

ORIGINAL ARTICLE

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Brainstem evoked potentials in three groups of prisoners after release from detention camp

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Abstract Brainstem evoked potentials (BAEPs) were determined in three groups of male prisoners of war (POWs) released from detention camps and a control group. The first group comprised 21 POWs in whom BAEPs were determined 10–60 days after release (group I). The second group comprised 24 POWs in whom BAEPs were determined 6–9 months after release (group II), and the third group comprised 22 POWs in whom BAEPs were determined 12–18 months after release (group III). The control group comprised 32 subjects. The following changes were found in relation to the control group: in group I significantly longer interpeak latencies (IPLs) P1–P3; in group II significantly longer IPLs P1–P3 and P3–P5; and in group III significantly longer IPLs P1–P3. The subjective symptomatology of the POWs and the results of a routine examination indicate subclinical functional changes of the central nervous system, reflecting the dynamics of these changes. It is suggested that the basis of these changes may be a demyelination intrathecal process, which occurred as a result of immunological changes during prolonged and intensive post-traumatic stress syndrome.

Key words Brainstem evoked potentials · Detention camps

Introduction

The majority of released prisoners of war (POWs) show signs of deteriorated physical and mental health (Kozarić-Kovačić et al. 1993; Solar 1993 a, b; Lončar 1993; Borčić et al. 1992; Novotny 1992; Brasko-Brnčić and Brnčić 1993; Vrca and Malinar 1995; Vrca and Bobić 1993). The aim of this investigation was to use brainstem evoked potentials (BAEPs) to follow-up the POWs in order to obtain evidence of the changes and to register their dynam-

ics (Gilmore 1988; Niedermayer 1990; Chiappa 1990 a). The evaluation of BAEPs is a very sensitive neurophysiological technique which almost entirely excludes the subjective factor and partially covers the so-called subclinical side in functional examination of the central nervous system (CNS) (Chiappa 1990b; McPherson and Starr 1993; Gilmore 1988; Niedermayer 1990).

Subjects and methods

Out of approximately 6,500 released POWs 1,500 were examined by a neurologist because of suspected neurological impairment, previous neurological disease, loss of consciousness and blows to the head and neck during imprisonment, 350 of them in the Outpatient Department of Neurology, Institute for Medical Research and Occupational Health.

Three groups of POWs were selected at random from the group of 350. The first group (group I) comprised 21 subjects in whom BAEPs were examined 10–60 days after release (mean 25.7 days, SD 16). The second group (group II) comprised 24 subjects in whom BAEPs were examined 6–9 months after release (mean 212.5 days, SD 43.55), and the third group (group III) comprised 22 subjects in whom BAEPs were examined 12–18 months after release (mean 437.7 days, SD 41.5). A control group comprised 32 subjects. All subjects were male, and groups were matched with regard to age and education. The mean age in group I was 34.6 years (SD 9), in group II 34.9 years (SD 8.3), in group III 34.2 years (SD 8.4), and in the control group 35.2 years (SD 7.5). The mean duration of education in groups I and II and the control was 10.5 years, and in group III 11 years. There were no significant differences with regard to the duration of imprisonment among the three groups of POWs (group I 173.7 days, SD 46.6; group II 193 days, SD 61.6; group III 189 days, SD 63.7). None of the subjects in the examined groups had previously been arrested or imprisoned, treated for chronic disease, suffered serious injury to the head and neck, or had lost consciousness prior to imprisonment in the camp. None had ever had hearing problems or any diseases of the ear. Medications had not been taken for long periods, and those taken were usually analgesics or antibiotics. All the POWs examined were referred to the institute because of reports of blows to the head and neck and/or loss of consciousness (25 cases) during imprisonment.

A Brain-Imager apparatus manufactured in 1988 by Medilog-Neuroscience was used in the study. BAEPs were determined by stimulating each ear separately with unstructured stimuli the "click" type (2048 times, frequency 15 stimuli per second, intensity 70 dB SPL). At the same time the other ear was subjected to

masked noise with an intensity of 50 dB SPL. Responses occurring within the first 10 ms after commencement of stimulation were recorded by a Cz electrode (10–20 international system). A reference electrode was attached to the earlobe. Filtration limits were 150–1500 Hz. During the recording the room was darkened and subjects were asked to keep their eyes closed. The temperature in the room ranged from 19 to 21°C. Amplitudes and latencies of waves P1, P2, P3, P4 and P5 and interpeak latencies (IPLs) P1–P5, P1–P3 and P3–P5 were analysed. For each subject the values for the right and left ear were added and divided by 2. Data were statistically analysed by the Mann-Whitney *U*-test and a result in which *P* was less than 0.05 was considered significant (Barry 1990).

Results

In group I no significant differences were found in the latencies and amplitudes of the examined BAEP waves in relation to the control group. However, significantly longer IPLs were found for P1–P5 and P1–P3 (Table 1). In group II no significant differences in amplitudes were found for the examined BAEP waves compared to the control group. However, significantly longer latencies were found for the P4 and P5 waves and significantly longer IPLs for P1–P3, P3–P5 and P1–P5 (Table 2). In group III no significant differences were found in the latencies of most of the BAEP waves, in relation to the control group. However, a significantly smaller amplitude was found for the P1 wave and significantly longer IPLs for P1–P3 and P1–P5 (Table 3).

When the numbers of pathological parameters for BAEPs were calculated, according to $\pm 2SD$ of the control group, there were 13.61% in group I, 8.33% in group II, 5.81% in group III, and 3.65% in the control group (Table 4).

The difference in the number of pathological parameters for each group of POWs in relation to the control

Table 1 BAEPs in a group of POWs 10–60 days after release from detention camps (group I) and in a control group (A wave amplitude, L wave latency, NS not significant)

Wave	Group I <i>n</i> = 21 (mean \pm SD)	Control group <i>n</i> = 32 (mean \pm SD)	Statistical significance
P1 A	0.29 \pm 0.11	0.30 \pm 0.12	NS
P1 L	1.78 \pm 0.19	1.85 \pm 0.19	NS
P2 A	0.21 \pm 0.10	0.20 \pm 0.90	NS
P2 L	3.02 \pm 0.23	3.03 \pm 0.18	NS
P3 A	0.25 \pm 0.10	0.22 \pm 0.08	NS
P3 L	4.07 \pm 0.25	4.02 \pm 0.19	NS
P4 A	0.23 \pm 0.12	0.18 \pm 0.10	NS
P4 L	5.32 \pm 0.33	5.24 \pm 0.25	NS
P5 A	0.38 \pm 0.12	0.36 \pm 0.10	NS
P5 L	6.14 \pm 0.31	6.03 \pm 0.24	NS
P1–P5	4.36 \pm 0.23	4.18 \pm 0.13	<i>P</i> < 0.01
P1–P3	2.29 \pm 0.19	2.18 \pm 0.14	<i>P</i> < 0.05
P3–P5	2.07 \pm 0.19	2.01 \pm 0.15	NS

Table 2 BAEPs in a group of POWs 6–9 months after release from detention camps (group II) and a control group

Wave	Group II <i>n</i> = 24 (mean \pm SD)	Control group <i>n</i> = 32 (mean \pm SD)	Statistical significance
P1 A	0.32 \pm 0.15	0.30 \pm 0.12	NS
P1 L	1.79 \pm 0.19	1.85 \pm 0.19	NS
P2 A	0.19 \pm 0.13	0.20 \pm 0.19	NS
P2 L	3.09 \pm 0.25	3.03 \pm 0.18	NS
P3 A	0.22 \pm 0.08	0.22 \pm 0.08	NS
P3 L	4.09 \pm 0.23	4.02 \pm 0.19	NS
P4 A	0.21 \pm 0.09	0.18 \pm 0.10	NS
P4 L	5.40 \pm 0.30	5.24 \pm 0.25	<i>P</i> < 0.05
P5 A	0.32 \pm 0.11	0.36 \pm 0.10	NS
P5 L	6.27 \pm 0.29	6.03 \pm 0.24	<i>P</i> < 0.01
P1–P5	4.47 \pm 0.22	4.18 \pm 0.13	<i>P</i> < 0.001
P1–P3	2.30 \pm 0.21	2.18 \pm 0.14	<i>P</i> < 0.05
P3–P5	2.18 \pm 0.22	2.01 \pm 0.15	<i>P</i> < 0.01

Table 3 BAEPs in a group of POWs 12–18 months after release from detention camps (group III) and a control group

Wave	Group III <i>n</i> = 22 (mean \pm SD)	Control group <i>n</i> = 32 (mean \pm SD)	Statistical significance
P1 A	0.41 \pm 0.18	0.30 \pm 0.12	<i>P</i> < 0.05
P1 L	1.75 \pm 0.14	1.85 \pm 0.19	NS
P2 A	0.19 \pm 0.11	0.20 \pm 0.19	NS
P2 L	3.05 \pm 0.18	3.03 \pm 0.18	NS
P3 A	0.22 \pm 0.08	0.22 \pm 0.08	NS
P3 L	4.05 \pm 0.20	4.02 \pm 0.19	NS
P4 A	0.20 \pm 0.13	0.18 \pm 0.10	NS
P4 L	5.27 \pm 0.18	5.24 \pm 0.25	NS
P5 A	0.35 \pm 0.11	0.36 \pm 0.10	NS
P5 L	6.08 \pm 0.18	6.03 \pm 0.24	NS
P1–P5	4.33 \pm 0.18	4.18 \pm 0.13	<i>P</i> < 0.01
P1–P3	2.30 \pm 0.17	2.18 \pm 0.14	<i>P</i> < 0.01
P3–P5	2.03 \pm 0.16	2.01 \pm 0.15	NS

group was statistically significant, while between group I and group II the difference was not significant. However, a significantly larger number of pathological findings for BAEP was found for group I than for group III (Table 4).

Discussion

In group I changes were indicated in the lower part of the brainstem and auditory nerves (Chiappa 1990a; McPherson and Starr 1993). In group II progressive changes were indicated in relation to group I. These changes were registered throughout the whole of the auditory pathway up to the level of the auditory colliculus in the mesencephalon.

Table 4 Number of pathological parameters according to $\pm 2SD$ of the control group in three groups of POWs and the control group

Wave		Control group <i>n</i> = 32 (parameters <i>n</i> = 576)	Group I <i>n</i> = 21 (parameters <i>n</i> = 378)	Group II <i>n</i> = 24 (parameters <i>n</i> = 432)	Group III <i>n</i> = 22 (parameters <i>n</i> = 396)
P1	A	2	3	3	9
	L	3	1	1	0
P3	A	2	4	3	1
	L	4	5	5	3
P5	A	2	4	5	3
	L	3	5	9	1
P1–P5		1	8	4	2
P1–P3		1	3	3	3
P3–P5		3	3	3	1
Total		21 (3.65%)	36 (13.61%)	36 (8.33%)	23 (5.81%)

Group I: control group $P < 0.01$, group I: group II NS, group I: group III $P < 0.05$, group II: control group $P < 0.05$, group II: group III NS, group III: control group $P < 0.05$

In group III the changes found indicate regression in relation to those found in group II, which only indicate changes in the lower part of the brainstem and auditory nerve, as in group I.

Although the changes found in this investigation cannot be considered specific, based on the results of previous studies (Vrca and Bobić 1993; Vrca and Malinar 1995) they suggest the possibility of a demyelination process. The demyelination process can most likely be attributed to an altered immunological status. This corroborates the findings of a study by Dekaris et al. (1993), who found significant immunological changes in POWs released from detention camps. We speculate that these changes can be attributed to post-traumatic stress syndrome in the POWs.

On the basis of anamnestic data and routine neurological examinations, it can be concluded that the BAEP changes are most likely subclinical (Chiappa 1990b; McPherson and Starr 1993). However, the question remains whether and to what extent these changes will be a health risk for the POWs in the future.

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