CHAPTER 30

Post-traumatic vegetative syndrome

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Even definition of the concept of a post-traumatic syndrome is difficult according to Teuber (1969). He cited Broadly, who included in the post-traumatic syndrome all possible sequelae of skull and brain injury, either open or closed, focal or diffuse. Broadly's concept of the post-traumatic syndrome included, therefore, the most severe defects which might follow damage to the skull or brain, such as the apallic syndrome, as well as mild subjective disturbances following concussion. In the Anglo–American literature, this unclear concept has recently undergone considerable change, so that the post-traumatic syndrome is now considered to comprise only conditions with few or no neurological symptoms and principally subjective complaints. The latter include headaches, nervousness and irritability, vegetative disturbances, such as vertigo, numbness, tiredness, sleep disturbances, alcohol intolerance, memory disturbances and inability to concentrate (Caveness 1966). Teuber (1969) considered that there was no justification for restriction of the concept of a 'post-traumatic syndrome' to subjective symptoms, and that it should also cover organic neurological sequelae of traumatic damage to the brain. However, he did not attempt exact delimitation of the concept. His suggestion that the posttraumatic syndrome was due to damage to the long tracts and midbrain structures with relative sparing of the cortex has not been verified by the present authors.

The present authors, in a review of the post-traumatic syndrome (Gerstenbrand 1969), showed that use of this term gave no indication of the severity, the symptomatology nor the localisation of the lesion, nor had it aided distinction between its acute, subacute and defect stages. For these reasons they argued that the term 'post-traumatic syndrome' should be employed only with addition of a more exact description, e.g. post-traumatic frontal syndrome, or post-traumatic vegetative syndrome following fronto-basal lesion, and that the acute and defect stages should be distinguished.

VEGETATIVE SYMPTOMS

Frowein and Harrer (1956) reported that vegetative disturbances might occur during the acute stage of traumatic brain damage, although it was difficult to judge their incidence and frequency. The assessment of vegetative disturbances is difficult, as only a small proportion may be detected by routine clinical examination, and so specific methods of investigation and complicated laboratory tests must be employed. In addition, the objective post-traumatic vegetative symptoms can not be compared with the subjective severity of the disorders, because of the variable nature of 'subjective tolerance'. On the other hand, negative results of investigations despite subjective symptoms cannot exclude a true post-traumatic

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vegetative disturbance, as the severity of the disorders is very variable and they may only appear under particular conditions and special stresses which may not be present at the time and under the specific circumstances of the examination. Finally, the causal relationship of objective vegetative symptoms to prior head injury may not always be obvious, as such disturbances may develop after other types of brain damage, such as encephalitis, and even on a constitutional basis, perhaps in combination with extracerebral diseases (Frowein and Harrer 1956).

The clinical features of post-traumatic vegetative disturbances, the post-traumatic vegetative syndrome, may best be discussed by a study of individual symptoms and their relationship to the different grades of intensity of brain injury. In order to try to correlate the Anglo–American literature with German and French experience, only mild and severe forms of brain trauma will be considered. The vegetative sequelae to secondary brain stem damage, in the sense of the old concept of 'compressio cerebri', or the complications following head injury with secondary brain stem damage, must be considered separately as will the vegetative components of the traumatic apallic syndrome.

The symptomatology of mild brain injury (commotio cerebri) includes loss of consciousness, amnesia and vegetative disturbances in the absence of morphological changes in the brain.

Severe brain injury (contusio cerebri) depends for its symptoms on damage sustained by the parenchyma, and particularly on the extent and localisation of the damage. In contrast to mild brain damage, the clinical features of severe brain injury are due to localised loss of brain tissue in uni- or multifocal lesions. Simultaneously the essential disorders, such as loss of consciousness, amnesia and vegetative disturbances are, in general, increased. In order to make the results of these injuries easier to understand, they have been subdivided when necessary in the following discussion into mild brain injury, moderately severe and severe injury. There are acute, subacute and defect stages of injury. The end of the acute stage is marked by fading away of the loss of vegetative control; and the end of the subacute phase is denoted by the appearance of stable residual symptoms, such as a variety of disturbed brain functions.

Secondary traumatic brain stem lesions must be differentiated from primary brain injury. These may result from complications of an injury, e.g. oedema, intracranial haemorrhage, producing a midbrain and bulbar brain syndrome, which can then lead to the traumatic apallic syndrome.

General and essential clinical features

In the acute stage after head injury, as in the other stages, there is onset of a specific vegetative symptom complex of varying severity which may last for a short or a long period. It appears in every patient with a certain degree of variability of symptoms. The clinical features include nausea and vomiting, fluctuation of blood pressure and pulse rate, a tendency to collapse and disturbances of body temperature and sweating. To these complications, Bodechtel (1959) added attacks of vertigo, and other workers have included an almost constant finding of headache. Post-traumatic vegetative essential symptoms and various other features of vegetative disturbances, such as abnormalities of blood sugar, basal metabolism, red blood cell count, haemoglobin concentration, white blood cell count, differential blood count, serum bilirubin, electrolytes, blood urea nitrogen and alkali reserve may all show fluctuations due to altered sympathetic and parasympathetic activity (Frowein and Harrer 1956). Unfortunately, this important hypothesis has neither been proven nor investigated subsequently by other workers (Cook et al. 1961; Gilbert and Glaser 1961; McLaurin 1966; Bouzarth et al. 1968; Carey et al. 1970; King et al. 1973; Smazknova et al. 1973; De la Torre 1974).

Whilst in the subacute stage of brain injury most of the symptoms are still explicable, this is true only for some of them in the defect stage. In part, the vegetative complaints of the defect stage correspond to the 'post-traumatic syndrome' described by Caveness (1966), which consists of headache, vertigo, giddiness, excessive fatiguability and sleep disturbance, but also intellectual and emotional disturbances. A similar set of complaints in the defect stage has been described by Frowein and Harrer (1956) as the post-traumatic subjective

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complaints syndrome (das posttraumatische subjektive Beschwerdesyndrom).

Nausea and vomiting are amongst the cardinal vegetative disorders that follow brain injury. They are explained by irritation of the vomiting centre in the lateral reticular formation in the medulla oblongata and may be due to vascular–circulatory disturbances following temporary basilar insufficiency, in which case they may have an extracerebral origin. Nausea and vomiting occur almost always in the acute stage of the post-traumatic syndrome and in it alone. If they continue into the subacute phase, a whiplash trauma of the cervical spine should be suspected, or some other independent cause such as electrolyte imbalance. Nausea and vomiting which start late may be caused by a rise in intracranial pressure due to a chronic subdural haematoma, or brain abscess.

Athough nausea for the most part is a subjective vegetative disturbance, vomiting can be more serious, particularly if it happens whilst the patient is unconscious, and failure to employ relevant first aid measures may have the most serious consequences, such as aspiration of vomitus with its consequent cerebral hypoxia and cerebral oedema. As always, the latter may result in an increase in volume, displacement of mass and tentorial and foraminal herniation. In this way patients with mild head injuries and no parenchymal damage may develop a fatal acute, irreversible bulbar brain stem syndrome, or very severe secondary damage to the cerebral hemispheres and the brain stem (apallic syndrome). It is imperative to treat the nausea with drugs, and if there is vomiting, to position the patient correctly and if necessary intubate him speedily.

Giddiness. In both the acute and subacute phases after head injury, nausea and vomiting may be associated with giddiness, which can have many different causes. Frenzel (1952) subdivided giddiness into the extreme type of vestibular giddiness and the diffuse vasomotor cerebral giddiness (Hirnschwindel). The vestibular type (vertigo) (Drehschwindel) is associated with pseudo-movement of the surroundings in the direction of the nystagmus (oscillopsia). In the acute stage, vertigo occurs as persistent giddiness, and in the subacute stage as a positional effect, or associated with change in position and movement. Later it is found as bouts or sudden attacks of giddiness. Vertigo is caused by a vestibular disturbance, according to Hoff et al. (1961), due to vestibular lesions in 90 % of cases, often as a consequence of fracture of the petrous temporal bone. Vertigo may also result from whiplash injury of the cervical spine, with a predominant disturbance of the circulation in the vertebral artery on one side (Scherzer 1968). Vertigo caused by central-vestibular disorders without a circulatory origin is unusual in the acute stage and absent from later stages. Giddy feelings, which may resemble vertigo, may be a manifestation of post-traumatic epilepsy (psychosensory attacks), and should be distinguished from vestibulogenic epilepsy (Orban and Lang 1963), i.e. epileptic attacks excited by caloric labyrinthine stimulation tests.

After head injury Scherzer (1968) reported that the commonest complaints were giddy feelings of swaying or groping (Schwank-, Tastschwindel), and a feeling of unsteadiness that was not true giddiness. The cause of these types of giddiness is usually a lesion of the vestibular system (commonly peripheral), but it may be psychogenic. Cases with feelings of swaying particularly have feelings of floating.

Diffuse vasomotor cerebral giddiness, which is also known as syncopal-like giddiness (Frenzel 1952), resembles in its aetiology a circulatory disturbance of the brain stem. However, it may be observed with vestibular lesions. In the subacute and defect stages, vasomotor cerebral giddiness may be associated with temporary loss of consciousness, which is known as cerebral syncope. Gastaut and Gastaut (1957), and Gastaut and Fischer-Williams (1956), have described the frequent misdiagnosis of post-traumatic epilepsy in cases of cerebral syncope following brain injury; the diagnosis was made more difficult in their patients because occasionally urine was passed and there were myoclonic attacks during the syncope. There is also the possibility that a symptomatic epileptic attack may be produced by cerebral syncopal episodes ('see-saw' reaction (Kippreaktion) of Selbach 1953).

It is usually impossible to distinguish absolutely between vestibular vertigo and diffuse vasomotor

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cerebral giddiness. Even though subjective giddiness is not life-threatening, its combination with vegetative symptoms, particularly vomiting, e.g. as in vertigo, or the subjective sensation of giddiness is a severe burden for the patient, and special treatment has to be undergone.

Vasomotor disorders which take the form of fluctuations in blood pressure and pulse rate, and a tendency to collapse under various forms of stress, are some of the principal features of the vegetative disturbance that may follow brain injury. Although these disorders occur particularly during the acute stage, they may persist until later, and even at the defect stage they and their complications may be a serious problem to the patient. They should be distinguished from those disturbances of blood pressure and pulse rate that are associated with an increase in intracranial pressure and produce the features of an acute secondary midbrain or bulbar brain syndrome.

Changes in the resting blood pressure and pulse rate occur immediately after trauma to the skull (Frowein and Harrer 1956). There is usually a more or less marked increase in systolic blood pressure. In milder cases who do not have complications it falls to below normal during the first day; in the following days, and certainly within the next 2–3 weeks, the blood pressure returns to normal. An increased blood pressure maintained for several days is suggestive (Wanke 1950) of a moderately severe brain injury (contusio cerebri). Tönnis et al. (1949) used Schellong's method to test circulatory function and found disturbances of regulation in two thirds of cases of recent brain injury. These workers, as well as Bürkle de la Camp (1951), Westermann (1954) and others reported that this method could be used to assess the severity of the cranio-cerebral trauma. The Schellong method of testing circulatory function involves continuous measurement of blood pressure in the erect patient for a period of 10 minutes. A fall of systolic pressure of more than 20 mm Hg below the initial value is indicative of a regulatory disturbance. Maintenance or an increase of diastolic blood pressure is indicative of a hypertonic regulatory disturbance, whilst falling diastolic and systolic pressures are due to a hypodynamic regulatory disorder. The latter is more serious. Quite often the pulse rate increases as the blood pressure falls ('tachycardio-regulatory disturbance' of Tönnis).

Hypertension after brain injury may be confirmed by measurement of retinal artery pressure. Raised diastolic pressure in the central retinal artery may give some indication of the side of the injury to the cerebral hemisphere. At the same time as the diastolic pressure is raised the systolic is depressed (Remky 1950; Frowein and Harrer 1956).

The circulatory disturbances associated with states of shock must be differentiated from the vasomotor disturbances caused by cranio-cerebral trauma. It is difficult to differentiate between centrally caused regulatory disturbance of the vegetative system and the peripheral vegetative disturbance of shock. In cases of head injury the various types of shock, such as haemorrhagic, hypovolaemic, toxic, cardiogenic and neurogenic shock of haemorrhagic or hypovolaemic type are of inportance (Kucher and Steinbereithner 1972). The latter occur mainly with additional injuries, particularly tissue bleeding. They may, as reported by Killian and Dönardt (1955), produce sympatheticotonic centralisation in order to maintain circulation in the important organs. If the response is excessive the blood pressure may collapse and then the patient usually dies. The principal signs of shock are thread-like pulse, tachycardia, fall in blood pressure, pallor and later disturbance of consciousness. The result of shock is a generalised reduction in tissue circulation with subsequent hypoxia and the development of cerebral oedema (Schneider 1964).

Disorders of circulatory regulation may be present for months after a brain injury and may even persist during the defect stage. In addition to orthostatic loading of circulatory function, other methods of investigation may be used, such as adrenaline and sympatol testing and examination of the excitability of the vasomotor centre by inhalation of CO_2 according to Raab's technique.

As has been shown by the many post-war investigations, mainly carried out in Germany, two types of regulatory disturbance of the circulatory system can be distinguished; the hyperregulatory syndrome (peaking of the blood pressure during circulatory function tests, increased responsive-

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ness of the vasomotor centre to CO_2 stimulation, increased blood pressure reaction during a sympatol test) and the hyporegulatory syndrome (inadequate response of the circulatory system to orthostatic stress, reduced excitation of the vasomotor centres by CO_2 stimulation, reduced response to sympatol). Frowein and Harrer (1956) reported that it was possible to place the lesions in certain regions of the brain. Frontal brain damage produces the hyporegulatory syndrome, whilst trauma to the parietal and temporal lobes produces a hyperregulatory syndrome.

These disturbances of circulatory regulation and instability and lability of the circulation may persist during the defect stage and may also be associated with other vegetative regulatory disturbances to produce the 'vegetative disturbance syndrome' (vegetatives Beschwerdesyndrom) (Frowein and Harrer 1950), or the 'post-traumatic syndrome' (Caveness 1966).

A post-traumatic disturbance of circulatory regulation may not only cause subjective symptoms, but it may, because of the increased tendency to produce collapse with loss of consciousness, even result in cerebral syncope. In addition to general disturbances in circulation, Reivich (1968) and Taylor (1969) have reported a disorder of autoregulation of the cerebral circulation during the acute phase after brain injury. This disturbance is probably functional and not due to a structural lesion. During the acute phase, changes in metabolism with an alteration in acid-base balance and in respiratory rate are caused, and may produce secondary tissue damage. An increase in circulation time may persist for years (Taylor 1969).

In the acute stage Brown and Shoemaker (1973) described reduction in cardiac output and stroke volume and tachycardia, as haemodynamic changes due to severe brain damage that would return to normal during the subacute phase. In patients who died there was also increased peripheral resistance, reduced central blood volume and a reduction in myocardial force. The haemodynamic alterations were considered to be due to an alteration in central regulatory mechanisms. In patients examined in this connection, however, there was probably a secondary mid-brain syndrome.

Electrocardiographic changes which result from brain damage are usually transitory and disappear once signs of central irritation have gone (Schönbrunner 1948, 1949). In severe cases, however, Schönbrunner reported ECG changes that persisted until death. He suggested that they were due to a fall in blood pressure caused by central nervous circulatory collapse and thus were secondary to a haemodynamic disorder. Electrocardiographic changes have also been described during experiments on monkeys (Fernando et al. 1969).

According to Hersch (1961), the most frequent changes in the electrocardiogram consist of a prolongation of the QT interval, and an increase in voltage of the P wave. Other changes were: increased QRS voltage, change of ST-segment, and an inverted T wave in precordial leads V_4–V_6 (Hersch 1961). In 5 out of 165 brain-injured patients, Julkunen et al. (1972) observed an initial high T wave, followed by an inverted T wave with a U wave, and a prolonged QT interval. The correlation of the ECG changes with the area and severity of the brain injury is still unclear. Further study of the prognostic significance of the ECG changes is necessary.

Disturbances of thermoregulation and sweating. Wanke (1948) reported hyperthermia in 30% of patients with cranio-cerebral trauma. Tönnis (1959) considered that it was caused by activation of 'heat centres' in the caudal hypothalamus. Kucher and Steinbereithner (1972) reported that, in addition to the initial rise of temperature, there was a second bout after 5–7 days, and a further attack after 2 weeks. In the present author's experience, hyperthermia after severe cranio-cerebral trauma is a symptom of basal damage, either fronto-basal or in the hypophyseal-diencephalic region, or it may be a feature of one of the phases of an acute secondary midbrain syndrome. In mild or moderate brain injuries, a rise in temperature of central (cerebral) origin is very uncommon.

Alterations in sweat secretion are quite often caused by constitutional vegetative lability (Frowein and Harrer 1956); this is true particularly of the subacute or defect stage after trauma. In addition, other causes of disturbance of sweat secretion should be excluded, such as hyperthyroidism.

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Although a rise in temperature during the acute stage of cranio-cerebral trauma may mark the onset of midbrain herniation, disturbances of heat regulation hardly ever arise in the later stages of brain injury. There have been very few detailed investigations of this problem, or of disorders of sweat secretion.

Respiratory disturbances may be included amongst the essential vegetative symptoms. It is, however, very difficult to correlate respiratory disturbances with the severity of the cranio-cerebral trauma. Frowein (1963) found that disorders of breathing could not be associated with brain damage at any particular site; indeed they were more common after diffuse lesions. However, respiratory disturbance is an important feature in the acute mid- and bulbar brain syndrome. Abnormalities of respiration secondary to metabolic changes must be distinguished from primary neurogenic dysfunction. The former are closely associated with changes in acid-base balance and O_2/CO_2 tension. Changes in blood chemistry, which are the primary result of brain injury, may in this way indirectly cause altered respiration. Respiratory disturbances without other features of an acute midbrain syndrome can be valuable indicators of any severe imbalance of blood chemistry.

Finally, a respiratory disturbance may be an expression of mechanical irritation of the respiratory tracts or the lungs, and be caused either by obstruction of the respiratory passages or by secondary blood gas changes. Obstruction of the air passages, particularly the upper segments, is usually due to aspiration which follows post-traumatic vomiting. A specific type of respiratory disturbance is caused by aspiration of hydrochloric acid (i.e gastric juice): the Mendelson syndrome (Mendelson 1946). This includes cyanosis, tachycardia, dyspnoea, asthmoid bronchospasm, pulmonary congestion and oedema, and a respiratory acidosis as well as a fall in pO_2. Some of the post-traumatic respiratory disturbances which have been described after moderate or severe brain injuries have really been part of this syndrome.

Another form of respiratory disorder is due to pulmonary oedema following traumatic shock. Many factors may cause pulmonary oedema and a different mechanism may predominate in each (Lutz and Schumacher 1965). Their characteristics permit easy differentiation between this and the neurogenic type of respiratory disturbance. The development of so-called central pulmonary oedema may parallel the clinical features of an acute midbrain syndrome (Steinbereithner and Kucher 1972).

Before the respiratory disturbance caused by a head injury can be relieved, it is first of all necessary to be clear about its aetiology, and then to treat it either by restoring to normal the changes in blood chemistry, or by use of respiratory aids, such as immediate reanimation.

Headache. Friedmann (1969) regarded headache as a general result of head injury and as only one facet of the symptoms of the post-traumatic syndrome. He reported that a variety of mechanisms that affect extra- and intracranial structures might be responsible for the development of post-traumatic headache.

According to Friedmann extracranial factors were most often the cause of such headaches and their persistence. They include muscular contraction in the nape of the neck and the scalp, local tissue scarring around the head, haematomata in the extra-cerebral, subdural- and subarachnoid spaces, injuries to structures around the neck, such as the musculature, the ligamentous apparatus, the intervertebral discs, bones, nerve roots, or irritation of the extracranial parts of the cerebral vessels. The only striking intracranial factors were dilatation of cranial arteries and changes in the microcirculation. Finally, Friedmann cited psychological factors as a cause of post-traumatic headaches.

According to the type of headache it should be possible to distinguish those that occur in the acute, subacute and chronic post-traumatic stages. The present authors consider that headaches in the acute stage may be due to meningeal irritation, consequent to traumatic lesion of the dura or leptomeninges; it may also follow traumatic subarachnoid haemorrhage. Furthermore, there is also acute traumatic vascular headache, which has a vasomotor origin, and acute extracranial headache caused by traumatic damage to the cervical spine or the neck, either as a result of whiplash injury or a local trauma. There is a subjective difference between the headaches produced by

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meningeal or vascular mechanisms; vascular headache is pulsatile in character, whilst the meningeal form is a constant pain. Pseudoradicular headache, which takes the form of a pressure headache (helmet feeling) after spinal trauma, differs from these two types.

In the acute and subacute stages, post-traumatic headache is a constant pain, aggravated by movement, as well as by organic or psychiatric stress, and thus made more intense than a throbbing vasomotor pain. Chronic post-traumatic headaches, however, occur at shorter or longer intervals. Two types of continuous headache may be distinguished: the pseudoradicular pain syndrome, which corresponds to the upper cervical syndrome, and vasomotor headache, which may be compared to cervical migraine and its corresponding associated vegetative disorders. Post-traumatic headaches due to local malgrowth of bone after skull fractures or arachnoid scars are uncommon.

Analgesics are the recommended form of treatment for post-traumatic headache in the acute phase. Rest obtained by a cervical collar combined with muscle relaxants may influence the spinal component of the headache; and compounds that regulate vascular tone, such as ergotamine preparations, may help with its vasomotor component. In the later stages the aim of treatment must be to attack the aetiological factors, such as mechanical or functional disturbances, and vasomotor factors, particularly of biochemical type, which may cause migrainous attacks. Contrary to the opinion of Friedmann (1969), the present authors consider that psychotherapeutic measures may be valuable.

Special features

The most important particular vegetative disturbances which may follow cranio-cerebral trauma are of certain metabolic functions. They can be investigated in the laboratory, and the results of such tests provide a useful guide to treatment and for determination of the prognosis. Of course, they are a mandatory requirement for the intensive care of any patient with a brain injury. Detailed information about the disturbances of water, electrolyte and acid-base balance, kidney function, carbohydrate, protein and energy metabolism, and also changes in haematology and endocrine function can be found in Vol. 23, Chapter 6 by McLaurin and King.

VEGETATIVE DISTURBANCES CAUSED BY LOCAL INJURIES

This section is concerned only with the vegetative disorders caused by local damage to the cerebellar hemispheres and the diencephalic-hypophyseal system. It must be carefully noted, however, that it is very difficult in individual cases to prove that vegetative disturbances are associated with damage to a particular cortical region. This is at least partly due to our considerable ignorance of the cortical representation of the vegetative system. Damage to the hypophyseal and diencephalic regions, respectively, is relatively uncommon because of their central position, as shown by the investigations of Sellier and Unterharnscheidt (1963); it is usually caused by centrifugal forces (Unterharnscheidt and Higgins 1969a, b), bone splinters or basal haematoma formation. Hypophyseal and diencephalic lesions are easier to distinguish, because of the measurable biochemical changes which they produce.

Local injury to the cerebral cortex

One of the most common sites of massive cortical contre-coup lesions is the fronto-basal region, and another is the basal part of the temporal lobe. Functionally, the closed association of both areas with the limbic system is important. The little understood vegetative disturbances associated with injury to the parietal lobes of the non-dominant hemisphere are less important. A control centre, believed to be responsible for the various vegetative functions, is present in the orbito-frontal cortex, the fronto-basal region and above all in the medial surface of the frontal lobes (Clara 1959). This cortical region affects both the central vegetative regulatory areas and all other similar cortical zones. The gyrus cinguli is another associated part of the cortex that is connected to the limbic system. The anterior temporal lobe is held to have a vegetative control function because of its association system with the cortico-hypothalamic tracts which pass to the orbito-frontal

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cortex and the temporal pole. From these anatomico-functional considerations it is apparent that damage to the fronto- and temporo-basal cortical regions may cause vegetative disorders. A further explanation for the occurrence of vegetative defects after cortical damage involves the anatomical association of the ascending reticular formation, which radiates diffusely into the cerebral cortex from various relay nuclei, and thence returns to the reticular formation of the brain stem. The reticular formation itself is responsible for the integration of respiration and vasomotor tone, the regulation of muscle tone and the level of consciousness.

The traumatic fronto-basal syndrome was described by Kretschmer (1949). In the acute stage it comprises restlessness, frontal symptoms and loss of vegetative inhibition, and in the subacute and defect stages a change in the higher brain functions, loss of emotional control, a tendency to motor overactivity, loss of control of libido with hypersexuality and bulimia, profiled frontal lobe symptoms and vegetative lability, such as changes in circulation, temperature regulation and increased sweating. As the authors have shown elsewhere (Haider et al. 1975), the fronto-basal syndrome is associated with increased basal metabolic rate, a rise in the secretion of catecholamines and a disturbed nitrogen balance. Clinically, in addition to these symptoms and signs there is a tendency to vascular hypertonia and a disturbance of the sleeping and waking mechanism. The lack of vegetative disturbances and the various psychiatric disorders (apathy, reduction of activity etc.) may permit differential diagnosis of the fronto-cortex from the above-described fronto-basal syndrome.

The vegetative disturbances of the fronto-basal syndrome have been treated successfully with sedatives, particularly phenothiazine derivatives, and also by mixtures of atropine and barbiturates.

The traumatic temporo-basal syndrome, which has only recently been differentiated (Gerstenbrand 1974) corresponds to the pseudopsychopathic syndrome (Peters 1969).

The clinical features comprise increased drive, disturbance of concentration with very easy distraction, euphoria, reduced anxiety, flattening of affect, loss of inhibition of libido causing hypersexuality, disturbance of social adjustment, primitive oral motor patterns and vegetative disorders, and in some cases temporal epilepsy. The acute stage may resemble in whole or in part the symptomatology of the Klüver–Bucy syndrome (Gerstenbrand and Lücking 1971). The principal vegetative disorders of the temporo-basal syndrome found by the authors was lability of circulatory regulation. There have been no reports of the symptoms that follow the temporo-basal syndrome. Treatment of the vegetative disorders is not necessary, but the epileptic fits should be controlled. Phenothiazine derivatives may be helpful in controlling the loss of inhibition of the libido.

Traumatic lesions of the parietal lobes. Lesions of the non-dominant parietal lobes, particularly Area 39, the angular gyrus, may produce vasoneural or central trophic disturbances. They are manifested as atrophy of the corresponding extremity, particularly in its distal part, especially in the upper limb, lividity and cyanotic discolouration of the finger- and toe-tips and secondary changes in the skin and joints. Patients also complain of severe pain, especially on passive or active movement, despite the concurrent sensory disturbances. No explanation for these central vegetative disturbances has yet been advanced, although it has been assumed that there is some central representation of vegetative functions in the non-dominant parietal lobe.

Pain must be treated symptomatically; vasodilators are usually ineffective; physiotherapy is useful for treating secondary changes in the joints and their associated contractures.

Traumatic lesions of the diencephalic hypophyseal system

Some of the vegetative disturbances produced by lesions in this region have already been described in Chapter 6 (Volume 23) on the metabolic effects of head injury by McLaurin and King.

Sarkissow (1967) stated that vegetative-trophic, neurohumoral and psychic functions were coordinated in the hypothalamus. There is a direct

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communication between the cortex and the paraventricular and supraorbital nuclei of the hypothalamus (Ward and McCulloch 1947). The latter are connected by efferent fibres to the hypophysis, which provides a pathway for control of hypophyseal function by the frontal cortex.

As mentioned above, local damage in the diencephalo-hypophyseal region caused by primary traumatic lesion is unusual. If such a lesion does occur, perhaps due to a foreign body such as a projectile or splinter, acute vegetative disorders ensue. Bodechtel (1958) described them in terms of Förster's theory of 'diencephalic crises' (Zwischenhirngewitter). The principal features of the syndrome are a raised body temperature, sweating attacks, circulatory disturbances, hyperventilation, hyperglycaemia and poly- or oliguria. Bodechtel (1958) mentioned the possibility of a counterregulation to a restorative phase. The fasting levels of blood sugar may be extremely high, even with concurrent glycosuria.

Damage to the diencephalic-hypophyseal system sometimes causes diabetes insipidus. It may be mild and transient and only apparent during stress (Sack 1947; Kornblum and Fisher 1969). It usually appears on the tenth to fourteenth day and is often accompanied by chiasmal damage (bitemporal hemianopsia). In some cases, symptoms are prolonged over several weeks or months. Hypophyseal symptoms such as acromegaly were described by Birkmayer and Winkler (1951) as a rare occurrence in association with diabetes insipidus. It is usually possible to treat these cases successfully by administration of pitressin.

Local hypophyseal-diencephalic lesions may be associated with endocrinological disorders, as has already been discussed by McLaurin and King, Volume 23, Chapter 6.

The acute, secondary mid- and bulbar brain syndrome

In patients with an acute mid- and bulbar brain syndrome, the totality of post-traumatic complications may amount to so-called 'compressio cerebri', an expression, which, despite much argument to the contrary (Bay 1953), is still used in clinical accounts of cranio-cerebral injury, particularly as no better term has been put forward. It must be noted in this context that the clinical diagnosis of primary brain stem contusion, which has been used for generations, is no longer considered valid after investigation of the pathomechanism of an impact on the head and especially after diverse morphological studies. In previous accounts by various workers of the problem of cranio-cerebral trauma, it was accepted that injury to the mesencephalic region could be caused by external trauma (Bay 1953; DeMorsier 1947a, b); but Sellier and Unterharnscheidt (1963) showed that local damage to these areas of the brain was very uncommon, except, perhaps, in association with centrifugal trauma. Peters (1966) and Jellinger (1966) have confirmed the rarity of primary traumatic lesions of the midbrain. Thus, in the majority of cases the development of an acute midbrain syndrome must be regarded as a secondary complication. Therapeutic nihilism in a case of acute traumatic midbrain syndrome is totally unacceptable. The clinical features of a primary, traumatic acute midbrain syndrome are virtually the same as those of the secondary syndrome, although the latter may develop physically and are not usually obvious.

There are two principal causes of post-traumatic complications which produce brain stem compression during the course of 'compressio cerebri', namely an acute intracranial haematoma (epidural, subdural or intracerebral), and acute diffuse, cerebral oedema. Both produce an increase in volume, mass displacement and tentorial and foraminal herniation.

Plum and Posner (1972) described them as uncal herniation which may be combined with central herniation. It is of clinical significance that uncal herniation may be more marked on one side and so cause lateralisation of the midbrain syndrome (Gerstenbrand et al. 1973).

The acute, secondary midbrain syndrome. Gerstenbrand and Lücking (1970) described four phases of the acute midbrain syndrome associated with bilateral herniation; in some ways they agree with the subdivisions into 'early' and late diencephalic stages of McNealy and Plum (1962) and of Plum and Posner (1972). During development of the phases of the acute midbrain syndrome, as well as in the phases of the acute bulbar brain syn-

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drome, it is best to differentiate the clinical symptomatology into the symptom categories of disturbance of consciousness, reaction to painful stimuli, emotional reactions, disturbances of eye and body movements, and disorders of vegetative function. Whilst the state of consciousness, reaction to external stimuli and emotional reactions become increasingly inhibited during development of the midbrain syndrome, eye, body and limb movements as well as vegetative functions become increasingly less inhibited. These effects become manifest as transitory constriction of the pupil, divergent position of bulbi and initial overshooting of still-coordinated mass movements of the extremities and the body, which increasingly develop into an extensor posture with stretch synergism. The loss of inhibition of vegetative functions includes a rise in pulse rate which may lead to tachycardia (160/min), an increase in respiratory frequency which may lead to a machine-like respiration, hypertension of more than a third of the initial values, and pyrexia to extreme values of more than 42°C (Fig. 1). In the fully developed stage of the acute midbrain syndrome, there is usually a marked increase in sweating and bronchial secretions. Table 1 summarises the symptomatology of the fully developed acute, secondary midbrain syndrome.

It is only recently that the vegetative disturbances associated with this syndrome have been systematically examined. Lorenz (1973) studied blood pressure and pulse rate and catecholamine, whilst Lausberg (1970, 1972) has investigated disturbances of temperature regulation.

The authors' own research group investigated metabolic disturbances by use of the respiratory quotient, respiratory minute volume, oxygen uptake and nitrogen balance; catecholamine secretion was also studied (Haider et al. 1975).

Pulse rate rose (Fig. 2) as the acute midbrain syndrome progressed (Lorenz 1973), an observation in agreement with those of Gerstenbrand and Lücking (1970) and Amann et al. (1971).

Systolic and diastolic blood pressures lagged behind the increase in pulse rate (Fig. 2), although there were regular episodic peaks during the extensor spasms of the fully developed acute midbrain syndrome (Lorenz 1973). Patients who were hypertensive before the head injury showed marked swings of systolic and diastolic pressure during development of the midbrain syndrome and of the pulse rate (Lorenz 1973). The quotient of pulse frequency over systolic pressure fell during progression of the midbrain syndrome (Lorenz 1973).

During the course of the acute midbrain syndrome following central herniation (possibly more marked on one side than the other), characteristic changes in respiration may be registered (Fig. 2). In the first phase respiratory frequency rises slightly and in the second, respiration becomes increasingly rhythmic. In the third phase there is a

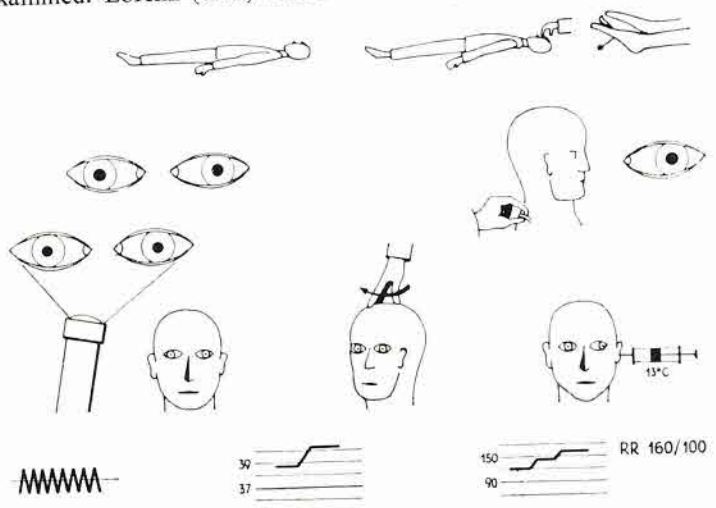

Fig. 1. Schematic representation of symptoms of the complete acute midbrain syndrome (MBS) (Phase 4). (For explanation see Table 1.) (Gerstenbrand and Lücking 1970.)

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TABLE 1

Symptoms of the acute midbrain syndrome (Phase 4) (see Fig. 1).

Unconsciousness
Absent blink reflex
Central positioning of eyeballs
Dissociated or absent eye movements
Moderately wide pupils with diminished response to light
Absent cilio-spinal reflex
Present corneal reflex
Oculo-cephalic reflexes diminished or abolished
Uncoordinated eye movement response to caloric stimulation
Muscle tone elevated (Decerebrate rigidity)
Extensor spasms spontaneously and in response to noxious stimulation
Hyperreflexia, sometimes not elicitable due to elevated muscle tone
Bilateral pyramidal signs
Tachypnea and machine-like respiration
Hyperthermia
Tachycardia
Hypertension
Increased perspiration

type of Cheyne–Stokes respiration, and in the fourth, with the acute midbrain syndrome fully developed, machine-like automatic respiration ensues with a frequency of about 40/min. The result is a reduction in arterial CO_2 tension and a respiratory alkalosis associated with a fall in pCO_2 in the CSF. This 'hyperventilation syndrome' results in changes in electrolyte concentration and increased production of lactate (Steinbereithner 1965a, b; Eldridge and Salzer 1967).

An increase in body temperature develops in parallel with the acute midbrain syndrome (Fig. 2), as observed by Lausberg (1972). The 'spontaneous hyperthermia' may cause an increase in temperature to more than 40°C; nowadays it is called 'central fever'. Lausberg (1972) considered it was due to a disorder of the heat loss mechanism caused by functional disturbances in the mesencephalon. On the other hand, Steinbereithner and Kucher (1972) agreed with Schneider that it was due to activation of irritable centres. Lausberg considered that the hyperthermia caused 'centralisation' of the circulation (i.e. circulation in cen-

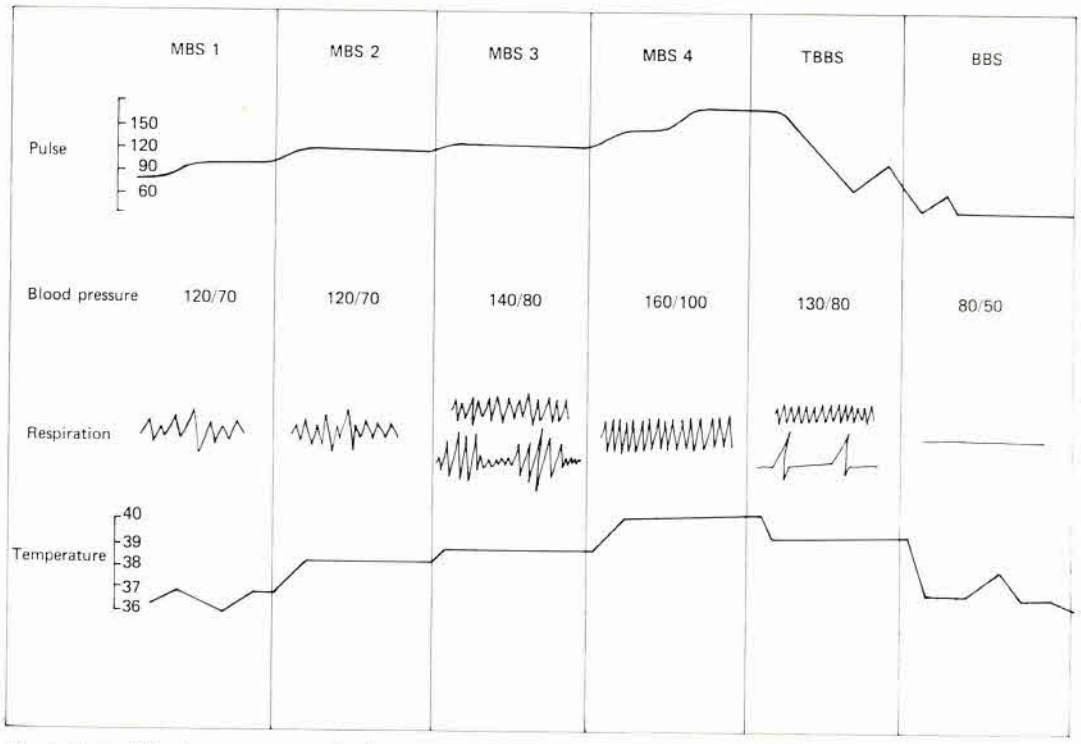

Fig. 2. Pulse, blood pressure, respiration and body temperature during the development of the acute midbrain syndrome (MBS) and the bulbar brain syndrome (BBS).

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tral core regions; Kreislaufzentralisation), which would further reduce heat loss. Mesencephalic hyperthermia may produce an additional increase in metabolic rate, and a further increase in the already elevated requirement for food calories.

Patients who have developed secondary midbrain damage, usually only after the development of the apallic syndrome, may suffer gastrointestinal haemorrhage from an acute ulcer. The stomach is most often affected, but the oesophagus and duodenum may also be disturbed. These complications are well known in neurosurgical patients (Cushing 1932), and were recognised even by Rokitansky (1856) as having a neurogenic basis. More extensive studies of the subject have been undertaken by Simpson (1940/41), Maciver et al. (1956) and Lewin (1966). The most detailed work was reported by Bischof (1965), who produced acute gastro-intestinal haemorrhages by brain injuries in experimental animals; in 50% of cases he found an association between pulmonary oedema and generalised hypoxia with gastrointestinal haemorrhage. The pulmonary oedema thought to be due to generalised shock and diversion of the blood circulation to the central body core, and to release of tissue thrombokinase, which caused thrombocyte aggregation, multiple emboli and consequent hypoxidosis.

In the acute, secondary lower brain syndrome there is virtual collapse of the motor and vegetative regulatory centres of the brain stem, due to damage to the medulla oblongata by interruption of the long tracts to the spinal cord caused by local pressure of herniated parts of the cerebellum. The chief effects comprise a reduction of extensor spasms, increased flaccidity of body posture, loss of muscle tone and failure of respiratory function.

The respiratory disturbance starts as gasping, changes to frog-like movements of the mouth (Mundbodenatmung) and finally breathing ceases (Fig. 2). At the same time as the respiratory disturbance, there is a fall in pulse rate, first to normal values and finally to bradycardia, and a reduction to normal levels of the raised blood pressure caused by the midbrain syndrome. The hyperthermia, too, ceases and normal body temperature reappears.

Lorenz (1973) made a systematic study of the disintegration of circulatory function, particularly the disturbances of pulse rate and blood pressure (Fig. 2). He found an increase in the pulse quotient and respiratory frequency in the bulbar brain syndrome, in contrast to the midbrain syndrome.

During the development of the lower brain syndrome, Lausberg (1972) showed that the temperature fell in steps. For obvious reasons investigations of metabolic balance, catecholamine excretion etc. have not yet been undertaken in patients with the lower brain syndrome.

Irreversible loss of brain function. If an acute lower brain syndrome lasts for more than 30 minutes, a condition develops which is known as 'irreversible loss of brain function' (Gerstenbrand and Lücking 1970). It is characterised by collapse of the vegetative regulatory system of the brain stem in addition to loss of cerebral function (see Table 2). In a high percentage of cases, deeply organised motor patterns are apparent (Gerstenbrand 1973; Gerstenbrand et al. 1975): the so-called 'spinal reflexes'. Following the onset of irreversible loss of brain function, there is a complete failure of circulatory regulation which, inter alia, causes a drop in blood pressure. The maintenance of adequate circulation to the organs then requires intensive pharmacodynamic measures. The disturbance of temperature regulation produces hypothermia, sometimes even lower than 33 °C (Lausberg 1972); the author has observed cases with body temperatures as low as 23 °C, or even room temperature. To maintain a patient in such a state of suspended animation, necessitates artificial methods of warming. In his account of the vegetative symptoms associated with irreversible breakdown of cerebral function, Kurtz (1973) stated that the fall in pulse rate, blood pressure and temperature described above did not occur in all cases, and that fluctuation of the patient's temperature with his surroundings was not an obligatory part of the disorder. It is clear from his reports, however, that in some cases cardiac arrest probably preceded the onset of brain death, so that the picture of irreversible damage of brain function described above never developed. Kurtz (1973) reported that one vegetative function, namely respiration, was always absent in these patients.

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TABLE 2

Symptoms of irreversible loss of brain function.

1) Clinical criteria.

Unconsciousness; no reaction to external stimuli
Loss of spontaneous motor activity
Loss of muscle tone
Maximally dilated, non-reacting pupils
Loss of brain stem reflexes (pupillary and corneal reflex; oculo-cephalic and vestibulo-ocular reflex; masseter-, retching-, swallowing-, cough- and tracheal-reflex).
Respiratory arrest
Hypothermia or poikilothermia
Deficient regulation of circulation
Spinal reflexes may be present:
'Fremdreflexe': tonic grasping reflexes of the toes; flexion (withdrawal) reflexes of legs and arms; priapism; contraction of vaginal muscles; contraction of muscles of the pelvic floor and abdomen; cremaster reflex; Galant reflex etc.
tendon reflexes: knee-jerk and ankle-jerk reflex (biceps reflex)
Mechanic muscle contraction (idiomuscular swelling).

2) Isoelectric EEG
more than 6 hours (registration for 20 minutes during every hour).

3) Angiographically proven cerebral circulatory arrest.

4) Loss of oxygen gradient between cerebral arteries and veins.

Syndrome following a secondary traumatic mid- and bulbar brain syndrome. During reversal of an acute, secondary brain stem syndrome which, at least in principle, is possible at any stage, there will be recovery of the vegetative functions corresponding to the disappearance of the disorders of consciousness and motor control. During remission of a fully-developed bulbar brain syndrome, which must occur within the first 30 minutes of its onset, loss of the inhibitory features of the acute midbrain syndrome is apparent first, particularly loss of extensor synergisms and of vegetative inhibition, followed by further phasic progression as consciousness is gradually recovered. A number of cases develop a traumatic apallic syndrome. There has only been sporadic systematic investigation of the vegetative symptoms that follow an acute mid- or bulbar brain syndrome. Reports in the literature have failed to distinguish between the effects of severe brain injuries and the secondary, acute midbrain syndrome and, in the French literature, also from the apallic syndrome which is usually termed 'coma prolongé'. The latter inaccuracy is also applicable to some extent to the Anglo–American literature.

As there are also primary traumatic lesions in the majority of cases of the acute secondary midbrain syndrome after head injury, the patients may have the subjective complaints syndrome as well as the concomitant complaints objective syndrome (see p. 591). The symptoms of an acute, secondary mid- or bulbar brain syndrome are very similar to the complaints which follow a severe brain injury, although they are more intense and more prolonged.

A disturbance of the circulatory system, in the sense of hypotonia of the circulation, may be seen in almost all patients within weeks or months of an acute mid- or bulbar brain syndrome. The resultant subjective disturbances demand appropriate medical treatment.

The present authors' work has shown a disturbance of energy metabolism (Haider et al. 1975), in which there may still be a marked rise in basal metabolic rate, even weeks after disappearance of an acute midbrain syndrome. No long-term controlled examinations have been undertaken, but clinical studies have suggested disappearance of the disturbance of energy metabolism after a fixed time of about 6 months. Vigouroux et al. (1972) have established that there are disturbances of

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basal metabolism in patients with the 'prolonged coma' (coma prolongé) type of acute midbrain syndrome, in the sense employed in the French literature, and have commented that the basal metabolism remains abnormal for some time.

Glucose metabolism may also be decompensated for long periods. Vigouroux et al. (1972) reported a rise in the rate of glucose metabolism. Haider et al. (1975) have shown that glucose utilisation is markedly increased. There are no data available about more prolonged disturbances of carbohydrate metabolism.

The disorders of water balance in patients who have suffered from acute, secondary traumatic mid- and bulbar brain syndromes have not been adequately investigated. It may be assumed that, in some patients with reported severe brain injuries who have disturbed water balance, this lesion has occurred as a result of an acute, secondary midbrain syndrome. Disorders of temperature regulation, as reported by Lausberg (1972) in the acute and subacute stages of the mid- and bulbar brain syndromes, have not yet been fully investigated. The present authors found that after midbrain symptoms have subsided there is no disturbance of normal temperature, although the response to heat stress may still be overshooting of temperature regulation. The level of thyroid hormone (thyroxin, T_3 and triiodothyronine, T_4) excretion shows no change in patients with mid- and bulbar brain syndrome (Haider et al. 1975).

Lorenz (1973) and Haider et al. (1975) found a marked increase in catecholamine secretion during the acute stage. The former worker reported a tendency for it to become normal within 3–4 weeks, but the latter (Haider et al. 1975) showed that catecholamine secretion fell to normal over a longer period.

No other systematic observations appear to have been made on disturbances of vegetative function associated with an acute mid- or bulbar brain syndrome.

THE TRAUMATIC APALLIC SYNDROME

Kretschmer (1940) described the apallic syndrome as a condition in which there was a loss of all the functions of the cerebral hemispheres and a reduction of the central nervous activity to the mesodiencephalic level. The symptom complex should not be understood as being due to morphological apallia, i.e. decerebration or decortication caused by parenchymal damage, but rather to functional loss of the physiological activities of the cerebral hemispheres. The most important consequence of this definition is the emphasis it places on the possibility of remission and recovery of any of the disorders of function. Various synonyms have been employed in the literature for the 'apallic syndrome', but none are really suitable to replace the term 'apallic', i.e. without cerebral hemispheric function. This applies, for example, to the terms used by French workers, such as 'coma vigile', 'stupeur hypertonique' etc., and by Anglo–Americans, such as 'prolonged coma', 'locked-in syndrome' etc. (see chapter by Bricolo in this volume).

Gerstenbrand (1967, 1975) and Avenarius and Gerstenbrand (1975) stated that all patients who developed an apallic syndrome also suffered vegetative dysregulation. This was usually manifested as a disorder of sleeping and waking rhythm, which was not regulated by the time of day, but by the time at which the patient felt tired. Additional vegetative changes may include marked changes in sympathetic and parasympathetic tone in all vegetative functions, which parallels the sleeping or waking state. Stimulation produces a form of emergency reaction and so leads to extreme responses, particularly of pulse rate and blood pressure. Chronic irritative states, such as are produced by the common decubitus ulcers or by chronic cystitis may lead to a type of chronic emergency reaction, and to hypertension, tendency to tachycardia etc.

Earlier descriptions of the traumatic apallic syndrome mentioned severe marasmus. It is probably caused by the increased, basal metabolic rate which produces a hypercaloric nutrition (Visalli 1972); 6000 calories per day are recommended in modern treatments of the apallic patient. A hypercaloric diet should be given in the initial stages of the traumatic apallic syndrome, i.e. by the second or third day after the injury, and, according to most recent research, even within the first 24 hours (Gerstenbrand and Galanti 1972; Haider et al. 1975). Haider et al. (1975b) confirmed the rise in metabolic rate in the apallic syndrome; it varied between 110 and 180% of normal (Fig. 3).

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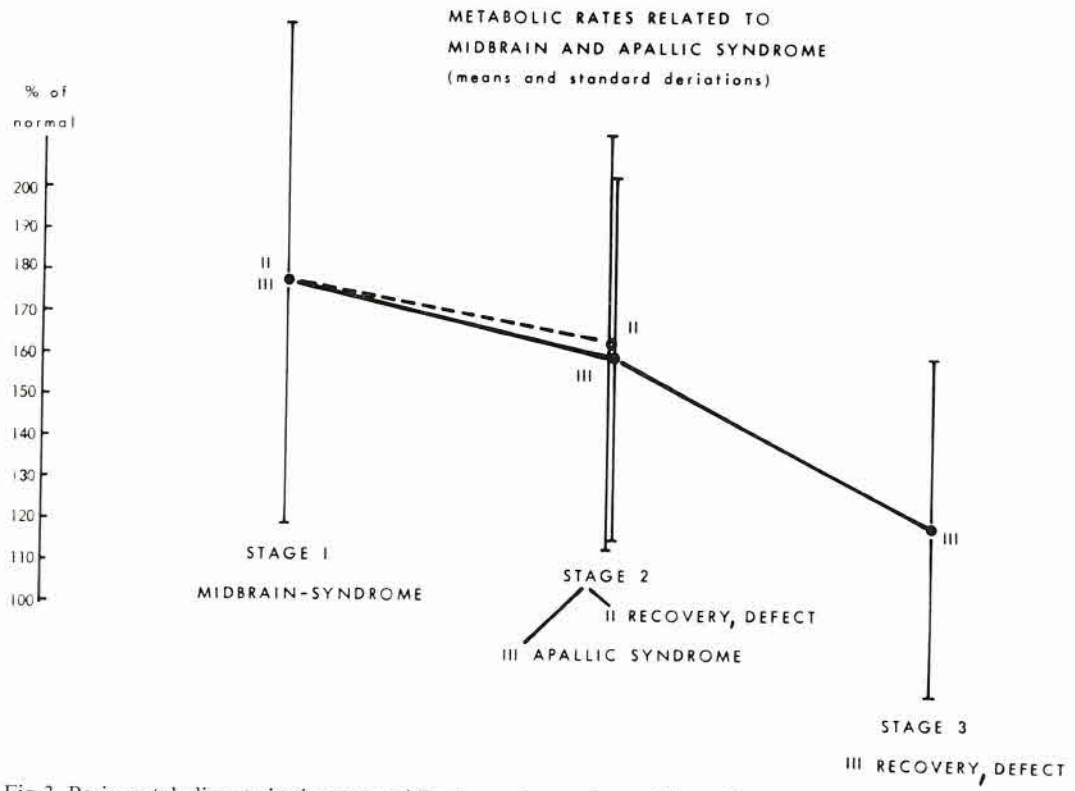

Fig. 3. Basic metabolic rate in the acute midbrain syndrome, the apallic syndrome and the defect stage (for explanation see text). (Haider et al. 1975b.)

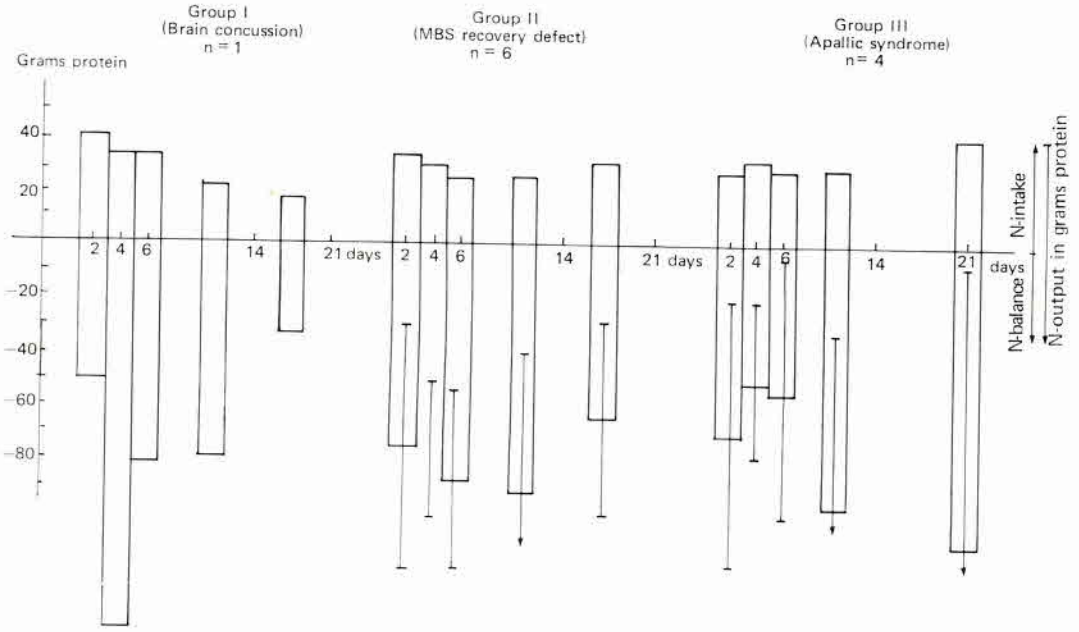

Fig. 4. Nitrogen balance in three groups of patients with traumatic brain damage (for explanation see text). (Haider et al. 1975a.)

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There is a negative nitrogen balance in apallic patients after brain injury, which is most marked in the fully developed cases of the syndrome. It disappears on remission (Haider et al. 1975), but if the apallic syndrome persists, the disturbance diminishes (Fig. 4).

Periarticular ossification, which may be observed in 25% of cases of the apallic syndrome (Gerstenbrand et al. 1970) is thought to be due to a vegetative disturbance, particularly disordered regulation of calcium metabolism. The essential basis of this complication appears, however, to be increased tone of the tonic musculature. No more detailed investigations appear to have been made of calcium metabolism in such patients. The authors' recent experiences show that by using high-dose cortisone treatment over a long time period, ossification can be markedly reduced. Previous to this discovery, surgery was the necessary method of treatment.

In about 60% of sufferers, the traumatic apallic syndrome shows remission to a defect state, which is mild in a third of cases. During the course of the remission there is a reduction in the functional vegetative disturbances. Some abnormalities persist during the defect state, particularly those affecting circulatory function, temperature regulation and generalised vegetative disorders, and notably those responsive to changes in climate. A further persistent change is an excessive vegetative response to bodily stress, a symptom complex which bears the same relationship to the totality of the patient's disorders as does the post-traumatic vegetative syndrome according to Caveness (1966). Pagni et al. (1974) described similar symptoms in children following traumatic apallic syndrome. A proportion of patients with a severe defect develop parkinsonian features, including the corresponding vegetative disorders. As in other forms of Parkinsonism, the latter become apparent as hypersalivation, mask-like face, increased sebaceous and sweat secretion and a disturbance of heat regulation. Furthermore, constipation can occur. The origin of the Parkinson syndrome after traumatic apallic syndrome is in the substantia nigra (Gruner 1965; Travenec 1965; Gerstenbrand et al. 1971).

Two special forms of an endocrine vegetative symptom complex, which follow an apallic syndrome of traumatic origin, must be mentioned. They are, to a certain extent, the result of local damage. We observed pubertas praecox in two cases of traumatic apallic syndrome. The symptoms developed in the defect stage. In both patients (girls of 3 and 8 years), the symptoms are still present, 3 and 4 years respectively after the acute stage. In both cases, the levels of 17 ketosteroids and ketogenous steroids were elevated.

The second form is hypertrichosis. Such cases have been observed by the authors and by Stöwsand (1974). Stöwsand observed alopecia at the same time as hypertrichosis. According to the present authors, an elevated production of gonadotrophic hormones may be the basis of the disease. Bues and Stange (1959) demonstrated elevated production of these hormones during the first 10 days in a case of severe brain injury. According to Stöwsand (1974) the symptoms are the result of a diencephalic lesion, although a diencephalic trophic hair centre has not yet been found.

Of interest is the concentration of homovanillic acid and 5 hydroxyindoleacetic acid (5-HIAA) in CSF (Porta et al. 1973). Their studies confirm certain therapeutic tendencies. They found a relative decrease of HVA level in a case with elevated 5-HIAA. These side-reactions disappeared in parallel with the improvement of the apallic syndrome; at the same time, the biochemical data indicated the effectiveness of the L-dopa therapy in cases of traumatic apallic syndrome. This treatment was first reported and advocated by Gerstenbrand (1967) and later confirmed by Bricolo and Dalle Ore (1970). In this connection, the change in amino acid levels in CSF in cases of traumatic apallic syndrome should be mentioned (Gründig and Gerstenbrand 1970). Glutamic acid diminished excessively, simultaneously with an increase of glycine, threonine, methionine and cystine. These changes correspond to the changes in amino acid levels observed in the Parkinson syndrome.

The vegetative symptoms in the traumatic apallic syndrome are mainly a result of functional disturbances in the meso-diencephalic region, or are due to lesions in this region. This is confirmed by dilatation of the IIIrd ventricle and the aquaeductes Sylvii which can be demonstrated by pneumoencephalography in the majority of cases (60%).

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SPECIAL POST-TRAUMATIC VEGETATIVE SYNDROMES

Two groups of syndromes consisting of complex vegetative symptoms or vegetative detail symptoms as they occur in the acute and subacute stages respectively, and a symptom complex characterised by particular vegetative disturbances, must be mentioned. In the first group, a specific localisation is not possible, but in the second, a circumscribed lesion in the meso-diencephalic region can be assumed.

Discussion of the following disease patterns is justified by their occurrence as unitary complexes and as fragmentary disorders, and on didactic grounds for better understanding of the rarely described and poorly differentiated vegetative consequences of brain injuries. Only after more thorough clinical studies and after systematic investigations using EEG and pneumoencephalography will it be possible to understand the vegetative disturbances, whose very existence has often been overlooked, whose aetiology has been misunderstood, to give a more accurate prognosis and to institute correct treatment. Finally, systematic consideration of the vegetative consequences of a brain injury is necessary for forensic reasons on the one hand, to help to put valid claims for disorders in their clinical setting, and on the other, to enable the manifold subjective complaints to be differentiated; i.e. complaints which are partly or wholly psychiatric in origin, as in a compensation neurosis in a litigant, or which are simulated as part of a deliberate deception.

Complex post-traumatic vegetative syndromes

Every disorder that belongs to this group consists of a number of vegetative symptoms which have marked subjective overtones. The symptoms may either be constant or may only appear in certain stress situations; they may occur as complex sets or only in partial form as groups of complaints.

Little attention has been paid to them in the German literature, perhaps because neurological disorders that are difficult to classify and cannot easily be verified by special investigations, are very rapidly diagnosed as psychiatric in origin. In addition, there is always the tendency to diagnose true subjective complaints as psychogenic. And – lastly – it is so difficult to assess accurately primarily subjective complaints, such as fatiguability, exhaustion and a tendency to tachycardia, that they may be ignored.

The subjective complaints syndrome (Subjektives Beschwerdesyndrom). Frowein and Harrer (1956) gave the following as typical of the subjective complaints syndrome: headaches, drowsiness, giddiness, difficulty in lifting and bending, intolerance of alcohol, nicotine and heat, dependence on the weather, excessive sweating, palpitations, tendency to fainting, disturbance of potency, gastrointestinal disorders, inability to sleep, sensitivity to noise, increased irritability and 'generalised emotional lability'. Most of these complaints are vegetative disorders and are probably due to changes in the integration centres of the diencephalon. The additional presence of psychiatric symptoms, such as increased irritability and sensitivity to noise, suggests primary or secondary involvement of the emotional integration systems of the temporal and limbic lobes.

As suggested by Frowein and Harrer, however, constitutional and psychiatric factors are involved in the sense of a secondary psychogenic superimposition. Important psychiatric factors include the patient's attitude to the trauma (Hoff and Solms 1952) and his mental orientation at the time of the injury (Reisner 1951).

The intensity and variability of the subjective complaints syndrome differ markedly in the individual symptoms. This means that quantitative measurement of them and their effects is very difficult, which, in turn, will influence attempts to assess the harm done. Attempts have yet to be made to correlate the symptomatology with the results of electroencephalography, or with the various quantitative batteries of psychological tests. It appears particularly difficult to differentiate secondary psychiatric disturbances from organically-based symptoms.

The symptoms may first be treated symptomatically by drugs to increase circulatory tone, psycho-sedatives or other proven combined preparations such as Belladenal® etc.

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The 'virtual' post-traumatic syndrome. This symptom complex has often been mentioned in the Anglo–American literature and was most recently discussed by Caveness (1966).

In the introduction to this article it was pointed out that the very existence of the diagnosis of 'post-traumatic syndrome' is uncertain, which has led to widespread misunderstanding of the diagnosis and of the phenotype of this type of disorder. Caveness (1966) characterised the symptoms as: headaches, vertigo, swaying, nervousness, irritability, reduced ability to remember and to concentrate, sleep disturbance and intolerance of alcohol. There was also a feeling of being unwell and a reduced pleasure in living. The principal symptoms were headache and emotional and intellectual disturbance (Denny-Brown 1945; Jacobson 1963). The pattern of the disease in general was consistent with the subjective complaints syndrome.

Caveness (1966) considered that the virtual post-traumatic syndrome did not develop until 3 months after the brain injury. Russell (1934) reported that if symptoms commenced at over the age of 50 years and persisted for more than 18 months, the prognosis was poor. If symptoms were present for a long time, permanent psychiatric defects should be anticipated (Denny-Brown 1945). Caveness and Liss (1961) also reported that there was no clear correlation between the severity of the head injury and the onset of the virtual post-traumatic syndrome. Russell and Smith (1961) drew a connection between the length of the amnesia and the onset of the symptom complex. The syndrome, according to Caveness (1966), is based on a diffuse, cerebral 'irritation' and is less likely to be a local disturbance.

The same therapeutic measures may be employed as for the corresponding subjective complaints syndrome.

The concomitant complaints syndrome. Whilst the above disorders are represented by constant symptom patterns, the concomitant complaints syndrome is a group of symptoms that appear only on stress (Frowein and Harrer 1956); in essence they correspond to complexes of the subjective complaints syndrome. The various symptoms may be excited by a variety of stress mechanisms, which may be considered objectively as of the vegetative type. Frowein and Harrer (1956) saw these symptom patterns as being due to a disorder in switching from the ergotropic to the trophotropic reaction level, which could be manifested as a false reaction, an uncontrolled partial reaction, undifferentiated mixed state or even as a 'catastrophic response'. The clinical symptoms are variable and differ in severity.

This concomitant complaints syndrome appears during the subacute phase of recovery from a brain injury. It has not been possible to correlate them with the severity of the preceding trauma. In addition to symptomatic measures, treatment of this syndrome should include vegetative damping and avoidance of particular stresses. Psychogenic mechanisms appear to play an even more important part in this syndrome than in the complex of subjective complaints syndrome, both in providing it and in its reinforcement.

Specific symptom complexes

As mentioned in the introduction to this section, the results of brain injury may include specific symptom patterns with vegetative features, in addition to the particular disorders mentioned above. To a large extent it is impossible to correlate the specific symptoms to the site of an injury and its severity. It must also be emphasised that certain parts of these symptom complexes are seldom diagnosed, particularly because their presence is not considered. It must also be noted that the necessary additional investigations and methods to obtain objective evidence are not often used and the literature is very scanty.

Disturbances of libido and potency are very common after cranio-cerebral injury. Mild to moderate cases recover within a short time; this is true particularly of younger patients. If the head injury were severe, the disorders may remain permanently. Until recently it was not possible to localise accurately disturbances of libido and potency. However, there are now indications of some association with lesions in the frontal lobes; lesions in the frontal cortex produce increased libido. This is true, too, of cases with a temporo-basal syn-

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drome. Details have already been discussed in the previous sections (p. 582).

Sleep disturbances. Wedler (1953) described sleep disturbances associated with brain stem injury and commented that they disappeared after a relatively short time. Roth (1962) has confirmed this in the majority of cases of post-traumatic sleep disturbances, but added a chronic form of hypersomnia of traumatic origin. The latter condition may present as prolonged hypersomnia (lethargic and somnolent types), periodic hypersomnia and sleep inversion. In contrast to Penta (1935), Roth (1962) reported that hypersomnia was less common than traumatic narcolepsy. However, of 93 patients with hypersomnia, he found only 4 of traumatic aetiology.

The hypersomnia follows directly on the recovery of consciousness; in many cases it rapidly gets better and disappears after a certain period. Its symptoms may present after a lengthy latent period.

Whilst the prolonged hypersomnia is manifested as a state of 'prolonged sleep' of variable intensity, or as 'prolonged somnolence', the periodic type of this disorder is characterised by attacks of prolonged sleep, which may last from hours to twenty days. Sleep inversion becomes apparent as reversal of the normal day-night rhythm.

In the majority of these patients, various neurological lesions in the diencephalon (endocrine disorders, obesity etc.) or the mesencephalic region (oculo-motor disturbances etc.) may also be found, which produce their own symptom complexes, such as the Kleine–Levin syndrome, consisting of hypersomnia and pathological appetite.

Pathological lesions have been found in the diencephalic region in most patients, and in the midbrain and thalamus, as well as in the pons, medulla oblongata and the cortex in a few cases (Roth 1962).

Post-traumatic vegetative attacks. Frowein and Harrer (1956) referred back to Pette (1942, 1943) for a description of vegetative attacks, which included sudden loss of consciousness either without warning or after some preliminary symptoms such as generalised exhaustion, inner tension, pressure on the head, giddiness with nausea and paraesthesiae. There is pupillary dilatation during the loss of consciousness; the unconscious state may last from a few minutes to hours. It may be followed by headache or vomiting. In the earlier literature (Schulte 1949; Birkmayer and Winkler 1951), variants of these attacks were distinguished and descriptions given of their associated vegetative occurrences.

Four different types of vegetative attacks have been described; faints, which take the form of orthostatic collapse with a fall in blood pressure; sympathetic or sympathotonic attacks corresponding to the 'diencephalic autonomic epilepsy' of Penfield; parasympathetic attacks of a Cushing type (Birkmayer 1949), which are accompanied by marked parasympathetic features; and, endogencyclical syncopal attacks as described by Schulte (1949).

Pette (1942, 1943) differentiated vegetative attacks from epilepsy, and Gottwald (1962) held that they never developed into epileptic attacks. Schulz (1972), on the other hand, distinguished epileptic from non-epileptic vegetative attacks but stated that there were no definite clinical criteria for differentiation of the two types, which could only be done by EEG studies. Shimoda (1961), too, described a method for their differentiation by similar means. Janz (1969), however, doubted the existence of diencephalic autonomic epilepsy.

Penfield-type attacks have been attributed to foci in the caudal hypothalamus and parasympathetic attacks to damage in the anterior hypothalamic region (Schulz 1972).

Narcolepsy is often due to head injury. Roth (1962) distinguished essential or idiopathic and secondary types of narcolepsy. He stated that the principal features of narcolepsy were cataplexy, symptoms of various types of sleep dissociation (so-called psychiatric sleep, somnabulism etc.) and disorders of night sleep. The associated neurological findings include oculopupillary disorders, acrovasal syndromes, vegetative disturbances, including abnormalities of water balance, obesity, signs of excessive idio-neural irritability (Chvostek's sign etc.), acromegaly and other endocrine disturbances. Secondary narcolepsy can usually be shown to be in direct time association with the acute stage of the pontine lesion. In

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contrast to essential narcolepsy, Roth (1962) has also found markedly atypical clinical features in the symptomatology and the course as well as significant neurological signs and changes in the pneumencephalogram.

There are characteristic EEG changes in some cases of secondary narcolepsy. Pathological lesions have been very uncommon in the essential type, but patients with secondary narcolepsy may show focal lesions in the meso-diencephalic region, particularly in the posterior hypothalamus (Roth 1962).

Tetany syndrome. Nevšímal and Roth (1963) have described a syndrome of tetany after traumatic damage in the meso-diencephalic region. Fünfgeld (1943) also described patients with cranio-cerebral injuries who developed tetany, but he suggested that the only association with the trauma was increased irritability of the CNS, which changed the rate of hormone production by the parathyroids.

Kogan and Kajschiwajew (1961) described a form of hypocalcaemic tetany that only presented years after the brain injury. They suggested that it was due to a disturbance of the hypothalamo-hypophyseal system.

CONCLUSION

It is very difficult to give a uniform account of the 'post-traumatic vegetative syndrome'. Important indications may be gleaned from the necessary subdivision of the vegetative disturbances caused by trauma into acute phase disorders and those that follow cranio-cerebral injury, as well as from the severity of the acute stage and its possible complications and from the severity and nature of any on-going damage.

A clinical classification of the acute stage has been advanced, as that in use at present is only partly successful. Knowledge of the very uniform symptom patterns of the acute mid- and lower brain syndrome, of their stages of development and of the traumatic apallic syndrome have been very useful additions to our knowledge. The type and course of the vegetative disorders associated with this most serious form of cranio-cerebral trauma provide valuable guides to diagnosis and prognosis.

Differentiation of the vegetative syndrome from associated psychogenic symptoms is very difficult and requires psychiatric investigation and the use of batteries of psychological tests. Wender (1965) believed it would still be possible to arrive at an exact diagnosis in some complicated cases with a negative EEG and negative pneumoencephalogram, employing the various methods for examining the vegetative disorders. The vegetative lesions caused by brain injury may be manifold and only in part can they be classified topically. This is particularly true of the disorders that may follow a primary brain injury with several foci of localised damage that are difficult to distinguish in position, as well as in patients with the traumatic apallic syndrome. As mentioned in the introduction, a better differentiation of persistent vegetative disorders after cranio-cerebral trauma would employ the term 'post-traumatic vegetative syndrome', if possible with topical classification, e.g. 'post-traumatic vegetative syndrome after fronto-basal lesion' etc.; or the name of the particular condition only would be given, such as 'post-traumatic narcolepsy'.

It must be recognised in work on vegetative disturbances that this area of neurotraumatology leaves many questions open. Systematic examination of this complaints complex may reveal new facts about the functional and anatomical basis of the vegetative nervous system.

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