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SOME OBSERVATIONS ON THE BLINK REFLEX IN POSTTRAUMATIC COMA

E. RUMPL, F. GERSTENBRAND, J.M. HACKL and M. PRUGGER

University Clinic of Neurology and Anaesthesiology, University of Innsbruck, A 6020 Innsbruck (Austria)

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Since Kugelberg's (1952) electromyographic analysis of the blink reflexes it has been known that the reflex, when evoked by mechanical tap or electrical current to the skin in the periorbital region, is composed of two discharges. One is the early unilateral oligosynaptic response R_1, the other the late bilateral polysynaptic response R_2. The common afferent limb of the reflex is formed by the sensory root of the trigeminal nerve. The facial nerve is the efferent limb. R_1 is transmitted through the pons and relayed via neurones at the level of the principal sensory nucleus of the fifth nerve (Tokunaga et al. 1958; Namerow and Etemadi 1970; Kimura 1973). The normal latency of R_1 is 10.6 ± 0.8 (2.5) msec (Kimura and Harada 1972; Kimura 1973). The trigemino-facial connections of R_2 are located in the lateral reticular formation in the lower brain stem involving the spinal trigeminal tract and the area of the spinal trigeminal complex (Ongerboer de Visser and Kuypers 1978). The latency of R_2 is 31 ± 3.3 (11) msec (Kimura and Harada 1972; Kimura 1973). In analogy to the pupillary light reflex, the ipsilateral component of R_2 will be referred to as the direct R_2 latency (31 ± 10 msec) and the contralateral components as the consensual R_2 (R_{2c}) latency (32 ± 11 msec) (Kimura 1973).

Although both the early and late reflexes are altered by brain stem lesions (Kimura 1970; Namerow and Etemadi 1970; Kimura and Lyon 1972; Ongerboer de Visser and Kuypers 1978; Straschill 1980), the late reflex is particularly vulnerable to altered states of consciousness and is either totally absent, or at best minimal in amplitude regardless of the site of the responsible lesion and the side and the intensity of stimulation (Kimura 1971; Lyon et al. 1972; Kimura 1973). This study correlates the blink reflex data with different stages of posttraumatic coma to determine their diagnostic and prognostic value in the evolution of a posttraumatic comatose patient.

Case material and Methods

A group of 25 patients (average age 23 ± 10 years) were investigated in the acute stage of posttraumatic coma (within the first 2 days after the brain injury). All had closed head injuries and demonstrated signs of brain stem dysfunction. The patients underwent the usual neuroradiological examinations (CT scan, cerebral angiography) and appropriate management in the intensive care unit. This study does not include patients under sedative drugs. Barbiturate anaesthesia for the treatment of intracranial hypertension was not used at the time the study was performed. Further all patients suffering from serious pulmonary, hepatic or renal complications and from periorbital haematoma were excluded.

A group of 26 patients (average age 25 ± 11 years) were studied in a prolonged comatose state (day 3–12 after brain injury). In 17 patients the blink reflex data of the acute stage could be compared with the data of the prolonged coma. Five patients were studied in brain death. The blink reflexes of 11 patients were recorded after recovery from coma, within 1 month after injury. A total of 67 blink reflexes were analysed. Sixteen healthy persons (average age 26 ± 8 years) served as a control group.

The patients were classified neurologically in stages 1–4 of the midbrain syndrome according to Gerstenbrand and Lücking (1970) and in stages 1

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and 2 of the bulbar brain syndrome described previously (Rumpl et al. 1979). These stages characterize the well-known rostral caudal deterioration in patients with supratentorial lesions (McNealy and Plum 1962; Plum and Posner 1966). Four categories — death, severe disability, moderate disability and good recovery — were used to define the outcome of patients (Jennet and Bond 1975). The category 'severe disability' includes patients in an apallic (vegetative) state.

However, many patients in posttraumatic coma show signs different from these classical stages. This may point to a primary brain stem lesion. The most consistent clinical findings which spell direct brain stem dysfunction are decerebrate posture and severe respiratory abnormalities despite a relatively intact optomotor system (Maciver et al. 1958). The CT scan is diagnostic and eliminates a supratentorial lesion causing secondary brain stem dysfunction. Twelve patients were seen with these signs combined with normal or only slightly abnormal CT scans (mild brain oedema) at the acute stage of coma. Lateralized neurological signs may be present and they hint at the original hemispheric or brain stem lesion.

During the prolonged comatose state, the patients are still in deep coma with eyes kept closed. A steady increase of chewing, a decrease of decorticate or decerebrate rigidity, an increase of extrapyramidal symptoms and normalization of reflex eye movements can be observed (Avenarius and Gerstenbrand 1977). The most important and characteristic feature of this stage is the onset of overactivity of the sympathetic nervous system (Hörtnagl et al. 1980), leading to extensive tachycardia. At the end of this stage, within 2 or 3 weeks, the survivors begin to open their eyes. The neurological signs are similar to those seen in the acute stage. These signs combined with CT scan examinations may help to separate these patients into two groups, which developed the prolonged coma after primary or secondary brain stem lesions. Fifteen patients were found to have taken this evolution after classical midbrain syndromes, while 11 patients were considered as being in prolonged coma after primary brain stem injury.

Electroencephalographic and behavioural signs of sleep are frequently seen in comatose patients after head injuries (Silverman 1963; Chatrian et al. 1963). These signs of sleep change to a more aroused state of consciousness. According to the observations of Silverman (1963) the comatose states of our patients were further divided by clinical and EEG changes in 'sleep' and more 'alert' states. During 'sleep' the patient was quiet with low heart and respiratory rates, when controlled respiration was not needed. These states were accompanied by spindles or alpha activity in the EEG. When 'alert,' head, facial and body movements, uncharacteristic movements of the extremities, but also decorticate and decerebrate responses appeared spontaneously or in response to acoustic or painful stimuli. Also movements of mastication could be seen. Heart and respiratory rate increased. The accompanying EEG was characterized by high voltage delta activity with complete loss of spindles. In response to stimulation, the EEG and the clinical pattern frequently changed from 'sleep' to 'alert'. These observations were made both in secondary and primary brain stem injuries. Patients in brain death fulfilled the clinical criteria of brain death accompanied by isoelectric EEG.

Electromyographic analysis of the blink reflexes was done by single mechanical and electrical stimulation during 'sleep' and 'alert' states. Mechanical stimulation was performed by tapping a circular metal plate, 2.3 cm in diameter, placed above the supraorbital foramen. The stimulus was therefore rather diffuse and not painful. The metal plate was used to avoid the development of a subcutaneous haematoma. A strong stimulus was applied by using a Medelec tendon reflex hammer with synchronizing contacts triggering the stimulus through the Medelec USC 6 stimulus control unit. Single supramaximal electrical shocks were first applied in the 'alert' state to the supraorbital foramen with a bipolar surface nerve stimulator (Medelec EL 212 M). Stimulus duration was between 0.2 and 0.5 msec, intensity varied from 120 to 220 V. Both kinds of stimulation were done on the right and left. The responses were recorded with silver disc surface electrodes on the lower orbicularis oculi muscles (nasal and temporal at a distance of 3 cm). The reflex activity was fed into standard amplifiers (Medelec MS 6 system) and

recorded. Sweep velocity was 10 msec/div, the typical gain 100 or 200 μV/div; the selected frequency range was between 16 Hz (LF) and 3.2 kHz (HF). Each stimulus was repeated 3 times and the shortest available latency was chosen for further calculation. The studies were done during EEG monitoring or immediately after an EEG recording when the difference between 'sleep' and 'alert' was quite clear. Sixteen normal persons were studied in the waking state using the same methods. Measurement of the latencies was made on photographs. Between the application of the mechanical stimulus and the trigger of the oscilloscope sweep there was a constant delay of 2 msec ($\pm$0.1). The delay was measured by simultaneously recording the hammer's impact on the Medelec tremor transducer and the closing of the synchronizing contacts triggering the oscilloscope sweep. Therefore all latencies after mechanical stimulation had to be increased by 2 msec. For statistical analysis the *t* test for paired samples was used.

Results

General observations

During 'sleep' states of acute coma the late responses were totally absent after both mechanical and electrical stimulation. In 'alert' states of acute coma R_2 and R_{2c} were practically absent after electrical stimulation. This finding was confirmed by a statistical analysis of all data, including zero latencies (no response), by the *t* test for compared samples in 17 patients, who were examined in acute as well as in prolonged coma. Mechanical stimulation rarely evoked R_{2c} but R_2 was found frequently (Table I). Besides the method of stimulation the excitability of the late reflexes was dependent on the moment of stimulation. In our cases mechanical stimulation at an aroused 'alert' state of coma significantly increased the excitability of the late responses. Further results are therefore discussed in the light of the best response after mechanical stimulation. The suppression of the late responses was markedly reduced in prolonged coma both after mechanical and electrical stimulation.

In 23 out of 25 patients in acute coma there were clear alterations of EEG and behavioural signs, when the patients changed from 'sleep' to an 'alert' state. In 23 patients the behavioural signs of sleep were accompanied by EEG sleep patterns (spindles or alpha activity). Only 2 patients showed no significant EEG changes during 'sleep.' In 17 out of 26 patients in prolonged coma spindles in the EEG accompanied the behavioural signs of sleep. No signs of sleep could be detected in 9 EEGs. Frequently spindles and alpha activity disappear during 'sleep' states in prolonged coma both after primary and secondary brain stem injuries (Steudel et al. 1979; Rumpl 1980). In cases with uncertain EEG signs of sleep only the clinical observation helped to ascertain 'sleep' and 'alert' states and therewith the time of stimulation.

Unilateral loss of one or more components of the blink reflex was found in 16 patients during acute and prolonged coma. No correlation could be established with neurological or EEG lateralized signs and with lateralized signs in the CT scan.

In comparison with normals the latencies of the late reflexes were delayed in the acute state of coma followed by slight decrease of latencies in prolonged coma (Table II, Fig. 2g). Latencies returned to normal during the state of recovery. In acute coma the mean values of R_2 and R_{2c} after electrical stimulation were impaired by the small number of available late responses (only two cases).

The amplitudes of the early and late responses were generally larger in the 'alert' than in the 'sleep' state. Amplitudes and also latencies fluctuated somewhat from time to time during each stage of coma. Asymmetries of amplitude were frequently seen but showed no correlation with clinical signs. The course of habituation was rapid in acute comatose states, with further diminution and abolition of already small amplitude late responses. In most cases R_2 was completely suppressed after a second or third stimulus at a 3 sec time interval. Therefore only a single stimulus with a long resting period was used in this study. In acute coma the amplitude of the early response was evidently larger than that of the late responses (Figs. 1 and 2). This difference was reduced or inverted in prolonged coma, when amplitudes of both the early and late reflexes clearly

TABLE I

Statistical analysis of all data, including zero latencies = no response, by *t* test for paired samples in 'alert' states of acute and prolonged coma. R_2 and R_{2c} are practically absent in acute coma after electrical stimulation. Mechanical stimulation rarely evokes R_{2c} but R_2 is found frequently. In prolonged coma these differences become less evident.

'Alert' coma (17)		Mechanical stimulation						Electrical stimulation					
		R_1		R_2		R_{2c}		R_1		R_2		R_{2c}	
		Right	Left	Right	Left	Right	Left	Right	Left	Right	Left	Right	Left
Acute coma	$\bar{X}$	9.24	8.94	8.71	6.65	2.44	2.00	8.24	8.59	0.00	0.00	0.00	0.00
	SX	4.83	4.59	19.97	15.96	10.37	8.25	4.49	3.95	0.00	0.00	0.00	0.00
Prolonged coma	$\bar{X}$	9.82	10.06	2.71	22.29	19.65	18.29	9.65	10.82	24.82	22.12	17.76	18.24
	SX	4.03	2.36	7.63	17.55	17.61	18.11	2.74	1.33	19.53	19.79	19.96	20.30
	Δ	1.00	0.35	8.71	6.65	2.44	2.00	0.18	0.76	2.12	0.18	1.88	0.06
	SX	5.16	3.00	19.97	15.96	10.37	8.25	3.13	2.28	15.69	13.41	13.32	10.85

increased. Patients who showed no or only minimal return of late responses within 1 month after brain injury carried a worse prognosis. These patients remained severely disabled. Patients under barbiturate anaesthesia or therapy with sedative drugs had no early and late responses. Also patients with severe metabolic complications showed no late responses at all.

The early component after electrical stimulation was usually more synchronous than after mechanical stimulation, when many of the R_1 components were polyphasic. The strong mechanical tap gave rise to spread of percussion to the opposite side in most cases and evoked a double R_1 (Figs. 1 and 2).

Special observations

Secondary brain stem lesions (classical midbrain syndrome): in the acute state of coma 13 patients showed the well-known signs of rostral-caudal deterioration. Seven patients were in midbrain syndrome (MBS) stage 2, three in MBS stage 3 and three in MBS stage 4. In MBS 2 the analysis of blink reflexes revealed one or more R_2 and R_{2c} in 6 cases (see Fig. 1a–d). No R_2 and R_{2c} were seen in one case. In MBS 3 a direct unilateral R_2 was seen in one case, the others had no R_2. In all cases of MBS 4 R_2 and R_{2c} were totally absent; in one case also no R_1 could be elicted. Fifteen patients were examined in prolonged coma after classical MBS. All late components were present in 3 patients in prolonged coma after MBS 2. After MBS 3 (7 patients) four had complete late responses, three incomplete ones. Four out of 5 patients with evolution from MBS 4 had totally absent R_2 and R_{2c}, one showed an unilateral direct R_2.

Primary brain stem lesions (atypical midbrain syndrome): 12 patients were found in this group by clinical and CT scan examinations. The CT scan was normal in 3 cases and showed mild brain oedema in 9 cases. Decerebrate posturing was seen in 8, decorticate posturing in three and decerebrate posturing confined to the legs in two cases. R_2 and R_{2c} were totally absent in 9 cases, uni- or bilaterally present in 3 patients (see Fig. 2a–g). Both R_1s were absent in two cases. Eleven patients were observed in prolonged coma after initial atypical MBS. The CT scan was normal in 5 cases, mild brain oedema was present in 4 cases, bifrontal small subdural hematomas without oedema were seen in 2 patients. Decerebrate posturing was seen in 7, decorticate posturing in 2, flaccidity was observed in 2 patients. All late responses were recorded in 6 patients; in three the late responses were incomplete. In two patients R_2 and R_{2c} were totally absent. R_1 was found in all cases, unilaterally in two cases.

TABLE II

Mean values of R_1, R_2 and R_{2c} latencies after mechanical and electrical stimulation in normal persons and in 'alert' comatose states. Note, that latencies increase in acute coma, decrease slightly again in prolonged coma and return to normal in the recovery state.

'Alert'	Mechanical stimulation			Electrical stimulation			Days after trauma
	R_1	R_2	R_{2c}	R_1	R_2	R_{2c}	
Normals (16) S.D.	8	26	26	10	30	30	
	1	2	2	1	4	4	
Acute coma (25) S.D.	11	41	38	10	43	45 *	
	2	11	5	2	7	6	1– 2
Prolonged coma (26) S.D.	11	34	33	11	36	36	
	2	6	8	1	6	7	3–12
Recovery (11) S.D.	8	29	30	10	31	33	
	2	3	3	1	3	5	13–30

* This observation is impaired by the small number of available late responses — 2 cases — after electrical stimulation.

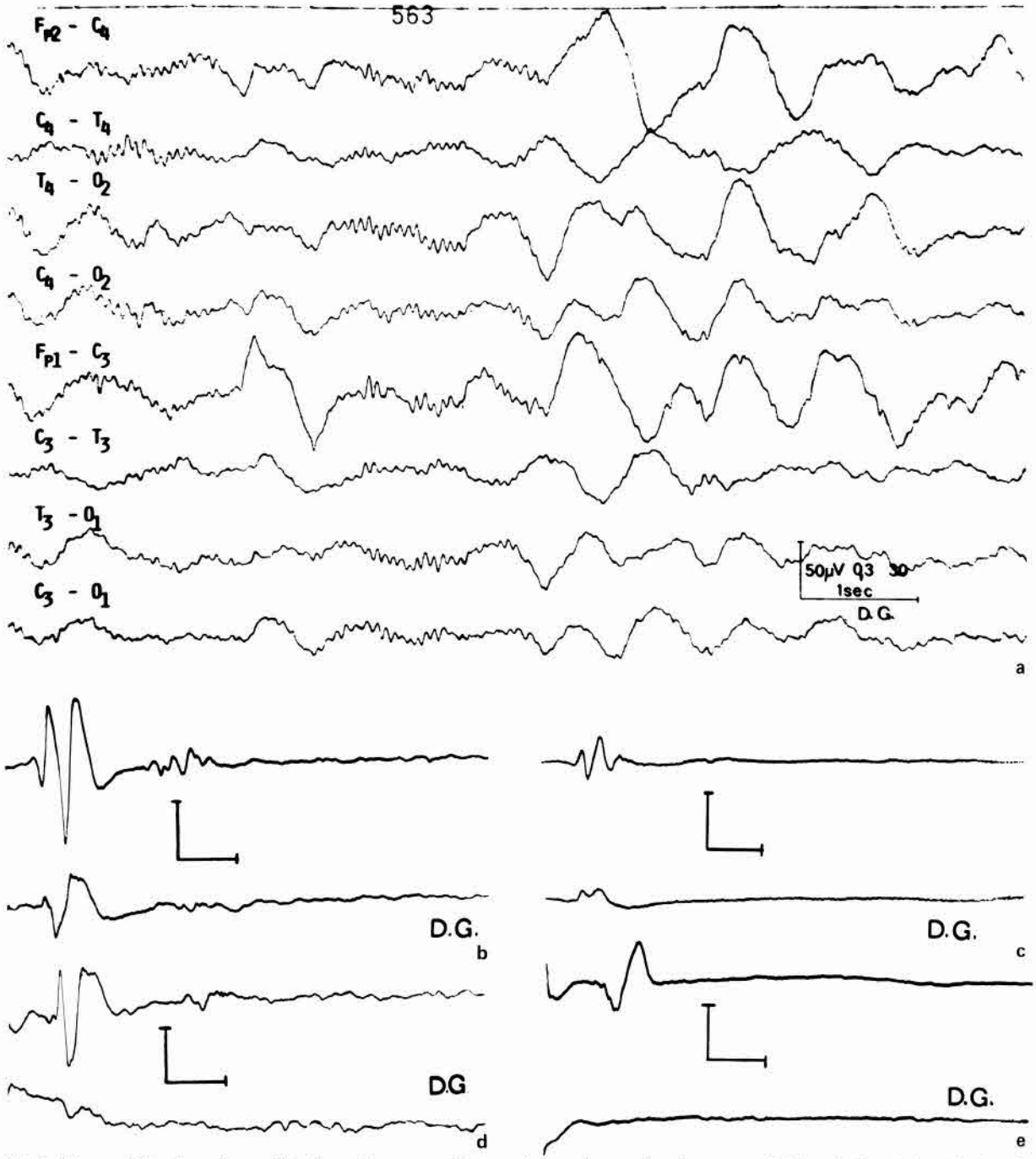
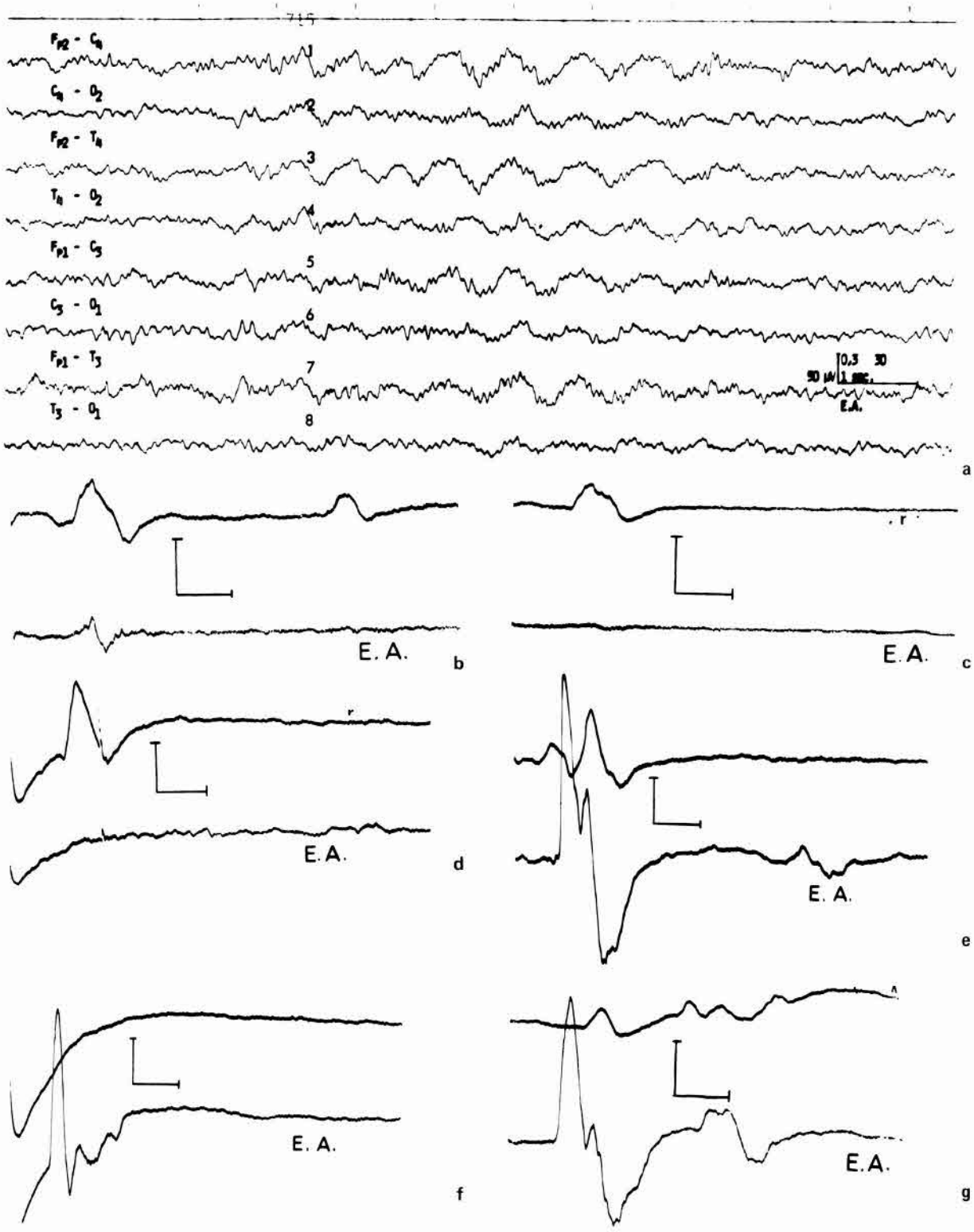


Fig. 1. 13-year-old male patient, midbrain syndrome stage 2, secondary brain stem involvement. a: EEG; spindles during 'sleep' and high voltage delta activity during 'alert'. b: blink reflexes; 'alert'; strong mechanical stimulus right supraorbital; large amplitude polyphasic R_1 and small R_2 (upper channel). R_{2c} hardly distinguishable from the baseline. Spread of R_1 to opposite side (lower channel). c: blink reflexes; 'sleep'; strong mechanical stimulus right supraorbital; small R_1, no R_2 and R_{2c}; spread of R_1 to opposite side. d: blink reflexes; 'alert'; electrical stimulation right supraoribtal; high voltage R_1 and minimal R_2, no R_{2c}; some spread of R_1 to opposite side. e: blink reflexes; 'sleep'; electrical stimulation right supraorbital; small R_1, R_2 and R_{2c} absent. Adequate changes when stimulated left supraorbital. Outcome: good recovery. (200 μV/div; 10 msec/div).


F_{P2} - C_4
C_4 - O_2
F_{P2} - T_4
T_4 - O_2
F_{P1} - C_3
C_3 - O_1
F_{P1} - T_3
T_3 - O_1
1
2
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5
6
7
8
0,3 30
50 µV 1 sec.
E.A.
a
E. A.
b
E. A.
c
E. A.
d
E. A.
e
E. A.
f
E. A.
g

TABLE III

The prognostic value of the late responses in acute and prolonged coma. All patients (one exception) with at least one R_2 in the acute state of coma recovered well. In prolonged coma the presence of R_2 was of less prognostic significance. In acute coma the 5 patients with absent R_2 and good recovery suffered from primary brain stem injuries.

Outcome	Good recovery	Moderate disability	Severe disability	Death	Total R_2
R_2 present					
Acute coma (25)	9	1			10
Prolonged coma (26)	9	3	5	3	20
Total coma (51)	18	4	5	3	30
R_2 absent					
Acute coma (25)	5		4	6	15
Prolonged coma (26)	1		1	4	6
Total coma (51)	6		5	10	21

Brain death

Five patients were studied in brain death. The early and late responses were totally absent. The accompanying EEG was isoelectric.

Recovery

In 11 patients the blink reflexes were controlled by EMG, when the patients had opened their eyes (within 1 month after injury). R_2 and R_{2c} were absent in 2 patients. Both patients showed no further recovery and remained at a state of severe disability resembling an apallic syndrome (Gerstenbrand 1967). In all other cases the early and late responses were present whether the patients recovered well (4 cases) moderately (3) or were severely disabled (2).

Prognostic value

Twenty-five patients were investigated in the acute state of coma (Table III). Ten patients were found to demonstrate direct R_2 responses on one or both sides. R_{2c} was unilaterally present in 4 cases, absent in the others. Nine out of 10 patients with one or more R_2 component had good recovery of all brain functions. One patient had moderate disability due to a local cortical lesion characterized by expressive aphasia and later confirmed by a local atrophy in the left temporal region in the CT scan (see Fig. 2a–g). Six of 15 patients without R_2 died, four evolved to severe disability. Five patients without R_2 showed good recovery; all were patients suffering from primary brain stem injuries. The blink reflexes of these patients were controlled in prolonged coma, demonstrating early complete recovery in 4 cases, the reappearance of a unilateral R_2 in one case. Two patients with no R_1 after mechanical and electrical stimulation died.

In 26 patients blink reflex studies were done in

Fig. 2. 41-year-old male patient, atypical midbrain syndrome, primary brain stem lesion, decerebrate position. a: EEG; alpha activity during 'sleep' and short-lasting higher voltage delta waves during 'alert'. b: blink reflexes; 'alert'; strong mechanical stimulation right supraorbital; higher voltage R_1 and delayed low voltage R_2 (upper channel); spread of R_1 to opposite side (lower channel). c: blink reflexes; 'sleep'; strong mechanical stimulus right supraorbital; low voltage R_1, no R_2. d: blink reflexes; 'alert'; electrical stimulation right supraorbital; more synchronous response of R_1, no R_2. e: blink reflexes; 'alert'; strong mechanical stimulus left supraorbital; high voltage R_1 and low voltage delayed R_2 (lower channel); spread of R_1 to opposite side (upper channel). f: blink reflexes; 'alert'; electrical stimulation; high voltage R_1, no R_2 (b–f 100 μV/div; 10 msec/div). g: blink reflexes; prolonged coma; 'alert'; strong mechanical stimulus left supraorbital; high voltage R_1 and low voltage R_2 (lower channel); R_{2c} and spread of R_1 to the opposite side (upper channel). Note increase in amplitude (200 μV/div; 10 msec/div), the shortened latency of R_2 and appearance of R_{2c}. Similar signs of recovery after electrical stimulation and when stimulated right supraorbital. Outcome: moderate recovery (expressive aphasia).

prolonged coma. Twenty patients showed one or more R_2 components. Nine of them recovered well, three developed moderate, five severe disability and three patients died. Fourteen patients of this group demonstrated a complete response. Six of these patients recovered well, two were moderately, four severely disabled and one patient died. Four out of 6 patients with totally absent R_2 died. One patient remained severely disabled. One patient made a good recovery. At the time the blink reflex was recorded in this patient the CT scan demonstrated a marked increase of brain oedema, which was successfully treated. Only this patient had a loss of R_2 in prolonged coma after a good response of the late reflexes in the acute state of coma.

Discussion

Kimura (1971, 1973) and Lyon et al. (1972) have postulated that alteration of the early component of the blink reflex is relatively specific for pontine lesions and that the absence of the late component in coma is due to diffuse suppression or inactivity of the polysynaptic reticular system regardless of the aetiology of coma. In all their comatose patients R_2 was absent or at best minimal in amplitude, often indistinguishable from the baseline regardless of stimulus intensity. Our results confirmed these findings when electrical stimulation was used, or when mechanical stimulation was performed during the 'sleep' state of acute coma. Unfortunately mechanical stimulation was not used in coma by other authors (Kimura 1971, 1973; Lyon et al. 1972; Serrats et al. 1976; Buonaguidi et al. 1979; Malin et al. 1980). A strong mechanical stimulus frequently evoked R_2 in our patients during an 'alert' state (Silverman 1963) of acute coma. According to Kugelberg (1952) the mechanical stimulus had to be classified as strong, because R_1 was frequently seen in both orbicularis oculi muscles, provoked by the spread of percussion to the opposite side. This was also seen in our normal persons. The spread of R_1 hampered further investigations in normal persons (Kugelberg 1952) but there seemed to be no influence on the R_{2c} responses in coma.

Our findings in comatose patients suggest that the blink reflex, particularly the late responses, is more easily elicted by mechanical than electrical stimulation. This should be especially true when the patient is in an 'alert' state of coma accompanied by easy contraction of the facial muscles. Slight voluntary contraction of the orbicularis oculi muscles increases the amplitudes of R_1 and R_2 in normal persons (Shahani and Young 1973). This was usually seen in our patients when they changed from 'sleep' to a more 'alert' state of coma. R_2 latencies in our patients were slightly increased after electrical stimulation, compared with the mechanically evoked late responses. In normal persons latencies of R_2 responses increase after weak stimulation (Penders and Delwaide 1973). These observations may lead to the conclusion that the mechanical stimulus was stronger than the electrical one. The mechanically stimulated area (2.3 cm in diameter) is certainly large and may be the more adequate stimulus for the cutaneous afferent fibres (Shahani and Young 1973).

There is evidence that in normal persons the late reflex is suppressed, but still frequently evoked by shocks of high intensity, in stages of synchronized sleep and freed from this suppression in stage REM (Kimura and Harada 1972). In addition to these findings Ferrari and Messina (1972) showed that the threshold for the blink reflex was higher in synchronized than in desynchronized sleep. The late reflexes were totally absent in acute coma in our patients when signs of synchronized sleep (spindles) were recorded in the EEG. This points to a strong inhibiting effect of sleep on the blink reflex in acute comatose states. In prolonged coma the late reflexes were delayed but rarely absent in stages of 'sleep', with or without spindles. It is noteworthy that spindles were less frequently seen, the activity of the sympathetic nervous system was increased and that the blink reflexes were more easily elicitable during 'sleep' states of prolonged coma. These observations show striking similarities to those made in REM stages of normal sleep.

The different stages of the traumatic midbrain syndrome (Gerstenbrand and Lücking 1970) characterize the increasing rostral-caudal deterioration

caused by supratentorial traumatic lesions and secondary brain stem involvement. In the acute stage of coma late responses were frequently seen in the early stages of the midbrain syndrome (MBS 2, MBS 3) but were absent in MBS 4. A similar suppression of the late responses was seen in prolonged coma at different stages of the midbrain syndrome. When the blink reflex does not recover in prolonged coma it is highly suspicious that a probably reversible functional neuronal block in the acute stage has changed to a local irreversible lesion. The absence of the early response indicates transtentorial herniation (Lyon et al. 1972). Finally, in brain death R_1 and R_2 are totally absent (Mehta and Seshia 1976). Our findings confirm the observations of Buonaguidi et al. (1979), who saw a similar decrease of late and early responses in the course of rostral-caudal deterioration. It is of prognostic significance that all patients with at least one late component in the acute stage of the classical midbrain syndrome showed good recovery.

Although primary brain stem damage rarely exists in pure form (Mitchell and Adams 1973) a primary brain stem lesion may be the principal cause of coma. A normal or slightly abnormal CT scan is diagnostic. In most of these patients the late reflexes were absent. Patients with absent R_1 and R_2 died. The early recovery of the blink reflex in prolonged coma was a favourable prognostic sign. In primary brain stem lesions the suppression of the late responses seemed more likely to be due to a functional disturbance of neurones than in secondary brain stem injuries. Because of the slight cortical impairment of these patients the prognosis was generally good when the brain stem lesions were reversible. Otherwise the clinical deficits resemble a state described as traumatically induced locked-in syndrome (Britt et al. 1977). Consistent absence of R_2, if prolonged after both primary and secondary brain stem lesions, indicated a bad outcome. The reappearance of the late responses was a favourable prognostic sign in cases with primary brain stem injuries, but was of less prognostic significance in secondary brain stem lesions.

The blink reflex is of high prognostic value in comatose patients, but the localizing value is of less significance. Clinical and electromyographic lateralized signs rarely show correspondence. In posttraumatic coma the EEG is more helpful in lateralization (Stockard et al. 1975; Rumpl et al. 1979). Local brain stem lesions become clinically clearer in the state of recovery. Now a closer correlation between clinical localization of brain stem lesions and lateralized blink reflexes can be established (Namerow and Etemadi 1970; Kimura 1970, 1971; Kimura and Lyon 1972; Ongerboer de Visser and Kuypers 1978). The blink reflex is of no use in patients undergoing treatment with barbiturates and other sedative drugs, or when metabolic disturbances interfere with the original brain lesions. Unfortunately also the significance of the EEG and of the neurological examination is clearly reduced in these cases.

Summary

Blink reflex studies were carried out on 51 comatose patients with signs of brain stem impairment due to head injury. Twenty-five patients were studied in acute coma on day 1 or 2 after trauma. Twenty-six patients were studied in prolonged coma during days 3–12 after brain injury. Brain stem involvement was divided clinically and by CT scan into secondary lesions due to supratentorial mass displacement and primary lesions due to direct violence to the brain stem. Further, the comatose states were separated by behavioural and EEG signs of sleep into 'sleep' and more 'alert' states.

The blink reflexes were recorded after mechanical and electrical stimulation. Mechanical stimulation appeared to be more effective in evoking late responses than the electrical stimulus. The elicitability of the late responses was also dependent on the time of stimulation. During 'sleep', usually accompanied by a spindle-EEG, the late responses were totally absent after both mechanical and electrical stimulation in acute coma. In more 'alert' states, usually accompanied by high voltage delta waves in the EEG, the late responses, especially direct R_2, could be frequently elicited by mechanical stimulation. These differences decreased during prolonged coma.

All patients with signs of secondary brain stem

involvement who had at least one late component in the acute stage of coma recovered well. So did all patients, with one exception, with primary brain stem injuries. Early recovery of the blink reflexes in cases of primary brain stem injury was a further favourable sign. Consistent absence of R_2 (and R_1) in prolonged coma indicated a bad outcome. The presence of all components in prolonged coma was of less prognostic significance than in the acute stage.

Résumé

Quelques observations à propos du réflexe de clignement dans le coma post-traumatique

Des études sur le réflexe de clignement ont été menées chez 51 patients comateux avec signes d'atteinte du tronc cérébral due à un traumatisme crânien. On a ainsi considéré 25 patients en coma aigu le 1er ou le 2e jour après le trauma, et 26 en coma prolongé entre le 3e et le 12e jour après leur accident. Parmi les atteintes du tronc cérébral on a distingué, sur la base des examens cliniques et par CT scan, des lésions secondaires dues au déplacement supratentoriel en masse, et des lésions primaires dues à l'atteinte directe du tronc cérébral. De plus on a séparé les états comateux en états de 'sommeil' ou 'plus éveillés' d'après des critères comportementaux et des signes EEG de sommeil.

Les réflexes de clignement furent enregistrés après stimulation mécanique et électrique. La première s'est montrée plus efficace que la seconde pour évoquer des réponses tardives. L'apparition de ces dernières dépendait également de la durée de la stimulation. Pendant le 'sommeil', généralement avec des fuseaux sur l'EEG, les réponses tardives furent totalement absentes, dans le coma aigu, aussi bien après stimulation mécanique qu'électrique. Dans les états 'plus éveillés', avec généralement des ondes delta de grande amplitude sur l'EEG, on a souvent pu obtenir des réponses tardives, par stimulation mécanique, en particulier les R_2 directes. Ces différences s'atténuaient pendant le coma prolongé.

Tous les patients avec signes d'atteinte secondaire du tronc cérébral qui avaient au moins une composante lente au cours du stade aigu du coma, se sont bien rétablis, de même que tous les patients — sauf un — avec atteinte primaire. Une restauration précoce des réflexes de clignement chez ces derniers était un signe favorable supplémentaire. L'absence persistante de R_2 (et R_1) dans les comas prolongés était de mauvais pronostic. La présence de toutes les composantes dans les comas prolongés avait une moins bonne signification pronostique que dans les états aigus.

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