

Brain death

F. Gerstenbrand, M. Marosi

University Klinik fur Neurologie, Innsbruck, Austria.

Due to the development of modern anaesthesiology and the proceedings of medical techniques with the increasing possibilities of intensive care, machine supported blood circulation and other organ functions proved to survive brain for a long period. Brain death and not the function of other organs defines the live end. Additionally to this the transplanted organs is compelling to an accurate definition of brain death criteria.

TABLE 1

DEFINITION OF BRAIN DEATH

- IRREVERSIBLE DAMAGE OF BRAIN AND BRAINSTEM
- CESSATION OF BRAINSTEM FUNCTIONS
- SPINAL REFLEXES CONTINUE TO EXIST

- DIFFERENTIATION
- LOCKED IN SYNDROME
 - APALLIC SYNDROME

Brain death is defined as the irreversible loss of all functions of brain and brainstem. (Table 1) The crucial point of this definition is the determination of the irreversibility.

One essential differential diagnosis to the cessation of brainstem functions is the locked in syndrome (Table 2). A differentiation to the cerebral death - the apallic syndrome - is only an interesting aspect.

The pathogenetic mechanisms causing the cessation of brain functions are multilocal damage of brain and brainstem or local primary or secondary lesions of several parts of brainstem. The latter caused by

increasing intracranial pressure with tentorial and foraminal herniation followed by a midbrain syndrome (MBS) and consequently by a bulbar brain syndrome (BBS). Table 3

TABLE 2

LOCKED IN SYNDROME

TOTAL MOTOR IMMOBILITY

EXCEPT - VERTIKAL EYE MOVEMENTS

- BLINKING REFLEX

- THREATENING REFLEX

NO LOSS OF SENSORY AND SENSITIVE FUNCTIONS

TABLE 3

AETIOLOGY OF BRAIN DEATH

1. SEVERE MULTIFOCAL DAMAGE OF BRAIN AND BRAINSTEM

- TRAUMATIC

- ENCEPHALITIC

2. DIFFUSE DAMAGE OF BRAIN AND BRAINSTEM

- EXOGENOUS / ENDOGENOUS INTOXICATION

- HYPOXIC

- DIFFUSE / LOCAL BRAIN EDEMA

- INTRACRANIAL HEMORRHAGE

3. LOCAL LESIONS OF BRAINSTEM

- PRIMARY (TRAUMATIC, TUMOR ...)

- SECONDARY (HERNIATION OF BULBAR BRAIN)

The irreversible breakdown of brain functions mostly shows a typical course. Especially in cases after brain injury this course is characterized by the symptomatology of a midbrain syndrome which could be followed by a bulbar brain syndrome. Fig. 1

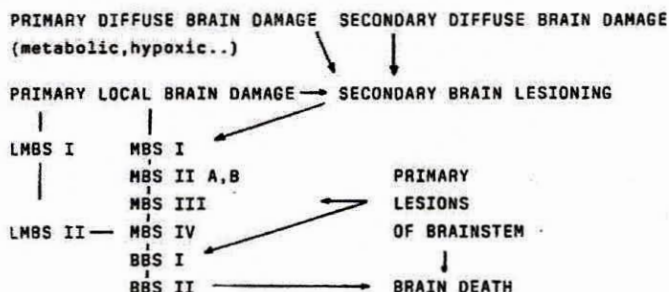


Fig.: 1 developments of brain death

LMBS - lateralized midbrain syndrome, MBS - midbrain syndrome BBS - bulbar brain syndrome

A crucial point for patients with deteriorating brain functions is the MBS IV and BBS I. An ongoing worsening with the symptomatology of a BBS II without a tendency of remission within 30 min. is the point of no return with the consequence of brain death.

TABLE 4

BULBAR BRAIN SYNDROME II

NO REACTION TO PAIN OR OTHER STIMULI
 DIVERGENT POSITION OF BULBI (FIXED)
 MISSING OCULAR MOVEMENTS
 MISSING BLINKING REFLEX AND THREATENING REFLEX
 PUPILS DILATED AND NONREACTIVE
 CILIOSPIINAL REFLEX MISSING
 VESTIBULO OCULAR REFLEX ABSENT
 ATONIC POSITION OF BODY AND LIMBS
 MUSCLE ATONIA
 AREFLEXIA
 RESPIRATION ARREST
 NORMOTHERMIA OR SLIGHTLY INCREASED TEMPERATURE
 HYPOTONIA
 BRADYCARDIA

TABLE 5

SYMPTOMS OF BRAIN DEATH

1. CLINICAL SYMPTOMATOLOGY
 COMA
 NO REACTION TO PAIN OR OTHER STIMULI
 NO SPONTANEOUS MOTOR ACTIVITY
 ATONIC POSITION OF BODY AND LIMBS
 MUSCLE ATONIA
 BRAINSTEM REFLEXES ABSENT
 - PUPILS MAX. DILATED AND NONREACTIVE
 - CORNEAL REFLEX ABSENT
 - OCULOCEPHALIC REFLEX MISSING
 - VESTIBULO OCULAR REFLEX MISSING
 - GAG REFLEX, SWALLOWING REFLEX ABSENT
 RESPIRATION ARREST
 POIKILOthermia
 HYPOTONIA - ARTIFICIAL SUPPORT NECESSARY
 SPINAL REFLEXES
 2. ISOELECTRIC EEG

The difference between the BBS II and brain death are the break down of vegetative functions and the occurrence of spinal reflexes. In about 60% of patients with the diagnosis of brain death mono- and polysynaptic reflexes are present. Spinal reflexes can be observed between 1 and 6 hours after the first symptoms of brain death. The occurrence of these motor reflexes render more difficult the diagnosis, but are also astonishing - for instance the tonic plantar flexion reflex which could be connected with a flexion of the homolateral lower limb and perhaps with an additioned flexion of the contralateral upper limb. Some of these reflexes are listed up in Table 6.

The existence of the spinal reflexes complicate and rattle the diagnosis of brain death. To assure the diagnosis auxilliary diagnostic means should be applied.

Till now the most important technique to assure the diagnosis of brain death is the use of EEG. According to international and also national recommendations of several EEG societies the EEG evaluation is done on fixed premises.

Concerning brain death examination we stick to well defined schedule and criteria. Table 7

TABLE 6

SPINAL REFLEXES (in 60% of cases)

all reflexes with \pm long latency after occurrence of first signs of brain death

POLYSYNAPTIC REFLEXES

- * TONIC PLANTAR FLEXION
- * FLEXION OF LOWER / UPPER LIMB
- * PRIAPISMUS
- * CONTRACTION OF INTROITUS VAGINAE
- * CONTRACTION OF PELVIC FLOOR
- * CONTRACTION OF ABDOMINAL MUSCLES
- * GALANT REFLEX

MONOSYNAPTIC REFLEXES

- * PATELLAR REFLEX, BICEPS REFLEX

TABLE 7

INNSBRUCK - BRAIN DEATH EXAMINATIONS

I. CLINICAL SYMPTOMATOLOGY

after diagnosis BBS I - clinical examination in a sequence of 2 hours

II. after diagnosis BBS II - EEG recording

III. first symptoms of brain death (clinic & EEG)
clinical examination & EEG all 6 hours

EXCEPTIONS

EXOGENOUS HYPOTHERMIA BELOW 35° C

INTOXICATION

SUBARACHNOIDAL HEMORRHAGE

NOT CERTAIN ETIOLOGY



PROLONGATION
OF
INVESTIGATIONS

Other possibilities to assure the diagnosis are adequate testing of apnea, electrophysiological investigations (MEP, BAEP, SSEP), monitoring of the intracranial pressure, angiography or Tc99 isotope angiography.

Ethical but also the medical consequences are compelling to fixed brain death definition. Given the above mentioned principles of brain death the physician uncompromisingly has to stick to these criteria. To continue life support measures in patients with proven brain death seems to be degrading. In all cases but especially if transplantation is contemplated, the diagnosis of brain death should be affirmed by

- three physicians not directly connected with the transplantation -
1. the physician of the intensive care (in most cases an anaesthesiologist)
 2. a neurologist for clinical evaluation
 3. a neurologist as specialist for the EEG evaluation.

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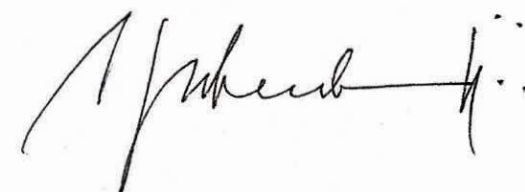
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