Arch Psychiatr Nervenkr (1982) 232:541–548

Archiv für Psychiatrie und Nervenkrankheiten
Archives of Psychiatry and Neurological Sciences

Evoked Potential Monitoring During Acute Occlusion of the Basilar Artery and Selective Local Thrombolytic Therapy

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Summary. Neurophysiological examinations of brainstem functions before, during and after selective interventional neuroradiology i.e. selective intra-arterial thrombolytic therapy of an acute occlusion of the basilar artery are reported. The success of the therapy was demonstrated by the reappearance of normal somatosensory potentials and of the blink reflex immediately after the thrombolysis. The BAEPs were continuously recorded during the thrombolysis. Normalization of the latencies and of the interpeak latencies at a very early stage of the therapy indicated the improvement of brainstem functions.

Key words: Interventional neuroradiology – Basilar occlusion – Local thrombolytic therapy – Brainstem monitoring

Zusammenfassung. Neurophysiologische Untersuchungs- und Überwachungsmethoden der Hirnstammfunktionen vor, während und nach selektiver Katheterangiographie i.v., selektive intraarterielle Thrombolysetherapie eines akuten Arteria basilaris-Verschlusses werden dargestellt. Der Erfolg der Therapie wurde anhand des Wiederauftretens normaler somatosensorischer Potentiale und aller Blinkreflex-Komponenten unmittelbar nach der Thrombolyse demonstriert. Die akustischen Hirnstammpotentiale wurden kontinuierlich während der Therapie überwacht: In einem sehr frühen Stadium der Thrombolyse zeigt die Normalisierung der Latenzzeiten und der Transmissionszeit die Erholung der Hirnstammfunktion an.

Schlüsselwörter: Therapeutische Katheterangiographie – Basilarisverschluß – lokale Thrombolysetherapie – Hirnstamm-Monitoring

Introduction

The use of brainstem auditory evoked potentials (BAEP), somatosensory evoked potentials (SEP), and the blink reflex in demyelinating diseases, in tumours of

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the cerebello-pontine angle, space occupying lesions of the posterior fossa and brainstem ischaemia is widely recognized (Dengler and Struppler 1981; Kimura and Lyon 1972; Noel and Desmedt 1975; Oh et al. 1981; Rowe 1981; Starr and Hamilton 1976; Stockard et al. 1978, 1980; Giblin 1980). Both BAEP and SEP are also used in brainstem monitoring during neurosurgical operations in the posterior fossa (Grundy 1980; Hashimoto et al. 1980; Starr et al., in press) and during interventional neuroradiology (Hacke et al., in press). We report here another most useful application of these electrophysiological methods, i.e. monitoring of brainstem functions during the local thrombolytic therapy of an occlusion of the basilar artery. This condition, caused by local arterial thrombosis or by embolisation from an occluded vertebral artery is known to have a poor prognosis, with a lethality between 60% and 80% (Dorndorf and Gänshirt 1972; Archer and Horenstein 1972; Thompson et al. 1978). We had the opportunity to study a patient with occlusion of the basilar artery before, during and after successful local thrombolytic therapy. The method of the selective thrombolysis of a basilar occlusion is reported elsewhere (Zeumer et al. 1982).

Case Report

A female patient aged 27 years, without any history of serious disease presented with transient headache, right hemihypaesthesia and hemiataxia immediately following a sudden turning movement of the head. On admission to our department the SEPs of the median nerve and the blink reflex showed no pathological changes. The initial diagnosis of a subarachnoid haemorrhage was ruled out by CSF examination. After a free interval of 6 h an acute deterioration occurred, the patient now presenting with downbeat nystagmus, left hemiparesis, right hemihypaesthesia, dysarthria and drowsiness. Transfemoral angiography was immediately performed and a distal occlusion of the right vertebral artery and a nearly complete obliteration of the basilar artery by a large thrombus was found (Fig. 1). The posterior cerebral arteries were supplied by the carotid circulation. Using an F3 catheter placed in the left vertebral artery, 200,000 IU of Streptokinase were infused continuously over 115 min. Angiographic control showed a recanalization of the lumen of the basilar artery. The following day 120,000 IU Streptokinase were applied locally. At the end of this procedure only a mild irregularity at the top of the basilar artery remained, both posterior cerebral arteries now being supplied by the basilar artery (Fig. 1). A CT scan at this time showed a small hypodense cerebellar lesion in the territory of the right superior cerebellar artery.

Neurophysiological Examination

The neurophysiological examinations were performed on admission to hospital, during the acute deterioration, during and after the first thrombolytic therapy and after the second thrombolytic procedure. All examinations were done with a mobile DISA 1500 four-channel recorder.

BAEP

For technical reasons, during the angiography the BAEP were examined by stimulation only of the left ear. Needle electrodes (DISA 13 L 26) were inserted at the positions C_z-Mst_i (ipsilateral mastoid process). The amplifier's bandpass was 200–2000 c/s; click stimuli at a rate of 10/s with an intensity of 80 dBSPL were present. During the 115 min of the first thrombolytic therapy the BAEPs were recorded continuously.

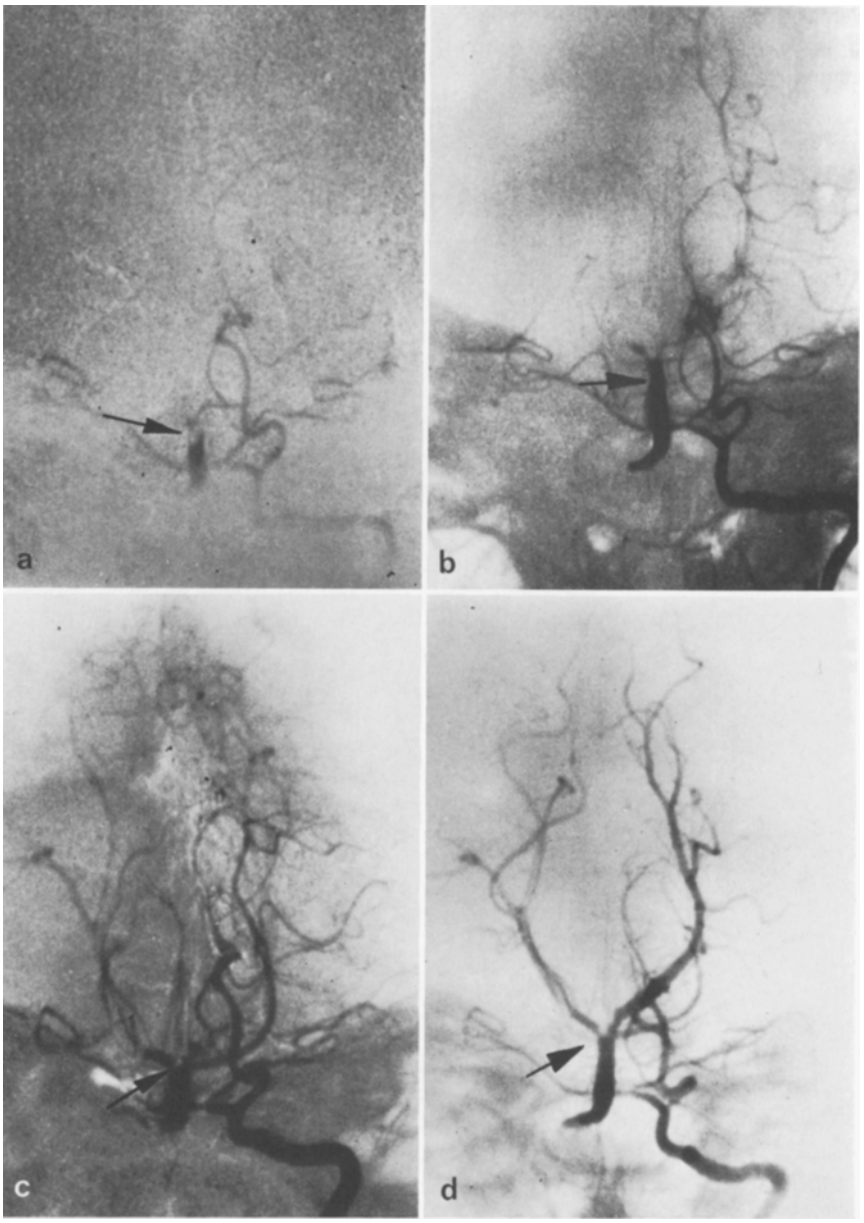

Fig. 1a–d. Acute basilar occlusion: Reopening of the basilar artery during local fibrinolytic therapy. **a** Occlusion of the basilar artery by a large embolus *(arrow)*. **b** First control after 200,000 IU Streptokinase within 2 h, the occlusion is partially reopened *(arrow)*. **c** and **d** Controls before and after another 120,000 IU Streptokinase 24 h later. Reopening of the basilar artery, only a stenosis of the left posterior cerebral artery remains *(arrow)*

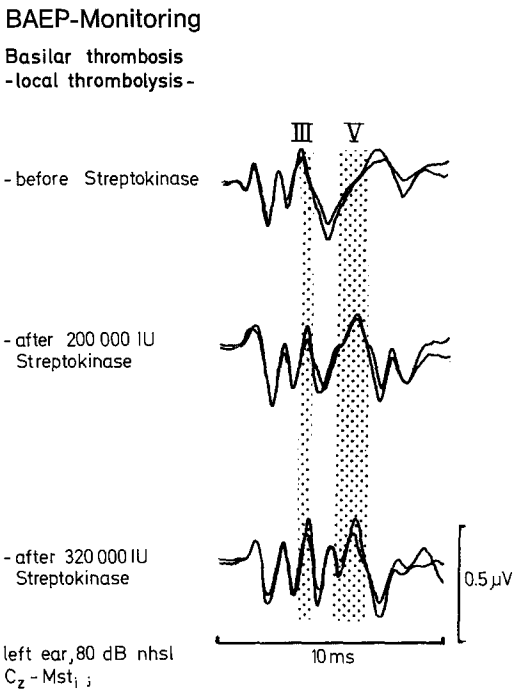

Fig. 2. Normalization of the latencies of the initially delayed component V and the interpeak latency I–V during Streptokinase therapy. The dotted stripes indicate latency norms for the components III and V

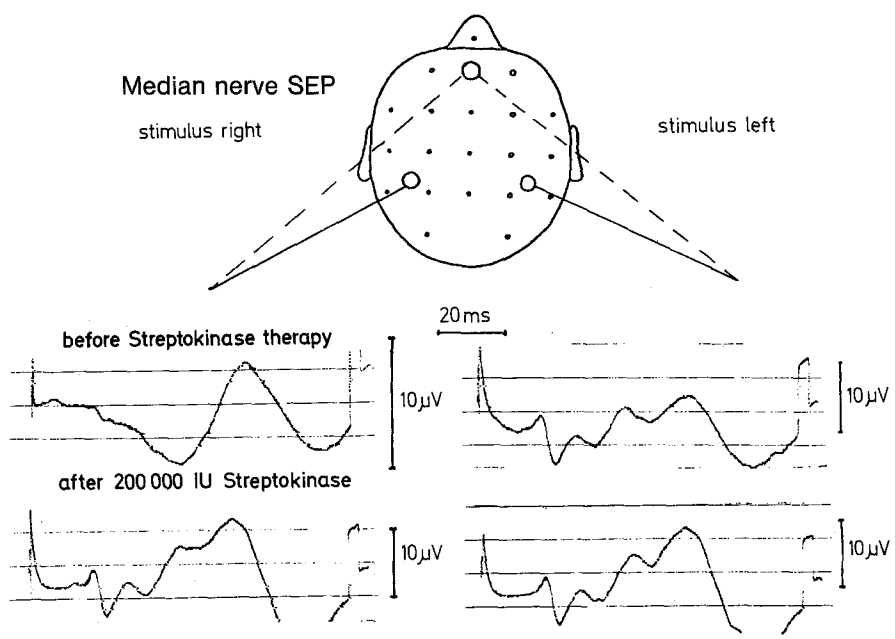

Fig. 3. Recovery of the initially pathological right median nerve evoked potential after thrombolysis

Median Nerve SEP

The SEPs were recorded with unipolar needle electrodes (electrode position F_{p2}-P_3/P_4). The median nerve was stimulated by square wave pulses (0.2 ms duration, 5 mA intensity; preamp-filters were 0.5–100 c/s; 128–256 responses were used for averaging). The narrow bandpass was chosen because the examination took place in the operating theatre. As the controls were similarly recorded, the observed changes of the potential were realistic.

Blink Reflex

The blink reflex was recorded simultaneously with surface electrodes from the orbicularis oculi muscles (stimulus: square waves pulses, 0.2 ms, 20 mA, at each supraorbital nerve). At least six responses were recorded and the average latency for each reflex component was calculated.

Results

BAEP

Before starting the thrombolytic therapy, the latencies of the peaks I–III were normal, but the components IV–V were delayed (IV = 6.05 ms, V = 7.22 ms, IPL I-V = 5.78). After the first phase of therapy (200,000 IU Streptokinase) the latencies of all peaks reached normal values (IV = 5.2 ms, V = 6.05 ms). The inter-peak latency I–V was still delayed (4.54 ms, upper limit 4.45 ms). At the end of the second session a normal BAEP was recorded (IV = 5.2 ms, V = 6.05 ms, IPL 4.40 ms) (Fig. 2), and at this time the BAEP evoked from the right ear was normal. On control examination one day later the BAEPs remained stable.

Median Nerve SEP

The median nerve SEP recorded on admission showed normal latencies and amplitudes of the early peaks. At the time of the clinical deterioration no early components of the SEP could be recorded by stimulating the right median nerve. A delayed reproducible negative wave appeared with a latency of 74 ms. The left median nerve SEP was normal. After the first 200,000 IU of Streptokinase all early waves were reproducible with normal latencies (Fig. 3). The late negative component now appeared at 63 ms. Control examinations 1 week later showed no changes in the SEP. We did not examine the SEPs during thrombolytic treatment because the patient was under anaesthesia.

Blink Reflex

Normal latencies of the electrically evoked blink reflex were recorded when the patient was admitted to hospital. Before starting the fibrinolytic therapy the R_2-component evoked by stimulating the right supraorbital nerve was significantly delayed (45.5 ms) and low in amplitude.

In addition, all late reflex components evoked from the left supraorbital nerve were slightly delyed and low in amplitude (R_1 = 12.6 ms, R_2 = 41.2 ms, R_2 = 43.0 ms). These findings suggested a diffuse disturbance of the reflex probably caused by two lesions, one in the spinal tract of the right trigeminal nerve and the second in the left dorsolateral meduallary region. Several hours after termination

of the first thrombolytic therapy the blink reflex was normal in latencies and amplitudes.

Discussion

Certain lesions of the brainstem lead to typical alterations of SEP. It has been hypothesized that the loss of early SEP components may be caused by a conduction block or by slowing in conduction of some fibers and by desynchronisation of the remaining afferent signals in the medial lemniscus, while brainstem lesions, sparing the medial lemniscus do not cause alterations in the SEP (Noel and Desmedt 1975).

The blink reflex evoked by tactile or electrical stimulation of the supraorbital nerve is transmitted via a well defined network through the dorsal brainstem including the spinal tract of the trigeminal nerve and the reticular substance of the brainstem. Lesions of pontine or meduallary brainstem areas are supposed to correlate with alterations of a certain component of the reflex (Dengler and Struppler 1981; Kimura and Lyon 1972; Ongerboor de Visser and Kuypers 1976), although blink reflex studies in patients with CT-verified brainstem lesions did not reveal the postulated close relationship in all cases (Hacke et al., in press; Fesefeldt et al. 1981).

The BAEP components correlate with the hierarchically organized brainstem structures of the central auditory pathways (Oh et al. 1981; Starr and Hamilton 1976; Stockard et al. 1978, 1980). The use of the BAEP in demyelinating diseases, in tumours of the cerebello-pontine angle and in other space occupying lesions of the posterior fossa is widely known (Rowe 1981).

In our patient the SEP, the BAEP and the blink reflex proved to be helpful in the anatomical localisation of the functional disturbances during the acute deterioration of the patient's state. We assume that there were lesions in the dorsolateral medulla oblongata on the left side and in the spinal tract of the trigeminal nerve of the right side, as indicated by blink reflex. Furthermore, a lesion affecting a part of the medial lemniscus, where afferences from the right hand are propagated is assumed. The BAEP indicated that the impulse transmission in the central auditory pathways was altered too. The clinically successful thrombolytic therapy confirmed by angiography was indicated at a very early stage during the therapy by the recovery of the BAEP. The monitoring of the BAEP during the thrombolysis provided continuous information about the increasing improvement of the propagation of impulses in the auditory pathways. On the other hand, this could have permitted early recognition of an eventual deterioration in the patient's condition during treatment (brainstem haemorrhage). Thus it was demonstrated that BAEP monitoring can serve as a control for brainstem function in patients during thrombolytic therapy in much the same way as it is already used during neurosurgical operations in the posterior fossa (Grundy 1980; Hashimoto et al. 1980; Starr et al., in press). The two sessions of the thrombolytic therapy were done under full anaesthesia, therefore the continuous monitoring of the BAEP gave the only information on the patient's condition during the treatment. After the first part of the therapy the patient's

consciousness had improved compared with the state before commencing the therapy. The supranuclear disturbances of ocular movements were nearly normalized. Right hemiataxia, left hemiparesis and dysarthria remained.

The other neurophysiological examinations, blink reflex and SEP, also showed rapid and impressive improvement. Immediately after the first thrombolysis blink reflex components normalized and the early waves (N 20, P 25) of the median nerve SEP reappeared with normal latencies. After the second part of the therapy there were no disturbances of the ocular movement left. The hemiataxia diminished to a mild dysdiadochokinesia and dysmetria of the right hand. The patient was fully awake, dysarthria and right hemiparesis were improved, and BAEP, SEP and blink reflex remained stable in relation to the good clinical condition. When the patient left the hospital 8 weeks later she had only a mild dysdiadochokinase of the right hand.

We have monitored BAEPs and SEPs in two more patients during a selective thrombolysis of the vertebrobasilar system. In the first case no alterations of the BAEP were recorded at the beginning of therapy and there were no changes in the BAEP during the effective thrombolysis of a large thrombus located in the intradural part of the right vertebral artery. In the other patient suffering from a 3-day-old bilateral vertebral occlusion (located distally from the inferior posterior cerebellar arteries), we only found a very small wave at the beginning of the therapy, indicating a diffuse brainstem dysfunction. No cortical SEPs could be recorded. During the thrombolytic therapy no changes occurred in the evoked potentials and there was no reopening of the left vertebral artery. The blink reflex recorded before starting the therapy showed only an isolated left R 1-component, while we could not obtain any late reflex components.

Acknowledgements. The authors wish to thank Ms. A. Spielmann (Neurophysiology) and Mr. W. Winkhold (Neuroradiology) for their efficient technical assistance.

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Received September 16, 1982