

# Brain Death

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## Key Words

Brain Death;  
Diagnosis of Brain Stem Death.

The problem of determining death has been fascinating humanity through the ages. Specific criteria have been changing with development of medical science and practice.<sup>1</sup>

In the eighteenth century, for example, the only certain sign of death was putrefaction! Until quite recently death was determined by the cessation of heart-beat and respiration. The invention of the mechanical lung ventilator, in the early fifties, radically changed the treatment of critically-ill patients. In cases of brain-damaged patients this only served to prolong the process of dying and to complicate the medico-legal definition of death. Most medical and legal authorities today identify death with brain death, in particular brain stem death.<sup>2</sup> Damage to cerebral cortex caused by hypoxia after cardiac arrest make the patient unconscious and unresponsive, but leaves the brain stem reflexes intact. This clinical condition is referred to as "appalic syndrome", "social death", or "persistent vegetative state".

It is also evident that co-ordinated reflex activity in the spinal cord can persist long after all the brain stem reflexes have disappeared.

For these reasons the modern concept of brain death focuses on the brain stem. Jennett's (1981)<sup>1</sup> concept "if the brain stem is dead, the brain is dead, and if the brain is dead, the person is dead" would seem to gain widespread acceptance.

There is accumulated evidence that it is the death of the brain stem which indicates that the process of death has passed the point of irreversibility.<sup>2</sup>

## Causes, circumstances and frequency of brain death

In the following three groups of patients extreme care should be taken before brain death can be safely

declared: 1. Patients poisoned by barbiturates, sedatives, narcotics, carbon monoxide, alcohol, and other CNS depressants; 2. patients with acute CNS infection and other intracranial conditions, such as tumour or abscess; 3. infants and small children.

Such patients can frequently recover following prolonged and appropriate treatment. Caution is also required to exclude other reversible causes of brain stem depression such as hypothermia and gross metabolic imbalance.

About 4000 diagnoses of brain death are made annually in Britain.<sup>3,4</sup> More than half of all cases of brain death are a result of head injury with obvious structural brain damage. Spontaneous intracranial haemorrhage accounts for another one-third of cases. The remaining cases are usually patients in whom treatment for other intracranial conditions such as brain abscess or tumour, has failed or patients who have not responded to resuscitation after cardiac arrest or profound hypotension.

Occasionally, persons may be found unconscious with no witnesses able to explain what happened and in spite of an obvious head injury it is prudent to wait long enough to allow exclusion of drugs as the cause of brain stem depression.

## Criteria for diagnosis of brain death

According to the most recent, so-called "U.K. criteria", the time of death is when brain death is diagnosed, not some time later when the heart stops.

The Harvard criteria require absence of all reflex motor activity which implies death of the whole central nervous system: cortex, brain stem and spinal cord.

It is now agreed that permanent functional death of the brain stem constitutes brain death.<sup>2</sup> It implies that brain stem death would always be followed by cessation of the heart-beat. This concept does not redefine death but only changes the criteria by which death can be identified. The "U.K. criteria" have two

components: the pre-conditions and the tests. It has to be emphasized that only when the pre-conditions have been satisfied is it appropriate to apply the tests.<sup>1</sup>

The pre-conditions require that the patient is: 1. in coma; 2. apneic; 3. has suffered irremediable structural damage of the brain; and that 4. reversible causes of brain stem depression have been excluded.

The tests are aimed to determine if there is any evidence at all of residual or returning activity in the brain stem. The following reflex responses are tested: response of the pupils to light; of the eyelids to corneal touching; responses of the facial muscles to painful stimuli applied to the face and forehead; responses of the muscles of the throat to movement of the endotracheal tube; injecting 20 ml of ice-cold water into the external meatus of the ears and observing for eye movements. If any of these responses is present, than the brain stem is still functioning.

The most important test, however, is to confirm that there is no return of respiratory drive from the brain stem. The patient is disconnected from the ventilator for long enough i.e. from 3 to 10 minutes, to ensure a threshold value of Pa CO<sub>2</sub> (c 8 kPa), whilst maintaining diffusion oxygenation to avoid hypoxaemia. This is the apnoea test. An important safeguard against a false-positive diagnosis is to permit sufficient time to elapse.

Provided enough time has passed to satisfy the pre-conditions, the tests can be repeated after an interval of 30 min. Important safeguards include also, the two following tests: angiography and monitoring the electrical activity of the cerebral cortex by EEG. If absence of blood flow to the brain for 15 min can be recorded it is considered that the brain is dead, but because of the equipment and expertise involved, angiography is regarded as impractical test for general use. The E.E.G. assesses function in the cerebral cortex whilst brain death depends on loss of function in the brain stem. There are quite a number of reports of patients recovering from deep coma whose E.E.G was at one stage iso-electric<sup>4</sup>. This is the strongest argument against its use and "U.K. criteria" explicitly state that E.E.G. is not needed for the diagnosis of brain death.

There are another three tests for diagnosis of brain death which are worth mentioning.

Failure to increase heart rate by more than five per minute following 1 mg atropine intravenously is compatible with brain stem death, as it shows that the

Vagus nuclei in the brain stem are not functioning. This is called the atropine test.<sup>5</sup>

Another simple and useful test is the so-called ophthalmic test.<sup>6</sup> 10% solution of fluorescein hydrochloride is injected in the cephalic vein of the arm and its appearance in the retina is noted. The normal arm-retina time is 10-15 sec. Delay of more than one minute in appearance of the fluorescein on fundoscopy indicate absence of blood flow in the brain. And finally, it has been suggested that the endogenous opiate system could be responsible for the breakdown in sympathetic discharge which may occur in brain death.<sup>7</sup> One of the authors (N.B.) has used and proposed the so-called naloxone test<sup>8</sup> for diagnosis of brain death, in order to determine whether the nociceptive system, which is a function of the brain stem has retained any activity.

In summary, the brain is the ultimate site of the human personality. It is also the target of resuscitation and various treatment modalities. Our main concern is the false-positive diagnosis of brain death in a patient who might survive with continuing mechanical ventilation and medical care.

In these controversy-laden and litigious times, it is prudent that physicians who are involved in making the diagnosis of brain death familiarize themselves with the material pertinent with the subject.

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