SPECIAL ARTICLE

CLINICAL ASPECTS OF BRAIN DEATH

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INTRODUCTION

The concept that death of the brain is death of the individual is an inescapable logic accepted by the medical profession and most of the public. If death is the permanent cessation of function then only brain death fits this definition. The certainty of death following stoppage of the heart beat is valid only in so far as the brain will die within minutes of circulatory arrest With present day resuscitation methods, the heart can often be restarted and the circulation artificially maintained by machine. During open heart surgery, the heart is routinely stopped for hours and circulation is maintained by cardio-pulmonary bypass. Similarly ventilation can be maintained for days and weeks with machines. The major impetus for the concept of brain death has been the need for transplantation of organs. Organs from a beating heart donor have much better chances of survival in the recipient than those after circulatory arrest. Doctors also need to know when the brain is dead so that relatives may be spared the anxiety and more attention may be given to those still alive. The shortage of beds, doctors and nurses in the intensive care ward means that only patients likely to survive will be admitted. Having a brain dead patient in the intensive care unit may cost the life of another patient.

WHAT IS BRAIN DEATH?

Brain death may be defined in one of two ways.
A) Anatomical death
B) Loss of all brain function

There are major difficulties in defining death only in anatomical terms i.e. pathological changes. The brain may be normal in appearance and on light microscopy when death occurs soon after cardiac arrest. And yet the brain is obviously dead. In one study (1) 10% of autopsies on brain dead patients had brains that were grossly normal. Permanent loss of brain function is an acceptable definition. However there are practical difficulties. It is impossible to test for all brain functions in a deeply comatose patient. (For instance, smell, coordination, vision and some higher cerebral functions can only be tested adequately in a conscious patient.) To avoid these difficulties most current criteria have emphasized the absence of brain stem function. For instance, The U.K. Colleges (2) have defined brain death as being constituted by death of the brain stem. I think it is essential that we understand and accept this definition. Death of the brain stem inevitably leads to cardiac standstill and total death. It makes no practical difference if some brain cells clusters are still alive at the time the brain stem is dead. Death is a gradual process. Total cellular death does not occur all at once. If we insist on total death then there is no way of determining this with 100% certainly. This concept of brain stem death however has

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yet to be fully accepted by the medical profession and the public. This became obvious following the BBC's panorama programme last year. This programme suggested that British criteria of brain death are not strict enough and that kidneys are possibly being removed from donors who would otherwise have lived. Three cases from the USA were illustrated — a man in coma after cardiac arrest, a woman with drug overdose and a man with severe injures — all of whom were thought to be brain dead and had recovered. Examination of the circumstances revealed the fact that none of the 3 patients met with the criteria of brain death as specified in U.K. Thus none of them would have been considered brain dead in Britain. The main criticism of the British criteria however relate to EEG. According to British criteria confirmatory investigations are not necessary, and yet US Collaborative Study had shown that some patients with apnoeic coma had EEG activity. The inference is that if EEG activity is present, then the cerebral hemispheres are not completely dead. I think that wether EEG activity is present or not is irrelevant if one accepts the concept of brain stem death. Whilst absence of EEG activity may be essential for total brain death, it does not contribute to the diagnosis of brain stem death.

There are other types of severe brain damage which can lead to prolonged unresponsiveness. Such patients are not brain dead however. Their brain stem is intact. The Quinlan case is such an example. Another type is the lock-in syndrome where as a result of massive destruction of the ventral portion of the pons, the patient is totally paralysed except for vertical eye movements and breathing. Such cases should be clearly distinguished from brain death.

WHAT ARE THE CRITERIA FOR BRAIN DEATH?

The first formal criteria was published in 1968 by the Ad Hoc Committee of the Harvard Medical School (3). Since then there has been modifications which have led to presently over 30 published sets of criteria in the diagnosis of brain death (4). It is important to appreciate that the criteria will continue to undergo modifications as medical opinion evolves. Any legal definition of brain death must take this into account. All the different sets of criteria have a common core made up of four parts (5).

A) There must be irremediable structural damage. The emphasis is on structural not functional or metabolic. In most cases the brain must have been destroyed by tumour, trauma or intracranial haemorrhage. The diagnosis of the disorder that can lead to brain death should have been fully established. The other key word is irremediable which implies that all therapeutic efforts have been exhausted. The first precaution is extremely important.
B) The exclusion of reversible causes of unresponsiveness. This follows from the first part and means exclusion of drug intoxication (including neuro-muscular blocking agent) and metabolic disease (including hypoxia and hypotension), primary hypothermia. There will be some cases where this will be in doubt.
C) Clinical tests to demonstrate absence of brain stem function and;
D) Confirmatory investigations

The clinical tests used in different countries vary slightly in operational detail. Basically they demonstrate absence of brain stem function and include the apnoea test. It is likely that the criteria which will eventually be adopted will be based on the UK criteria with certain modifications. Diagnostic tests for absence of brain stem function may be carried out no sooner than 6 hours after the onset of coma and apnoea. When the cause is cerebral anoxia or severe shock or embolism, then a much longer period of observation may be necessary. In most instances the tests have to be repeated no sooner than 2 hours after the first. These are:

A) Loss of all responsiveness to the environment. There is no response to externally applied stimuli, no response to commands and no phonation.
B) No spontaneous muscular movements, no postural activity or shivering (in the absence of muscle relaxants).
C) Pupils fixed to light. A bright light should be used. It is not necessary for pupils to be dilated.
D) Corneal reflexes absent. Use a stronger stimulus than is customary, such as the tie string of a sterile surgical mask. It has been suggested that corneal sensitivity decreased markedly with prolonged eye closure of 60 hours or more (6).
E) No motor responses within the cranial nerve distribution can be elicited by adequate stimulation of any somatic area.
F) No gag reflex or reflex response to bronchial stimulation by a suction catheter passed down the trachea.
G) No vestibulo-ocular reflexes to stimulation of the ears with 20 mls of ice cold water.
H) No respiration occur when the patient is disconnected from the ventilation for long enough to ensure the pCO2 rises above the threshold for stimulation of respiration (pCO2 50 torr) Hypoxia during disconnection is prevented by delivering 02 at 6 1/min into the trachea. If there is history suggestive of dependence on a hypoxic stimulus or ventilation (eg a chronic obstructive lung disease patient) the paO2 must be less than 50 torr. It should be noted that spinal reflexes can persist or return after an initial absence in brain dead patients, relatives should be told this to avoid any misunderstanding.

Confirmatory Tests

It is generally accepted that once the clinical criteria are satisfied without doubt, then confirmatory investigations are not essential. However it may be advisable to have some documentary proof when there are medico-legal implications such as may happen in organ transplantation or when the relatives are litigation conscious. An EEG which shows electrocerebral silence — defined as absence of cerebral electrical activity above 2 uv — and properly recorded provides easily visible and acceptable evidence to the public and the courts (7). An even more reliable method, one which has no false positives and applicable to brain death whatever the cause is demonstration of absence of intracranial circulation. Thus if bilateral carotid angiography shows no intracranial circulation then the brain is dead. The problem about this method is the transportation of patients to the neuroradiology facility and the possible harmful effects of injecting contrast media. The current view is that if there is any doubt either do not diagnose brain death or confirm with EEG or angiography in order to be trebly sure.

The clinical tests should be carried out by two physicians of at least consultant status and familiar with the management of such patients. They must not be part of the transplant team. Once the diagnosis is established, then death should be certified. It is only after this has been done that the patient is disconnected from the ventilator.

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