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Some Comments on the Clinical Use of Evoked Potentials

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Summary. In this survey we describe the uses of somatosensory, visual, and auditory evoked potentials (EPs), adding some critical comments on the values and pitfalls of these methods. It must be stressed that the application of EPs is most valuable when combined with a thorough neurological examination. There is general agreement that EP measurement is one of the best techniques for objective, noninvasive study of brain function in humans.

Key words: Evoked potentials – Somatosensory evoked potentials – VEP – AEP

Introduction

In the last decade a larger number of papers dealing with various aspects of evoked potentials (EPs) has been published [4, 5, 8, 18, 19, 24, 26, 27, 45, 63], and EPs have in recent years become increasingly popular. Several books on the subject appear annually, describing a widening range of possibilities for application. Some applications of visual (VEP), somatosensory (SSEP), and auditory (AEP) EPs have already been delineated, while others are still under investigation [70].

Concerning the Contingent Negative Variation (CNV) most investigators are interested in the relation between the CNV amplitude and psychopathology, mainly concentrated on schizophrenic psychosis, in which for the most part lowered amplitudes have been found.

Research on P300 demonstrated that this slow potential is mainly determined by the subjective intention of the subject existing before the information (stimulus) concerned is offered. It seems reasonable to assume that the take-off point of CNV is influenced by the distribution and the amplitude of P300. Some authors believe in a hypothetic relation between decreased CNV amplitude, increased arousal, and psychosis. In this way research and registration of CNV and several late components such as P 300 could become a real diagnostic instrument in clinical psychiatry [67, 68, 107, 108, 111].

The combination of low amplitude CNV and long duration of the Spiral After Effect (SAE) illusion of movement characterizes patients with florid schizophrenic symptomatology. The SAE returns to normal with remission of symptoms, thus qualifying as a marker of the schizophrenic state. The status of CNVs as a trait marker has been reinforced by the observation that patients with lower amplitude CNVs had a very much poorer clinical and social outcome than others with CNVs in the normal range [1].

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Shagass et al. [92] reported differences in the topography of the different SSEP components in patients with psychotic depression and chronic schizophrenia in relation to normals.

There is a large field of literature regarding VEP applications in functional psychiatric disorders [93]. Possibly the VEP can be used to predict worsening or improvement of the psychiatric disease [91]. An extensive description of the effects of drugs on EPs, especially in psychiatric patients, has been provided by Shagass and Straumanis [94].

One of the most widespread applications of multimodality VEPs, SSEPs and AEPs has been in the evaluation of multiple sclerosis [6, 9, 61]. These potentials have also proven to be clinically useful in judging the prognosis of comatose patients, in differentiating between toxic and metabolic causes of irreversible coma [43, 59, 60, 71, 80, 97, 99, 100]. The tests have also been used in monitoring the reversibility of neuronal dysfunction in scoliosis surgery of spinal cord, posterior fossa, and optic pathway [37, 55, 57, 69, 102, 112], and for the follow-up of patients with Friedreich's ataxia [78, 79].

In this article we would like to evaluate the importance of the clinical application of the three main EP modalities.

Clinical Application of Somatosensory EP

SSEPs can be derived from the spinal column, the brainstem and the cerebral hemispheres [35] (Fig. 1). A critical review of the early and middle latency SSEP has been published by Celesia [16] and Stockard [101].

In clinical practice, application of SSEP has proven useful in two distinct areas. In the first place it enables examination of sensory propagation along the myelinated pathway from the point of stimulation right up to the cerebral cortex [33, 41, 54]. This is particularly important in diseases where lesions may be found in the peripheral nerve, the plexus, the cervical roots, the cervical column, or the brainstem [3, 56, 82, 117].

Somatosensory EPs may be derived from different points along this pathway and can, if necessary, be compared to EPs derived after stimulation of the contralateral side of the body. Thus, in these cases the primary specific component of the SSEP as an indication of a lesion in the white matter is important. Bearing this in mind, one is able to locate lesions in this pathway and obtain an impression of their severity [56, 104].

In the central nervous system, the method can be applied to detect clinically silent lesions in suspected multiple sclerosis, thus confirming what may be a difficult diagnosis by providing evidence of multiple lesions [21, 61, 68]. Moreover, in patients with sensory disturbances due to central pathology

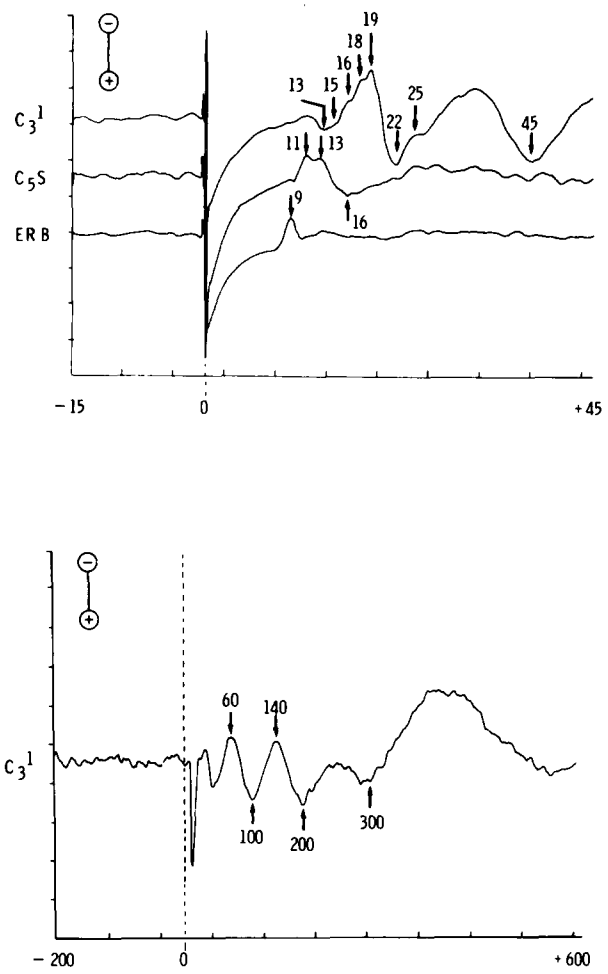

Fig. 1. Example of a median nerve SSEP, derived from C_3^1–A_2. *Upper trace* short latency, *lower trace* long latency. The short latency SSEP was elicited after 1000 electric stimuli, 0.1 ms duration, twitch level, 3.1/s, regular stimulation, bandpass 30–3000 Hz sample frequency 8000 Hz. The long latency SSEP was elicited after 100 random stimuli, 0.1 ms duration, twitch level, 0.3/s, bandpass 1–250 Hz and a sample frequency of 600 Hz

the SSEP findings can help locating lesions in the nerve or subcortical regions, and in the latter can help to define the extent to which the cauda equina [36] or lemniscal fibres [74, 76, 95] are involved. In conjunction with other electrophysiological EP techniques, it may in the future also be of value in determining the prognosis for ultimate recovery in unresponsive, comatose patients, but here more research is required [44].

In the second place it enables one to examine the propagation of sensory information over the cortical hemispheres. Pathology of the cortical and subcortical neurons ("gray matter") causes changes in the late components of the SSEP. In diseases where diffuse dysfunction of the cortical gray matter exists, the changes in the late components of the SSEP may appear to be more extensive than the alterations in the EEG, e.g., in mucopolysaccharidosis, ceroid lipofuscinosis, or Huntington's chorea [2, 22, 42, 77]. Studies of EPs have indicated their usefulness in brain maturation studies and have made possible the identification of central dysfunctions which otherwise would have become clinically obvious only at a later stage of development [62, 84, 90, 98]. The somatosensory responses are reduced or disappear in dorsal column lesions, but remain almost unaffected in patients with loss of pain and thermal sensations [17, 73]. The early components of the SSEP are unchanged in hypnotic or hysterical anesthesia [65, 66]. The responses can be reduced or enhanced without change in latency in certain parietal lesions [73]. More investigative work is required to help differentiate response characteristics which are associated with the various clinical conditions involving the somatosensory system [34].

Clinical Application of Visual EP

Visual EPs are derived from the occipital region after flash or checkerboard stimulation, and originate from the eye, the optic pathway, and the brain (Fig. 2).

Visual EPs may be helpful in detecting retinal function abnormalities, and anterior visual pathway and retrochiasmal pathway diseases [15]. Local cortical abnormalities may also be detected by VEP [32].

Subclinical involvement of the chiasma and optic nerve, especially in cases that are not always detectable by means of routine ophthalmological examination, can often be indicated by VEPs [46, 47, 48, 50, 51, 75]. Visual EP abnormalities are not specific to any particular disorder and are certainly not pathognomonic of multiple sclerosis. The same abnormalities can be encountered in patients with compressive lesions of the anterior pathways [52]. Changes seen in these patients – i.e., abnormal peak latency, a typical wave form, and a reduced amplitude – are different from those seen in patients suffering from a primary demyelinating disease, where the wave forms are typical or show prolonged latency but otherwise have a normal morphology and amplitude [109]. Visual EPs obtained by pattern-reversal are a reliable and sensitive tool for detecting lesions of the optic nerve [12, 14].

Laboratories employing this technique should establish their own standards because of the great variability in commercially available visual stimulating systems [49, 96]. However, one should bear in mind the fact that the clinical significance of an abnormal VEP will depend on the context in which the examination has been performed [50, 88].

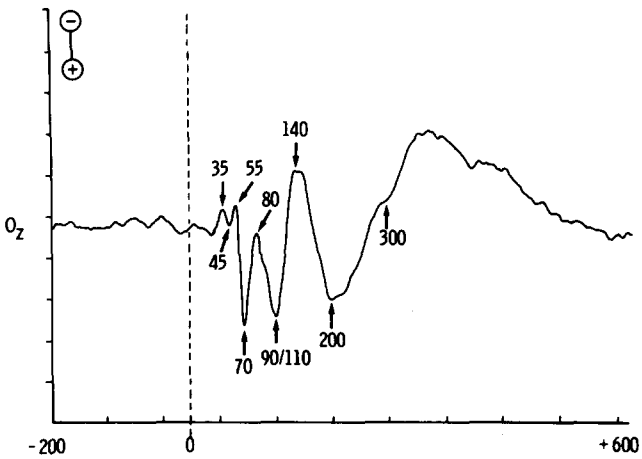

Fig. 2. Example of a flash VEP, derived from O_Z–A_2. The VEP was elicited after closed eye stimulation with flashes of 0.2 ms duration, 0.17 at 30 cm, random at 0.3/s, bandpass 1–500 Hz and sample frequency of 600 Hz.

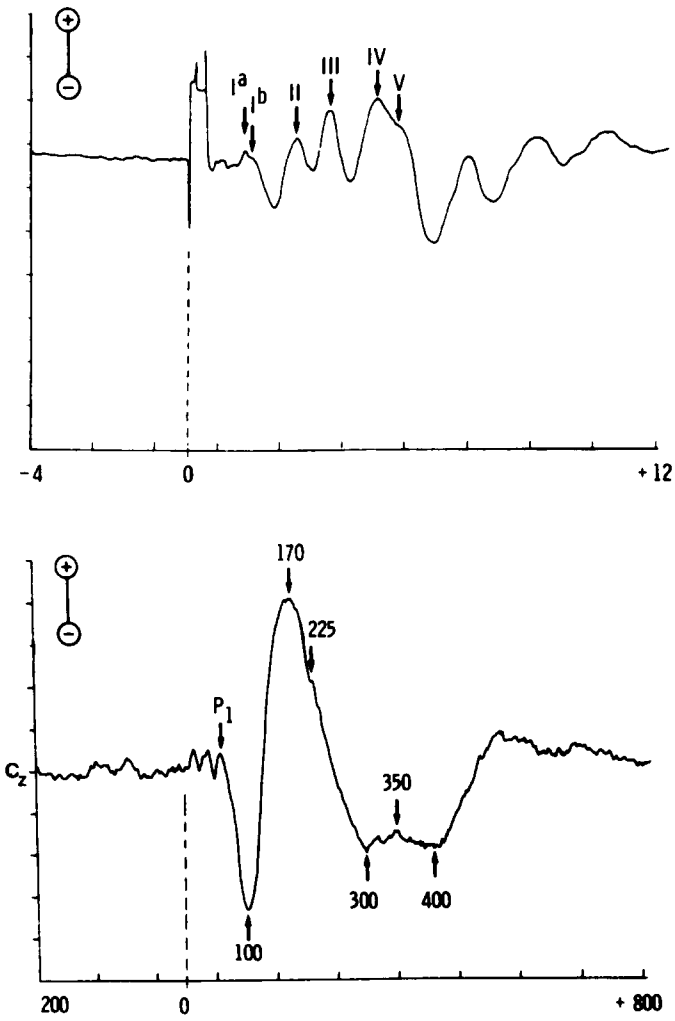

Fig. 3. Example of an AEP. *Upper trace* BAEP, *lower trace* LLAEP, derived from C_2–A_{ipsi}. The brainstem AEP was elicited after 1000 0.1 ms rarefaction click stimulation at 80 dB, regular at 11.1/s, bandpass 150–3000 Hz, sample frequency at 30000 Hz. The LLAEP was elicited after 100 0.1 ms rarefaction click stimulation at 80 dB, random at 0.3/s, bandpass between 1–250 Hz, sample frequency at 500 Hz

Clinical Application of Auditory EP

Auditory EPs can be derived from the auditory nerve, the brainstem, and the cerebral hemispheres. From a neurological point of view, the brainstem responses up to now have been clinically by far the most important components of the AEP (Fig. 3). Auditory brainstem responses can help in evaluating the condition of patients in various neurological diseases [40, 118]. In particular, structural damage to the auditory brainstem pathways due to tumors, demyelination, loss of circulation, infarction, contusion, and inflammatory lesions, which usually fail to show in radiological examination, can be detected by looking for abnormalities in amplitude and latency of the various components [53, 60]. When the structural integrity of the brainstem is not compromised, the responses are normal in toxic and metabolic conditions. The most important clinical applications of AEP in neurology are the evaluation of the cause and reversibility of coma of traumatic origin, the early diagnosis of multiple sclerosis and the detection and location of posterior fossa tumors [13, 29, 38, 58, 81, 99, 110, 112, 114]. Furthermore, it has proven useful in the case of brainstem infarctions and other brainstem lesions such as syringobulbia and hereditary ataxia [30, 89]. For instance, when EEG activity and auditory waves II to VII are absent, irreversible coma is invariably established [43, 44, 83, 97, 110].

Computerized axial tomography and conventional brain scans have their highest incidence of false-negatives in the pathology of the posterior fossa. Brainstem auditory responses can therefore supplement or suggest selective radiologic screenings for lesions in this area. Auditory EPs have proven to be especially useful in maturation studies [87].

Auditory brainstem responses offer a new method for objective measurement of the deep structure of the brain, and can be used to complement clinical evaluation [38, 60]. The clinical usefulness of middle and late AEPs is still being evaluated [72, 116].

The registration of auditory evoked brain responses at term and in young infants gives good information about their clinical condition, and about the maturation of the brain [39, 85, 86].

Pitfalls

In interpreting EP registrations, one must be conscious of the fact that false-positive and false-negative results may occur. The frequency with which this happens depends largely on the investigator's experience in recording and interpretation. False interpretations can be avoided by considering the EP tests as an extension of the neurological examination. The criteria for abnormality must therefore be critically and quantitatively defined and be based on adequate normal control data of one's own laboratory values [34].

This intriguing problem is caused by the need for quantitative normative or reference values. Every EP appliance has its own characteristics. Slight differences in filter capacity and time measurement facility between different appliances cause differences in shape and latency of EP components. Ideally, normative values ought to be obtained for all the various EP components and modalities inherent to every part of EP equipment. This is, of course, impossible in practice and reference values are generally taken from literature. This may, however, lead to false clinical conclusions. The best way to get around this problem is to establish one's own normative values for EPs which are used routinely in one's daily practice, while for other EPs the heterolateral side is always used as a control.

Various EP characteristics must be considered when defining abnormality. Since EPs are elicited after sense-organ or nerve stimulation, latency and shape are also dependent on the side of stimulation. In SSEP variations in body or ambient temperature, nerve entrapments, or inaccurate stimulation may particularly alter the latency of the early components. Peripheral hearing impairment (e.g., which may occur during a cold) may give rise to abnormalities in the AEP. Appropriate stimulation is, of course, absolutely essential. In SSEP the ulnar nerve may erroneously be stimulated during median nerve stimulation. In AEP the heterolateral ear, if not masked by noise, may be stimulated via bone conduction, and in checkerboard VEP fixation may be impossible.

Opinions are still divided regarding the best location for the reference electrode. Noncephalic electrodes, placed far away from the active electrode, e.g., on the feet, contaminate

highly significant information with muscle artifacts and ECG artifacts. In the routine clinical setting distant electrodes cannot be used in restless patients or in tense patients, or in situations in which the signal is diminished by disease. In our opinion the best site for placing the reference electrode in the various EP modalities (excluding the brainstem AEP) is the area around the ear.

Characteristics of the EP to be considered when defining abnormality are e.g., the absence of components consistently recorded in normal subjects, increased peak and interpeak latencies, decreased amplitude, and increased dispersion of components. These are often not all abnormal in the same patient. Some characteristics such as increased peak and interpeak latencies can usually be reliably identified. Component amplitude can be measured, but normal variation must be taken into account, because the establishment of the base line may be impossible. Sometimes it is rather difficult to define component dispersion in technically suboptimal recordings [49, 113].

One must be aware of the restrictions of the applied method. When abnormal, EPs identify nonspecific disturbances of peripheral or central nervous systems, resulting from either demyelination or axonal degeneration; EPs cannot always be used to locate a lesion anatomically, because the neural pathways have not been unreservedly identified [3, 25, 28, 64, 103, 115]. It is an oversimplification to ascribe a given EP component to a specific neural generator, because some EP components probably reflect sequentially activated dipoles of uncertain origins and terminations. Additionally, some EP components arise from multiple generator sources. Correlation between CT and EP may help answer these questions but only in a limited fashion. Caution is required because CT provides an anatomical slice frozen in time, while EPs assess activity in fibre tracts and synaptic activity of inhibitory and excitatory cellular groups that are probably too small to be visualized by any current imaging technique [34]. Perhaps in the future nuclear magnetic resonance will resolve some of these problems.

We must express caution concerning misinterpretation of peaks and troughs of EPs in drowsy patients, especially in young children if there are many slow waves in the EEG, which could give misleading artifacts [18]. Also in children with subclinical seizures spike-wave complexes are easily misinterpreted as EPs [23].

Conclusions

The recording of EPs has developed into an extremely useful method for investigating the electrophysiological correlates of sensory processing, while the clinical applications in neurology are still being explored.

Particular emphasis should be placed on topics which will probably become the focus of future EP studies in clinical neurology. These topics include applications of EPs in the study of pain [7, 11], and the study of possible correlations between brainstem AEP abnormalities and the sites of lesions in the brainstem or posterior fossa.

The study of EP component distributions over the brain may give more relevant information when compared with standard EP measurements [20, 31, 92].

Special attention should be given to the results of multimodal EP studies in the field of detecting subclinical lesions of the sensory pathways, and the use of serial recordings of EPs in follow-up studies in multiple sclerosis. Preliminary studies suggest that EPs are of definite diagnostic value in such disorders, provided they are carefully correlated with the clinical findings [9, 10, 61]. Indeed, EP recordings cannot be interpreted when isolated from the clinical examination. Visual EPs cannot, with certainty, differentiate between a tumor or a demyelinating lesion of the optic nerve, and SSEP abnormalities found in patients suffering from a demyelinating disease are indistinguishable from those found in patients suffering from focal and diffuse pathology of the nervous system [6, 105, 106].

EPs have opened up a lively field of fruitful research. However, clinical enthusiasm must be tempered by the cost escalation and exhaustion of laboratory time resulting from too much emphasis on these tests. EPs should be obtained only when there is a reasonable possibility that new and relevant information provided by it might influence management or treatment of the patient.

However, the use of EPs is justified in order to elicit more information about the underlying diseases or pathophysiological mechanisms in relation to clinical research. We hold the view that measurement of EPs is one of the best electrophysiological techniques for objective, noninvasive studies of the function of the human brain.

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