5. The Apallic Syndrome in Metabolic Disorders of the Brain

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Introduction

Cerebral symptoms caused by insufficiency of brain metabolism in endogenous and exogenous metabolic disorders have been known for a long time (Frerichs, 1860). Pentschew mentions in his article (1958) that Jackson in 1882 already understood such neurologic pictures as a "stepwise disintegration of the cerebral integration efficiency." Nevertheless, the nonspecific term "metabolic encephalopathy" is widely used, as in the subtile presentations describing cerebral symptoms occurring in metabolic disorders in diseases of the liver (Summerskill et al., 1956; Penin, 1967), of the kidney (Tyler, 1968; Prill, 1969), in diabetes mellitus (Pentschew 1958; Levine, 1969), as well as in cases of anoxia (Strauss, 1931; Weinberger et al., 1940). Numerous other publications report disorders in serum electrolytes (Riecker, 1967), intra- and postoperative casualties (Gerstenbrand et al., 1968), as well as intoxications with narcotics (Victor and Adams, 1962), psychopharmaca (Corcoran et al., 1949; Bruck and Gerstenbrand, 1967), methanol (Plum and Posner, 1966), ethanol (Kieser, 1957), cyanide (Butenuth, 1970), paraldehyde (McDougall and Wyttie, 1932), water (Swanson and Iseri, 1958), carbon monoxide (Mumenthaler, 1961), and carbon dioxide (Sieker and Hickam, 1956). The above-listed references are limited to the given topic; severe brain damage in connection with metabolic disorders in the widest sense are reported.

The various authors describe similar psychopathologic transitory syndromes of the acute exogenous reaction type, without comprehension of the pathophysiologic connection between damage and respective central nervous reaction patterns. The published neurologic reports range from a slight reduction of the cortical level with varying disorders of consciousness and cortical dropout, to severe and partly irreversible damage of the brain and brain stem.

Comparison of clinical and experimental parameters with pathophysiologic analysis until a few years ago showed little unity and apparently divergent results (Erbslöh, 1958; Hoff and Jellinger, 1967; Jakob, 1964; Müting, 1962; Zysno et al., 1966; and many more). A few authors (Gerstenbrand et al., 1969; Tyler, 1970) have only recently started to re-emphasize the functional connections of the integration output of the central nervous system by pointing out similarities between the various neurologic findings in the development of the comatose syndromes on the one hand and the phylogenetic or ontogenetic stage of maturity of the central nervous system on the other.

At the same time it was possible to work out a uniform pathophysiologic concept of the comagenesis by using differentiating experimental and clinical tests to evaluate the brain metabolism. The energy requirement of the brain for the synthesis of the so-called

energy-rich phosphates, which maintain the active intraextracellular exchange of potassium and sodium ions as a prerequisite for the excitability of the cells, is mainly supplied by the aerobic catabolism of glucose (Fazekas and Bessman, 1953; Gottstein, 1966; Sherlock, 1957; Tyler, 1968; Zysno et al., 1966). Two types of disturbances of energy metabolism can be discussed. First, a reduction of the substrate and/or oxygen may arise, caused by a deficiency involving the whole metabolism, or insufficient blood circulation of the brain (for both, the possibility of a limited compensation is given), and secondly, either a disturbance of the utilization of the substrate through a decrease in enzyme activity, a change in the acid-base balance (pH) and electrolytes, or the presence of nonmetabolized toxic substances may be the cause. A disturbance of acid-base balance may be evident at the blood-brain barrier (Fishman and Raskin, 1967; Freeman et al., 1962), as well as intracellularly (Fazekas and Bessman, 1953; Schenker et al., 1967; Sokoleff, 1971). In some comatose conditions differences in the chemical composition of serum and spinal fluid were observed, with a change in the normal distribution of the individual chemical substances (Agrest et al., 1962; Cowle et al., 1962; Müting, 1962; Plum, 1971; and others).

In extreme glucose deficiency the brain cells are capable of catabolizing, within limits, substrates such as amino acids and lipoproteins. This emergency oxidation of the cells' own substances occurs, according to Sokoleff (1971), in comatose cases which are irreversible even after sufficient supply of glucose. Since histopathologic examinations are primarily impossible in processes like this, discrepancies between clinical and pathologic reports may arise.

Finally, it is of significance (Erbslöh, 1958; Hoagland et al., 1939; Weinberger, 1940) that the energy turnover is less in ontogenetic older parts of the central nervous system and subsequently not as sensitive to glucose or oxygen deficiency.

The desire for a more distinctive clinical syndromatology of comatose states (Gerstenbrand et al., 1969; Jouvet and Dechaume, 1960; Polli, 1971) to avoid the nondistinctive term of encephalopathy is therefore not only justified, but also necessary for a more exact diagnostic and prognostic evaluation of the individual cases. In accordance with other reports (e.g., Conomy and Swash, 1968; Mumenthaler, 1961; Engerth and Winkler, 1933), it was found that daily intensive neurologic examinations are essential.

The literature reports that, especially with hepatic coma and hypoglycemic coma, lesional changes in the brain stem appear, upon analysis of the reports, to be regarded as an acute midbrain syndrome (Sherlock, 1957; Swanson and Iseri, 1958), rarely also as an apallic syndrome (Conomy and Swash, 1968; Cravioto et al., 1960) or bulbar-brain syndrome (Ziegler et al., 1965; Sieker and Hickam, 1956). In fact, with sufficient neurologic examinations of the patients, single phases are distinguishable in the transient phases in the development of the cerebral coma symptomatology. These phases are of changing and varying duration; a phase may be omitted or may overlap into the next, persist in any one stage, and also occur partly in reverse order or be remittent.

Approximately 160 patients were observed with a severe comatose course of different etiology. Four of these cases may serve as examples here.

Case Histories

Two weeks later she was rehospitalized because of ascites and confusion in the morning, unexpected vomiting and within 30 min unconsciousness as well as a flexed/stretched position of the limbs accompanied by flex/stretch cramps of the extremities, as well as further symptoms of an acute midbrain syndrome of the third phase (Gerstenbrand and Lücking, 1970). After 30 min, there was spontaneous remission of midbrain symptomatology to a psychoorganic syndrome. On the following days, she presented symptoms of a diffuse brain damage with ever-increasing reflexes, frontal lobe signs, and slight-to-moderate dementia, corresponding to the state prior to the midbrain syndromatology. There was improvement of the pathologic findings of the liver by constant therapy with spirolactone, potassium, and Rocmalin treatment.

Eight days later, without external cause, renewed unconsciousness occurred within a few minutes, simultaneously with a stretch position of all extremities, as well as all signs of an acute midbrain syndrome in the full state. The stretch cramps subsided after 10 min, but, continuing increase of tonus, hyperreflexia, and pyramidal signs accompanied by simultaneous appearance of primitive motor patterns (spontaneous chewing automatism, oral and grasping patterns, mental reflexes, postural reflexes), as well as coma vigile, were seen. The position of the bulbi was divergent and the extremities were in the flex/stretch position. After 12 h, there was optic fixation and optic following, as well as primitive emotional reactions. This was followed 24 h later by the acute state symptoms of a Klüver-Bucy syndrome with simultaneous increase in voluntary movements. Twelve hours later, there was a transition to a Korsakoff syndrome followed by the development of a psychoorganic syndrome to the previous state of a diffuse cerebral lesion. After 8 weeks and intensive treatment of the liver, the patient was released from the hospital.

A few days later, she was rehospitalized with a fracture of the pubic bone caused by a fall in a state of confusion. Upon admission, she showed symptoms of a paranoid hallucinatory picture with intensive motor unrest, and also marked neurologic disorders exhibited by hyperreflexia, pyramidal signs, slight increase in tonus, as well as primitive motor patterns, primarily oral and grasping ones. With intensive treatment of the liver, remission into a neurasthenic state of the exogenous reaction type (Bonhoeffer, 1910) and transition into the previous state of cerebral defect, she was released from the hospital after several weeks of treatment.

Two days later she was readmitted to the hospital with suddenly appearing flashes of consciousness and strong motor unrest with mass and roll movements. Within 3 h an acute midbrain syndrome in the full state developed, lasting only 3 h, followed by subsidence of the stretch cramps and development of coma vigile. After approximately 6 h, the full state of an apallic syndrome with all characteristic symptoms appeared. After 20 h, there was sudden renewed stretch position of all extremities with stretch synergism and coma, and disappearance of the primitive motor patterns. After 6 h the stretch cramps subsided with simultaneous onset of gasping-breathing and finally breathing with only base-of-mouth movements. Within the next hour breathing was arrested, posture was atonic, reflexes were absent, and the pupils wide and without reaction. Twenty minutes later, cardiac standstill and death.

Neuropathologic findings (Neurologisches Institut, Vienna): Older walnut-sized hemorrhage of the white matter with perifocal necrotic edema parietooccipital dextra, slight diffuse brain atrophy (pre-aging glial and axonal dystrophy, as in chronic liver damage, no signs of acute brain edema with herniation).

Summary: The 52-year-old female patient, a chronic alcoholic, developed several times an acute midbrain syndrome, which transformed itself into an apallic syndrome. Remission followed through a Klüver-Bucy syndrome, as well as a Korsakoff symptomatology, into a psychoorganic syndrome to a defect state which existed previous to the acute incident. In between, an exogenous psychosis developed, in the course of which the patient fell, breaking her pubic bone. Death resulted during an apallic syndrome when a renewed acute midbrain syndrome developed, followed by a bulbar brain syndrome. Autopsy showed diffuse cerebral damage, but, without signs of an acute midbrain or bulbar brain herniation.

Case 2. Uremic coma (T.K., age 61, female): After repeated hospitalization because of shrinking nephropathy and high blood pressure, the patient was rehospitalized because of unrest and depression, anxiety, lack of drive, disturbed concentration, pain sensation, re-

symptoms, slight primitive motor patterns, as well as the symptoms of a polyneuropathy. Twenty-four hours after admission, she exhibited severe motor restlessness and fear, marked paranoid ideas, and optic and acoustic hallucinations. At the same time there was moderate intensification of the neurologic symptomatology, with slight pyramidal signs and a marked reduced convergence reaction of the bulbi as well as increased amimia and akinesia. In the course of 36 h, the restlessness subsided, as did the paranoid thoughts, but hallucinations continued, together with increased confusion with Korsakoff symptoms and a moderate increase in the neurologic symptomatology. After 12 h, she was comatose, and showed undirected movements of defense. Within the next 4 h lower extremities were in the stretch position, the arms were flexed, there was marked increase in muscle tonus, hyperreflexia, on both sides a positive Babinski sign, further divergent position of the bulbi with sluggish reaction to light and moderately marked primitive patterns (oral reflexes, tonic grasping, asymmetric tonic neck reflex).

Within the following 12 h coma vigile occurred with typical sleep-awake rhythm, lack of emotional reaction, divergent position of the bulbi, moderately marked oculocephalic reflexes, medium-wide, left somewhat smaller, pupils with delayed reaction to light, positive reaction to pain, flex/stretch position of the extremities with increased tonus, hyperreflexia, pyramidal signs on both sides, mass movements to intensified pain stimulation, primitive motor patterns (chewing automatism, oral reflexes, tonic and phasic grasping, asymmetric tonic neck reflex of both sides), as well as moderate amimia, a tendency to tachycardia, and other signs of autonomic disinhibition.

For 30 h there was no change. Then the coma vigile disappeared, the legs assumed the stretch position with undirected mass movements of the arms upon pain stimulation, and there was beginning disinhibition of the autonomic functions. Two hours later there was coma, flexed position of the arms, stretched position of the legs, and divergent position of the bulbi. One hour later the acute midbrain syndrome with all typical symptoms appeared, especially stretch cramps. After 3 h without change gasping-breathing began with a simultaneous decrease in muscular hypertonus, as well as hyperreflexia, and finally, atonic posture of the upper extremities while the legs were in a stretched position. The pupils were dilated with only a moderate reaction to light, and there was increased divergent position of the bulbi. This state continued for over 24 h, followed by atonic posture of body and limbs, areflexia, disappearance of pyramidal signs, marked divergence of the fixed bulbi, dilated pupils, no reaction to light, slight dyspnea, drop in temperature to 36°C, and bradycardia. Death after 1 h was caused by cardiac arrest. There was continuous increase in BUN and creatinine unchanged by therapy during the comatose state.

EEG (at the stage of the beginning of the bulbar brain syndrome): diffuse general changes, without side signs or focus.

Neuropathologic findings (Pathologisch-Anatomisches Institut, Vienna): Brain edema with slight basal pressure signs, recent damage of the white matter.

Summary: In this 61-year-old female patient suffering from shrinking nephropathy and high blood pressure, during an exogenous type of reaction according to Bonhoeffer (1910), a hyperemotional weakness state appeared first, which finally developed into a paranoid hallucinatory state and later into a marked psychoorganic syndrome with symptoms of severe diffuse cerebral damage. Upon intensification, an apallic syndrome in the full state developed, from which an acute midbrain syndrome evolved, finally progressing to the transitory phase of an acute bulbar brain syndrome which was shortly followed by the death of the patient. The neuropathologic findings showed brain edema with slight signs of herniation.

Case 3. Diabetic coma, hyperglycemia, uremic coma with diabetic nephropathy (Kimmelstiel-Wilson) (M.L., age 72, female): Diabetes with insulin therapy for over 20 years, for 3 years increased BUN. The patient was admitted because of decompensation of carbohydrate metabolism and pneumonia with a "comatose state." Upon pain stimulation, she exhibited undirected defense movements with latency, flexion tendency of the arms, flex/stretch position of the legs, increased muscular tonus, hyperreflexia, decreased Achilles tendon reflex, slight pyramidal signs, divergent position of the bulbi, oculocephalic reflex slightly releasable, moderately dilated pupils with decreased reaction to light, and slight reaction to pain, primitive motor patterns partly releasable (grasping reflexes, oral pattern,

somewhat deeper rhythmic breathing, tachycardia, BP 100/70; blood-sugar 944 mg%, no ketone bodies in the urine, and acidosis. Within 24 h metabolism was balanced with normal blood sugar and acid-base balance. Twelve hours after commencement of therapy there was a passing intensification of the primitive motor patterns, especially of the chewing automatism, disappearing coma vigile, optic fixation, afterwards optic following, beginning of undirected spontaneous mass movements, increase in the grasping reflexes with tendency to grasp objects, sound reply upon calling; 24 h after admission, turning-toward, as well as performing simple tasks, remains of a stretch position of the lower extremities with slight increased tonus of the arms and legs, slight divergent position of the bulbi upon limited convergence, directed voluntary movements, slight primitive motor patterns of the oral sense and of grasping. Six hours later the psychoorganic syndrome, with increase in orientation was observed, with slight neurologic symptomatology with hyperreflexia, slight increase in muscle tone, as well as slight cerebral reduction signs, and further symptoms of polyneuropathy.

After another 7 h there was rapid development of motor restlessness and fear, followed 2 h later by acoustic and optic hallucinations, simultaneous increase in the neurologic symptomatology with hyperreflexia, and slight tonus increase, oral and grasping reflexes. After 1 h the hallucinations subsided, but anxiety, depression, and severe restlessness continued. The neurologic symptomatology decreased.

Three days after admission there was a rapid decrease in diuresis, finally polyuria, an increase in BUN to 124 mg%, creatinine to 2.6 mg%, and potassium to 6.3 mVal/1, followed 1 day later by increased motor restlessness, logorrhea, and increased Korsakoff symptomatology. There was a simultaneous increase in the primitive motor patterns, hyperreflexia and slight pyramidal signs. After 12 h symptoms of a Klüver-Bucy syndrome with further intensification of the neurologic symptoms appeared: marked flapping tremor, myoclonia on the face as well as the upper and lower extremities. On the 6th day of treatment there was marked limitation of consciousness with a decreased reaction to external stimuli, slight divergent position of the bulbi, flexed position of the upper extremities and a flex/stretch position of the lower extremities, increase in muscular tone and reflexes, and pyramidal signs. After 8 h the patient exhibited coma vigile, lack of emotional reaction to pain stimulation, latent onset of undirected mass movements, flex/stretch position of the extremities with increase of tonus, hyperreflexia and pyramidal signs, marked primitive motor patterns, including the tonic asymmetric and symmetric neck reflexes, and marked autonomic overexcitability. Twelve hours later there was rapid development of coma with stretch position and stretch synergism of all extremities. The bulbi were divergent, with decreased reaction to light, tachycardia, tachypnea, as well as all symptoms of an acute midbrain syndrome. After 4 h the full state of a bulbar brain syndrome appeared; 15 min later the patient died of cardiac failure.

Summary: In this 72-year-old female patient coma developed with hyperglycemia with all symptoms of an apallic syndrome, which on admission showed the remaining symptoms of an acute midbrain syndrome. After normalization of blood sugar and of the acid-base balance, remission began. Still in the phase of a psychoorganic syndrome, an exogenous type of reaction started which developed increasingly into a state of hallucination. After renewed remission a Korsakoff syndrome developed, followed by a Klüver-Bucy symptomatology and again an apallic syndrome. During this time a disturbance of kidney function developed, with a severe increase in BUN and creatinine as well as potassium. The apallic syndrome developed into a renewed acute midbrain syndrome and finally into a bulbar brain syndrome ending in the patient's death.

Case 4. Hypoxic coma (K.B., age 11 months, female); (cf. Amann et al., 1971): During an examination of the mouth with a spatula because of laryngitis sudden heart and breathing arrest occurred, followed 5 min later by setting in of the heart's action, and after 15 min spontaneous breathing. Within 2 days heart arrest occurred 2 more times. Upon admission the patient was in acute midbrain syndrome with coma, showed divergent position of the bulbi, and stretch cramps of all extremities. Because of repeated heart arrest, thoracotomy and manual heart massage was done, but cardiac arrest occurred again. After stabilization of the heart's function, the acute midbrain syndrome continued. Three hours later the arms

of the arms and legs in the posture pattern as described previously. On the 8th day symptoms appeared of a transitory stage into the apallic syndrome with increasing primitive motor patterns, primarily of the chewing automatism, with simultaneous decrease in the myoclonia. Three weeks after onset of the disease, the patient exhibited the full state of an apallic syndrome with coma vigile, typical sleep-awake rhythm, lack of emotional reactions, flex/stretch position of the extremities, marked primitive motor patterns, including the Babinski sign, as well as severe autonomic lability. Five weeks after the start of the disease with unchanged neurologic state, a pontine crying pattern developed. Death was caused by irreversible failure of the circulatory system, 57 days after onset of the disease.

EEG (16 days after heart arrest): Extremely flat curves with slight theta and beta activity, no noticeable side difference.

Neuropathologic findings: Extreme form of postanoxic encephalopathy with extensive necrosis of the cerebral cortex, symmetric necrosis of almost all central gray matter, diffuse total atrophy of the cerebellar cortex and symmetric focal necrosis in the mesencephalon and pontine tegmentum. Secondary embolic-metastatic focal encephalitis was also found.

Summary: In this 11-month-old girl cardiac and respiratory arrest set in during an examination of the pharynx. After reanimation an acute midbrain syndrome developed, which changed only after 8 days into the transitory stage to an apallic syndrome and finally into the full state of this syndrome. The apallic symptomatology was maintained without noticeable changes until death, 57 days after the acute incident. Besides the characteristic midbrain symptoms, rhythmic myoclonia of the face and extremities developed which could not be observed in the full state of the apallic syndrome. During the course of the apallic symptomatology a pontine crying pattern developed. In this case, massive hypoxic damage to almost all gray matter and also edematic damage to the white matter occurred. Further, a diffuse total atrophy of the cerebellar cortex and of the nucleus dendatus, as well as of the lower oliva, developed. At the end of the course of the disease a metastatic focal encephalitis existed.

Discussion

Actual knowlege of cerebral metabolic disorders in coma of various genesis allows the following hypothesis:

The cerebrum shows a decreasing sensitivity towards an impaired energy supply, conditioned through its phylogenetic functional planes (Richardson et al., 1959). The supply and utilization of glucose, essential for the functions of the central nervous system, depend in comatose states upon the respective damage. If the energy turnover is lowered to such an extent that the output for a central functional level is insufficient, the cerebral integration output will be reduced to the phylogenetic older functional plane still sufficient for energy supply, whereby the rostrally situated parts of the brain then cease to function (Fazekas and Bessman, 1953; Gottstein, 1966; Tyler, 1970; and others). This process was termed a functional cerebral disintegration independent of its etiology.

Biochemical parameters or the encephalogram (Conomy and Swash, 1968; Polli, 1971; Tyler, 1968; Kitani, 1959; Penin, 1967; Parsons-Smith et al., 1957; and many others) are only a rough aid in the therapeutic and prognostic evaluation. Strict neurologic and psychiatric observations during the whole course are important in differentiating certain functional syndromes which may be brought into relation to the central disintegration levels. Not every

glycemic and other forms of coma, as well as postoperative hypoxemic complications, show that the various disintegration levels may be present in all comatose states.

Several authors (Erbslöh, 1958; Prill, 1969; Stefenelli, 1962) have pointed out that the clinical course is subject to the acuity of the decompensation process. Depending upon whether the brain has enough time for a relative adaptation, the acute and subacute courses (electroencephalographic; Penin, 1967) may be differentiated.

In the development of the coma syndrome, the variable acute midbrain syndrome (Cases 1-4) dominates with a characteristic disorder of consciousness, a flexed-stretched position of the extremities and the trunk, which may be intensified by pain stimuli, increased muscle tone, increased reflex action, positive pyramidal signs, disorder of the optomotor system and autonomic disinhibition. With an intermittent bulbar brain syndrome (muscle atonia, loss of reflexes, light-rigid mydriasis, disintegration of autonomic functions), this picture, if it persists, may lead to death. Other cases may go into remission shortly after direct cessation of the midbrain symptomatology—very rarely, however, with a brief previously occurring bulbar brain or apallic syndrome. The severity of the functional disintegration depends upon how acute the comatose state appeared (Case 4). If the comatose state can be controlled therapeutically, late damage may often result. In contrast to the reports of Plum and Posner (1966), personally observed cases show neurologically complete remission, even after an acute midbrain syndrome in its full state, caused by metabolic disorders as well as traumatic events. In lethal cases, especially in younger patients who die during one of the acute phases, the brain shows conditioned basal pressure signs or even transtentorial or foraminal herniation (Case 2). A causal connection between herniation and clinical course, however, cannot be established, since the actual intracerebral metabolic or other disorders causing local changes (Weinstein, 1947) cannot be differentiated clinically in the individual case.

In contrast to this, the subacute "coma forms" in their various central disintegration phases are more easily distinguishable. This is shown in our second case, where it was possible to analyze some symptomatologically different disintegration phases, namely, a neurasthenic phase (Penin, 1967) showing cortical disintegration with loss of vitality; pain sensation; irritated, anxious, depressive disposition; an exogenous paranoid-hallucinatory phase; a phase of a Korsakoff syndrome followed by an apallic syndrome which was interrupted by different phases of the acute midbrain syndrome (Gerstenbrand and Lücking, 1970). This was followed by a transient prephase of the bulbar brain syndrome and the full picture of a bulbar brain syndrome, which soon after led to death.

Effective therapy of the basic metabolic disorder will favor a stepwise reintegration of the higher central function levels in the remission course of the apallic syndrome with an increasing stabilization of the energy balance. Here, too, the various phases may be omitted in individual cases or may not be noticed because of their short duration. The explanation for the nonappearance or short duration of the individual transitory phases may lie in the condition of the patient (e.g., age, varying degree of disturbance of the various central structures, local vascular disorders, previous cerebral damage, rate of metabolic restabilization).

The remission as well as the disintegrating course corresponds for the most part to the course of the apallic syndrome of traumatic genesis. Clinical details are given in Chapter 2. Particular attention is given to the Klüver-Bucy phase (Case 1) which shows in disintegration as well as in remission hypersexuality, lack of shame reaction, complex primitive motor

times bulimia. The Korsakoff syndrome, with massive amnestic disorders, disturbed sense of time, logorrhea, euphoria, and rapid tiring combined with a disappearance of the primitive motor patterns and improvement of the spontaneous voluntary movements, is well known as a transitory phase in the development and remission of comas of various etiology. A defective state marked by organic dementia, affective disinhibition and/or persisting extrapyramidal, spastic or cerebral symptoms, appears in a certain percentage of the cases in the apallic syndrome with metabolic or hypoxic or traumatic genesis.

Through the indicated analysis and the symptomatologic classification of a coma syndrome of various etiology, a deeper understanding of the cerebral functions may be gained. Adequate consideration of the patient's age, duration and severity of the central disintegration, and the acuity of the dominating functional disorder allows a faster and more reliable prediction of the effectiveness of modern therapy. Furthermore, a better prognosis of individual cases can now be made than was previously possible from laboratory tests or electroencephalographic reports. However, many questions remain, such as the manner and exact location of the damage causing the coma and its pathophysiologic pattern. Improved routine laboratory tests on the spinal fluid, for example, may further assist the clinician to supplement the neurologic reports and possibly aid in the commencement of additional therapeutic measures on the central energy metabolism–as an aid in the analysis of individual factors outside the acute metabolic disorder requiring therapeutic attention. Finally, the analysis of function as well as brain morphology and ontogenesis of the primitive motor patterns will prove useful.

It should also be mentioned that chronic central cerebral damages caused by endogenous or exogenous disorders of metabolism may possibly lead to an apallic syndrome. These disorders are, for example–besides the causes already mentioned–diabetes mellitus and chronic alcoholism, hyperthyroidism, chronic lead and mercury intoxication, and usually congenital disorders of lipoid or amino acid metabolism in children. The clinical symptomatology of the apallic syndrome caused by these diseases has, as far as we know, not been described, with the exception of mercury poisoning.

Summary

The central nervous structures show a varying degree in rate of energy turnover, depending upon their phylogenetic age. The disturbances in oxygen utilization in comatose states of various genesis is the most important and last step in cerebral metabolic disorders, whereby a stepwise progressive disintegration of the cerebrum occurs with a uniform central remission pattern, independent of the coma etiology, in the levels of the cortex, limbic system, midbrain, and lower brain stem. Four characteristic cases serve as examples of these reaction patterns and the development of the apallic syndrome. Laboratory tests and the electroencephalogram are only a rough aid in evaluating therapeutic measures and prognosis in the individual coma cases, but they may be significantly supplemented by keen neurologic observations.

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