

Pathological Changes of the Spinal Cord after Brain Death*

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Summary. The characteristic pathological changes of the spinal cord after brain death of 15 cases are reported. In some cases of a sudden ischaemic or hypoxic accident the spinal lesions occur simultaneously with the ischaemic infarction of the brain. In most cases of cerebral death reactive lesions of the primary intact spinal cord upon the autolytic disintegration of the brain occur. The segments C2/C3 became necrotic in nearly all cases, thus representing a demarcation between the autolytic brain and the spinal cord. Necrotic cerebellar tissue was displaced into the lower parts of the spinal subarachnoid space and produced inflammatory reactions of the meninges and meningeal vessels. The reactive meningeal angiitis and the mechanical impairment of the venous drainage by the cerebellar detritus seem to be the main factors contributing to the severe circulatory disorders in the spinal cord including roots and spinal ganglia. It is assumed that the lesions of the spinal cord are at least partly responsible for autonomous spinal activities of brain death patients.

Key-Words: Brain Death — Demarcation — Circulatory Disturbances, Spinal — Foreign Body Reaction — Spinal Autonomy.

Brain death is the result of a complete and permanent intracranial circulatory arrest, caused by extreme brain oedema, induced by various agents (trauma, tumour, metabolic disorders etc.). Brain death can be regarded as an infarction of all structures enclosed by the skull. If the organism is kept alive, it reacts in all cases identically upon brain necrosis: 24—36 h after the stop of cerebral circulation demarcation develops in the anterior pituitary lobe, in the upper cervical segments of the spinal cord and in the optic nerves [19]. Due to the extreme brain edema axial distortion of the brain stem occurs and caudal parts of the cerebellum are pressed into the foramen occipitale magnum. The resulting cerebellar pressure cone may be cut off and displaced into the spinal subarachnoid space, lying as a cuff around the spinal cord [19, 20, 21].

The spinal cord below the segments C2—C3 is in most cases not directly affected in cases of brain death. The transitory loss of spinal functions, observed during the incidence of cerebral death, corresponds to the spinal shock after a transverse lesion. Later on the isolated cord can regain some autonomous functions, if the organism is maintained alive by intensive treatment [20, 4, 16, 21]. The spinal mechanisms may appear even earlier and with more intensity in cases of cerebral death than in patients with transverse lesion what can lead to diagnostic confusion [4]. No systematic study has been published so far concerning the underlying morphological changes of the spinal cord which determines the further clinical course of brain death patients.

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Material and Methods

The following is the report of pathological changes of the spinal cord observed in 15 consecutive cases of brain death. In the Table the essential data of the primary disease, the survival time, the clinical observations of spinal mechanisms, and the spectrum of the spinal morphological changes are summarized.

Clinical diagnosis of brain death was confirmed by loss of all cerebral functions, electrical silence (flat EEG) and proof of intracranial circulatory arrest by angiography.

The typical findings in the necrotic brain have been described extensively in previous papers [19, 20, 21]. The massive impaction of the autolytic brain, the increased brain weight, fresh thromboses in the superior sagittal, transverse, sigmoid sinuses and in the bulb of jugular vein, and the demarcation in the bordering tissues of the necrotic brain must be regarded as the morphological equivalent of the permanent intracranial circulatory arrest. Brain and spinal cord were prepared from dorsal and removed in connexion. The transitional zone between brain and spinal cord was examined with special attention. Sagittal sections through the brain stem and the upper cervical cord allow the best insight and demonstration of the demarcation zone in C2—C3 (C1—C4) (Fig. 2).

Specimens of the cord for microscopic examination were taken after transverse and longitudinal sections.

Observations

The lesions of the spinal cord in cases of brain death can be classified as follows:

- I. Lesions caused by primary disease.
- II. Lesions occurring as reactions upon brain necrosis (demarcating reactions).
- III. Lesions caused by interaction of primary disease and reactions upon brain necrosis.

This classification partly anticipates the interpretation of the lesions observed in our cases. The combination of different agents in the single case, however, justifies this distinction.

I. Spinal Lesions Caused by Primary Disease

In this group post mortem analysis revealed the simultaneous damage of cerebral and spinal structures after a sudden hypoxic-ischaemic accident. In the two cases mentioned here the ischaemic accident due to cardiac arrest was followed by temporary residual functions of the brain and finally by extinction of all cerebral activity ("intervallary course").

Fig. 1 shows the spinal cord of a 65-year-old man admitted because of septicaemia (Case No. 14). Cerebral death occurred after a cardiac arrest lasting 5—10 min. Reanimation was continued for 50 hrs.

Cross sections of the spinal cord revealed haemorrhages in the grey matter extending to the caudal segments which were more intense in the thoracic and lumbar regions. Histologically multiple, partly confluent pericapillary and perivenous haemorrhagic extravasates were found, combined with endothelial swelling, necrosis and rupture of vessel walls. Shrinkage of the neurons, edema of the grey substance and the bordering white matter as well as granulocytic infiltration demonstrated the severe microcirculatory disturbances developing after the transient ischemia. The displaced cerebellar tissue in the subarachnoid spinal case extended to C8. The upper cervical segments showed only discolouration and softening in the grey and white matter.

Similar lesions were found in a case of HCN-intoxication, complicated by a transient cardiac arrest, as reported in a previous paper [4]. In this case ischemic alterations of the neurons and a beginning glial and capillary proliferation in the intermediate grey matter of the lumbar cord were observed.