Workshop A/2a:
Treatment and Rehabilitation after Cerebral Sequelae of Accidents and Cerebral Fits

48 New Aspects in Rehabilitation of Patients in an Apallic State

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Long term survival after a severe brain injury is not uncommon nowadays, when intensive care prevents patients from dying within the first days. During those very first days after onset of coma it is difficult or impossible to identify the future state of the patient and the development of an apallic syndrome cannot be excluded. The explanation of these difficulties lies in the pathological basis of the posttraumatic comatose state, which usually consists of badly damaged cerebral hemispheres combined with secondary brainstem lesions of different degree which are clinically well determined by the different stages of the acute traumatic secondary midbrain syndrome.

Fig. 1 demonstrates the development from the acute midbrain syndrome to the apallic syndrome. The transition stage to the apallic syndrome usually lasts 10 - 20 days and can be separated into three periods, coma prolonge, parasomnia and the period of akinetic mutism. During the first two periods the patient is still in a comatose or sleep like comatose state, while at the state of akinetic mutism the patient usually starts to open his eyes. At the full stage of the apallic syndrome the patient is awake with eyes open but shows no reaction to external stimulation.

Fig. 2 shows the clinical picture of such a patient at the full stage of an apallic syndrome. This clinical condition may be persistent but in 80 % of these patients signs of recovery appear.

Fig. 1 (right side): This recovery is called the remission stage of the apallic syndrome. Increased mental awareness is associated by optical contact with the sourroundings and reappearance of voluntary motor movements, usually first seen in the fingers. At the time first emotional reactions may be observed. The following step of remission is marked by the symptomatology of the Klüver Bucy-Tertian-Dalte-Ore Syndrome. The main symptoms are grasping and bringing to the mouth anything followed by biting and swallowing movements. The patient's mood is euphoric, and hypersexuality, lack of fear and rage reactions are commonly seen. The voluntary movements show a progressive recovery.

When the development of speech and other higher brain functions has reached a high functional level the symptomatology of the Korsakoff syndrome may occur. Besides the cerebellar dysfunction voluntary movements may be normal at the time. However, symptoms of local brain lesion can now be clearly determined and may influence the patient's further development. Further recovery may be characterized by a psychoorganic syndrome of different severeness and may include symptoms of organic dementia, spasticity, cerebellar ataxia, parkinsonism and hyperkinesia combined with symptoms of focal brain lesion, metabolic disturbances may influence recovery and reintegration may stop at any step of the recovery. However, when the stage of Korsakoff syndrome is reached, the prognosis for further recovery is good.

In order to avoid secondary lesions of the central and peripheral nervous system, rehabilitation of the apallic syndrome has to be started at the transition stage to the apallic syndrome. This stage is characterized by the onset of an overactivity of the sympathetic nervous system, probably a nonspecific response of the central nervous system, when cortical neurons are not integrated and the brainstem is out of control.

Fig. 3 (from the paper Hortnagl et al 1980) shows the plasma level of norepinephrine and epinephrine and the heart rate of a patient in a midbrain syndrome stage 3. With the onset of the transition stage (marked at arrow) there is a significant increase of the norepinephrine level accompanied by a parallel increase of the heart rate. Treatment with bupranolol, a beta blocking agent, and debrisoquine, an alpha adrenergic neuron blocking agent, reduced both the heart rate and the plasma levels of norepinephrine. The advantage of this therapy is the avoidance of myocardial damage and the suppression of the catabolic drive in the course of severe head injury leading to marasm and peripheral and central secondary nervous lesions. This development was also seen in patients treated with high caloric nutrition. There is certainly an important impact of beta blocking agents on the lipid and carbohydrate metabolism. However, the influence of beta blocking agents on proteins is doubtful. Therefore human growth hormone was thought to be of additional value in the therapy of the disturbed metabolism in apallic patients.

Fig. 4 (from Hackl 1980): Plasma levels of human growth hormone (HGH) were low in all patients without specific stimulation. After arginine stimulation the release of HGH increased in patients with midbrain syndrome stage 2 and 3, but no or less increase of HGH was seen in patients with midbrain syndrome stage 4 and with the apallic syndrome. In the light of these findings HGH was introduced in the therapy of the full stage of the apallic syndrome. Although the number of cases treated with HGH is small, first results suggest a new successfull therapy for these patients.

Fig. 5 (from Gerstenbrand et al. 1980) demonstrates the prognosis of patients in an apallic state. A total number of 441 patients in an apallic state was observed in Vienna and Innsbruck in the last 24 years. 39 % of the patients returned to work. The patients of the group Innsbruck may be of special interest. They were treated by a standardized therapy including the use of beta blocking agents and were under continuous control of a well trained intensive care staff. The good recovery of 56 % of these patients should suggest that the outcome of these patients largely depends on early effective and comprehensive therapy. The use of beta blocking and adrenergic neuron blocking agents has already brought significant help for these patients. HGH seems to be an additional successful therapeutic step. A study just carried out in Innsbruck in these days seems to support this view.

Literature

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Figure 1

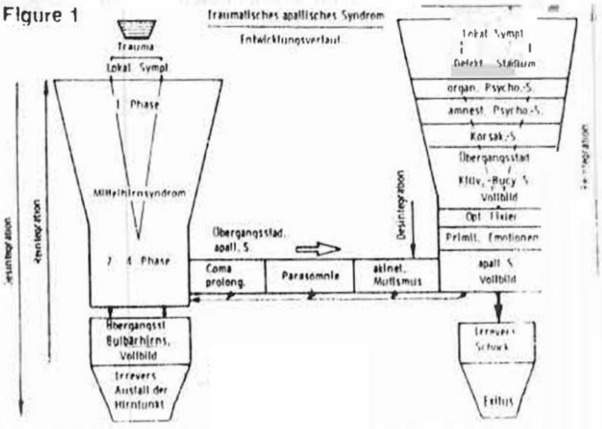

Figure 2

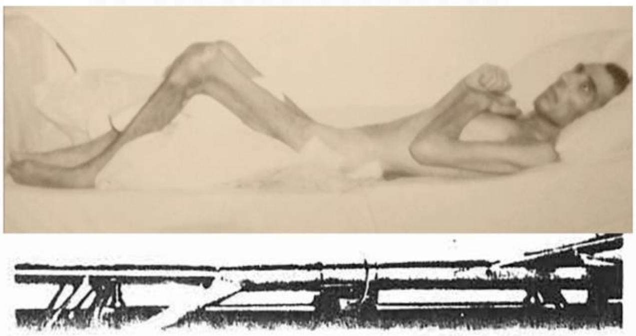

Figure 3: Plasma levels of NE and E and the heart rate in case 4. For explanation, see legend to Figure 1. The patient received bupranolol p.o. (B) and debrisoquine (D) ** approximate onset of remission.

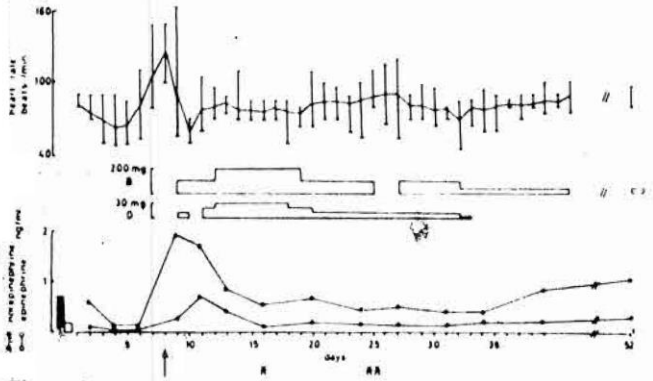

Figure 4

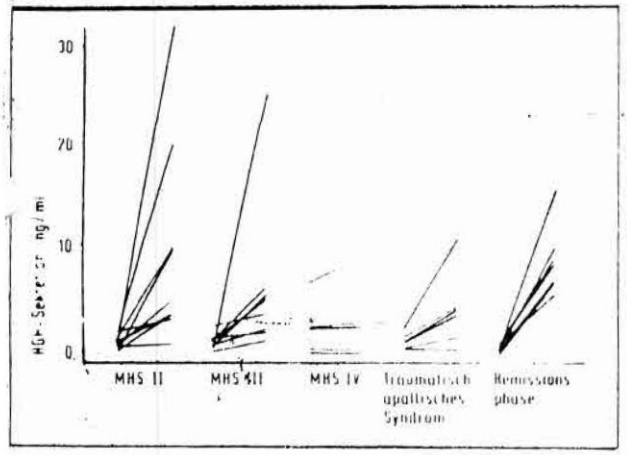

Figure 5

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1957-1966 Wien/1	74	29	2	7	36
1967-1971 Wien/2	106	44	16	10	36
1972-1975 Wien/3	68	30	11	9	18
1966-1975 Innsbruck/1	100	26	21	27	26
1976-1977 Innsbruck/2	43	24	5	11	3
1977-1979 Innsbruck/3	50	20	7	9	14
Zusammen	441	173	62	73	133

49 The Value of a Behaviour Management System in Rehabilitating the Brain-Injured Patient

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Behaviour disorders which can continue beyond the early recovery phase constitute a major problem in the rehabilitation of the head-injured patient. They can impede or prevent co-operation with the therapy staff and interfere in the retraining of functional skills. There are basically four areas of behaviour disturbance which can disrupt the patient's participation in a rehabilitational programme.

Aggression:

This can be of two kinds. First there are the explosive aggressive outbursts which usually have an epileptic basis, following damage to the temporal lobes during injury, and are characterised as uncontrolled, poorly directed, and of short duration. Secondly, we have the habitual aggressive behaviours, which are frequently learned during the post-traumatic confusional period, and which are maintained as an attention seeking phenomenon, very often involving premeditated aggressive assaults on patients or staff, designed to achieve the maximum effect because of the attention they receive from members of staff.

Disinhibited:

Such behaviours are usually seen as part of a frontal lobe syndrome, very common in closed head injury, and involve both sexually and socially inappropriate behaviours which are unacceptable to the open community.

Uncooperative:

This involves the rejecting or insightless behaviour frequently seen as a consequence of severe head-injury, which makes the implementation of therapy difficult, either because the patients reject the treatment or reject that they have any problem which requires treatment. Another feature of unco-operative behaviour which may involve insightlessness is manipulation of staff or other patients for the individual's personal gain. This is often seen as part of a dissociative or "hysterical" personality change in which a patient will demonstrate vicarious motivation and try to achieve certain objectives which are entirely at odds with those established by the treatment team. It is not unusual for such patients to actively sabotage a treatment programme.

Driveless:

Poorly motivated behaviour with severe apathy and lethargy is a frequent residual feature of severe head-injury. This significantly restricts the efforts of the rehabilitational team because the patients are unwilling to exert the kind of effort necessary for progress, simply because they see the amount of effort required as unrelated to the kind of reward available to them as a consequence of that effort. It is important to note that this kind of negative behaviour change is not simply a psychological reaction to injury, but is mainly an imposed and organically determined condition.

Patients with such disturbances of behaviour often fail to receive "proper" rehabilitation because they are either regarded as being unmanageable, due to their disruptive and threatening behaviour; or because their lack of effort and enthusiasm dampens the motivation of the therapy staff to persist in a rehabilitation programme with patients who offer so little reward. These behaviour problems need not affect the clinical or social outcome of such patients, however. A very effective system of behaviour management has long been established, and recently extended to facilitate the rehabilitation of patients with acquired brain injury (Wood and Eames, 1981). A behaviour management programme aims to increase constructive and applied behaviour, while eliminating disruptive or undesirable behaviour, by linking such behaviours to a series of contingent rewards or punishments called "reinforcers", (for a complete description see Bandura, 1969; Kanfer and Philips, 1970). This is best achieved in an environment where there is a general attitude of positive social reinforcement in which the patient receives as much encouragement and praise as is realistically possible for effort towards some rehabilitational goal.

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