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MEDIAN NERVE SEP AFTER A HIGH MEDULLARY LESION

PRESERVED N18 AND ABSENT P14 COMPONENTS

Case Report

G. M. MANZANO, R. R. SCHULTZ**, O. G. P. BARSOTTINI**, E. ZUKERMAN***, J. A. M. NÓBREGA****

ABSTRACT - Median nerve SEPs recorded from a patient with a high medullary lesion are described. The lesion involved the anteromedial and anterolateral right upper third of the medulla, as documented by MRI. Forty one days after the lesion, left median nerve SEP showed preserved N18 and absent P14 and N20 components; stimulation of the right median nerve evoked normal responses. These findings agree with the proposition that low medullary levels are involved in the generation of the N18 component of the median nerve SEP.

KEY WORDS: median nerve, somatosensory evoked potential, medulla, subcortical.

PESS do nervo mediano após lesão bulbar alta: componente N18 preservado e P14 ausente. Relato de caso

RESUMO - Descrevemos os potenciais evocados somatossensitivos obtidos por estimulação dos nervos medianos em um paciente apresentando uma lesão localizada, envolvendo as porções anteromedial e anterolateral do terço superior do bulbo, documentada por ressonância nuclear magnética. Quarenta e um dias após o estabelecimento da lesão os potenciais evocados por estimulação do nervo mediano esquerdo evidenciaram ausência dos componentes P14 e N20 e preservação do componente N18; após estimulação do nervo mediano direito as respostas apresentaram-se normais. Estes achados estão de acordo com a sugestão de que as porções baixas do bulbo estão envolvidas na geração do componente N18.

PALAVRAS-CHAVE: nervo mediano, potencial evocado somatossensitivo, bulbo, subcortical.

The N18 component of the median nerve somatosensory evoked potential (SEP) was described in 1981[1]. Since then substantial controversy has developed in relation to its generator structure. While during the eighties, generator structures were suggested to be located from the frontal cortex[2] to the midbrain[3], during the nineties discussions have been focused from the midbrain[4-6] to the medulla[7-14]. While the first discussion was solved by the clear documentation of persistence of the N18 component in cases with hemispherectomies[3], the second discussion (i.e., from the midbrain to the medulla) is still going on. The recent descriptions of selected cases with well defined lesions at the high medulla, showing preservation of the N18 component[9,13], suggest that indeed the lower medulla is capable of generate the N18 component. However, some contradictions still persists[15] and this point does not seem to be universally accepted[16]. In view of this picture we believe that it is pertinent to report the SEP findings of another case with a lesion at high medullary level.

Neurology and Neurosurgery Department, Escola Paulista de Medicina, UNIFESP: *Professor Substituto; **Pos-Graduando; ***Professor Adjunto.Aceite: 8-dezembro-1998.

Dr. Gilberto Mastrocola Manzano - Rua Berta 204 - 04120-040 São Paulo SP - Brasil Fax 011 814 3966. E-mail Jnobreg@ibm.net

CASE REPORT

A 31 year old man, woke up feeling nausea, dizziness and paresthesias / weakness on the left side of the body. He sought medical attention, was submited to a computerized tomographic (CT) scan of the head, received an "intravenous medication" and, after experiencing some improvement, was sent home. On this same day in the afternoon, he had one episode of vomiting and noticed that the left side of the body became completely paralyzed. At this point he was admitted to a hospital. At admission the patient was oriented, mildly dysarthric and presented a spastic hypertonia, hemiplegia and enhanced tendon reflexes at the left side of the body. Babinski sign was observed bilaterally as well as an upbeat nistagmus. He also showed to have positional and vibratory sensory loss and some reduction in tactile and pain sensibilities on the left side of the body. A magnetic resonance image (MRI) of the head showed an infarct zone involving the right anteromedial and anterolateral artery territories limited to the high meddullary region (Fig 1). The patient was medicated with anticoagulants and showed a progressive improvement. Forty one days after the ictus, the patient was sent for a SEP study.

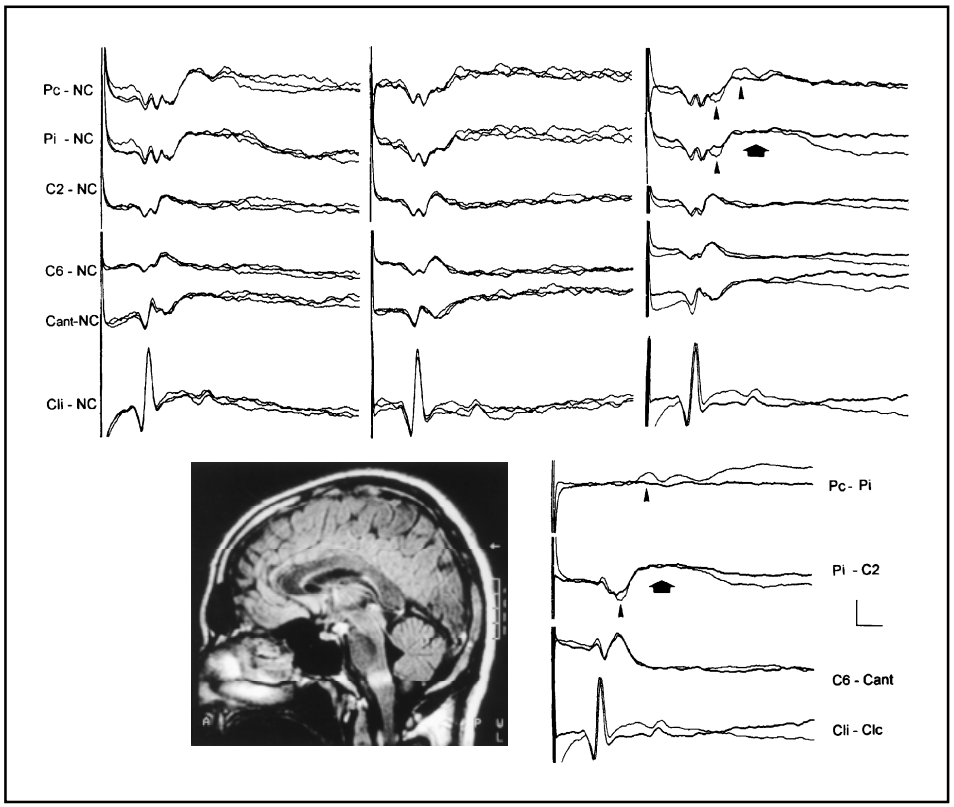

Fig 1. The left group of curves displays the responses evoked after stimulation of the right median nerve. The group at the center shows the curves obtained after stimulation of the left median nerve. The upper right group shows the mean responses of the replications after stimulation of the right (thin traces) and left (thick curves) median nerves, superimposed. The lower right group of curves shows bipolar montages derived from the mean of the original recordings, responses from the right and left median nerves are superimposed. The MRI shows the upper and lower limit of the lesion at the midline. Arrow heads point to P14 and N20 components, thick arrows point to the N18 component. Pc and Pi refers to the parietal electrodes related to the stimulation of the contra and ipsilateral median nerves respectively; NC refers to non-cephalic reference. Calibration: 2μV and 5ms.

METHOD

Somatosensory evoked responses were recorded through surface electrodes (Ag/AgCl) fixed with conducting paste to the skin, which was cleaned and subjected to a mild abrasion, giving an inter electrode impedance of 5 KOhms or less. Electrodes were placed over both parietal and both frontal regions (F3, F4, P3, P4 of the 10-20 international system), at the posterior surface of the neck at the level of the second (C2) and the sixth (C6) cervical vertebrae, at the anterior surface of the neck (Cant) at the level of the cricoid cartilage and over the left (Cli) and right (Clc) clavicles. Montages were F4-Clc, F3-Clc, P4-Clc, P3-Clc, C2-Clc, C6-Clc, Cant-Clc and Cli-Clc. Ground electrode was applied over the forehead (Fpz of the 10-20 international system).

The median nerves were stimulated independently at the wrist, with the cathode proximal (bipolar saddle type electrode). Stimulus consisted of a rectangular electrical pulse of 200 μs duration delivered at a frequency of 5/s, with the intensity defined by the sum of the sensory and motor thresholds. Responses where recorded at a Nihon-Khoden machine, model 5508, in eight simultaneous channels with 1024 points per channel over an analysis time of 100 ms. Filters were set at 5 and 3000 Hz and 1000 responses were averaged and replicated twice after each median nerve stimulation.

RESULTS

After stimulation of the right median nerve, N9, P9, P11, P14, N13 at C2 and C6, P13 at Cant, N18 and N20 were recorded with latencies within normal limits (Fig 1). After stimulation of the left median nerve, components N9, P9, P11, N13 at C2 and C6, P13 at Cant and the N18 component were normal, however P14 was not identifiable as well as the cortical activities at the contra-lateral parietal electrodes (Fig 1). For clarity, bipolar montages reconstructed from the original data are also shown on Figure 1.

DISCUSSION

As documented by the image studies, the lesion was located at a high medullary level. The unilateral involvement of the medial lemniscus lead to abolition of the P14 component, however the N18 component remained. These findings are in agreement with the recent cases described[9,13,15].

The case reported by Sonoo et al.[13] and two cases reported by Hashimoto et al.[15] documented the full preservation of the component, however in the two other cases reported by Hashimoto et al.[15] the component was either absent or reduced in amplitude, it should be noted that the recordings presented of the case with absent component are severely distorted by stimulus artifact. In the case reported by Noël et al.[9] the component showed a different profile although the amplitude could not be considered definitively reduced and the authors believed that this was related to poor technical conditions of the recording which was carried out on an adverse environment.

In our case, although the profile of the component was different from the contralateral side it could still be recorded. Therefore, the present case and the others described with similarly located lesions favors of the proposition that implicates low medullary levels in the generation of the N18 component of the median nerve SEP.

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