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LA SINDROME APALLICA

Gli Autori, dopo essersi soffermati sul concetto di "sindrome apallica" quale entità nosografica carica di significati più funzionali che morfologici, e che comunque va sempre fatta risalire a lesione e/o a sofferenza della reticolare attivante del diencefalo e del mesencefalo rostrale, ne descrivono le diverse fasi cliniche che sfumano dalla sindrome diencefalo-mesencefalica grave a quella mesencefalica e mesencefalo-pontina, le quali, di fatto, costituiscono i limiti entro i quali va confinata la sindrome apallica vera e propria.

Largo spazio viene dedicato a sfatare il concetto d'ineluttabilità, forse ancora diffusamente radicato, e a sottolineare la necessità di rigorose pratiche terapeutiche e riabilitative che mirino alla superficializzazione del livello di coma, con l'intento di poter alla fine reinserire, là dove sia possibile, il paziente nel proprio ambiente.

Vengono infine descritti alcuni criteri cui ci si dovrebbe ispirare nella costituzione di centri riabilitativi, affidati a gruppi di medici specialisti, logo e fisioterapisti, assistenti sociali e terapisti occupazionali, ai quali collettivamente spetta il compito del recupero e della risocializzazione del paziente apallico.

COMPLICAZIONI CEREBRALI IN POLITRAUMATIZZATI

Fase della Compressione del Tronco Cerebrale	SINDROME MESENCEFALICA				SINDROME BULBARE	
	1	2	3	4	5	6
Vigilanza	leggera sonnolenza	profonda sonnolenza	Coma	Coma	Coma	Coma
Reattività a stimoli sensoriali	ritardato	diminuita	assente	assente	assente	assente
Motilità spontanea	normale	ridotta	accentuata	assente	assente	assente
Reazione motoria a stimoli dolorifici	normale	ridotta	assente	assente	assente	assente
Tono muscolare	normale	aumentato (agli arti inf.)	aumentato (in senso generale)	molto aumentato	normale flaccido	flaccido
Ampiezza pupillare	media	costrizione	costrizione accentuata	dilatazione media	dilatazione	dilatazione massimale
Reazione pupillare alla luce	normale	ritardata	lenta	diminuita	accennata assente	assente
Motilità del bulbo oculare	pendolare	disconiugata	assente	assente	assente	assente
Riflesso oculo-cefalico	assente	presente	presente	assente	assente	assente
Riflesso vestibulo-oculare	normale	aumentato	tonico	dissociato	assente	assente
Respiro	normale	irregolare	irregolare	iperpnaico	periodico	assente
Temperatura	regolare	aumentata	aumentata	aumentata	aumentata	irregolare
Frequenza del polso	regolare	regolare	irregolare	irregolare	irregolare	irregolare
Pressione arteriosa	normale	normale	aumentata leggermente	notevolmente aumentata	diminuita	molto diminuita

SINTOMATOLOGIA DELLA COMPRESSIONE PROGRESSIVA DEL TRONCO CEREBRALE

THE APALLIC SYNDROME

Introduction

From the type of the motivating disease as well as from the course of the first observed cases of an apallic syndrome described by KRETSCHMER (1950), the conclusion can be drawn that the term «apallic» was conceived as denoting a functional disorder of the brain, while it did not intend describing the actual brain damage on a morphological basis. This fact shows that in the apallic syndrome a functional dynamism exists and that in some cases a remission is possible.

The apallic syndrome can develop in two different ways:

1. Following an acute brain process which affects the total cerebrum or which interrupts the ascending or descending pathways as a consequence of a local lesion in the midbrain. A combination of both conditions is possible and that, indeed, represents the highest degree of brain damage. To this group belong cerebral hypoxidosis of various origins, brain oedema caused by an allergic reaction, diffuse encephalitis, metabolic disturbances, exogenous intoxication, brain injury as well as processes of the midbrain. In this type of apallic syndrome a remission is possible sometimes, although the full syndrome can in others persist until the death of the patient without the slightest tendency towards remission.
2. A diffuse progressive brain damage affecting the cortex, the white matter or both. Neurological diseases which can determine an apallic syndrome of that kind are, for instance, senile or pre-senile atrophy, diffuse brain sclerosis, bubacute sclerosing panencephalitis («SSPE»)

Key words: apallic syndrome, acute midbrain syndrome, therapeutic community.

and the Marchifava-Bignami syndrome. The apallic syndrome developing within a progressive process of the brain represents an irreparable final condition.

The apallic syndrome develops in certain stages. In acute brain processes an initial stage is the rule, showing the various phases of the acute midbrain syndrome; in a much smaller number off cases, the acute bulbar-brain syndrome, followed by a transient stage, which gradually develops towards the full apallic syndrome.

In the remission phose several steps in the reintegration of higher brain functions can be observed.

The symptomatology of the apallic syndrome through all its course can be divided into the symptom categories of the disturbance of consciousness (activity and content of consciousness), as well as of vigilance, of the reaction to external stimuli, of the emotional reaction, of the optomotor function, of the motor function of face and throat, of the body posture, of the primitive motor patterns and finally of the autonomic functions.

The initial phase

In the initial stage of the traumatic apallic syndrome there are constantly present the various phases of an acute mid-brain syndrome, or, less frequently, of the transient or full stage of the bulbar-brain syndrome. It should be pointed out that, according to recent findings concerning direct or indirect traumatic lesion of the brain, most frequently a primary stem lesion can rapidly lead to a breakdown of vital functions. Peters (1967), Mayer (1967-68) and Jellinger (1968) could demonstrate from a wide range of autoptical material that patients with primary traumatic brain-stem lesions die soon after their brain damage, mostly within one hour from the accident, a great number at the same moment. This knowledge leads to the most responsible task of handling every case showing symptoms of a beginning midbrain syndrome after a brain injury, trying to investigate wether the patient is developing an intracranial haematoma or the brain-stem damage were simply secondary to the brain oedema.

Together with Dr. Lücking, from Munich University, we were able to describe the different stages of the acute secondary mid-brain and bulbar-brain syndrome in more than seven hundred cases who showed a mid-brain symptomatology. We found two ways of development: the central and lateral herniation.

In the first stage of the acute mid-brain syndrome, the most striking symptoms are a change in vigilance and the type of reaction to external stimuli, as well as the occurrence of spontaneous mass movements and tossing movements. There are, too, some slight pathological signs in the autonomic reactions.

The second stage is marked by symptoms such as somnolence, increased reaction to external stimuli, stretch position of the legs, persistence of mass movements in the arms with undirected warding off movements in response to pain stimulation, together with an increased stretch position of the legs. There are signs of a dysinhibition in the tonus regulation system of the midbrain, such as increased muscle tonus, hyperreflexia and pyramidal signs. The optomotoric system is altered with modification in the eye position as well as a disregulation of ocular movements and a dysinhibition of the vestibulo-ocular reflexes. Disturbed appear the autonomic regulations.

During the third stage, the symptomatology is uniform and highly typical. The patient is unconscious and lays with arms flexed and legs stretched. In the anglo-american literature this is designated decorticate rigidity posture. There are increased signs of dysinhibition of the tonus regulating system as well as of the autonomic system.

In the full stage, the symptomatology appears remarkably uniform as during the stage during which there is a stretch position of all extremities the so-called decerebrate rigidity of the anglo-american literature. There is a relevant dysinhibition of the motor and autonomic systems.

During the transition stage towards the secondary bulbar-brain syndrome, the fifth stage in the course of the evolving brain-stem symptomatology, the main symptoms are a decrease of the dysinhibition in the mid-brain level, that is to say that stretch cramps gradually decrease, and the autonomic regulation begin to show a breakdown. The opto-motor system is practically out of function. On the onset of such a stage a dangerous error can eventually be made on account of the decrease in stretch cramps; it can lead to to the hasty and misleading conclusion of an increase in the conditions of the patient.

The acute bulbar-brain syndrome is charachterized by an almost total breakdown of brain-stem functions. The patient lays in a flaccid position so far as extremities and body posture are concerned. The autonomic regulations are aut of function, showing breathing arrest and automatization of blood circulation.

There is, at this time, a further possibility for remission, unless bulbar brain symptomatology lasts more than thirty minutes, in which case

remission would no longer be possible. There is no great difference in comparison to the bulbar-brain symptomatology were it not for the altered blood circulation and the onset of spinal reflexes. An important examination which should not be discarded in this period is the EEG recording, showing no sign whatever of electrical activity.

As above mentioned there is, in the development of the acute midbrain syndrome, the possibility of a lateralization, although this cannot be discussed in details here; two stages should be pointed out: the first, during which a deviation of head and eyes to the apposite side can be observed, as well as a flexed position of the arm and stretched position of the homolateral leg. The opposite extremities are animated by mass movements; vigilance is diminished. During the second stage, the deviation is increased, homolateral extremities show a stretched position, the controlateral arm is flexed, the leg stretched. The patient is somnolent.

In the further course, the patient develops the so called acute midbrain symptomatology which was described before. We need here emphisizing that both stages in the period of lateralization can show a remission. If a remission does not take place, that means a traumatic pallic syndrome is developing, during which we can observe the evolution from the transitional to the full stage of symptomatology.

The transitional stage from the midbrain syndrome to the traumatic apallic one can be divided into three different phases: the phase of coma prolongé, of parasomnia and od akinetic mutism, characterized by a change to the coma vigile and the persistence of disinhibition in the motor and autonomic systems.

The symptomatology of the full stage

This develops as described below:

Coma vigile with sleep-wake regulation controlled by exhaustion. The patient is awake although having no conscious activity nor capacity. The threatening reflex cannot be evoked, but the blinking one is present. During the state of awakeness the autonomic functions are in a sympathetic tonus, whilst they are in a parasympathetic one during sleep.

The patient shows no reaction whatever to external stimulation: pain stimuli, arouse, after a latency period, mass movements of the extremities. No emotional reaction is present.

Eyes are in a divergent position as well as anisocoric, nor whatever convergent reaction is possible. The oculo-cephalic reflexes (the so-called « doll-head» phenomenon) are reduced; the vestibulo-ocular reflex shows a tonic reaction. The light reaction is diminished and tonic, while slightly positive apperars the pain reaction.

The tonus of the masseter muscle con be increased, especially in apallic cases following brain hypoxia. Tyjical appears the posture of the extremities, with slight flexion of elbows and knee joints, an adduction of upper and lower extremities, a fist position and a plantar flexion of the foot angle. The trunk lays in extention. Sometimes the posture of the asymmetric tonic neck reflex with deviation of the head to one side and a stretched position of the chin extremities and flexion of the other side can be observed.

The muscle-tonus of the extremities and trunk appears increased showing signs of spasticity and rigidity (rigido-spasticity of NARABAYASHI. All tendon reflexes are accentuated, although there may well happen sometimes that they are diminished on account of contraction of joints, periarticular ossification or, last not least, a highly increased muscle tonus. Pyramidal signs can be present.

The primitive motor patterns are remarkable symptoms of the full stage, the most important of which are: the sucking-chewing automatism, the tactile adjusting oral mechanisms, the bulldog reflex and the snout one. Occasionally, breast-searching can be observed; this, indeed, occurs especially in cases of progressive brain damage or after a diffuse encephalitis.

In some cases yawning is present, entirely similar to the yawning of the brain-damaged newborn infant. The grasping reflex is either phasic or tonic. The tonic one, which is evoked by a proprioceptive flexion of the fingers, can be followed by a flexion of the elbow joint and by adduction of the whole arm. An accentuated tonic grasping reflex makes it possible to pull the patient up. In rare cases the CHODZKO reflex can be elicited.

The so called mental reflexes — whose denomination is explained by the contraction of the mental muscles — first described by MARINESCO and RADOVICI (1920) are constatly present in the apallic syndrome. There are several forms, the commonest of which is the palmo-mental reflex which can be elicited by scratching the palm by a blunt object. This does not only provoke the contraction of the homonateral mental muscles; indeed, the reflexogenous areas often can spread to the whole arm and even to the trapecius muscle.

The same pathophysiological basis as in the mental reflexes group underlines the BABKIN reflex, which is elicited by a simultaneous vigorous pressure of both palms, followed by opening of the mouth. Among the so-called fright reflexes, there may be present the glabellar, the head-re-

traction as well as the total fright-reflex. In some cases only climbing movements of the legs are present and the GALANT reflex (scratching one side of tre trunk, the homolateral hip moves towards the stimulated side).

The asymmetric and symmetric tonic neck reflexes are elicitable in nearly every case more or less intensively. These occur with a lapse of latency and it is perhaps worthwhile pointing out their importance for the earliest stage of physiotherapy in the so called method of the tonus-regulationg reflex therapy.

The disinhibition of autonomic functions plays a relevant role on the conditions and the further evolution of the apallic patient. As mentioned above, during the awake state, all signs of a sympathetic tonus regulation may be found, shifting over, after the beginning of the sleeping period, to a parasympathetic regulation. The period of the predominance of the sympathetic tonus regulation leads to a chronification of an emergency reaction and can explain the need for a hypercaloric nutrition. In connection with the regulation of the autonomic system, complication such as bedsores, marasmus and so on, may be observed.

Within the course of autonomic disregulation, incontinence of bladder and rectum must be cited. Of main importance appears the decreasing resistance against alla sorts of infections. The increased activity of some glands accompanied by hypersalivation, ointed face and sweating involves a parkinsonian symptomatology, as well as the presence of akinesia and amimia.

The remission stage

During that stage a reintegration of the higher brain functions appers as well as a decrease in the primitive motor patterns which do integrate into increasing voluntary movements and a recovery of the previously disinhibited function.

If we look at the remission phase from a functional point of view, two stages may be distinguished: that during which the patient establishes contact with the surrounding world, combined to an increasing mental awareness and a reintegration of the turning-towards movements and another characterized by the disappearance of the persisting disinhibitions symptoms while do reappear the voluntary motor functions as well as the highest brain functions. Clinically, the remission stage can be divided into several phases. The esordium is indicated by emerging primitive emotional reactions, by a change in the sleep-wake rythm influenced by the nictoemeral regulation, and by a regression of the chewing-sucking automatisms. All the above signs can either occur after several days of a lasting full stage or after some months, and the phase itself can last from several days to several months or even represent a final one.

During the further course, optical fixation occurs and there is an increase in sleep-wake regulation in parallel with a progressive disappearance of the coma vigile. This phase, too, can last for a varyng lapse of time or represent the end stage of remission. Emotions determine positive effects with slight signs of recognition of special persons and the first signs of a smiling reaction. Usually, the first voluntary movements occur in the fingers. Towards the end of this phase, the patient performs very simple orders, like opening mouth or closing the eyes. It is during this stage that the superimposed focal lesions of the brain or the cerebellum may be found (hemiparesis, etc.).

The following phase is marked by the symptomatology of the KLUVER - BUCY - TERZIAN - DALLE ORE syndrome whose main signs are grasping and bringing to the mouth almost everything, followed by biting and swallowing, by a change in emotional reactions towards more positive effects, the arousal of an euphoric mood, hypersexuality with hetero e/o homosexual tendencies as well as lack of shame reactions during masturbation and lack of fear reactions. Rage reactions appear sometimes as well as bulimia and lack of memory. The primitive motor patterns show a further differentiation, some of them disappearing. Quite often hyperreflexia and piramidal signs can be found as well as symptoms of parkinsonism.

The symptomalotolgy can be explained by the rising of the functional *niveau* to the gigher level of the disinhibited limbic system.

In the further course of the remission the symptomatology of a Korsakow syndrome occurs, but, in the meatime, redevelopment of speech and other higher brain functions take place and voluntary movements normalize. Significant signs of an emotional lability appear, and along the further recovery a psycho-organic syndrome develops followed by a defect stage. This is signed by a group of symptoms such as:

Predominance of an organic dementia combined to a special emotional disinhibition.

Predominance of spastic symptoms with pseudobulbar paralitic symptomatology.

Predominance of a cerebellar symptomatology.

Predominance of a parkinsonian symptomatology.

Predominance of an extrapiramidal hyperkinetic symptomatology.

Such a symptomatology, which is typical of the defectual stage of the apallic syndrome, may be combined to symptoms of uni or multifocal superimposed brain lesions. It may, perhaps, be useful here to enphasize that in rare cases the apallic syndrome is likely to appear as a transient syndrome unaccompanied by any of the defectual symptoms, or these may well be very slight.

The exitus of an apallic patient after an acute brain damage may be determined by an irreversible breakdown of the circulatory system, or by a renewed secondary midbrain syndrome, followed by a bulbar-brain syndrome. Death of the apallic patients of the firts group usually occurs during the full stage or, possibly, during the early period of the remission stage. Once the patient enters into the Kluver-Bucy-Terzian-Dalle Ore syndrome, the prognosis for survival is favourable.

The therapy of the apallic traumatic syndrome

The treatment of traumatic apallic cases should be divided into early treatment and the actual treatment of the syndrome. The first one shoul be started during the initial phase, that is during the stage of the acute midbrain and bulbar-brain syndrome, and consists of the necessary oxigen supply and the treatment of the symptoms of the acute bidbrain syndrome. In cases of brain oedema, intensive dehydration must be started and Valium can be employed or new antispatic drugs of the meprobamate structure in order to relieve stretch cramps.

The employment of ipercaloric feeding ought to be started thirty six hours after the arousal of a midbrain syndrome. During the first days the rate should range around 3.000, 4.000 calories, increasing it, during the transient phase, even to 5.000. It is possible the employment of a mixture of fat carbohydrates and proteins administered either by infusion or tube-feeding. In recent years a mixture of polypeptides has brought a new trend to the nutritional system.

Besides the feeding problem, in the early stages of a traumatic apallic syndrome a special physioteraphy needs to be started, beginning during the transional stage. Availing ourselves of the posture reflexes, i. e. the tonus regulating reflexes, it becomes possible to activate movements of the limbs as well as of the neck and body muscles. Such a passive physiotheraphy should be combined to passive movements of the joints.

This special therapeutic activity can prevent the development of periarticular ossifications and contraction of various joints, but it may also exercise an indirect activation of the ascending reticular system, playing a relevant role during the remission stage. It has not been pointed out that regular posture modification together with the employment of special pillows and cripple-beds are necessary in order to prevent bedsores.

Many attemts are known so far as the therapeuthic problems of the apallic syndrome are concerned. In some cases the employment of L - DOPA seems to activate the remission course; our daily dosage varies froom 250 to 750 mg. Indeed, we made ours the suggestion which came from Bricolo, who could evaluate the eventual DOPA action by repeated EEG recordings.

Hassler, in cooperation with Dalle Ore and co-workers, tried in some cases a stereotaxic stimulation on the internal *lamella* pallidi and of the contralateral nucleus *anteroventralis* with the aim of activating the unspecific projection system in the forebrain. It was possible, in one case, to observe a transient activation of the patient's stage. In one of the four cases the remission stage appeared to be increased. A new trend in the therapy of apallic syndromes appears to be the employment of MIF (melanocyte inhibiting factor). This polipetide while exercising an antiparkinsonian effect, seems as well to exercise a good effect on the level of vigilance.

A prevalent role is played by rehabilitation tecniques which need being started indirectly since the transient phase, that phase which was defined, so to speak, « preparatory » by Gerstenbrandt in 1967, and during which all precautions should be taken in order to prevent cachexia, bedsores, contraction of joints as well as periarticular ossification.

During the following stage, i.e. the stage of active rehabilitation, the patient must be moved to a special rehabilitation unit, but this must be done only if the patient is in good general conditions. No complication connected to an eventual tracheotomy should interfere with the first stage of remission, which represents the beginning of contacts with the surrounding world. Moreover, patients in a full apallic condition ought to be moved to the rehalitation unit only whenever a complete chek-up examination (EEG, PEG, Xenography, EMG) authorize a favourable prognosis. How to deal with those patients showing no sign of recovery, is, as can be expected, still an open problem.

According to our experience, a rehabilitation unit for patients with severe brain damage, should consist of no more than 18 - 20 patients of both sexes selected independently from the degree and etiology of their pathology. The patients share a community area complete with sleeping,

living, working and gymnastic rooms. A rehabilitation center may well consist of several of such units, each one looked after by a special staff of specially trained medical and nursing personnel. The medical staff should include, besides neuropsychiatrists, a psychologist, an orthopaedist, a neurosurgeon, an oto-laryngologist and finally an ophthalmologist. It would seem useless to underline the close cooperation which should exist whith the anaesthesiologist. The staff must obviously include a logopedist, physiotherapist, social workers, occupational therapist and a vocational guide.

Every rehabilitation unit must be devided into functional subgroups, the mobilization group and the special therapeutic or achievement group. Once admitted, the newly arrived patient, whatever the severity of his or her brain damage, enters into the mobilitation group which consists mostly of five patients, all af whom living however in close contact with the patients, of other subgroups.

The patient newly arrived to the mobilization or the rehabilitation units should afford, besides the normal clinical examinations, a battery of psychological tests in order to obtain a performance profile, including his various attitudes, such as general knowledge, memory, motor and speech functions. An individual rehabilitation programme should, then, be conceived having in mind the total scores.

The maintainance of a hypecaloric diet is necessary even once the treatment at the rehabilitation unit is started, although the aim should be replacing it as soon as possible by spoon-feeding. For that purpose, oral reflexes may well be profitable. The increasing remission, especially the entrance into the Kluver-Bucy phase, helps this recovery. Of main importance appears, during the rehabilitation period, the motherly role of the attendant nurses for regaining a profitable contact with the surrouding world. It must not be undervalued the importance, even during this part of the treatment, of all those measures such as the prevention of secondary peripheral nervous lesions as well as the employment of a special physiotherapy, with increasing use of the active methods. The logopedist ought to begin his task as soon as possible, before which it can, however, be profitable to teach the apallic patient a sort of code-system (like twinkling eyes, etc.) in order to help him to answer.

In analogy to the mother role of the nurses, especially the head-nurse, the physicians have no lesser part in playng the role of fathers so far as the social relations of the patient are concerned.

Praise and admonition, as well as constant encouragement and exhortation are necessary. Eventually, patients already in a much improved condition or those suffering from a lesser form of damage can be invited to cohoperate in the general care of other apallic patients, or might be encouraged their spontaneous offers of help. They could help in feeding or doing other simple tasks like assisting the logopedist and physiotheapist.

At the end of the KLUVER-BUCY phase, the apallic patient who has been able to leave the bed, can enter the achievement group, at first just to be present during the common singing or reading exercises or in order to watch television, later on sharing actively the exercises. The rules and functions of such a therapeutic community are fully applied. Owing to the different grades of brain damage of group members and the wide variety of their cerebral failures, and because of newly idmitted « recruits » an « ordering of achievement » arises which places « archievement demands » upon the individual patients. That who is not yet very active can be looked after by another in a more advanced state of remission, so receiving an increased impulse to activity. The difference among the regained disturbed brain functions and the possibility to compare one's own improvement with the other's is most likely to act as an incentive, developing compensatory actions among the members of the community. A patient unable to deambulate will be assisted by a motor semirehabilitated person or by an aphasic. Owing to such a method the number of paramedical persons comes to be increased.

The efficiency of each patient is stimulated my means of group-dynamics since each one would like to do better than the other; performance differences provide incentive and encourage the more disabled to increase their efforts in order to attain the standars set by their fellow- patients. The group incentive must be directed not only towards achievement demands and results, but above all towards a positive cohesion within the group itself through reciprocal aid and support.

According to his premorbid personality and dependent on the grade of his cerebral damage, every patient is entrusted with certain tasks within the group community; subgroups are formed, inspired by the clinical picture and professional interests. Patients belonging to other rehabilitation units can be added to special subgroups.

The achievement group has a fixed daily programme defined by the patients themselves; it includes the schedule for physiotheraphy, training of higher brain functions, not forgetting leasure time activity. One of the patients should be responsible for the daily programme; he should also refer on behalf of the whole group. Obviously, a patient should be chosen who were at an advanced stage of rehabilitation and particularly suited for such a task.

Between the whole rehabilitation group and the staff team a dynamic relationship should be created, whose responsibility might be shared by the head of the unit and the head-nurse. The relative roles within the staff team must be clearly balanced, since inconsistency immediately leads to negative affects. The conditions and progress of each patient must be valued weekly during regular staff sessions, and regular checks including EEG, laboratory tests and a psychological profile ought to be carried out.

A further factor of main importance within the group dynamics is represented by the necessary inclusion of the patients relatives and by personal friends e/o friends of the professional staff. All of them should be acquainted with the type of brain damage and with the expected modifications in the patient's personality. It is necessary to inform them as much as possible, in order to produce a positive influence on the disabled person.

In the rehabilitation of the apallic patients, the therapeutical community creates a useful framework and it may be regarded, especially for cases of severe brain damage, as a temporary social environment preparing the patient to readapt himself to normal society.

Summary

The clinical symptomatology of the traumatic apallic syndrome is described. In the TAS an initial phase is obligatory showing the symptoms of an acute midbrain an bulbarbrain syndrome. The same can be found on apallic patients of other origin. A transitory phase leads to the full stage. The remission stage shows different phases like the phase of the primitve emotion and optic fixation and the phase with the symptoms of the Kluver-Bucy-Terzian-Dalle Ore-Syndrome. Sixty percent are agoing this remission, some of them come to a complete resocialisation. No morphological problems are discussed. In the chapter of therapy two ways of treatment are described, the acute and the rehabilitation. The acute therapy has to be started in the initial phase. Most important is the hypercaloric nutrition. Summerising, the rehabilitation of an apallic syndrome has to start a soon as possible also in the first period in order to prevent complications. In the full stage the patient has to be transferred to a special unit where the rules of the therapeutic communitiy are followed. A rehabilitation programm has to be performed in every case with the aim of resocialisation. A resignation can take place only after all possibilities have been tempted and not before four month. A control of the brain damage using all modern methods as EEG, PEG and, last not least, the computer tomography of the brain is necessary.

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