Clinical and Topical Diagnosis in Traumatic Brainstem Disorders

F. Gerstenbrand and E. Rumpl

Introduction

While clinical guides to diagnosis of traumatic brainstem lesions are frequently given in acute stages of traumatic coma (12, 22, 27), there is surprisingly little interest in description of brainstem lesions of the chronic condition that emerge after severe brain injury. The lack of interest to describe brainstem lesions in these patients may be probably seen in the fact, that

a maximal brain damage happens early probably within minutes or hours after the accident, and that the main prognostic variables were intentionally restricted to the findings of the first neurological examination, because it has been shown, that most severely injured patients who survive do not have dramatic changes in the principial neurological signs in the first week (25). However, changes in neurological signs will appear in the further course of a traumatic brainstem disorder.

Abnormal postures, movements and motor response are one of the most important elements in the examination of patients in acute coma after head brain injury. Therefore special interest was taken to study these motor patterns in chronic traumatic brain disorders. It is the aim of our paper to show, which abnormal motor patterns can be seen in acute as well as in chronic traumatic brainstem disorders, and which tures and movements are of prognostic and localizing value.

The Neurological Features of Acute Traumatic Coma

Three different pathways have been reported to chause loss of consciousness and brainstem impairment. First, lesions producing bilateral impairment of either the cortical mantle or it's underlying white matter with bilateral involvement of the brainstem for example in the case of diffuse brain edema. Second, hemispheric mass lesions, when they either encroach directly upon deep diencephalic structures or secondarely compress diencephalic and mesencephalic structures in the process of transtentorial herniation. Third, lesions which directly involve the brainstem. Head injury often damages both brainstem and supratentorial functions simultaneously. Although brainstem injury rarely exists in pure form (23, 26) a primary brainstem lesion may be the principal cause of coma.

In cases of symmetrical rostral-caudal herniation and finally central herniation at tentorial clinical signs are rather typical (12, 27). Increasing involvement of diencephalic and mesencephalic structures produce somnolence, sopor and coma and a characteristic change in body position and spontaneous limb postures, which reach from non – stereotyped agitated movements to decorticate and decerebrate posturing (Table 1). Also the eye position, the pupil size and reaction show characteristic changes in the course of increasing intracranial pressure. Alterations in the oculocephalic, oculovestibular and ciliospinal responses and, most prominent, changes in respiratory pattern further indicate increasing impairment of midbrain and bulbarbrain structures and thereby the deepening of coma. These changes characterize the four stages of midbrain syndrome and two stages of bulbarbrain syndrome (12).

In cases of lateral or uncal herniation due to hemispheric mass displacement clinical signs differ from the signs of central herniation. Commonly with hemispheric lesions, there is a preexisting hemiparesis contralateral to the original focal brain lesions. At the mesencephalic level however, the patients exhibit signs of lateralisation, which also may be divided in two stages (13). In the first stage of lateralized midbrain syndrome the patient may show decorticate posture homolateral to the brain lesion and deviation of the head and eyes to the side of the lesion. Mass movements may be seen contralateral to the brain injury. The most consistent finding is an enlarged pupil homolateral to the side of the lesion, but also the

Table 1 Clinical signs in post-traumatic comatose patients at the different stages of midbrain syndrome and bulbar brain syndrome. Note the characteristic changes in motor patterns with increasing intracranial pressure (after Gerstenbrand and Lücking) (from Rumpl, E., et al.: Electroenceph. clin. Neurophysiol. 56: 420–429, 1983)

	MBS 1	MBS 2	MBS 3	MBS 4	BBS 1	BBS 2
Spontaneous limb postures	Non-stereotyped movements in the arms and legs	Non-stereotyped movements in the arms, Extension of the legs	Decorticate posturing	Decerebrate posturing	Flaccidity	Flaccidity
Motor response to pain	Non-stereotyped withdrawal of the limbs	Non-stereotyped withdrawal of the arms, Extensor response of the legs	Decorticate response	Decerebrate response	Decerebrate response	No response
Eye position	Roving movements	Roving more irregular movements	Immobile straight ahead	Immobile divergent	Immobile divergent	Immobile divergent
Pupil size and reaction	Normal Reacting	Normal small Reacting	Small Small range of contraction	Enlarged Small range of contraction	Large Unreacting	Large Unreacting
Respiration pattern	Normal	Cheyne-Stokes	Cheyne-Stokes, Rapid regular hyperventilation	Regular hyperventilation	Ataxic	No respiration

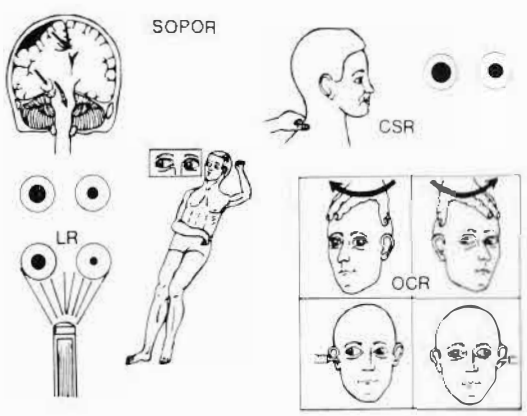

Fig. 1 First stage of lateralized midbrain syndrome (early uncal herniation). The soporose patient shows decorticate position homolateral, mass movements of extremities contralateral to the space occupying lesion. The pupil also is enlarged homolateral (from Gerstenbrand, F., E. Rumpl: Das prolongierte Mittelhirnsyndrom traumatischer Genese. In Neumärker, K.J.: Hirnstammläsionen. Hirzel, Leipzig 1983, pp. 236–248)

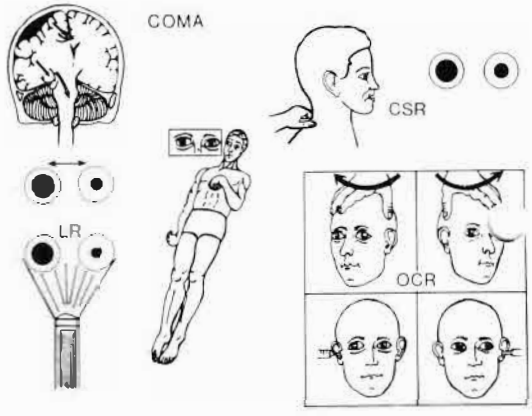

Fig. 2 Second stage of lateralized midbrain syndrome (late uncal herniation). The comatose patient demonstrates decerebrate position homolateral, decorticate position contralateral to the space occupying lesion. The pupil is large and unreacting (from Gerstenbrand, F., E. Rumpl: Das prolongierte Mittelhirnsyndrom traumatischer Genese. In Neumärker, K.J.: Hirnstammläsionen. Hirzel, Leipzig 1983, pp. 236–248)

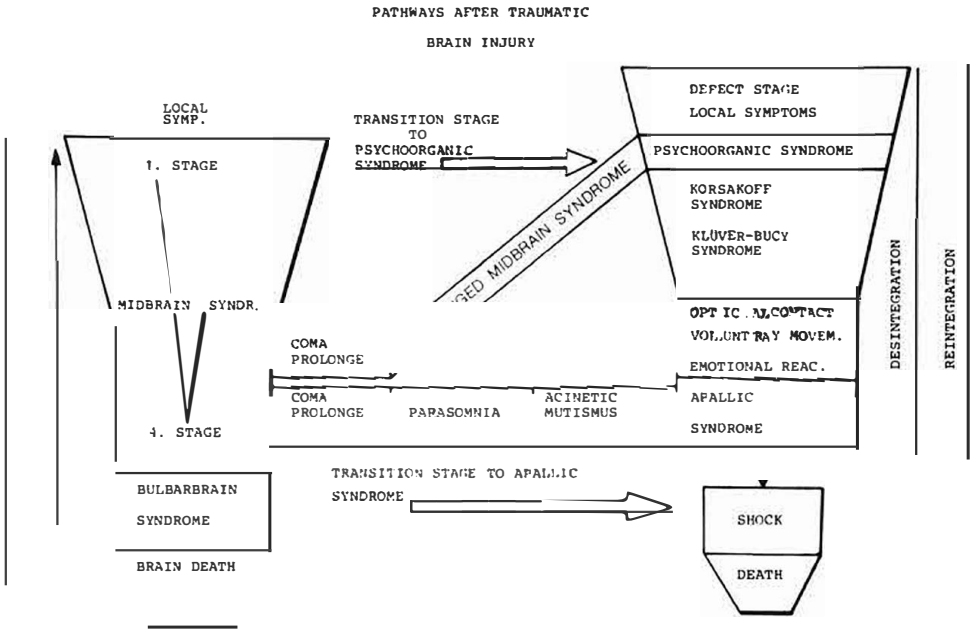

Fig. 3 Pathways of evolution after severe traumatic coma. 1. Patients may recover within few hours or days. 2. Rostral-caudal deterioration will continue and finally results in bulbarbrain syndrome and brain death. 3. Patients may show symptoms of early midbrain syndrome over weeks – prolonged midbrain syndrome – and emerge from this state to a psychoorganic syndrome. 4. Patients take development from late stages of acute midbrain syndrome to the apallic syndrome passing the transition stage. This stage usually lasts two or three weeks and can be separated in three periods, coma prolongé, parasomnia and period of acinetic mutism (after Gerstenbrand – Rumpl)

oculovestibular and the oculocephalic reflexes may be altered (Fig. 1). With increasing lateral involvement of the brainstem due to uncal herniation symptoms of the second stage of lateralized midbrain syndrome are evident. The patient becomes comatose and demonstrates decorticate position contralateral and decerebrate position homolateral to the space occupying supratentorial lesion. The homolateral pupil is large and unreacting. The oculovestibular and oculocephalic reflexes are poor (Fig. 2). Eventually signs of lateralisation will disappear and the patient shows late midbrain syndrome stage 4, characterized by decerebrate posture.

Neurological signs different from this expected rostral-caudal deterioration may be found in cases of primary brainstem injuries. Most frequently decerebrate posture and ataxic breathing is combined with a relatively intact optomotoric system (21). The CT scan is diagnostic and eliminates a supratentorial lesion causing secondary brainstem dysfunction.

In early stages of midbrain syndrome patients may recover from coma within a few hours or days. In some patients rostral-caudal deterioration will continue and finally results in bulbarbrain syndrome and brain death. Other patients will continue to show symptoms of early midbrain impairment over weeks – prolonged midbrain syndrome – and emerge from this state by appearance of a psychoorganic syndrome of varying severeness. Patients in late stages of midbrain syndrome usually take development to apallic syndrome (Fig. 3).

The Neurological Features of the Transition Stage to the Apallic Syndrome and the Full Stage of the Apallic Syndrome

The term apallic syndrome was introduced in 1967 (10) to describe this chronic condition after traumatic brain injury. The term apallic syndrome was proposed in 1940 by Kretschmer (20) to describe patients who were openeyed, uncommunicative and unresponsive from a

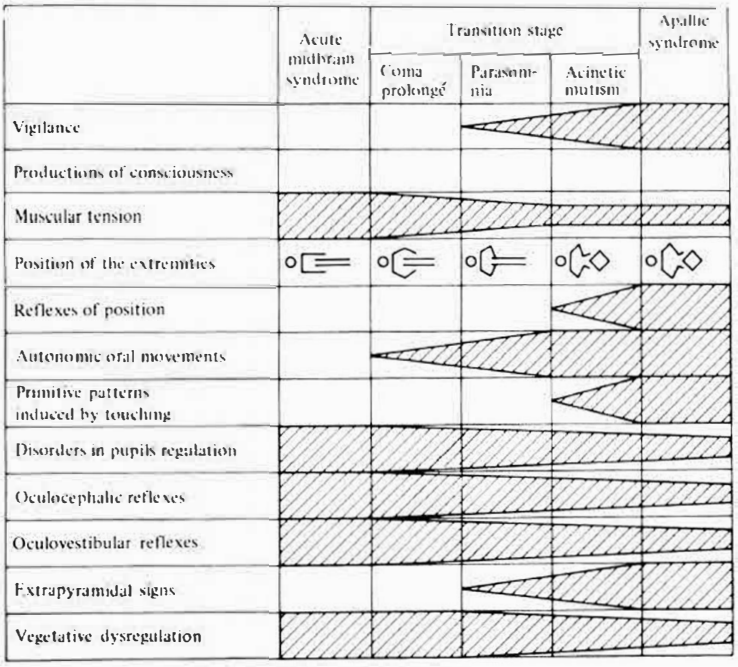

Fig. 4 Graphic representation of the most important symptoms of patients of the transition stage of the apallic syndrome. The most interesting point concerning motor patterns are the changes in position of body and extremities. In many cases decerebrate position slowly changes to a rather fixed flexion position (flexion tetraparesis) (from Avenarius, H.J., F. Gerstenbrand: The transition stage from midbrain syndrome to the traumatic apallic syndrome. In Dale Ore, G., F. Gerstenbrand, C.H. Lücking, G. Peters, U.H. Peters: The Apallic Syndrome. Springer, Berlin 1977, pp. 22–25)

variety of cerebral lesions, including cerebral arteriosclerosis, lues, encephalitis and gun shot wounds.

The transition stage to the apallic syndrome usually lasts two or three weeks and can be separated into three periods, coma prolongé, parasomnia and period of acinetic mutism (1). During the first two periods the patient is still comatose or sleep- like comatose, while at the stage of acinetic mutism the patient usually starts to open his eyes (Fig. 4). The muscle tonus continues to be increased, but now shows signs of spasticity and of rigidity (24). The most interesting point concerning our considerations is the significant change in posture of body and extremities. In many patients the decerebrate posture of acute coma will slowly change to a flexed position (flexion tetraparesis), which eventually is most prominent at the full stage of the apallic syndrome. Postural reflexes may appear spontaneously or may be elicited in more or less intensive form. In cases of spontaneous appearance of an asymmetric neck response this posture may dominate other movements and emerge to a rather fixed position („frozen" neck response). Otherwise, these reflexes are of great importance for early physiotherapy using the method of the so-called tonus regulating reflex therapy (Figs. 5 and 6). Primitve motor patterns of oral sense, such as chewing, sucking, and the grasp reflexes can increasingly be elicited by touching. Disorders in pupil's regulations, and disturbances of the oculocephalic and vestibuloocular reflex responses will decrease, but will be still present at the apallic state. Overactivity of the sympathetic nervous system characterize the vegetative dysregulation and will continue over weeks and months without special treatment (16).

The term apallic syndrome only describes a clinical condition and does not imply a typical neuropathologic abnormality. The most prominent symptoms of this disorder are summarized in Table 2. The patients are awake but not aware (Coma vigile). There is lack of emotional

Table 2 The most prominent symptoms of the apallic syndrome. Coma vigile: the patient is awake, but has no conscious activity nor any conscious capacity

Coma vigile (awake but not aware)
Lack of emotional reactions
Appearance of primitive motor patterns
Fixed body posture
Extreme overactivity of the sympathetic nervous system
Sleep regulated by exhaustion

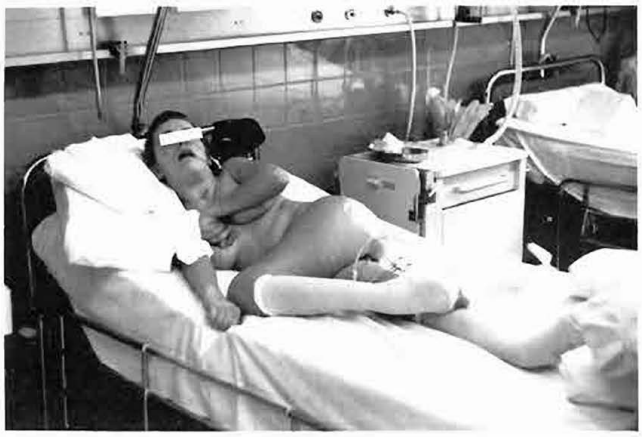

Fig. 5 "Frozen" Magnus-de Kleyn reflex seen in a 22-year old female patient. The patient was immobile in this position

Fig. 6 Using the method of tonus regulating reflex therapy a significant improvement of body position was noted after months of training

Table 3 Involuntary abnormal motor patterns (postures and responses) and limb movements seen both in acute traumatic coma and in chronic traumatic brainstem disorders

Turning, rolling movements
Decorticate posture
Decerebrate posture
Normal/abnormal movements combined with abnormal posture
Decorticate combined with decerebrate posture (Early and late third nerve state) (First and second stage of lateralized midbrain syndrome)
Flaccidity

reactions. primitive motor patterns appear and posture of body and extremities is rather fixed. There ist extreme overactivity of the sympathetic nervous system leading to catabolic drive. A sleep – wake cycle exists, but sleep is regulated by exhaustion (10).

Involuntary Abnormal Motor Patterns in Acute and Chronic Traumatic Brain Disorders

Involuntary abnormal motor patterns and limb movements, which can be seen in both acute coma and chronic traumatic brainstem disorders are listed in Table 3. Turning and rolling move-

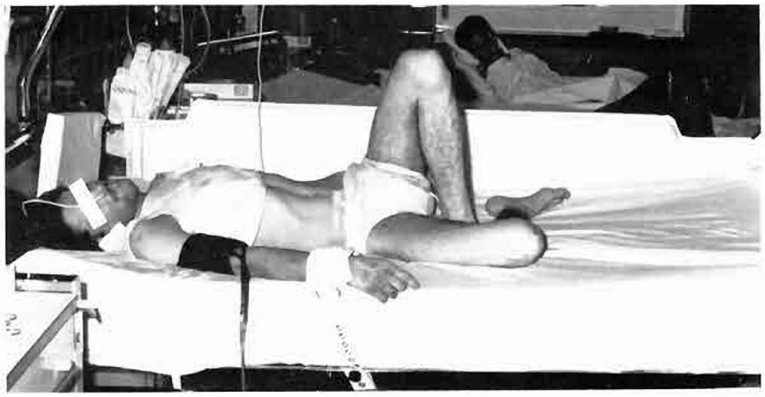

Fig. 7 Turning and rolling movements in a 28-yearold male patient in a prolonged stage of midbrain syndrome. These movements may be continued over weeks, but are not seen in patients in an apallic syndrome

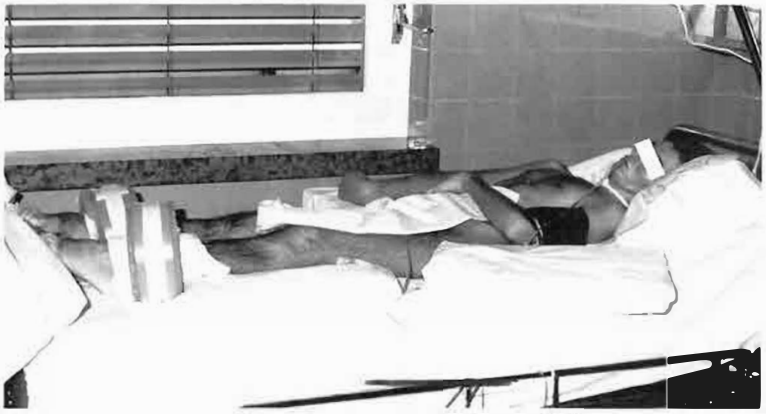

Fig. 8 Initial posture of the second stage of lateralized midbrain syndrome in a 24-year old male patients months after the traumatic brain injury. This posturing indicates uncal herniation. Indeed the patient had left epidural hematoma, which was removed late, hours after the accident

ments (Fig. 7) appear in early stages of midbrain syndrome and are suggested to be coordinated within the reticular formation of the brainstem (11). Usually these movements characterize a prolonged midbrain syndrome. More severe patterns of abnormal postures or abnormal postural responses to pain include decorticate rigidity, better termed flexor spasm, decerebrate rigidity or extensor spasm, flaccidity respectively. Normal (localizing pain) or abnormal (paretic) movements may be combined with decerebrate posture on the other. The persistence of these motor abnormalities in chronic stages allows to classify the grade of the initial brain injury, even months after acute trauma (Fig. 8, see also Fig. 2). Turning and rolling movements and unilateral normal or slightly abnormal movements are usually not present in patients at an apallic state.

Involuntary Abnormal Motor Patterns Characteristics for Chronic Brainstem Disorders

Abnormal motor patterns totally different from those seen in acute coma are listed in Table 4. Immobility due to a rather fixed neck respons is classified as a "frozen" neck response (see Fig. 5). Posture reflexes, such as the symmetric or asymmetric tonic neck response can be elicited in most patients, but the degree of the response may differ strongly from case to case.

Table 4 Involuntary abnormal motor patterns characteristic for chronic traumatic brainstem disorders. These motor abnormalities are not seen in acute coma

"Frozen" neck response
Flexion position (Flexion tetraparesis)
Stereotyped movements combined with abnormal postures
Palatal myoclonus with related myoclonus (of eyes, face, extremities)
Primitive motor patterns of oral sense (Chewing, sucking, swallowing)
Grasp reflexes

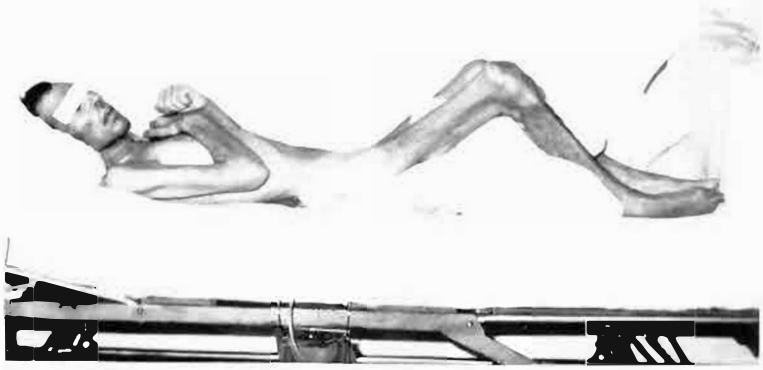

Fig. 9 One of the most prominent posture in apallic patients: Flexion position (flexion tetraparesis). The 34-year old patient further shows signs of marasm, due to continued overactivity of the sympathetic nervous system

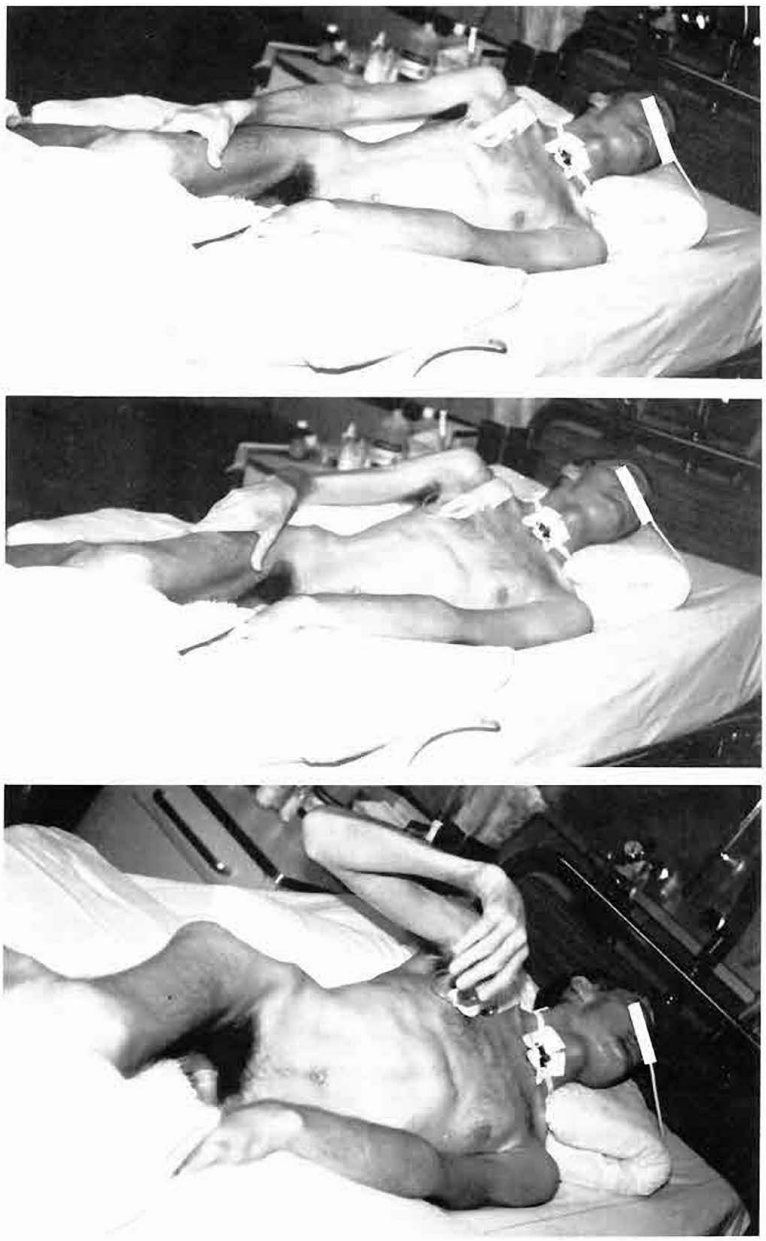

Fig. 10
a) This 24-year old apallic patient demonstrate a stereotyped voluntary movement of the right hand. The left extremities are extended. He starts his stereotyped movement from the knee

b) and moves his right hand gently to his hip

then elevates his arm to turn back to the knee. These movements lasted over hours and could not be stopped to command

The flexion position (flexion tetraparesis) is one of the most prominent postures in apallic patients (Fig. 9). Stereotyped movements combined with abnormal postures is another characteristic motor pattern (Fig. 10 a, b, c). The stereotyped movements are frequently repeated over hours and can neither be started nor stopped to command. In patients, who regain awareness, parts of these movements can be used to train the very first voluntary movements.
Palatal myoclonus with related myoclonus of the eyeballs, face and extremities is part of a chronic brainstem disorder, which appears after degeneration of connections between the cere-

bellar dentate nucleus, the red nucleus and the inferior olivary nucleus (The Guillan-Mollaret triangle) with consequent hypertrophic degeneration of the inferior olivary nucleus (5, 9, 14). The myoclonus is independent of cortical mechanism, and therefore also can be seen in apallic patients. Primitive motor patterns of oral sense and the grasping reflexes usually accompany other abnormal involuntary movements and disappear with increasing recovery of the higher cortical functions. In contrast, these primitive motor patterns persist in apallic states.

Frequency, Prognostic and Localizing Value of Involuntary Abnormal Motor Patterns

The frequency of postures, movements and motor responses found in 53 patients with severe disability outcome investigated three weeks to six months after brain injury is given in Table 5. 52 of the 53 patients had abnormal postures of extremities at least on one side. Decorticate posture, posture of second stage of lateralized midbrain syndrome, flexed position and flaccidity were the most prominent abnormal motor patterns. At the same period of time we could observe 14 patients with good or moderate disability outcome. Nine of them demonstrated abnormal postures (Table 6). However, there are striking differences in some types of posture between patients with good/moderate disability outcome and severe disability outcome. No patient with good or moderate disability outcome showed the posture of the second stage of lateralized midbrain syndrome, decerebrate posture or flexed position, but nearly half of the patients with severe disability outcome, including apallic states, did so (Table 7). Decerebrate posture (extensor spasms) is a constitutional sign of midbrain affection due to transtentorial herniation (13, 22, 27) but also seen in cases of primary brainstem injury (6, 21). Since extensor spasm were also seen in localized brainstem lesions with intact cortical functions (3, 15, 28) the term decerebrate posture is intrinsically fallacious (7). Clinical observations in patients with locked-in syndrome suggest that extensor spasms are stimulus-sensitive composed reflexes built – in within the bulbar brain and released by interruption of inhibiting pyramidal influences somewhere above midbrain level (3). The second stage of lateralized midbrain syndrome (decorticate combined with decerebrate posture) appears if a space occupying lesion makes the temporal lobe sliding medially squeezing directly the midbrain and pressing the contralateral cerebral peduncle against the opposite tentorial edge. Thereby decerebrate posture develops ipsilateral to the expanding supratentorial lesion. Compression of the ispilateral third nerve leads

Table 5 Frequency of involuntary postures, movements and motor responses in 53 patients with severe disability outcome in a chronic state after head brain trauma

Rolling, turning movements	1
Normal/abnormal movements comined with abnormal posture	8
decorticate posture	11
Decorticate posture combined with decerebrate posture	10
Decerebrate posture	4
flexed position	10
flaccidity	7
"Frozen" neck response	1
Decerebrate posture – arms	1
Flexion – legs	1
Abnormal postures: 52	

Table 6 Frequency of involuntary postures, movements and motor responses in 14 patients with good/moderate disability outcome in chronic condition after head brain injury

Normal	1
Rolling, turning movements	4
Normal/abnormal movements combined with abnormal postures	5
Decorticate postures	3
Flaccidity	1
Abnormal postures:	9

Table 7 Striking differences in motor patterns between patients with good/moderate disability outcome and severe disability outcome. None of the patients with good or moderate disability outcome showed postures of the second stage of lateralized midbrain syndrome, decerebrate posture or flexion position

	% Observation	
	Good/ moderate	Severe
Decorticate posture, combined with decerebrate	0	19
Decerebrate position	0	8
Flexed position	0	19

to an ipsilateral dilated pupil. While decerebrate posture does not allow a clear localization of the brain lesions, that underly the pathogenesis of this posture, the posture of second stage of lateralized midbrain syndrome is rather characteristic for uncal herniation. Flexion position (flexion tetraparesis) usually emerge after decerebrate position in apallic patients. Therefore the localizing value of this posture is as low as decerebrate posture. Muscle flaccidity with little or no response to stimulation, correlates most strongly with damage to the brainstem lying within or distal to the lower pontomedullary region (27). However, one has to keep in mind, that severe polyneuropathy due to malnutrition can provoke a similar clinical picture. While in the former case the prognosis is literally poor, there will be impressive improvement in the later case, if peripheral denervation is reversible (one case, see Table 6).
Rather rare observations are "frozen" neck responses or decerebrate postures of the arms accompanied by flexion or flaccidity of the legs. In the latter case the structural damage involves the pontine tegmentum (27).
The abnormal postures mentioned above, reflect the severity of brain lesions and contribute information about the degree of chronic brainstem impairment. Thus, these motor patterns provide as useful information about the final outcome of patients, as the relationship between early motor responses in coma and the final outcome (18, 25).
Decorticate posture (abnormal flexor response in the the arms with extension of the legs) is another motor pattern, which hardly can be localized accurately to one brain abnormality responsible for this posture, or hardly can be differentiated from lesions that cause decerebrate posture in man. Clinical observations have long recognized, that decorticate posture carries less serious prognosis than decerebrate posture in acute coma (19, 25). Our study confirms this impression in chronic traumatic brainstem disorders. We found patients with good or moderate disability outcome, who have demonstrated decorticate posture even in chronic conditions.
Rolling and turning movements reflect the lacking control of the reticular formation of the brainstem by the pyramidal tracts. Usually these movements decrease with increase of voluntary movements. The prognosis of these patients is generally good, but the localizing value of these movements is poor. However, they point to a relatively intact brainstem reticular formation.
Normal or slightly abnormal movements of extremities on one side combined with abnormal posture on the other, characterize the early stage of uncal herniation. In contrast to the persistent posture of the second stage of lateralized midbrain syndrome, the neurological deficits are frequently reversible in these cases. Therefore the first stage of lateralized midbrain syndrome marks the critical point of time, at which supratentorial space occupying masses should be removed at the latest, to assure a fair chance for the patient's recovery.

The localizing value of abnormal postures is high in cases with flaccidity, with second stage of lateralized midbrain syndrome or in patients demonstrating decerebrate postures of the arms and flexion or flaccidity of the legs. Another involuntary movement disorder of high localizing and prognostic value is seen in more chronic traumatic brainstem disorders weeks or months after the injury. This disorder is characterized by various types of segmental myoclonus. A palatal myoclonus was found to be accompanied by involuntary ocular movements best classified as opsoclonus. Another type of segmental myoclonus was seen in upper extremities supplied from cervical spinal cord structures. If all forms of segmental myoclonus were fully present, the prognosis was generally poor, because the patient remained severely disabled. Pathologic findings indicate, that this myoclonic disorder is attributed to lesions of the inferior olive, dentate nucleus and central tegmental tracts (5, 8). The hypertrophic degeneration of the olives due to the process of transsynaptic degeneration has been reported to play an important part in the production of palatal myoclonus (9). Therefore this myoclonus syndrome reflects irreversible damage of the brainstem. Primitive motor patterns of oral sense such as chewing, sucking and yawning are interpreted as automatisms generated within the brainstem and released due to the lacking influence of the cortical inhibiting system (4). There is no localizing value and also the prognostic significance is poor. However, the persistence of these movements in chronic states indicates the continued lack of cortical inhibition. Therefore they are characteristic symptoms in patients in an apallic state.

Except in cases with uncal herniation, flaccidity, extensor spasms of the arms and flaccidity of the legs and except in cases with segmental myoclonus neither experimental nor human post mortem studies provide a precise anatomic basis for the origins of the abnormal postures and movements described above. However, the abnormal postures reflect the severity of brain lesion and contribute information about the degree of chronic brainstem impairment. Certainly, abnormal postures, movements and motor response can not characterize a chronic traumatic brainstem disorder alone, and pupil's size and reaction, the oculocephalic and oculovestibular reflexes are of localizing, diagnostic and prognostic value. However, except pupil's reaction to light no other symptom carries a prognostic significance as high as abnormal postures (2). Although many of the abnormal postures and movements seen in acute traumatic coma can also be observed in prolonged traumatic disorders, there are several involuntary motor patterns, which are rather specific for chronic conditions. Generally, the prognostic significance of abnormal postures, movements and responses in chronic traumatic brainstem disorders seems to be as high as in the acute state of coma.

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Edited by
K. Kunze, W. H. Zangemeister, A. Arlt

With Contributions by

A. Arlt
H. Buchner
J. H. Buckley
U. W. Buettner
W. Bushart
G. D. Cascino
K. H. Chiappa
J. E. Desmedt
G. Deuschl
M. Dieterich
A. Ferbert
F. Gerstenbrand
W. Hacke
Ch. Hamburger
H. C. Hansen
I. Hashimoto
R. W. C. Janzen
K. Jellinger
K. Kunze
P. Langhorst
N. A. Lassen
R. J. Leigh
D. E. Levy
C. H. Lücking
C. H. Markham
B. W. Ongerboer de Visser
S. Otto
M. S. Pessin
G. Rancurel
E. Rumpl
L. Stark
A. Starr
W. I. Steudel
A. Thagavy
W. H. Zangemeister
St. Zschocke

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