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Average Evoked Responses (AER) and Homovanillic Acid (HVA) in Cerebrospinal Fluid (CSF) in a Psychotic Patient with Addison's Disease*

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Summary. Serial determinations of averaged evoked responses (AER) and homovanillic acid (HVA) in cerebrospinal fluid (CSF) were made during the psychotic period and recovery phase in a patient with Addison's disease. The patient was treated with cortisone continuously, 2 series of Uni-ECT without effect in the early case history and neurolepties (haloperidole), giving complete remission, at the end.

Clinical recovery was accompanied by a 7 fold increase of HVA in CSF, AER-amplitudes increased with about 30% and AER-latencies diminished with about 7%.

Key words: Average Evoked Responses (AER) — Homovanillic Acid (HVA) — CSF — Addison's Disease — Psychosis — Electroconvulsive Treatment (ETC).

Zusammenfassung. Mehrfache Registrierungen der gemittelten, visuellen und somatosensorischen evozierten Hirnpotentiale (AER) und der Homovanillinsäure (HVA) im Liquor cerebrospinalis (CSF) wurden während einer Psychose und nachfolgender Besserung bei einem Patienten mit Addisonscher Krankheit durchgeführt. Der Patient war mit Cortison kontinuierlich und anfangs mit zwei Serien von Elektroschocks ohne Erfolg behandelt. Mit Haloperidol wurde eine vollständige Remission erreicht.

Die klinische Besserung war von einer siebenfachen Erhöhung der HVA im Liquor gefolgt, die AER-Amplituden wurden um 30% größer und AER-Latenzzeiten um 7% verkürzt.

Schlüsselwörter: Gemittelte, visuelle und somatosensorische evozierte Hirnpotentiale (AER) — Homovanillinsäure — Liquor cerebrospinalis — Addinsonsche Krankheit — Psychose — Elektroschock.

Introduction

We have elsewhere (Mattsson, 1974) reported in detail the case history of a patient with Addison's disease who was treated at the Psychiatric Clinic in Umeå for a psychotic episode.

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In this article we wish to draw attention to serial findings concerning averaged evoked responses (AER) and homovanillic acid (HVA) in the cerebrospinal fluid (CSF), obtained during different phases of the disease and its treatment, and after recovery from the psychotic episode.

Case History

The patient, a 20-year-old student, fell ill in November 1970 with increasing stomach pains, fatigue, a feeling of sickness and a tendency to faint. He was admitted to the Medical Clinic in December the same year. Tests showed low hydrocortisone values in plasma, no response to ACTH loading, low values for 17-keto-17-hydroxysteroids in urine; Kepler's test positive. Immunological examination showed the existence of antibodies to adrenal cortex, whereupon Addison's disease of the so-called auto-immune type could be diagnosed. The patient received substitutive cortisone treatment with 50 mg/day cortisone and 1 mg/day fluorhydrocortisone. In February 1971 the patient developed a psychotic picture with confusion, desorientation, inadequate responses with long latency period. He was admitted to the Psychiatric Clinic and he deteriorated with delusions of allusive and persecutory nature; felt he was being listend to by the radio and observed by TV. Contact became increasingly insufficient; the patient showed splitting and virtual blocking. Hydrocortisone/plasma was within normal variation limits. He received, as well as cortisone treatment, 5 unilateral electro-convulsive treatments (Uni-ECT) which were followed by a temporary improvement. Five further Uni-ECT were administered about 1 month later, with subsequent clinical deterioration, but haloperidole 2 mg/day led to a pronounced improvement and final discharge. At out-patient controls, most recently August 1973, the patient has shown complete recovery from the psychotic syndrome.

Investigations

Among the investigations performed, a serial study of AER to both visual and somato-sensorial stimuli was made. These investigations were carried out on the following occasions: on admission, after the first Uni-ECT, after the third Uni-ECT, after completed Uni-ECT treatment and at an out-patient control in October 1971. The usual recording technique was used, already described elsewhere (Perris, 1974). In addition the HVA concentration in liquor was spectrophotometrically determined (Korf, 1971) with the cooperation of Dr. Sven-Åke Persson of the Department of Pharmacology, Umeå University. These determinations were made before Uni-ECT, immediately after the first 5 Uni-ECT and at an out-patient control in October 1971, the last occasion 6 months after Uni-ECT, and when continuously on haloperidole and cortisone treatment (see Table 1; test occasion 1, 4, 5). The patient participated, voluntarily in the investigations.

Results

Averaged evoked responses to visual stimuli (V.AER) and those to somatosensory stimuli (SS.AER) were numbered in accordance with current international norms (Figs. 1 and 2), and from these amplitudes

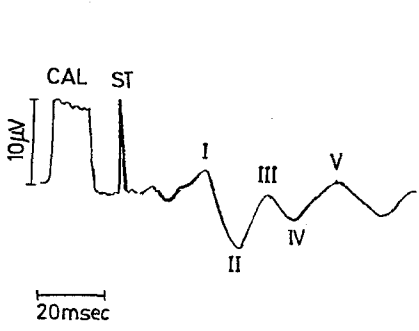

Fig. 1

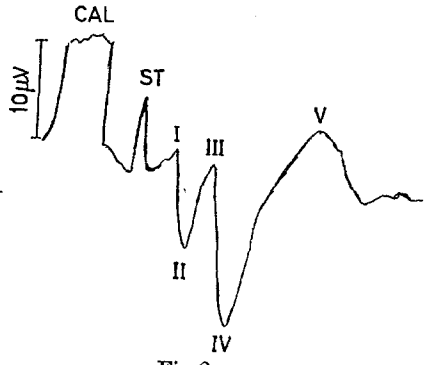

Fig. 2

Fig. 1. A typical visual AER showing the numeration of the peaks
Fig. 2. A typical somatosensory AER showing the numeration of the peaks

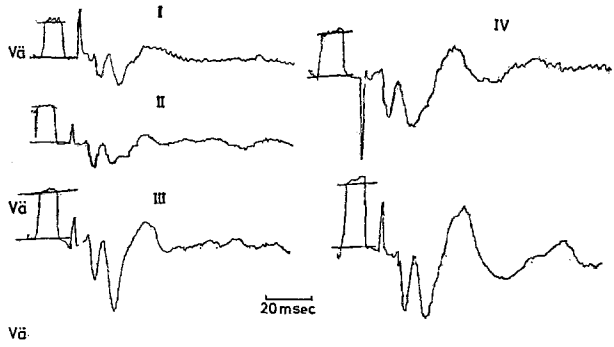

Fig. 3. Visual evoked responses from the patient at the different test occasions corresponding to those indicated in Table 2. Calibration 10 μV as in Figs. 1 and 2

and latencies were calculated. Results are shown in Fig. 3 and Tables 1 to 2. From these it emerges that the amplitudes for both V.AER and SS.AER increased progressively with the psychic improvement at the same time as the latencies diminished from before treatment up to the out-patient control when the patient was completely recovered. Similarly, the level of HVA in CSF showed a progressive increase. SS.AER amplitudes on test occasion 4 were somewhat higher than on occasion 5, in contrast to the HVA-level which was higher at the final control. On the later occasion, however, the patient was being treated with haloperidole. The differences between V.AER amplitudes were continual, and most obvious for the first two components.

Table 1. Over all results of SS.AER measurements[a], amount of HVA in CSF, treatment and psychic condition at the test occasion

Test occasion	Amplitude (μV) I-II	II-III	III-IV	IV-V	Latency (mm[b]) to peak 1	2	3	4	5	HVA ng/100 ml	Treatment	Psychic state	Observ.
1	6.5	6.5	8.5	11.0	10	14	19	25	41	3	cortisone	psychotic	
2	7.5	6.5	6.5	8.5	9	13	18	23	43		+1 ECT	psychotic	
3	9.3	7.8	14.3	16.4	8	12	17	23	42		+3 ECT	psychotic	
4	10.0	7.4	10.0	17.3	8	12	16	23	41	11	+5 ECT	improved	
5	9.7	9.0	10.0	18.3	8	12	17	23	41	23	+halo-peridole	recovered	

[b] 10 mm = 12.5 msec

[a] Since the pat. received Uni-ECT treatment on the right side, measurements of amplitudes and latencies have been consequently made on the left side both as concerns SS.AER and V.AER (Table 2).

Table 2. Over all distribution of amplitudes and latencies of V.AER

Test occasion	Amplitude (μV) 1-II	II-III	III-IV	Latency (mm[b]) to peak 1	2	3	4	HVA ng/100 ml	
1	(7.0)	(6.0)	(7.0)[a]	—	—	—	—	3	psychotic
2	8.0	6.7	6.3	22	30	39	53		
3	10.0	7.9	5.9	21	31	38	53		
4	10.0	8.2	4.6	21	29	38	53	11	improved
5	13.4	12.0	10.6	19	27	47	59	23	recovered

[a] Depending upon low amplication responses at the first test occasion were of extremely low amplitude and difficult to assess in detail.

Discussion

In the patient described there seems to have been a connection between clinical improvement, increasing amplitudes, diminishing latencies of evoked potentials and raised HVA-level in CSF. The discrepancy between SS.AER-amplitude and HVA-level at the fourth and fifth investigations may be accidental, but the recently completed electroconvulsive treatment may have contributed to the higher amplitude on test occasion 4. The patient was moreover being treated with haloperidole on the final test occasion; haloperidole is known to increase the concentration of HVA in CSF, as a consequence of a dopamine receptor-blocking effect. V.AER and HVA show a somewhat more clear-cut relation, which can be explained by the fact that V.AER comprise a more complicated reaction system involving several pathways. Possible relations between AER and the metabolites of the catecholamines have been pointed out in another connection by Goodwin *et al.* (1970). Our findings seem to be in line with the results of these authors, and may provide stimuli for further studies leading to possible causal connections.

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