*Electroencephalography and Clinical Neurophysiology*, 1979, 46: 487—497



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# THE EEG AT DIFFERENT STAGES OF ACUTE SECONDARY TRAUMATIC MIDBRAIN AND BULBAR BRAIN SYNDROMES

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(Accepted for publication: August 28, 1978)

Most investigations concerning EEG and coma have been carried out in patients in posttraumatic coma (Chatrian et al. 1963; Cadilhac et al. 1966; Naquet et al. 1967; Bricolo et al. 1971; Arfel 1975; Courjon and Scherzer 1975; Lorenzoni 1975). Although many authors follow the classification of Fischgold and Mathis (1959), comparison of the clinical findings is difficult. A detailed neurological description of patients in coma is necessary to define coma accurately (Fischer 1969). In posttraumatic coma the development of an acute secondary midbrain and bulbar brain syndrome is a precise clinical parameter of the increasing intracranial pressure, i.e. the grade of disintegration of brain functions (McNealy and Plum 1962; Plum and Posner 1966; Gerstenbrand and Lücking 1970).

From the EEG point of view, 2—5 stages have been described in posttraumatic coma (Silverman 1963; Passouant et al. 1964; Cadilhac et al. 1966; Naquet et al. 1967; Bricolo et al. 1971; Arfel 1975). The EEG changes show only bewildering and unclarified correlations with the different stages of coma. One reason for the confusion in this field is the multitude of classifications of coma and EEG patterns.

The present paper aims at a systematic correlation of the different stages of acute traumatic midbrain or bulbar brain syndromes and the accompanying EEG patterns.

## Case material and methods

A group of 113 patients varying in age from 6 to 74 years were investigated. All had closed traumatic head injuries and developed secondary brain stem dysfunctions. The patients underwent the usual neuroradiological examinations and the appropriate management in the intensive care unit. A total of 130 EEGs were analysed.

According to Gerstenbrand and Lücking (1970), the patients were classified neurologically in 6 stages:

*Stage 1 (midbrain syndrome, MBS 1)*: the most striking symptom is a change in vigilance. Mass movements occur spontaneously or as a reaction to external stimuli. Autonomic reactions show some slight pathological signs.

*Stage 2 (MBS 2)*: this stage is marked by stupor or coma, decreased reaction to external stimuli, extension of the legs, continuation of the mass movements in the arms with non-specific warding off movements in response to pain stimulation, together with increased extension of the legs. Signs of disinhibition of the tone regulating system of the midbrain are increased myogenic tonus, hyperreflexia and pyramidal signs. Signs of a disturbed oculomotor system are changes of eye position and irregular movements, as well as disinhibition of the vestibulo-ocular

reflexes. The autonomic regulation is disturbed.

*Stage 3 (MBS 3)*: the symptomatology is uniform and highly typical. The patients are unconscious and show a flexed position of the arms and extension of the legs, decorticate rigidity with synergism. There are also signs of disinhibition of muscle tone regulation as well as of the autonomic system.

*Stage 4 (MBS 4)*: the remarkably uniform symptomatology of this stage includes extension of all limbs and decerebrate rigidity with synergism. There is great disinhibition of the motor autonomic system.

*Stage 5 (BBS 1)*: the transitional stage to the secondary bulbar brain syndrome (BBS) is characterized by decreasing decerebrate rigidity. Autonomic regulation begins to break down. The oculomotor system is out of action.

*Stage 6 (BBS 2)*: the full stage of the bulbar brain syndrome is marked by complete breakdown of all brain-stem functions. The myogenic tone of the patient is flaccid; no abnormal posture can be observed and the autonomic regulation of breathing does not function.

In the early stages of the acute midbrain syndrome lateralized neurological signs may be present and they hint at the original hemispheric lesion. Such signs are contralateral deviation of the head and ipsilateral decorticate position of the extremities. In the second stage of the midbrain syndrome, the accompanying lateralized signs consist of deviation of the head, ipsilateral extension and flexion-extension of the contralateral extremities (Gerstenbrand et al. 1973). At this stage, a unilaterally dilated pupil due to ipsilateral 3rd nerve compression marks the uncal herniation (Plum and Posner 1966).

EEG patterns were classified in 5 grades: grade 1: predominant alpha and little theta activity; grade 2: predominant theta and little delta activity; grade 3: predominant diffuse high voltage rhythmic and arrhythmic delta and subdelta activity; grade 4: diffuse, mostly low voltage, delta and subdelta activity and low voltage cerebral activity only recognizable with increased amplification (3.5 $\mu$V/mm); grade 5: isoelectric record.

Apart from these grades, the following signs were noted in the EEG: (1) superimposed fast activity (6—18 c/sec) localized over the frontal regions or diffusely spread; (2) normal looking sleep records; (3) diffuse slowing accompanied by 'typical sleep potentials' (spindles, vertex sharp waves, K complexes); (4) diffuse slowing accompanied by altered 'sleep potentials' listed under 'atypical sleep potentials'; (5) spontaneously alternating EEG patterns with rapid succession of low voltage delta activity and high voltage slow waves; this activity was classified as delta bursts (0.5—2 sec), short (2—5 sec) and long (more than 5 sec in duration) runs of delta waves with frequencies of 0.5—4 c/sec; (6) 'lateralized signs': unilateral predominant slow activity, unilateral low voltage output, unilateral depression of superimposed fast activity listed under the heading 'unilateral predominant slowing'. Further lateralizing signs were asymmetries of 'typical or atypical sleep potentials' and asymmetries of response to external stimuli; (7) 4 types of reaction to external stimuli: (a) appearance of widespread 1—7 c/sec activity; (b) blocking of slow activity; (c) appearance of alternating EEG patterns; (d) appearance of 'typical or atypical sleep potentials'.

Usually, these external stimuli were visual (passive opening of the eyes), acoustic (clapping) or nociceptive.

Gauze covered silver electrodes were routinely placed in pairs on the left and right frontal, central, parietal and occipital, and anterior and posterior temporal areas. Bipolar longitudinal and transverse montages were used. In most cases artefacts compelled the use of high frequency filters (30 Hz). The filtering of high frequencies may be accepted because the evaluation of this activity is of little importance in comatose patients (except in cases with epileptic activity or with cerebral activity which is only recognizable with increased amplification). The use of a time

constant of 0.3 sec was tried in each recording. Artefacts, especially respiratory, frequently forced a time constant of 0.1 sec. In these cases we were aware that most of the slow activity might be attenuated beyond recognition.

The EEGs were recorded from a few hours to seven days after brain injury. Records of patients in the transition or full stage of a traumatic apallic syndrome following a midbrain syndrome were eliminated, because these conditions did not belong to the acute stages of the traumatic midbrain syndrome (Gerstenbrand 1967).

The circulatory and respiratory regulation were controlled by measurement of blood pressure (hypotension lower than 100, hypertension higher than 140 mm Hg systolic pressure), pulse rate (bradycardia $<$ 60/min, tachycardia $>$ 100/min) and registration of breathing pattern (normal, Cheyne-Stokes, regular hyperpnoea, assisted respiration, controlled respiration with or without relaxing drugs). Further analyses of blood gases ($pO_2$ and $pCO_2$), blood pH, electrolyte and metabolic parameters were carried out. In the following, only abnormal values will be recorded.

## Results

*Acute traumatic midbrain syndrome — stage 1*

Twenty-one EEGs were recorded in 19 patients with MBS 1. Fourteen EEGs showed mixed alpha and theta activity (grade 1), the most common pattern in this group. Delta bursts (9 tracings) and short runs of delta activity (6 tracings) were the commonest types of alternating pattern. In 3 records the activity could not be differentiated from physiological sleep phases I—III by conventional visual evaluation. Two out of 3 tracings with theta and little delta activity (grade 2) were accompanied by lowered $pO_2$ and raised $pCO_2$ respectively. One patient with delta and subdelta activity (grade 3) suffered from uraemia. In this tracing spindles and potentials resembling spindles were noted. Signs of lateralization consisted of focal slow activity in 7 and predominant unilateral slowing in one record. One patient with consecutive brain death showed a grade 1 EEG pattern at this stage. One patient with myoclonic jerks showed no paroxysmal discharges in the EEG.

The reactivity was examined in 13 tracings. In 6, high voltage delta activity was induced, in 2 cases intermixed with typical spindles. In 7 records stimulation led to blocking of slow activity.

Because of pulmonary complications, assisted respiration was necessary in 1, respiration and relaxation in 2, patients. Tachycardia was noted in 3 patients. 3 patients died later, one from primary brain death.

*Acute traumatic midbrain syndrome — stage 2*

Fig. 1 shows the most characteristic EEG pattern in MBS 2, 23 EEGs were in grade 3, 9 in grade 2, 4 showed a normal looking sleep pattern. Fast activity appeared over the frontal areas in 5, diffusely spread in 13 cases. 'Sleep and sleep-like potentials', confined to

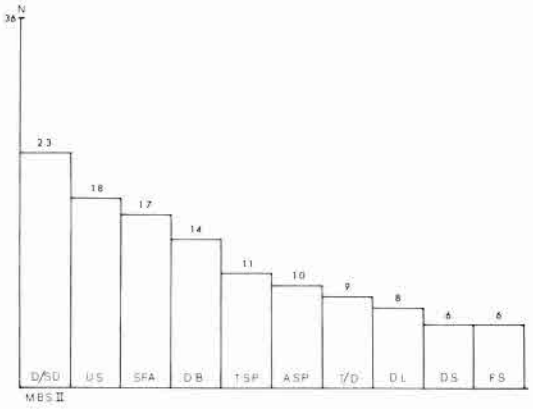


Fig. 1. EEG patterns in 36 records of 31 patients with midbrain syndrome, stage 2. The records were listed under one or more of the following categories: ASP, atypical sleep potentials; DB, delta bursts; DL, long runs of delta; DS, short runs of delta; D/SD, predominant diffuse delta and subdelta; FS, focal slow; SFA, superimposed fast activity; T/D, predominant diffuse theta and delta; TSP, typical sleep potentials; US, unilateral predominant slowing.

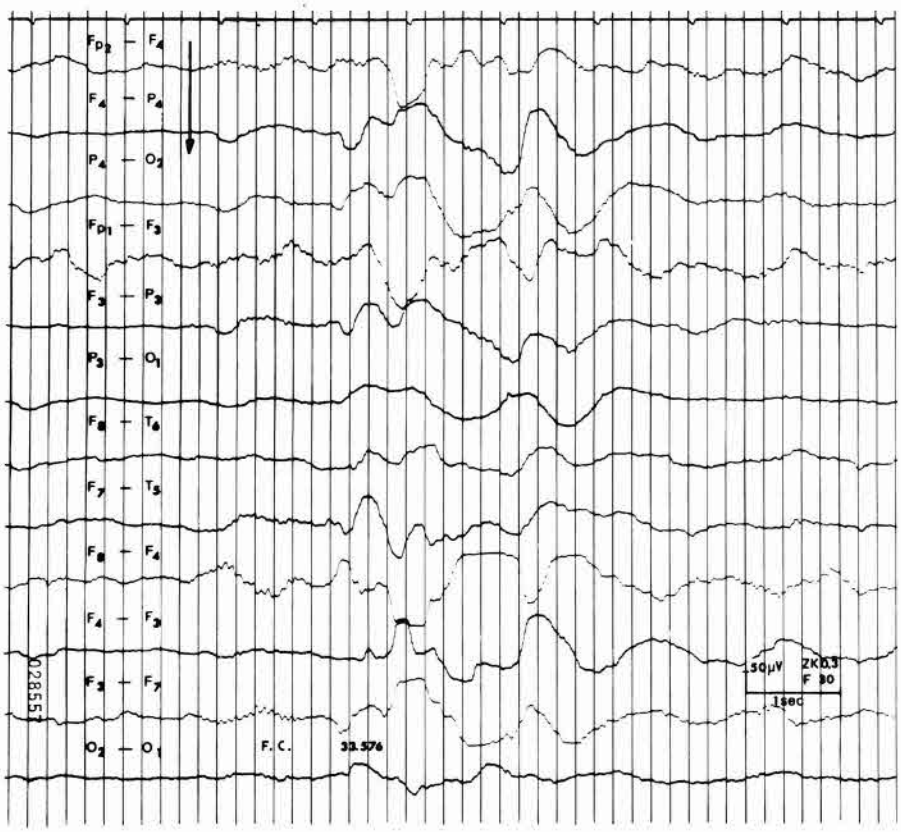


Fig. 2. Twelve-year-old girl, midbrain syndrome stage 2, no neurological signs of lateralization, diffuse delta and subdelta activity; with acoustic stimulus (noise shown by arrow) widespread high voltage delta and subdelta activity preceded by spindle activity; no EEG signs of lateralization.

one hemisphere, appeared in 9 tracings, accompanied by contralateral predominant slowing in 8 cases. In 17 tracings the reactivity to external stimuli was examined. No reaction could be observed in 4 tracings of 2 patients with uraemia and 1 patient with rupture of the spleen. Reactivity consisted in the appearance of spindles in 4, and widespread delta activity in 9 records (Fig. 2). Asymmetry of reactivity was observed in 3 tracings. One of 2 patients with consecutive brain death had 'atypical sleep potentials' in the EEG.

Assisted respiration was needed in 8, controlled respiration in 2, patients. The pH value was slightly alkaline in 14 patients. Blood pressure was hypertensive in 16, hypotensive in one, case. Bradycardia appeared in 9, tachycardia in 12 patients. Two patients died later of consecutive brain death. Cardiac, pulmonary and renal insufficiencies were the cause of death in 6 patients. An apallic syndrome developed in 6 patients.

*Acute traumatic midbrain syndrome — stage 3*

The main types of EEG pattern are listed in Fig. 3. The predominant activity was delta and subdelta (grade 3) in all records. Fast activity was superimposed over the frontal regions (12) or diffuse over all areas (11). 'Sleep or sleep-like potentials' were asymmetrical in 5 EEGs, 2 of them showing contralateral predominant slowing (Fig. 4).

Stimulation was carried out during 23

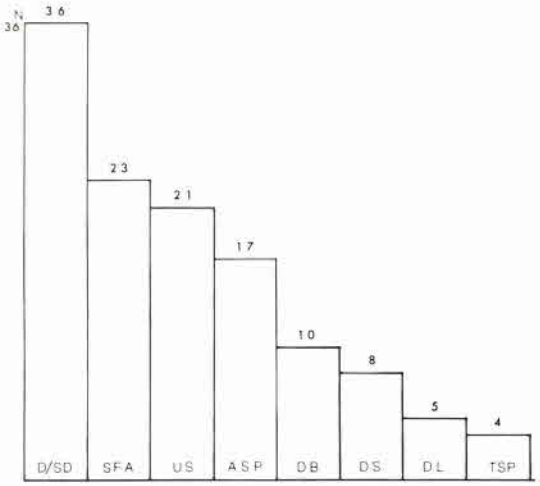


Fig. 3. EEG patterns in 36 records of 33 patients with midbrain syndrome stage 3. Abbreviations as Fig. 1.

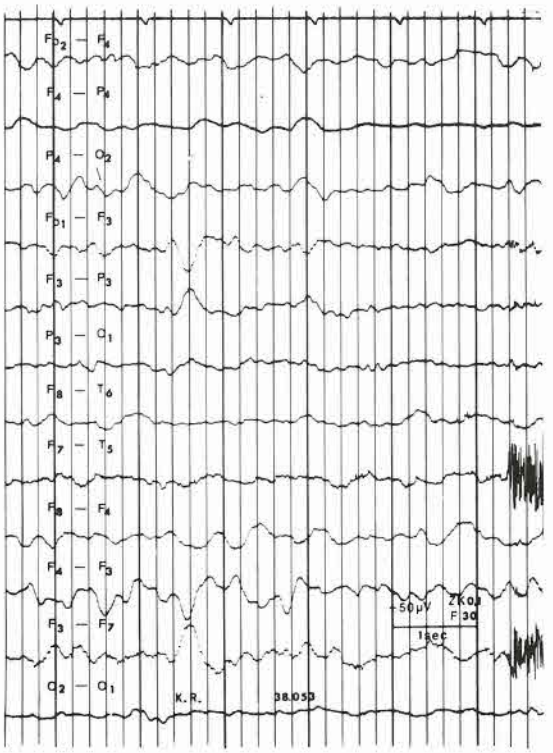


Fig. 4. Nineteen-year-old male patient, midbrain syndrome stage 3 with neurological signs of right hemisphere lesion. Diffuse delta and subdelta activity over the left hemisphere. Note single high voltage delta wave over left anterior region, regarded as an atypical vertex wave. The superimposed fast activity is absent over the right hemisphere.

recordings. Reactivity consisted in widespread delta activity in 15 cases. Long runs of delta waves occurred in 6 records. Asymmetries in reaction were observed in 2 cases (Fig. 5). 'Atypical sleep pattern' was seen in 2 out of 5 patients dying of brain death.

There was regular hyperpnoea in 2, assisted respiration in 2, controlled respiration in 3, patients. Twelve patients were slightly alkalotic. One patient suffered from hyperammonaemia. Hypertension was observed in 16 and hypotension in 4, patients. Pulse rate was bradycardic in 4 and tachycardic in 13 cases.

Two out of 3 patients with focal epileptic seizures showed contralateral rhythmic high voltage activity in the EEG. 5 patients died of brain death. 3 of these suffered also from septicaemia or pneumonia. 1 patient died of pulmonary insufficiency, another of bacterial meningitis. 25 patients developed a traumatic apallic syndrome.

*Acute traumatic midbrain syndrome — stage 4*

Fig. 6 demonstrates the EEG patterns in this group. Delta and subdelta activity in grade 3 was observed in all records. Superimposed fast activity occurred over the frontal areas in 9 cases, diffusely spread in 3 cases.

Stimulation was carried out during 10 recordings. No response was observed in 6 cases. In one case widespread delta activity appeared contralateral to low voltage activity.

The respiration was assisted in 11, controlled in 4, patients. The $pO_2$ was raised in 4 patients. Blood pressure was hypertonic in 8 and hypotonic in 3 cases. Bradycardia appeared in 4, tachycardia in 8, patients. The pH was slightly alkaline in 8 patients. Electrolyte or metabolic balance was greatly disturbed in 3 patients. In them the EEG showed uniform low voltage delta activity, and subdelta activity in one case, accompanied by

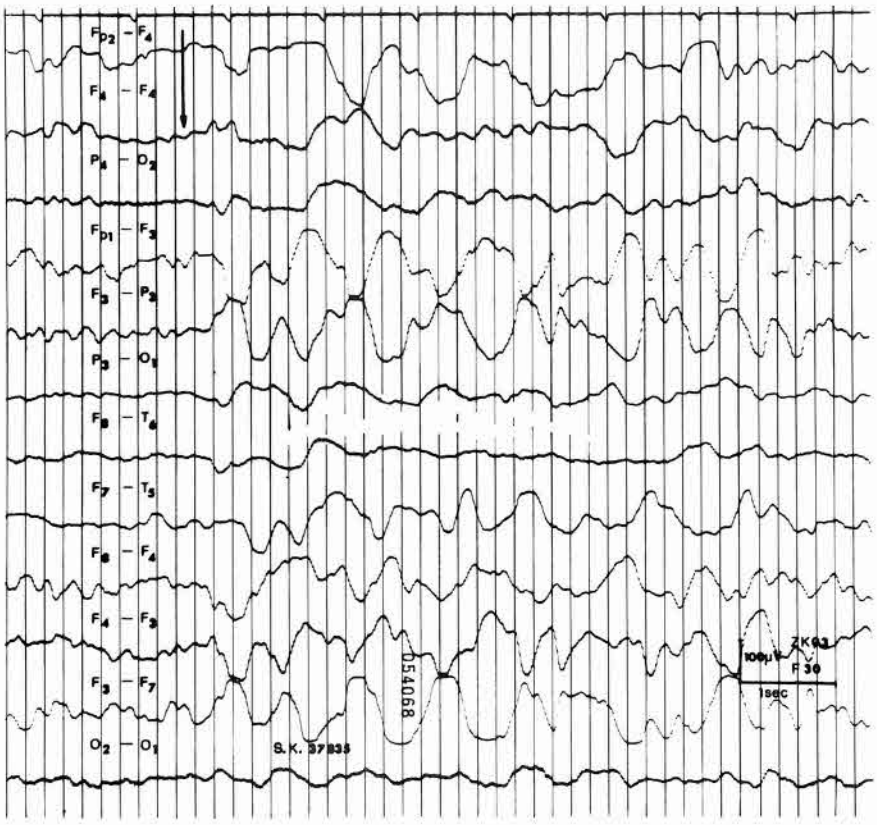


Fig. 5. 18-year-old male patient, midbrain syndrome stage 3 with neurological signs of right hemisphere lesion. With acoustic stimulus (clapping, shown by arrow) high voltage regular slow waves on the left, lower voltage and more irregular slow waves on the right. Asymmetry in reactivity indicates right hemisphere lesion.

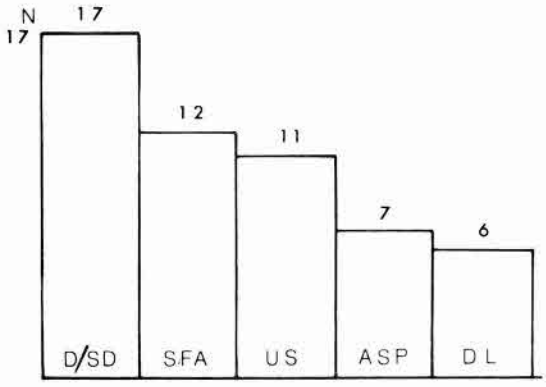


Fig. 6. EEG patterns in 17 records of 15 patients in midbrain syndrome stage 4. Abbreviations as Fig. 1.

scanty frontal superimposed fast activity. Myoclonic jerks were seen in 3 patients, in one timelocked to EEG paroxysmal discharges. 8 patients died of renal or hepatic failure. Transition to a traumatic apallic syndrome was observed in 7 patients.

*The transition stage of the secondary traumatic bulbar brain syndrome*

Seven EEGs were recorded in 6 patients with BBS 1. 5 EEGs showed delta and subdelta activity in grade 3. Subdelta activity was predominant in 2 cases. In 2 EEGs cerebral

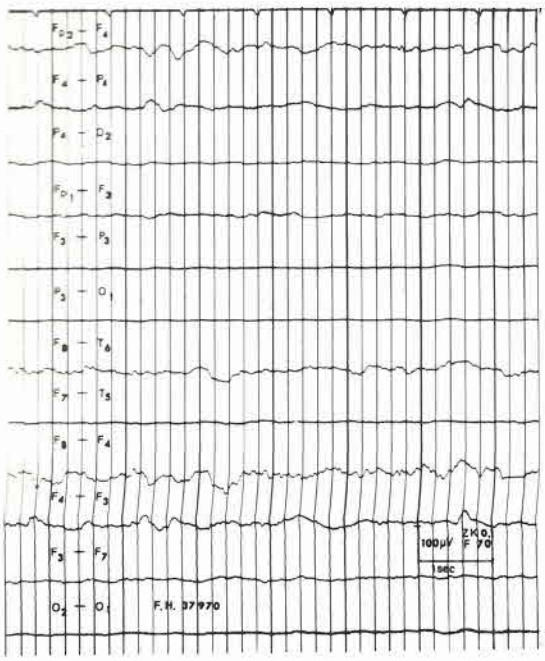

Fig. 7. 18-year-old male patient, bulbar brain syndrome stage 1 following midbrain syndrome stage 2 with signs of left hemisphere lesion. The markedly reduced voltage and abolished fast activity over the left hemisphere indicate the original lesion.

activity was only detectable with increased amplification (grade 4). Frontal superimposed activity appeared in 2 cases, unilateral predominant slowing in 3 cases (Fig. 7). Stimulation was carried out during 6 recordings; there was no reaction in any case.

Controlled respiration was necessary in all patients. A great disturbance of the electrolyte and metabolic balances occurred in 4 patients. The EEGs of these patients showed uniform low voltage delta and mainly subdelta activity (grade 4) with frontal superimposed 4—7 c/sec activity in one case. 2 patients died of primary brain death. One patient died of renal insufficiency, another of myocardial infarction. 3 patients developed an apallic syndrome.

*Acute traumatic bulbar brain syndrome (full stage)*

Twenty tracings from 17 patients in BBS 2 showed isoelectric patterns (grade 5).

The typical result of blood gas analysis was a remarkably lowered $pCO_2$ and a rise of $pO_2$ to a high level, indicating a decrease of cerebral oxygen uptake. All patients showed loss of respiration, and life was sustained by controlled respiration and artificial maintenance of blood pressure. All patients died of irreversible loss of brain functions.

*Lateralisation in the EEG in midbrain and bulbar brain syndrome*

Fig. 8 demonstrates the correlation between lateralizing signs in the EEG and the neurological examination. In MBS 4 and BBS 1, the neurological examination revealed no lateralizing signs at all. The EEG gives the only hint to a local cerebral lesion confirmed by previous, or later neurological signs or autopsy. The significance of the above men-

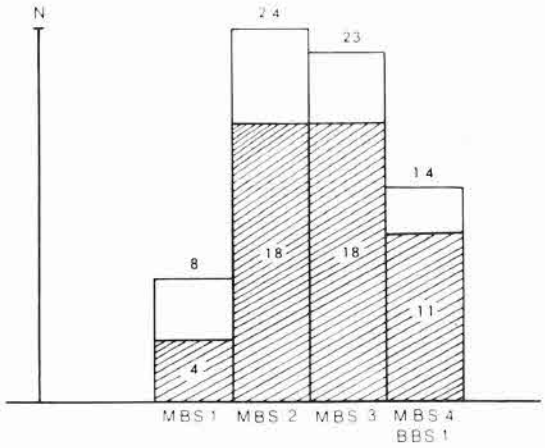


Fig. 8. Correlation of lateralizing signs in the EEG with the neurological or postmortem examinations. Dark columns demonstrate corresponding neurological and EEG signs of lateralization in midbrain syndrome (MBS) stages 1, 2 and 3. In midbrain syndrome stage 4 and bulbar brain syndrome (BBS) stage 1 (listed in one column), results of the autopsy have to be partly used to determine lateralization.

tioned correlation in MBS 4 and BBS 1 is hampered by the different stages at which the EEG and the neurological examination are performed.

## Discussion

The pathomechanism of traumatic coma follows the well known rostro-caudal deterioration, which was first described by McNealy and Plum (1962), Plum and Posner (1966) and modified by Gerstenbrand and Lücking (1970). The neurological symptoms allow a clear and pathophysiologically well established statement about the depth of coma.

Different parameters have been used in the breakdown of EEG signs in comatose states (Fischgold and Mathis 1959; Chatrian et al. 1963; Silverman 1963; Hockaday et al. 1965; Bricolo et al. 1970; Kubicki et al. 1970; Prior 1973; Arfel 1975; Courjon and Scherzer 1975). In order to increase the value of the EEG we tried to integrate all these parameters and to correlate them with the stages of rostro-caudal deterioration.

In MBS 1 the EEG is slightly or moderately abnormal, in accord with the mildly diffuse cortical disturbance. Stimuli to diencephalic or rostro-mesencephalic structures (Bricolo 1975) may induce delta bursts and short runs of delta waves. Local slow activity can only be seen in an otherwise mildly abnormal EEG, while increasing diffuse slowing may overwhelm local abnormalities (Hess 1961). Borderline normal EEGs occur in MBS 1, but not in deeper stages of MBS, as seen by Bricolo et al. (1971). In MBS 2 the amount of slow activity increases. We refrain from speculating whether this increasing abnormality is due to a direct cortical disturbance (Hess 1965) or to a more remote effect from deeper structures (Lücking 1970; Bricolo 1975). Both mechanisms may be involved. However, 'sleep or sleep-like potentials' indicate a relatively intact cortex (Silverman 1963). In MBS 2 the EEG shows the widest range of different EEG patterns. In MBS 3 a clear reduction in the number of EEG patterns can be noted. A decrease of spontaneous alternating patterns and 'typical sleep potentials' is claimed to indicate an increasing disturbance of the diencephalic and mesencephalic systems (Chatrian et al. 1963). The EEG patterns are still more simplified in MBS 4 and BBS 1. The absence, reduction and deterioration of sleep potentials or alternating patterns are suggestive of marked damage at the diencephalic level (Naquet et al. 1967; Bricolo et al. 1968). The telencephalon (Arfel 1975) must also be involved, considering the diffuse brain oedema in advanced stages of rostro-caudal deterioration.

No electrical cerebral activity can be observed in patients in BBS 2. The isoelectric EEG indicated both failure of the intrinsic cortical activity (Henry and Scoville 1952) and loss of cortical reactivity to stimuli from deep seated structures, which may also be self-limiting. From which it follows that BBS 2 is totally different from isolated focal pontine lesions, accompanied by a good deal of cerebral activity up to normal-looking EEGs (Loeb and Poggio 1953).

Mathis et al. (1957), Fischgold and Mathis (1959) and Bricolo and Turella (1973) described lateralizing EEG signs in coma, which were also studied in this investigation. Asymmetry in reactivity (Courjon et al. 1971; Courjon and Scherzer 1975) was also considered a reliable lateralizing sign. The EEG supports the neurological symptoms of lateralization in MBS 1-3. In MBS 4 and BBS 1 neurological signs of herniation override preexisting hemispheral signs. Therefore, only the EEG demonstrates signs of lateralization in these stages.

The variety of EEG patterns, especially 'sleep or sleep-like' activities and alternating patterns, decreases with the grade of rostro-caudal deterioration. This observation confirms the majority of results in the literature (Chatrian et al. 1963; Silverman 1963; Bricolo et al. 1968). The reaction to external stimuli further characterizes the depth of coma (Arfel

1975; Courjon and Scherzer 1975). In MBS 1 sensory stimulation may briefly block the slow activity. In further stages of the midbrain syndrome, reactivity consists in widespread delta activity, alternating patterns and 'typical or atypical sleep potentials'. In BBS 1 and BBS 2 no reactivity can be observed.

Metabolic derangements certainly have an important impact on coma and accompanying EEG abnormalities (Wilson and Sieker 1958; Goulon et al. 1959; Silverman 1962; Naquet et al. 1967; Merill and Hampers 1970; Arfel 1975). The number of metabolic disturbances is significantly higher in patients in MBS 4 and BBS 1. Most of these patients suffer from polytrauma including the liver, kidneys and lungs. The metabolic disturbance causes an additional diffuse encephalopathy which interferes with the original brain damage and consecutive herniation. This combination carries the worst prognosis.

In conjunction with the neurological examination, cranial computed tomography can detect descending transtentorial herniation (Osborn 1977; Stovring 1978). The scanning method easily identifies the space-occupying lesion and the displacement of the brain stem, but fails to give information about the degree of cortical activity. Therefore, the EEG is still important in identifying acute secondary traumatic MBS and BBS, especially MBS 4 and BBS 1.

## Summary

One hundred and thirty EEGs were analysed from 113 patients with acute secondary traumatic midbrain and bulbar brain syndromes. The EEG pattern was related to the stage of the midbrain syndrome caused by supratentorial brain shift. A decrease in the number of different EEG patterns was associated with increasing intracranial pressure. Unfavourable prognosis was indicated by the disappearance of sleep or sleep-like activities, alternating pattern and loss of reactivity. Lateralization by the EEG proved to be superior to clinical findings in full stages of the midbrain syndrome. EEG abnormalities due to the herniation itself interfered with EEG changes due to secondary circulatory, respiratory and metabolic encephalopathies. In such cases, the above mentioned regularities were blurred.

## Résumé

*EEG recueillis à différents stades du syndrome traumatique aigu mésencéphalique et bulbaire*

Une analyse de 130 EEG de 113 traumatisés crâniens qui présentaient un syndrome aigu post-traumatique mesencéphalique ou bulbaire a été réalisée. La morphologie des tracés EEG a correspondu au stade du syndrome mésencéphalique provoqué par le déplacement supratentoriel du cerveau. L'appauvrissement du nombre de configurations EEG était proportionnel à l'augmentation de la pression intracrânienne. La disparition des activités de sommeil ou apparentées, des tracés alternants et la perte de la réactivité est apparue comme de mauvais pronostics. La latéralisation de l'EEG s'est révélée plus valable que les données des examens neurologiques, au stade profond du syndrome mésencéphalique. Aux anomalies EEG liées au traumatisme lui-même se sont ajoutées des détériorations dues à des complications circulatoires, respiratoires ou métaboliques. Dans de tels cas, les constatations EEG ci-dessus étaient masquées.

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