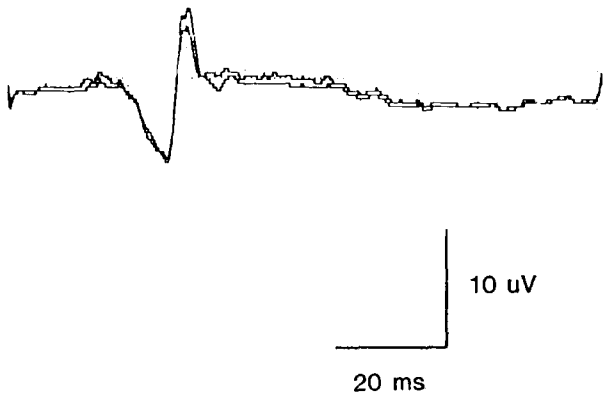

Fig. 1. Motor evoked responses recorded from the epidural space of the cauda equina at L2-3 following transcranial electrical stimulation of the motor foot area. The onset latency of the first initial negative wave – depicted downward – is 21 ms. The stimulus strength was 450 V. Two recordings are superimposed; 5 signals were averaged for each

cultural investigations for neurotropic infections of serum and CSF revealed no abnormalities. Electrophysiological examination of the sensory pathways showed absent neck and scalp responses to median and tibial nerve stimulation. There were also no EMG responses of the thenar and anterior tibial muscles on either side to peripheral nerve stimulation or transcranial electrical stimulation. With the latter method, no responses could be detected along the thoracic and lumbar spine using EMG surface electrodes. Therefore, we tried to record potentials from the cauda equina using a bipolar cardiac pacing electrode, which was inserted into the epidural space at L2-3 using a Tuohy needle. At this recording site, distinct potentials with a latency of 21ms were obtained following transcranial electrical stimulation of the motor foot area (Fig. 1). During the following weeks neurological deficits improved, and the patient was able to move arms and legs and could be mobilized. Respiration became sufficient so that the tracheostoma was closed on 5 September 1987.

Discussion

Non-invasive assessment of the descending pathways by means of transcranial excitation of the motor cortex has been available since 1980 using electrical stimulation [7], and since 1985 using magnetic stimulation [1]. So far there have been reports on the use of motor evoked potentials in electrophysiological evaluation of demyelinating diseases and other neurological disorders [3, 4, 8–11] as well as in intraoperative monitoring during orthopaedic [2] and neurosurgical [5, 6] operations on the spinal cord. Mills and Murray [8] examined 21 patients with early Guillain-Barré syndrome and found muscle weakness to be caused by a proximal conduction block between the spinal cord and the axilla, as demonstrated earlier by Brown and Feasby [3]. In the case presented here with examination when tetraplegia had developed, EMG responses of the thenar and anterior tibial muscles on both sides were not obtained, either by peripheral nerve or by transcranial stimulation. However, we did find distinct potentials epidurally at L2-3. The unusually long latency of these respones (21ms) contrasts with our experience in intraoperative MEP monitoring, where latencies of cauda equina potentials were found to range from 11 to 13ms. Additional recording along the spinal cord would have been helpful to evaluate conduction velocities in central and peripheral pathways. Furthermore, it cannot be decided whether cauda equina responses in our case represented neural or muscular activity. However, the absence of EMG responses in recordings along the thoracic and lumbar spine using surface electrodes seems to indicate neural activity, since our experience shows that muscular activity is more easily recorded along the spinal surface than epidurally.

In our opinion, epidurally recorded motor evoked responses allow electrophysiological assessment of the descending pathways in severe cases of Guillain-Barré syndrome when conventional measurement of distal motor latencies fails. Further observations will be necessary to decide whether the presence of cauda equina responses might contribute to a more accurate prediction of outcome in these cases.

References

1. Barker AT, Jalinous R, Freeston IL (1985) Non-invasive magnetic stimulation of human motor cortex. Lancet I: 1106–1107
2. Boyd SG, Rothwell JC, Cowan JMA, Webb PJ, Morley T (1986) A method of monitoring functions in corticospinal pathways during scoliosis surgery with a note on motor conduction velocities. J Neurol Neurosurg Psychiatry 49: 251–257
3. Brown WF, Feasby TE (1984) Conduction block and denervation in Guillain-Barré polyneuropathy. Brain 107: 219–239
4. Cowan JMA, Dick JPR, Day BL, Rothwell JC, Thompson PD, Marsden CD (1984) Abnormalities in central motor pathway conduction in multiple sclerosis. Lancet II:304–307
5. Levy WJ (1987) Clinical experience with motor and cerebellar evoked potential monitoring. Neurosurgery 20:169–182
6. Levy WJ, McCaffrey M, York DH, Tanzer F (1984) Motor evoked potentials from transcranial stimulation of the motor cortex in humans. Neurosurgery 15:214–227
7. Merton PA, Morton HB (1980) Stimulation of the cerebral cortex in the intact human subject. Nature 285:227
8. Mills KR, Murray NMF (1985a) Proximal conduction block in early Guillain-Barré syndrome. Lancet I:659
9. Mills KR, Murray NMF (1985b) Corticospinal tract conduction time in multiple sclerosis. Ann Neurol 18:601–605
10. Rossini PM, Caramia MD, Zarola F (1987) Mechanisms of nervous propagation along central motor pathways: noninvasive evaluation in healthy subjects and in patients with neurological disease. Neurosurgery 20:183–191
11. Snooks SJ, Swash M (1985) Motor conduction velocity in the human spinal cord: slowed conduction in multiple sclerosis and radiation myelopathy. J Neurol Neurosurg Psychiatry 48:1135–1139

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