CLINICAL EVALUATION OF THE SEVERE HEAD INJURY

F. Gerstenbrand[1], L. Saltuari[1], M. Kofler[1], R. Formisano[2]

Critical review

SUMMARY - Clinical evaluation and classification of patients suffering from severe head injury are essential for an effective treatment and rehabilitation of these patients. The subdivision of the midbrain syndrome in four separate stages and of bulbar brain syndrome in two stages is a valid method to describe patient's level of cerebral dysfunction.

The diagnosis of midbrain syndrome I and II usually allows a favourable prognosis. In the stages III and IV also influences of other factors have to be considered in defining the patient's outcome. In these cases the Innsbruck Coma Scale can be a useful tool to establish a valid prognosis.

Key words: head injury, midbrain syndrome, bulbar brain syndrome, Innsbruck coma scale

Introduction

The first description ever of a neurological disease is documented in the so-called Smith papyrus from 1700 B.C. Smith found the characterization of the neurological sequelae after a severe head injury of an Egyptian soldier who was hurt in a war.

The increasing incidence of traffic accidents after the Second World War led to a progressive increment of patients suffering from severe head and brain injury. It became the most frequent cause of disability in young male adults. One out of twenty head traumas is so severe to cause immediate or secondary disturbance of consciousness with potential coma (Morosini 1983). Evans et al. reported on approximately 140,000 admissions to hospitals due to head and brain injury in Great Britain per year, 7,000 of them being discharged severely handicapped and not fully able to work, a few hundreds being completely disabled. Most of the patients with severe head and brain injury belong to the age group between 15 and 19 years or are older than 75 years, with a distinct majority of the male sex, except for the older age group (Evans et al. 1982).

Similar data (200-250 admissions to hospitals due to head trauma per 100,000 inhabitants) were published by Strang et al. (1978), Kalsbeek et al. (1980), and Aitken et al. (1982). Already in 1968 Lewin calculated that 0.4% of patients admitted to hospitals due to head traumas (14-22 cases per 100,000 inhabitants) developed a prolonged coma with a duration of more than one month (Lewin 1968).

1. Department of Neurology, University Hospital, Innsbruck, Austria

2. Department of Neurology, University "La Sapienza", Rome, Italy

Different Forms of Head and Brain Injury

The term "commotio cerebri" is reserved for the loss of consciousness due to head and brain trauma without any structural cerebral lesion and with prompt recovery. "Contusio cerebri" and "concussio cerebri" are used for head and brain injuries of different degree of severity, but from the neuropathologist's view it seems questionable to distinguish between them. Both are characterized by a longer period of unconsciousness and by signs of brainstem dysfunction.

Although skull fractures are often associated with brain lesions they do not always accompany serious head and brain traumas.

A supratentorial intra- or extracerebral space-occupying lesion may lead to a displacement of the cerebral tissue, thus resulting in different types of cerebral herniation (cingular, tentorial or foraminal). Involvement of the brainstem may primary be due to the direct mechanical impact or hemorrhage (acute phase), secondary due to a downward displacement and direct compression or ischaemic lesion because of progressively increasing intracranial pressure (acute or subacute phase), and tertiary in the case of pontine myelinolysis.

Compression of the mesencephalon towards the tentorium due to a supratentorial space occupying lesion is of particular importance, especially because of the involvement of the oculomotor nerve (in sense of paresis) and the posterior cerebral artery (in terms of compression and occlusion) on the side homolateral to the lesion. Clinical signs developing with compression of the brainstem include a sequence of pathologic motor patterns, optomotoric disturbances, respiratory and vegetative dysregulation and dysfunctions of the ascending reticular formation, indicating a gradual impairment of the midbrain and the bulbar brain.

The prognosis of patients suffering from a serious head and brain injury depends to a high degree on the severity and duration of the initial coma, on patient's age and on the increase of the intracranial pressure.

The Acute Mesencephalic Syndrome

According to Gerstenbrand and Lücking (1971) the acute mesencephalic syndrome may be subdivided into four stages of midbrain syndrome and into two stages of bulbar brain syndrome (Figure 1).

Midbrain syndrome, stage I: The most important clinical signs are an attenuation of the sensorium and a reduction of vigilance in terms of somnolence, with spontaneous movements of the limbs and body, and with finalized responses to the external stimuli. Occasionally slight hyperreflexia and positive pyramidal signs may occur (Figure 2).

Midbrain syndrome, stage II: In this phase alterations of the sensorium are more pronounced in a sense of sopor or coma. Responses to painful stimuli are either retarded and slowed down (stage IIa) or not finalized with a tendency to flexion of the upper limbs (stage IIb). Muscle tone in the lower limb extensors may by slightly increased and intensified after the nociceptive stimulation. The bulbi tend to be slightly divergent with a transient swaying movement. Pupillar reaction to light is somewhat decreased. The deteriorating dysfunction of the mesencephalon entails also an elevation of muscle tone, hyperreflexia and positive pyramidal signs. The vegetative disturbance is reflected by an increase in respiratory and heart rates and by an increase in the arterial blood pressure (Figure 3).

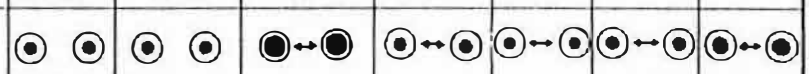
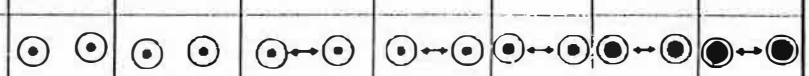
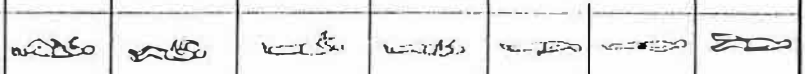
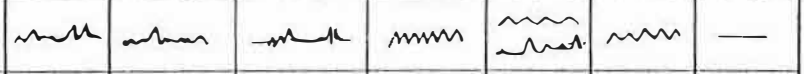
Stages of brainstem lesion due to increased supratentorial pressure		Acute midbrain syndrome				Acute bulbar brain syndrome		
		I	II a	II b	III	IV	I	II
Vigilance		somnolence	sopor	coma	coma	coma	coma	coma
Reaction	to acoustic stimuli	slightly delayed						
	to nociceptive stimuli	prompt finalized defence reaction	delayed non purposeful defence reaction	rest of a non finalized defence reaction	flexion of upper, extension of lower extremities	extension of upper and lower extremities	rest of extension of upper and lower extremities	absent
Oculomotricity	position of bulbi	normal swaying	slightly divergent oscillation	divergent dysconjugated	fixed divergent absent	fixed absent	fixed absent	fixed absent
	pupillary diameter							
	reaction to light							
Motility	posture							
	spontaneous movements	mass movements and rotations	mass movements of upper limbs extension	mass movements of upper lims, extended position of lower limbs	flexion of upper, extension of lower limbs	extension of upper and lower limbs	rest of extension of upper and lower limbs	flaccid posture
	muscle tone	normal	slightly increased in lower limbs	increased in lower limbs	increased	markedly increased	slightly increased	flaccid
	pyramidal signs							
Vegetative parameters	respiration							
	heart rate	slightly increased	normal	increased	increased	markedly increased	increased	decreased
	blood pressure	normal	normal	normal	slightly increased		normal	decreased
	body temperature	normal	normal	slightly increased	increased	markedly increased	increased	normal or decreased

Figure 1. Main neurological signs of different stages of the mesencephalic syndrome.

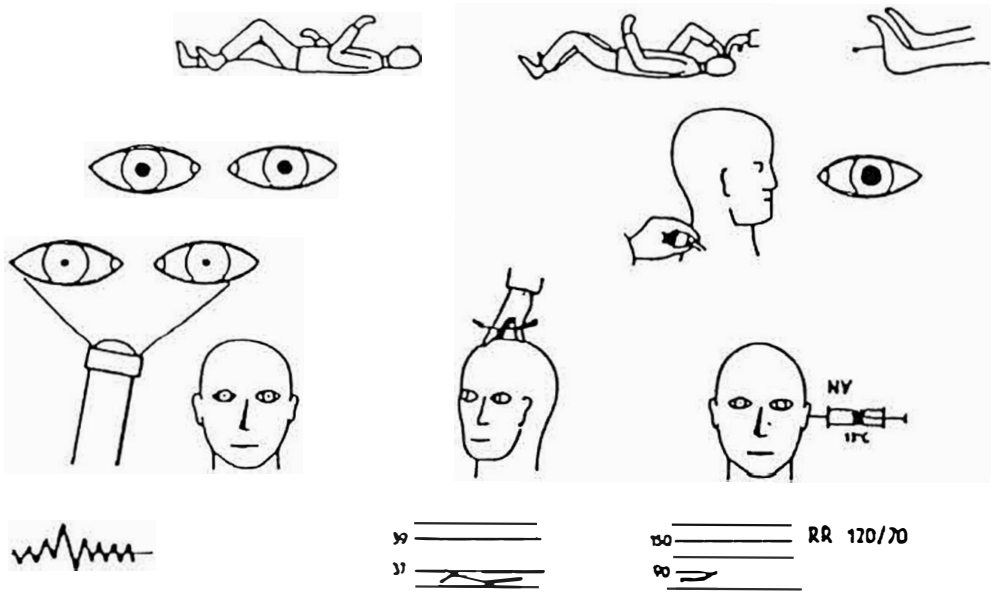


Figure 2. Midbrain syndrome, stage I.

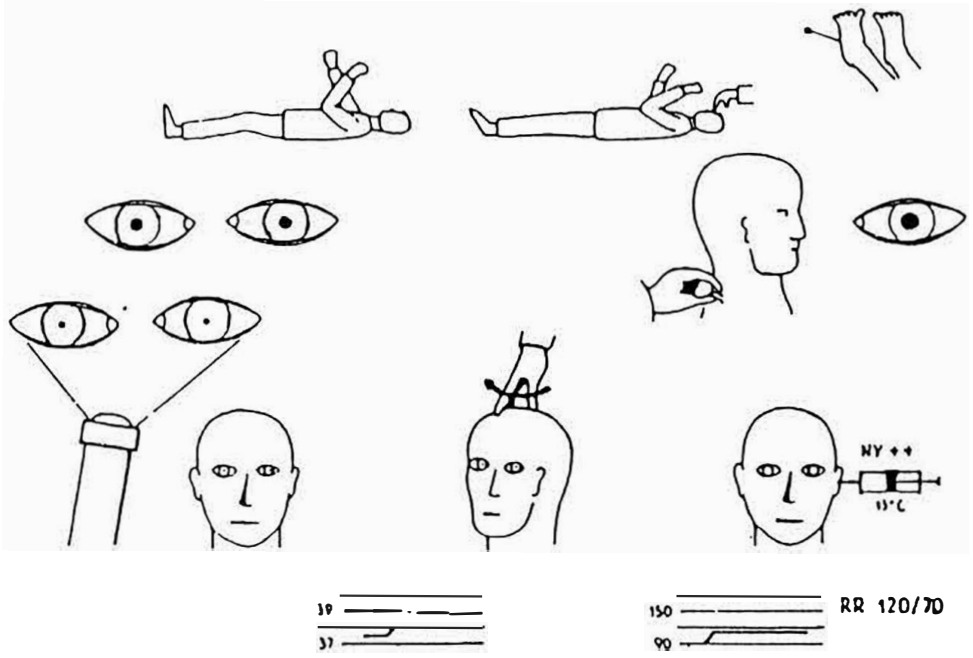


Figure 3. Midbrain syndrome, stage II.

Midbrain syndrome, stage III: Typical feature of this stage is motor pattern of decortication - (flexion of the upper and extension of the lower extremities) - which is intensified by painful stimulation. Patients are comatous, with markedly increased muscle tone, hyperreflexia and positive pyramidal signs. The pupillary diameter may be normal or smaller with a reduced reaction to light. The bulbi are divergent. Respiratory rate is increased but rhythmic. Heart rate, arterial blood pressure and body temperature are increased (Figure 4).

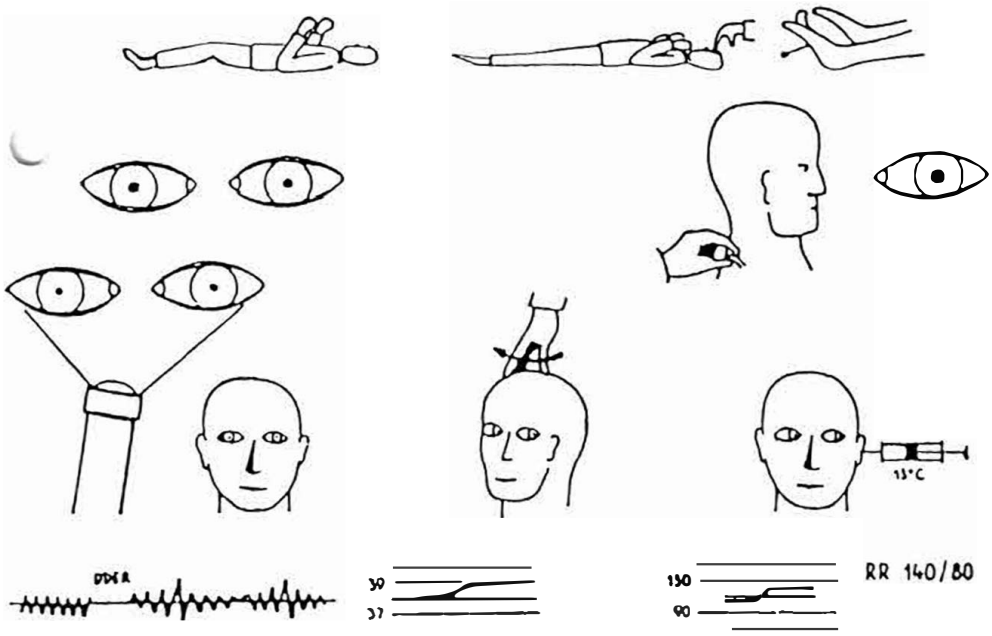


Figure 4. Midbrain syndrome, stage III.

Midbrain syndrome, stage IV: Progressive motor dysfunction becomes obvious. Typical decerebration posture, occurring spontaneously and increasing in intensity after nociceptive stimuli is evident. Muscle tone of the upper and lower limbs is elevated to the degree that tendon jerks may not be elicited. Pyramidal signs are highly positive. The pupils show no reaction to light or painful stimuli, and their diameter is often irregular. The bulbi are fixed in a divergent position. Abnormalities of the vegetative parameters are more pronounced than in the preceding stage (Figure 5).

Bulbar brain syndrome, stage I: This stage is characterized by continuation of coma and absence of any reaction to the external stimuli and by slight diminution of the increased muscle tone and of brisk tendon reflexes. Pyramidal signs are reduced too. Additional features are: bulbi fixed in a divergent position, mydriasis and absence of pupillary reactions to light. Heart rate, arterial blood pressure and body temperature tend to be low (Figure 6).

Bulbar brain syndrome, stage II: In this stage all brainstem functions cease. Patients are in a state of deep coma, their muscles are flaccid, tendon jerks are absent, but pyramidal

signs are sometimes present. The bulbi are fixed in a divergent position and the pupils are maximally mydriatic without reaction to light or pain. Respiration subsides, whereas heart rate, arterial blood pressure and body temperature drop to normal values (Figure 7). Brain death occurs in approximately twenty minutes.

Brainstem reflexes, such as ciliospinal, oculocephalic (doll-head phenomenon) and vestibulo-ocular reflex play an important role in determining the localisation of the brainstem lesion and its extent. E.g. the vestibulo-ocular reflex (cold calorization) shows tonic conjugated movement of both eyes in stage III of the midbrain syndrome, dissociated movement in stage IV of the midbrain syndrome, and no reaction in the bulbar brain syndromes.

The most relevant clinical features for classification of brainstem lesions are pathological motor patterns of either decortication or decerebration, and serious respiratory disturbances, even in case of intact oculomotor reflexes.

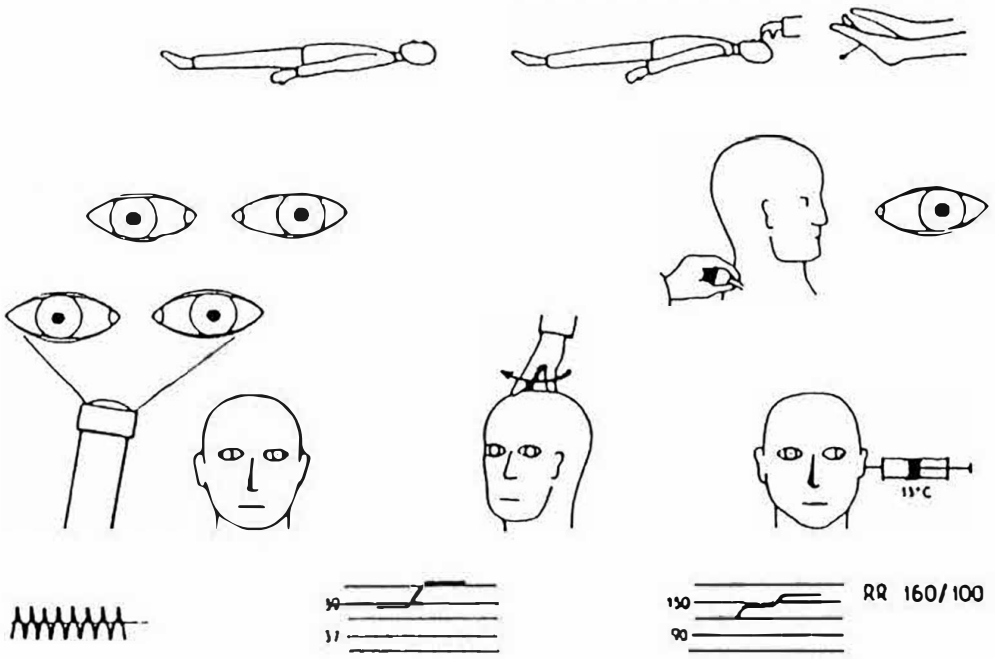


Figure 5. Midbrain syndrome, stage IV.

Furthermore Gerstenbrand et al. (1973) suggested the distinction between two different mesencephalic syndromes: the medial and the lateral mesencephalic syndrome. The first follows deterioration in the rostro-caudal direction as described above (midbrain syndrome, stages I-IV). Patients with the lateral mesencephalic syndrome, however, show typical features of a unilateral supratentorial space occupying process with predominantly unilateral compression of the brainstem. It appears in two clinical pictures of different severity.

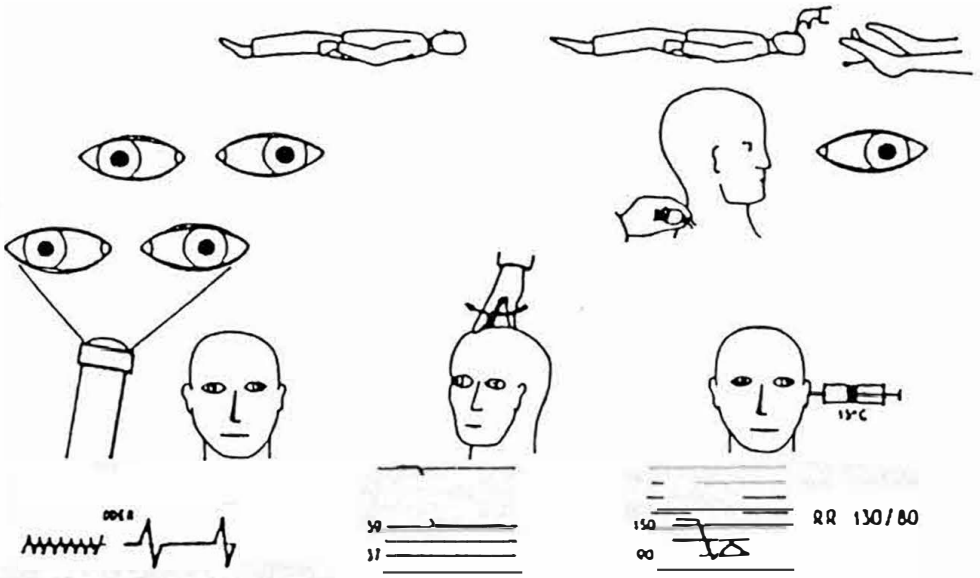


Figure 6. Bulbar brain syndrome, stage I.

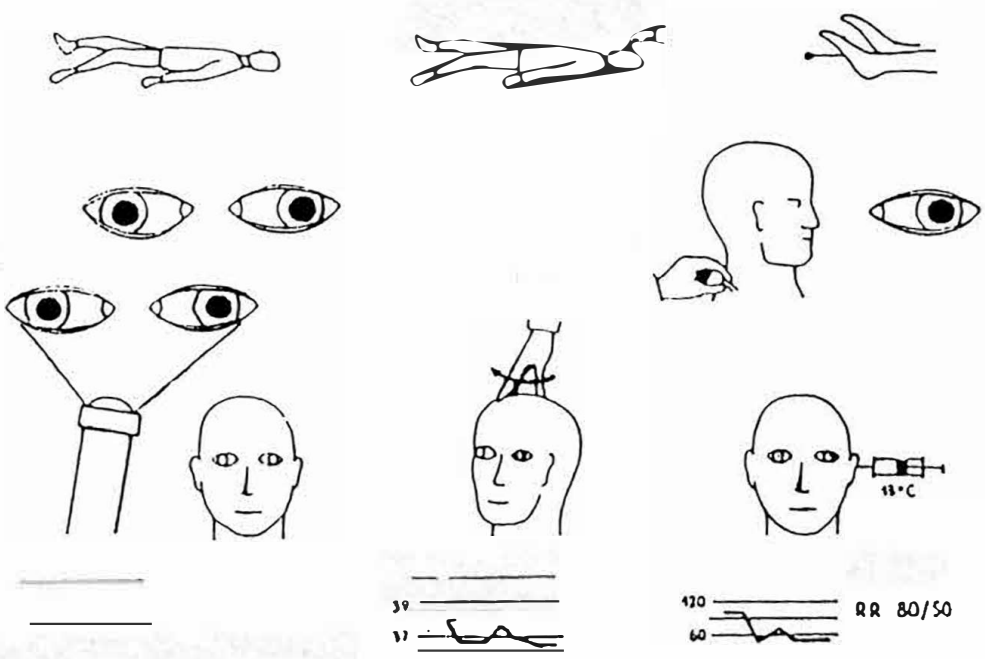


Figure 7. Bulbar brain syndrome, stage II.

Lateral mesencephalic syndrome - stage I: Patients are somnolent and show posture typical of decortication on the side homolateral to the space occupying supratentorial lesion. Muscle tone and tendon jerks are increased on that side, pyramidal signs are positive. Painful stimuli lead to nonpurposeful defense reactions on the side contralateral to the lesion and to a homolateral intensification of the decortication posture. Head and gaze are deviated to the side of the lesion. Ipsilateral oculomotor nerve is paralyzed due to its compression on the tentorium what results in a dilated pupil with a sluggish reaction to light. Vegetative parameters are analog to those of the medial mesencephalic syndrome stage II (Figure 8).

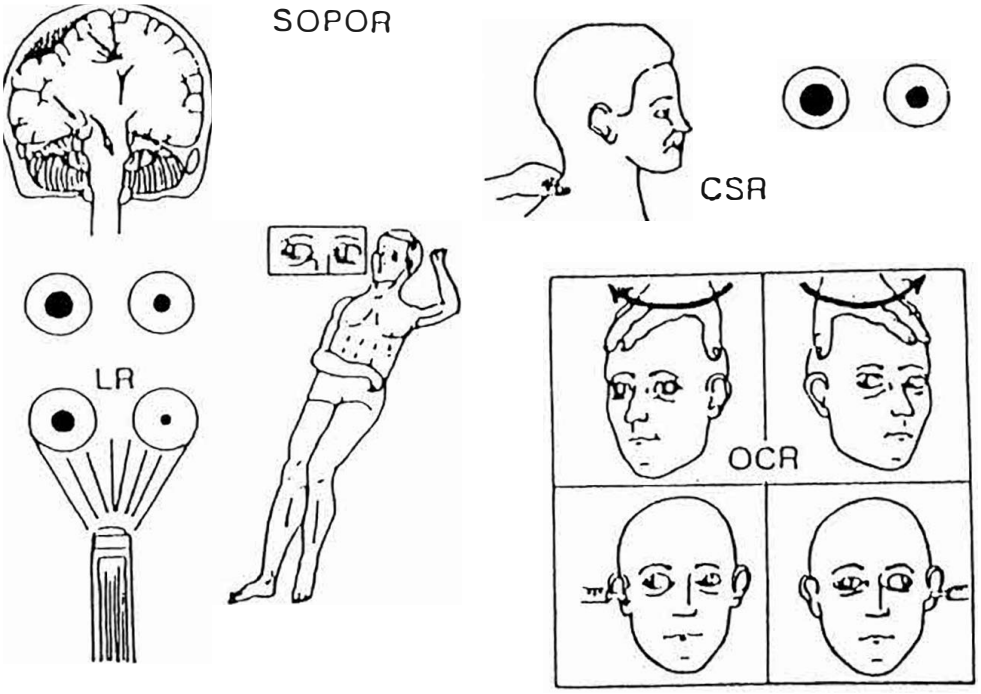


Figure 8. Stage I of acute lateralization of the mesencephalic syndrome.

Lateral mesencephalic syndrome - stage II: Patients are comatous, with the decerebration motor pattern homolaterally (extended upper and lower extremities) and the decortication pattern contralaterally (flexed upper and extended lower extremities) to the space occupying supratentorial lesion. Muscle tone and tendon jerks are exaggerated on both sides, but are more pronounced on the side homolateral to the lesion. Head and gaze are in a fixed position deviated to the side of the lesion, with the divergent bulbi. The pupil contralateral to the lesion is narrow with clearly diminished reaction to light, while homolateral pupil is dilated. Vegetative symptoms resemble those described in stage III of the medial mesencephalic syndrome (Figure 9).

With further deterioration midbrain syndrome stage IV develops, but with the maintenance of anisocory.

The described subdivision of the mesencephalic syndrome into different separate stages allows rather accurate classification of the cerebral dysfunction in the acute phase of

head injury. Daily monitoring of these patients, however, requires a simpler scale, that can easily be applied also by the nursing staff. Since the widely used Glasgow Coma Scale includes too few and partly inappropriate items, e.g. verbal responses, the Innsbruck Coma Scale was developed (Table 1).

The Innsbruck Coma Scale includes evaluation of the following parameters: patient's response to both acoustic and nociceptive stimuli, spontaneous body posture and movements, position of the eyelids, and position or movements of the bulbi. Particular attention is paid to the optomotorics (pupillar diameters and their reaction to light) which is an important brainstem function. Evaluation of the oral automatisms appears to be useful especially in case of post-traumatic coma prolongé in which the described features as well as automatic chewing movements occur 3-7 days after the acute phase.

According to the Innsbruck Coma Scale the severity of each sign is graded from 0 to 3. In patients who show asymmetricity of signs, the better one is chosen for quantification. Four or more examinations are performed each day to detect any tendencies towards improvement or particularly towards deterioration to be able to react adequately in appropriate time.

None of the comatous patients admitted to the intensive care unit of the University hospital of Innsbruck in the last year survived if he/she had not reached an initial score of at least 6 points. All patients with an average value of less than 11 points also died, what points out good correlation of the low Innsbruck Coma Scale score and unfavourable prognosis (Gerstenbrand, Rumpl 1982).

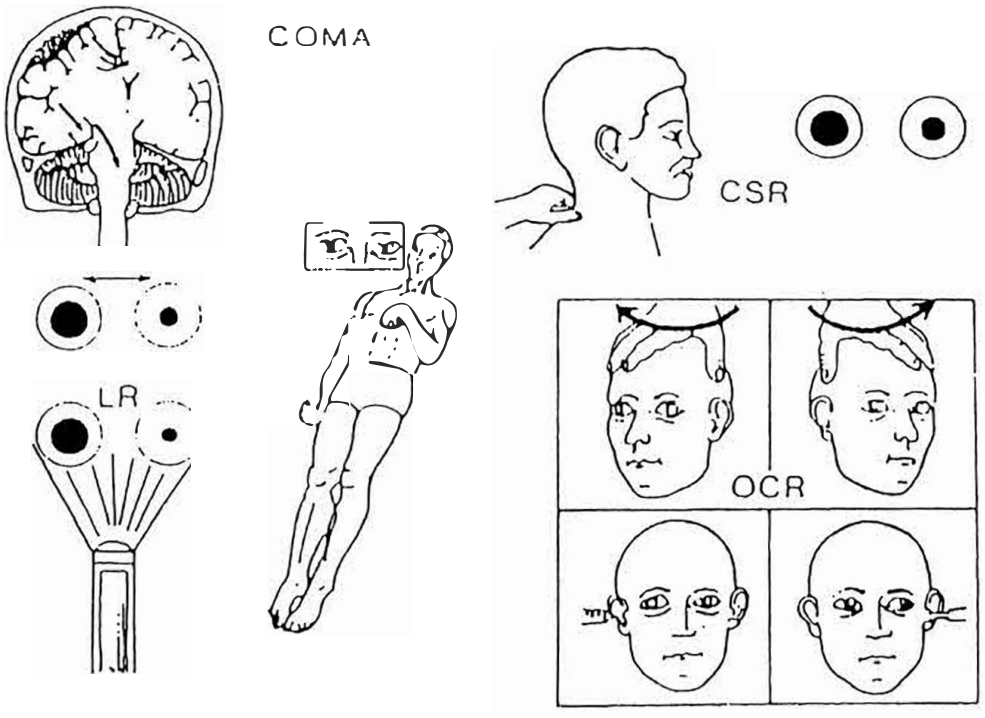


Figure 9. Stage II of acute lateralization of the mesencephalic syndrome.

Table 1

The Innsbruck Coma Scale

	Date Time	
Reaction to acoustic stimuli	Finalized Better than extension Extension No reaction	3 2 1 0
Reaction to nociceptive stimuli	Finalized defense reaction Better than extension Extension No reaction	3 2 1 0
Posture and body movements	Normal Better than extension Extension Flaccid	3 2 1 0
Position of eyelids	Opening of eyes spontaneously Opening of eyes to acoustic stimuli Opening of eyes to nociceptive stimuli No opening of eyes	3 2 1 0
Pupillary diameter	Normal Reduced Dilated Minimally dilated	3 2 1 0
Pupillary reaction to light	Prompt Small range of contraction Minimal Absent	3 2 1 0
Position and movement of bulbi	Optical following Swaying of bulbi Divergent, variable Divergent, fixed	3 2 1 0
Oral automatismus	Spontaneous Due to external stimuli None Maximal scores	3 2 1 0

According to Gerstenbrand and Rumpl (1982) a step-wise recovery is possible from each stage of the mesencephalic syndrome. The death of patients with acute head and brain injury may be due to either cardiovascular arrest or to secondary midbrain syndrome deteriorating gradually to the bulbar brain syndrome and terminating with brain death.

The Transition Stage from Midbrain Syndrome to Post-traumatic Apallic Syndrome

Patients presenting with primary or secondary lesions of the brainstem who do not recover may develop a prolonged post-traumatic coma, with a particular clinical picture of mesencephalic syndrome which is called the transitional stage to an apallic syndrome (Avenharius and Gerstenbrand 1975). It may be subdivided into three different phases: the coma prolongé (Fau 1956, Vigouroux et al. 1964), parasomnia (Jefferson 1944), and the akinetic mutism (Cairns et al. 1941, Cairns 1954).

Coma prolongé: Approximately 3 - 5 days after the acute incident the patient develops extensor spasms, initially elicited only by painful stimuli, but by the time occurring more and more spontaneously. Tendon jerks are increased and pyramidal signs are present bilaterally. Profound disturbance of consciousness without any reactions to the external stimuli persists. The bulbi remain in a divergent position, but are not fixed any longer. The oculocephalic or vestibulo-ocular reflex are hardly present. Ciliospinal reflex is markedly increased. Pupillary reaction to light is sluggish. Vegetative functions tend to be stabilized. If this stage lasts for 3-7 days, spontaneous chewing movements may occur, which are interpreted as primitive oral patterns (Poeck and Hubach 1963).

Parasomnia: Patients still remain comatous with their eyes closed. There is no reaction to external stimuli except for a tendency to flexion or extension of the extremities on the nociceptive stimulation. Coma appears to be more superficial and reminds of a status similar to sleep (parasomnia). In this stage head and gaze deviations may be present. There is continuous hyperreflexia, increased muscle tone and positive bilateral pyramidal signs. The bulbi show horizontal nystagmus with a small rotatory component.

Oculocephalic reflex can be elicited and vestibulo-ocular reflex shows tonic reaction. Vegetative parameters become more and more stabilized, although painful stimuli evoke an "emergency reaction" with the elevated sympathetic drive. Chewing movements appear either spontaneously or following external stimuli. At the end of this stage, which lasts 3 - 5 days, the so-called snout reflex may be elicited.

Akinetic mutism: In this stage patients open their eyes. This happens for the first time in the course of the transition to a post-traumatic apallic syndrome. Initially the awake periods are short, but extend with time. Blink and threatening reflexes fail to be elicited. In this phase the patient shows an increased tendency to respond to the external stimuli with the intensified vegetative reactions. Posture is the same as in the midbrain syndrome from which the transition to the apallic syndrome started. Pronounced flexion of all four limbs at this instant is a sign of an unfavourable prognosis. The bulbi are in a divergent position, but spontaneous horizontal movements are carried out. Oculocephalic and vestibulo-ocular reflexes are as in the preceding stage. Ciliospinal and snout reflexes are clearly evident as are also palmomental and naso-glabellar reflexes. They are present in almost all patients.

The persistence of a post-traumatic coma prolongé for more than 7 days as well as an interval of more than 5 days between the first two stages of transition to the apallic syndrome implicates an unfavourable prognosis.

Prolonged midbrain syndrome: If the transition from an acute midbrain syndrome to a post-traumatic coma prolongé lasts more than three days patients may pass directly into the so-called prolonged midbrain syndrome. Recovery from the disturbance of consciousness leads directly to a psycho-organic syndrome of variable duration (Gerstenbrand 1983).

Apallic Syndrome

Terminology: Numerous designations stand for the "apallic syndrome", terms which are not accepted everywhere (Peters and Gerstenbrand 1977, Peters and Rothemund 1977).

The first description of this syndrome is probably due to Rosenblath (1899). He reported on a case of a 15 year old male patient suffering from post-traumatic coma with extensor cramps. Some weeks later the patient was described to be "peculiarly alert". The condition remained unchanged until the patient died 8 months later. Now classical, historic report of an "apallic syndrome" was published by Kretchmer (1940), who defined the syndrome as an extensive and diffuse dysfunction of the entire pallium. The original description by Kretchmer referred to a marantic patient suffering from subacute sclerosing panencephalitis. He was awake with his eyes open but without contact with the environment. However, he was unable to comprehend any verbal orders and incapable of focussing his gaze and his attention on any object. Absence of any response, even emotional, characterized patient's condition. In spite of his awakeness he could neither speak nor recognize or carry out any previously learned behavioural patterns. Kretchmer noticed in his patient some vegetative sings and primitive patterns like yawning, sucking and grasping. He differentiated the apallic syndrome from the "usual coma" and from the dementia, and interpreted it as a blockade of the brain's functional capacity in toto, comparable to a panagnosia associated with panapraxia. Several other authors described similar clinical pictures of variable etiology (cerebral trauma, neurolues, severe cerebral arteriosclerosis, following the drainage of an epidermoid cyst at the bottom of the third ventricle, occlusion of the basilar artery etc.). Very different terminologies were used for rather similar clinical pictures: coma vigile (Mollaret and Goulon 1959), parasomnia (Jefferson 1952), hypersomnia (French 1952), hypersomnia prolongata (Facon et al. 1958), akinetic mutism (Cairns et al. 1941, Cravioto et al. 1960, Lhermitte et al. 1963), post-traumatic encephalopathy (Trillet 1940, Chavany et al. 1955, Dechaume et al. 1962, Jellinger 1965), post-traumatic edematous leukencephalopathy (Osetowska 1964), progressive post-traumatic encephalopathy (Kramer 1964), syndrome of decerebration or of rigidity due to decerebration (Mumenthaler 1961), prolonged unconsciousness (French 1952), post-traumatic catatonia (Jellinger 1965), hypertonic post-traumatic stupor (Fishgold 1957), severe dementia following cranial trauma (Strich 1956), progressive dementia with cachexia (Gruner 1965), vita reducta (Castaigne et al. 1962), persistent vegetative state (Jennet and Plum 1972). The classical definition of the apallic syndrome by Plum and Posner (1972) reads as follows: It is a diffuse progressive cerebral dysfunction involving the cortex and the white matter, affecting patients with an intrinsic degeneration of neurons and glia. Examples include senile or presenile cerebral atrophy, diffuse cerebral vascular sclerosis, subacute sclerosing panencephalitis, the most severe forms of multiple sclerosis and the syndrome of Marchiafava-Bignami. In each of these cases the apallic syndrome is considered to be an irreversible and terminal condition.

In our opinion the apallic syndrome may also develop secondary from an acute or subacute cerebral dysfunction. This may be caused by an inner cerebral lesion or by an interruption of the afferent and efferent pathways due to an organic or functional lesion at any level between the cortex and brainstem. Examples include hypoxia of different etiology (strangulation, obstruction of airways, accidents at anaesthesia, cardiac arrest, etc.), cerebral

edema due to allergic reactions, diffuse encephalitis, gas embolism, metabolic disorders (hepatic coma, uremic coma, etc.), prolonged insuline shock, exogenous intoxications (carbon monoxide, mercury poisoning, etc.), cranio-cerebral traumas, subarachnoid hemorrhages with obstruction of the basal cisternae; mesencephalic tumors and vascular disorders.

Symptomatology: Fully developed apallic syndrome shows remarkable uniformity in symptomatology independent of its etiology. Symptoms are due to the impaired cerebral functions at the meso-diencephalic level. Apallic syndrome could be, as already mentioned, the terminal stage of a progressive degenerative disease, but also a reversible transient syndrome in the course of an acute cerebral lesion with the potential for complete or incomplete recovery. Clinical picture of the fully developed syndrome consists of a coma vigile with the eyes open, absent threatening reflex, not frequently encountered blink reflex and finally with lack of reaction to external stimuli except for coarse movements of body and limbs following repetitive painful stimuli. Another feature is the recovery of a sleep-awake rhythm, independent of the day-time but controlled by patient's exhaustion (Gerstenbrand 1967). That the brainstem is also impaired is evident from the divergent position of the bulbi, from the asymmetry of the pupillary diameter, from the exaggerated brainstem reflexes, e.g. of the masseter reflex, from the body posture due to decortication or decerebration, and finally from the elevated muscle tone in terms of rigidity and/or spasticity. Tendon jerks may be brisk or, in case of contractures or periarticular ossifications of the major joints, absent (Narabayashi 1962).

Primitive oral automatisms, stereotyped chewing movements, swallowing, sucking and showing the teeth in association with rhythmic and repetitive movements of the tongue, also contribute to the clinical picture of the apallic syndrome. One may also observe an exaggeration of the snout reflex or the so-called bulldog reflex (following objects with the mouth with repeated attempts of "oral grasping"). Yawning always accompanies these primitive motor patterns what reminds of the symptomatology of the brain-damaged neonates. The so-called mental reflexes are obligatory in the apallic syndrome. Palmomental reflex is elicited by scratching the thenar with a blunt object, evoking a contraction not only of the homolateral mental muscle, but possibly also of the trapezius muscle and even of the entire upper extremity, indicating markedly enlarged reflexogenous zones. The same mechanisms underlie also the Babkin reflex, which can be elicited by vigourous and simultaneous grip of both hands resulting in an opening of the mouth. Masseter reflex may be brisk, especially if the apallic syndrome is due to the cerebral hypoxia. Dysregulation of the vegetative nervous system is reflected in tachycardia, tachypnoe, profuse sweating and seborrhea, localized mainly in the face.

Sympathetic drive is elevated most of the time. This could be interpreted as a "chronified emergency reaction". Haider et al. (1975a, b) reported on increased basic metabolic rate in the apallic syndrome with values exceeding 110-180 % compared to normals. In particular he found elevated turn-over of fat and marked increase of the glucose utilization. Hörtnagl et al. (1980) found elevated plasma noradrenaline concentrations beginning with the transition from the acute midbrain syndrome to the apallic syndrome. He suggested the use of beta-blockers to protect peripheral organs from the consequences of long-term hyperactivity of the autonomic nervous system.

Vegetative symptoms of the posttraumatic apallic syndrome are considered to be caused by organic and/or functional lesions in the mesodiencephalon.

Patients may die either at a time when the apallic syndrome is fully developed or within the first stages of remission. The prognosis quo ad vitam becomes better when patients enter in the phase of Klüver-Bucy-Terzian-Dalle Ore.

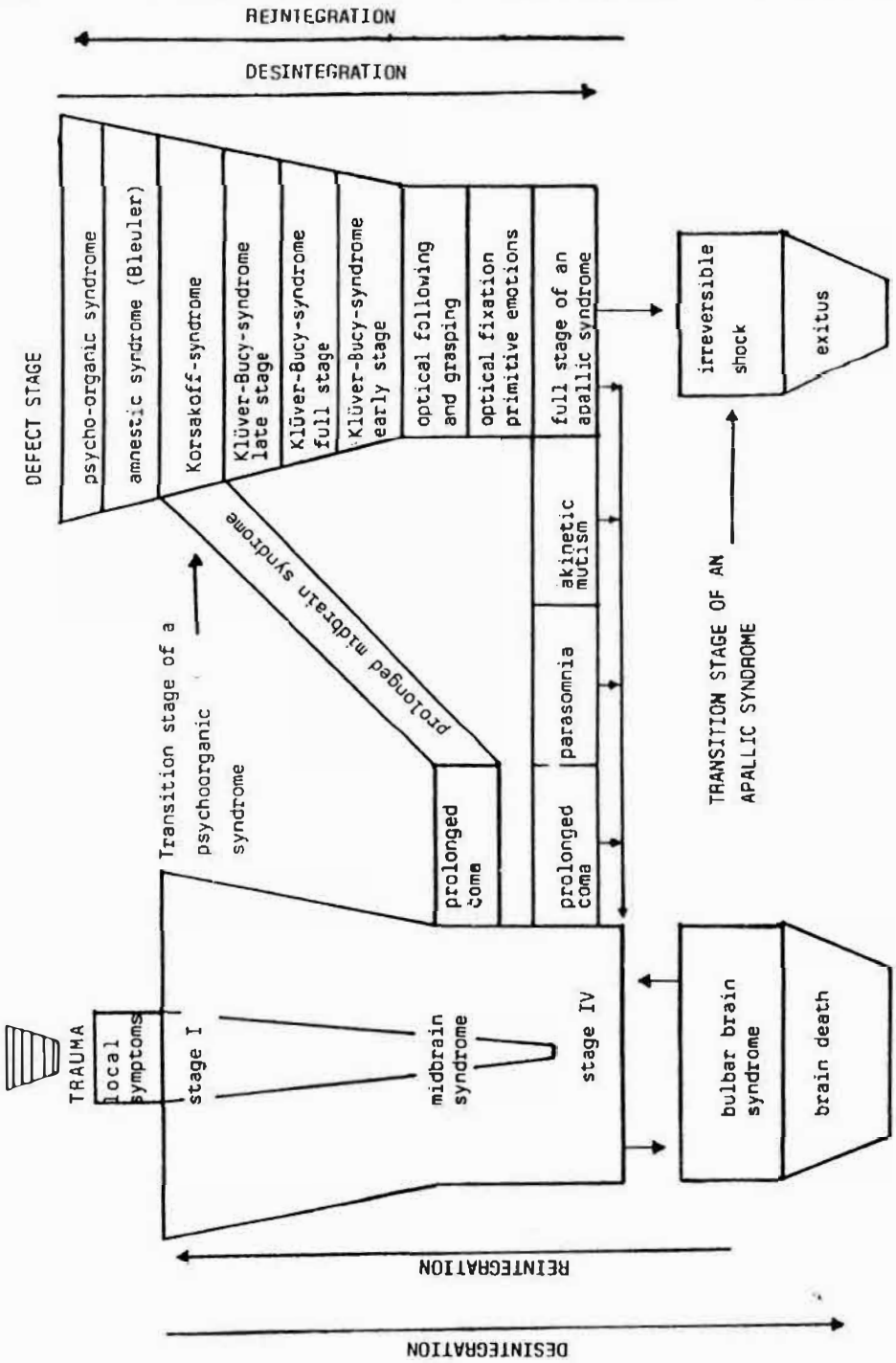


Figure 10. Possible development of an acute midbrain syndrome.

Stages of Remission from Post-traumatic Apallic Syndrome

Stages of remission from the apallic syndrome are characterized by gradual recovery of higher cortical functions accompanied by subsidence of primitive motor patterns until their complete disappearance (Figure 10).

The first signs of remission are in fact reduction of chewing automatisms and of sucking, and transition of the independent sleep-awake rhythm to a more regular one. The leading features, however, are the appearance of primitive emotional reactions following painful stimuli and recovery of optic fixation (stage I of remission).

Following this, differentiation of emotional reactions in regard of recognizing familiar persons and reacting with smiling occurs. Furthermore patients start to follow objects with their eyes and perform coarse movements spontaneously (stage II of remission). These movements mainly consist of repetitive and stereotyped grasping of objects. At the end of stage II some of the patients are already able to carry out simple orders.

Stage III of the remission is the so-called early stage of Klüver-Bucy syndrome. Patients tend to touch their genitals in a repetitive and stereotyped way, but remain without any obvious contact with the environment.

Stage IV of the remission, the fully developed Klüver-Bucy syndrome, is characterized by symptoms resembling the syndrome of Klüver-Bucy-Terzian-Dalle Ore. These authors described the conduct of monkeys after bitemporal lobectomy. They presented with marked oral tendencies, hypersexuality, the so-called psychic blindness, placidity, loss of fear and hypermetamorphosis.

The main symptoms of patients recovering from the apallic syndrome and passing into Klüver-Bucy syndrome are: grasping of all kinds of objects and raising them constantly and repeatedly to their mouth, with a tendency to bite and to swallow them, bulimia with unflagging demand for food, hypersexuality with tendency to masturbate, verbal stereotypes, and some extrapyramidal signs such as amimia, hypokinesia, hypersalivation and seborrhea. In this phase the capability of verbal communication reoccurs. The symptomatology of the Klüver-Bucy syndrome could be interpreted as an elevation of the level of functioning from the brainstem level to the level of disinhibited limbic system.

Late stage of the Klüver-Bucy syndrome (stage V of remission) maintains some features of the preceding phase, namely the demand for food, hyperphagia and the tendency to explore objects orally. Hypersexuality is either hidden or directed towards the nearby subjects, revealing itself with tenderness and caress, touching and kissing (kissing patterns).

Stage VI of the remission resembles the syndrome of Wernicke and Korsakow. Characteristic signs are emotional lability and impaired recent memory. Amnestic deficit is compensated by means of confabulation. Speech and other higher cortical functions are recovering which can last for more than one year.

The next step of the remission is the amnestic phase according to Bleuler (stage VII of remission). The main symptoms are disturbances of attention and of recent memory, impairments of the abstract and associative thinking and tendency to perseveration. A psychomotor deceleration or in some cases an attitude of hypomania, always associated with the emotional incontinence, may also be present in this stage.

The last phase in the remission from post-traumatic apallic syndrome is the evolution of psycho-organic syndrome (stage VIII of the remission). In this stage dysfunctions of recent memory and abstract thinking persist but are less obvious, while emotional lability, symptoms of depression, anxiety and in some cases major irritability, may be present.

The process of recovery ends in the defect stage and may find its expression in different clinical pictures:

1) Predominance of extrapyramidal symptomatology: amimia, sialorrhea, seborrhea, akinesia, rigidity, flexed posture, reduction of swinging movements of the upper limbs during walking, and diminished reaction to convergence.

2) Predominance of intellectual impairment in a form of organic dementia, frequently associated with psycho-organic syndrome and emotional disinhibition (affective incontinence, deficit of mainly recent memory).

3) Pseudobulbar symptomatology with spastic tetraparesis, dysarthria, dysphonia and positive pyramidal signs.

4) Predominance of the cerebellar symptomatology with ataxia, intention tremor and dysarthria, most often observed in children and young adults after severe head injury with the primary brainstem involvement.

It has to be mentioned that focal cerebral lesions may cause additional symptoms in the defect as well as in the preceding stages, e.g. spastic hemiparesis or other focal neurological signs.

It is also important to emphasize that post-traumatic apallic syndrome may be transient in some cases with complete recovery later. More frequent, however, is a less satisfactory outcome, which still allows reintegration into the family and/or society.

Thus, clear classification and thorough follow-up of patients suffering from severe head and brain trauma are of primary and fundamental importance for establishing valid prognosis and for organizing adequate and effective individual rehabilitation programmes.

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Editors:
Tine S. Prevec and Janez Zidar

University Institute of Clinical Neurophysiology,
University Medical Centre of Ljubljana