



Consensus Statement

On

Brain Death

2003



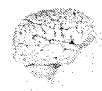
Ministry of Health
Malaysia



Academy of Medicine
of Malaysia



MALAYSIAN SOCIETY OF NEUROSCIENCES
persatuan neurosains malaysia



◀◀ Foreword ▶▶

A committee was formed in 2002 to review the Consensus Statement on Brain Death published in 1993. This committee comprised of senior clinicians in fields involved in the diagnosis of brain death and many of them were also members of the initial committee in 1993.

As done by the previous committee, extensive literature review was undertaken to look at the current standards and contemporary practice. We are happy to report that the practice has not differed significantly. The basic concept and understanding have not changed. The cardinal features required in the diagnosis of brain death and the clinical testing have remained the same. Interestingly too and also of significance is that there have been no landmark clinical studies on brain death published within the review period. Countries that have statutes on brain death have also not made amendments to what have been promulgated.

The revised guidelines on brain death only made minor changes to the original document without altering the concept and core principles. We have expanded some areas to give more details and clarity, namely in clinical testing, requirements in children, influence of drugs and ancillary tests. We hope this information will be useful to a wide spectrum of clinicians. In conclusion, it is important to emphasise that the diagnosis of brain death remains a clinical one with strict adherence to the inclusion criteria, exclusion criteria and clinical testing.



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(Chairman)

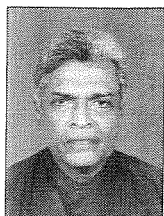
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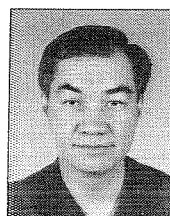
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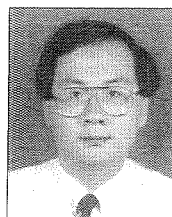
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Consensus Statement On Brain Death

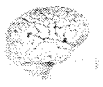
1. INTRODUCTION

The Brain Death Committee was formed in late 1992 to make recommendations regarding brain death and prepare guidelines for use in the country. Members comprised of specialists in relevant fields and representatives of medical organisations. The committee submitted its report to the Director General of Health and the Master of the Academy of Medicine in January 1993. The guidelines were then circulated to all major government and private hospitals and the three medical faculties then in existence. Members of the committee also gave lectures and explained the guidelines on brain death nationwide. The concept of brain death was accepted by the medical fraternity at a consensus meeting organised by the Ministry of Health and the Academy of Medicine on 12th December 1993.

2. BACKGROUND

Traditionally, death is recognised by the permanent cessation of the cardiovascular and respiratory functions. Until a few decades ago, this had served well in all situations. However medical knowledge has advanced, enabling these two vital functions to be supported and taken over by drugs and machines. The traditional definition of death will be inappropriate in such situations, and a different method to ascertain death is therefore required.

In general terms, death is the permanent cessation of the coordinated function of the organism as a whole.^{1,2} The use of circulation and respiration as markers of death is valid because permanent cessation of these two functions will lead to the inevitable permanent loss of yet another vital function, i.e. the function of the brain. In fact, the organ that determines whether the organism functions as a whole or not, is the brain.³ It receives stimuli which it processes, integrates and responds. As opposed to the functions of the heart and lung, these functions cannot be taken over by machines. The brain is also the centre for respiration, vasomotor, neural, hormonal and neurotransmitter control.⁴ It is therefore the ultimate organ that determines life and death.³



3. NEED FOR BRAIN DEATH CONCEPT

Brain death is a term that simply means that a person is recognised as dead, based on the examination of the nervous system. This method of ascertaining death is only limited to patients in the Intensive Care Units (ICUs) who are deeply unconscious and whose cardiopulmonary functions are supported by machines. It accounts for less than 1% of all deaths.⁵ The certification is only done by doctors experienced in the diagnosis, and strict guidelines are used. The reasons for the need to recognise brain death can be divided into:

- Ethical
- Human
- Intellectual
- Utilitarian

3.1. Ethical

Brain death is a definite clinical state. Adults with brain death will develop asystole within a week, regardless of what treatments are given.^{6,7} Magnetic resonance imaging (MRI) of the brain shows diffuse swelling with tentorial and foraminal herniations⁸ while various angiographic methods show absent blood flow.⁸⁻¹¹ In over 2,000 well documented cases of brain death, nobody has survived.² At postmortem, there is widespread necrosis and the brain hemispheres and brainstem are swollen and soft, with fragments of brain lodged in the spinal cord¹²⁻¹⁵ a situation totally incompatible with life.

It is therefore a matter of good medical practice to recognise brain death.¹⁶ In an era of rising medical cost, private health care and insurance, non-recognition either by ignorance or choice can be construed as unethical.

3.2. Human

Every man has a right to dignity and respect at death, and the pronouncement of death should not be unduly delayed. To continue ventilating the body whose brain is dead and undergoing liquefaction is an affront to this dignity.¹⁷ The heart may take up to a week to stop^{6,7}, and during this time, the family waits in immense distress for

the inevitable. Some may in fact be cruelly persuaded by the earnest attention of medical staff to believe the patient may still survive!

3.3 Intellectual

Certification of death by doctors has always been by brain death.¹ To start with, the patient is always unreceptive, unresponsive and not moving (*but these are taken for granted*). The doctor examines the pulse, heart, respiration and may even do an ECG. Convinced of the absence of these two vital functions, he would then examine the pupils. Fixed unreactive pupils (*usually dilated*) confirm his diagnosis. Thus, some of the basic criteria for brain death have always been used by doctors to certify death. The absence of a heart beat and respiration is actually a marker heralding the inevitable irreversible damage of the brain which is the ultimate organ that determines death. It is therefore a matter of intellectual progression to recognise brain death.^{1,3}

3.4. Utilitarian

Treating patients in ICU is costly. The number of ICU beds and ventilators is also limited. It is morally and economically unjustifiable to keep ventilating a brain dead patient in ICU, thereby depriving patients with better prognosis from availing themselves of these facilities.^{17,18}

Medical progress has also made possible organ transplantation which is now an accepted mode of treatment for chronic organ failure. This is a major problem worldwide and to which the profession has a duty to respond in the most appropriate manner. However, organ survival is only good when taken from a brain dead patient prior to circulatory collapse. Acceptance of brain death therefore, will be an important step for cadaveric organ transplant programmes.¹⁹⁻²¹

4. CRITERIA OF DIAGNOSIS

The criteria for diagnosis of brain death have evolved for more than 30 years.^{2,7,16,17,22-30} Over this period refinements have been made. However, the basic core features have remained. The crux of the criteria is deciding what needs to be present or demonstrated when the brain is dead. These refinements include additional safeguards and exclusions which preclude a proportion of apnoeic coma patients in the ICU, while at



the same time enabling an earlier diagnosis. The practices in various countries are also fairly similar, with minor variations only in details.^{7,16,17,23-31} This concurrence is indeed remarkable, bearing testimony to a definite clinical entity. These criteria are all very stringent so as not to allow any possible error of diagnosis.

Based on these contemporary medical practices and the situation in the country, the committee drew up the guidelines on brain death. Essentially, it is a clinical diagnosis, but brain death can only be certified when the diagnosis of irreversible brain damage is absolutely certain, and metabolic factors are not the cause of the state the patient is in. The patient must be apnoeic and properly ventilated, be totally unresponsive and unresponsive and the brain stem reflexes are absent. The loss of the inherent ability to breathe is further ascertained by the apnoea test. If there is any doubt in the diagnosis, the patient shall not be examined.

5. RECOMMENDATIONS

- 5.1 That the concept and entity of brain death be recognised and accepted; and that brain death means death.
- 5.2 The diagnosis of brain death is a clinical one and no confirmatory test is necessary. The exception to this is only for children because of the greater ability of the child's brain to withstand damage.
- 5.3 Two specialists who are experienced in diagnosing brain death are qualified to certify.
- 5.4 Doctors involved in organ transplantation are not allowed to certify brain death.
- 5.5 Hospitals where brain death is being certified, shall have a committee that functions as a coordinating body and is responsible for general policies, training and accrediting staff, counseling and overseeing the facilities available.
- 5.6 The brain death guidelines shall be reviewed every 5-10 years to accommodate new knowledge and contemporary practice.

Brain Death Guidelines

1. DEFINITION

Brain death is a state when the function of the brain as a whole, including the brain stem, is irreversibly lost.

2. RECOGNITION AND ACCEPTANCE

A person certified to be brain dead is dead.

3 DIAGNOSIS OF BRAIN DEATH

3.1. Preconditions (*All to be fulfilled*)

3.1.1. Patient is in deep coma, apnoeic and on ventilator, for at least 12 hours.

3.1.2. Cause of coma fully established and sufficient to explain the status of the patient.

3.1.3. There is irremediable brain damage.

3.2. Exclusions

3.2.1. Coma due to metabolic or endocrine disturbance, drug intoxication and primary hypothermia (*defined as a core temperature of 32°C [90°F] or lower*).

3.2.2. Certain neurological disorders namely, Guillain-Barré syndrome and locked-in syndrome.

3.2.3. Coma of undetermined cause.

3.2.4. Preterm neonates.



3.3. Diagnostic Criteria (*All to be fulfilled; see appendix I*)

- 3.3.1. Deep coma, unresponsive and unreceptive, Glasgow coma score (GCS) 3/15.
- 3.3.2. Apnoea, confirmed by apnoea test.
- 3.3.3. Absent brain stem reflexes confirmed by the following tests:
 - 3.3.3.1 Pupillary light reflex
 - 3.3.3.2 Oculo-cephalic reflex
 - 3.3.3.3 Motor response in cranial nerve distribution
 - 3.3.3.4 Corneal reflex
 - 3.3.3.5 Vestibulo-ocular reflex (*Caloric Test*)
 - 3.3.3.6 Oro-pharyngeal reflex
 - 3.3.3.7 Tracheo-bronchial reflex

3.4. Assessment and certification

- 3.4.1 The assessment of brain death is to be carried out by two specialists. A repeat assessment and certification must be carried out at least 6 hours after the first, not necessarily by the same pair of specialists.
- 3.4.2 The brain death certification is for 2 tests to be done 6 hrs apart. The repeat test should still be performed regardless of whether the patient will or will not continue to be an organ donor.
- 3.4.3 The "Brain Death Certification" form is filled up by the first set of doctors (*Doctor A and B*) and completed by the 2nd set of doctors (*Doctors C and D*)
or
Doctors A and B if the same doctors are performing the repeat test. The time of death will then be declared by the doctors performing the repeat test.
- 3.4.4 The time of death is at the time of the 2nd testing. If for any reason, the 2nd test is unable to be carried out 6 hrs later, e.g. patient is unstable, then the time of death will be when the test is next repeated. Should the patient's heart stop before the repeat test, that will be taken as the time of death.

4. OTHER CONSIDERATIONS

- 4.1 During the period of observation, the patient shall remain deeply comatose with no respiratory effort, no abnormal posture or movements in cranial nerve distribution.
- 4.2 Patients who do not meet all the above criteria shall not be considered for brain death certification.
- 4.3 For children additional guidelines required are (*see appendix II*):
 - 4.3.1 The interval between 2 examinations is lengthened depending on the age of the child.
 - 4.3.2 An ancillary test (EEG) is recommended for those less than one year old.
 - 4.3.3 No recommendations are made for newborns or preterm infants.
- 4.4 Pitfalls in diagnosis may occur, especially if certain aspects of the clinical tests cannot be reliably performed (*or evaluated*) (*see appendix III*). Ancillary laboratory tests (*not usually mandatory*) may be useful in these situations and in certain instances where children are involved (*see appendix IV for details of tests*).

5. QUALIFICATION OF DOCTORS

- 5.1 Each hospital must have a subcommittee to appoint and review doctors authorised to certify brain death in that hospital.
- 5.2 Two specialists who are competent (*at least 3 years of postgraduate clinical experience and trained in brain death assessment*) in diagnosing brain death are qualified to certify brain death. They should preferably be anaesthesiologists, physicians, neurologists and neurosurgeons.

6. QUALIFICATION OF HOSPITALS

Brain death certification must only be done in areas of the hospital with full facilities for intensive cardiopulmonary care of comatose patients.



Appendix I

Assessment of brain death

Description:

Brain death is a CLINICAL DIAGNOSIS.

The standard for the determination of brain death is the clinical neurologic examination performed with precision, to ascertain the irreversibility of the loss of brain function, having established the proximate cause of coma.^{32, 35} All clinical tests described below are needed to declare brain death:

1. COMA OR UNRESPONSIVENESS

Confirm unresponsiveness and an absence of coordinated eye movement and motor activity, including decorticate and decerebrate posturing. Spontaneous motor activity (*excluding spinal reflexes*) and seizures must be absent. There is no motor response to pain in all extremities (*nailbed and supraorbital pressure*).

2. ABSENCE OF BRAINSTEM REFLEXES

Measures reflex pathways in the mesencephalon, pons and medulla oblongata (*in a rostral-to-caudal direction*).

2.1 Pupillary reflex

- No response to bright light in BOTH eyes.
- Pupillary size may be mid-position (4-6 mm) to dilated (9 mm)
- Round, oval or irregularly shaped pupils are compatible with brain death.

Pitfalls:

Many drugs can influence pupil size but light response remains intact. Pre-existing anatomic abnormalities of the iris or effects of previous surgery should be excluded.

Topical ocular installation of drugs & trauma to the cornea or bulbus oculi may cause abnormalities in pupil size and can produce non-reactive pupils.

2.2 Oculocephalic reflex - Doll's eye response

- Testing is done ONLY when no fracture or instability of the cervical spine is apparent and in patients with head injury, the cervical spine must be imaged to exclude potential fractures or instability.
- The oculocephalic response is elicited by fast, vigorous turning of the head from middle position to 90° on BOTH sides.
- Any eye movement must be absent.

Pitfalls:

After closed head injury or facial trauma, lid oedema and chemosis of the conjunctivae may restrict movement of the globe.

2.3 Corneal reflex

- To be tested with a cotton swab.
- No blinking response seen.

2.4 Facial motor response

- Pressure stimulus is applied to the supraorbital nerve, deep pressure on both condyles at the level of the temporo-mandibular joint or on the nail bed.
- No grimacing is seen.

2.5 Vestibulo-ocular reflex - Caloric test

- Test should not be performed if there is a perforated tympanic membrane.
- Caloric testing to be done with head elevated to 30° during irrigation of the tympanum on each side with 50 ml of ice water.
- Allow 1 minute after injection and at least 5 minutes between testing on each side.
- Tonic deviation of the eyes directed to the cold caloric stimulus is absent.

Pitfalls:

Clotted blood or cerumen may diminish the caloric response.

Basal fractures of the petrous bone abolishes the caloric response only unilaterally and may be identified by an ecchymotic mastoid process (Battle's sign).



Drugs (sedatives, aminoglycosides, tricyclic antidepressants, anticholinergics, antiepileptic drugs and chemotherapeutic agents) can diminish or completely abolish the caloric response

2.6 Oro-pharyngeal reflex

- The gag response tested by stimulation of the posterior pharynx should be absent.

Pitfalls:

In orally intubated patients, the gag response may be difficult to interpret.

2.7 Tracheo-bronchial reflex

- A suction catheter is passed down through the endotracheal tube to the level of the carina or beyond.
- Lack of cough response to bronchial suctioning should be demonstrated.

Pitfalls:

Severe facial and neck trauma may limit the interpretation of all brainstem reflexes.

3. APNOEA - CONFIRMED BY APNOEA TEST

3.1. Prerequisites

The patient must be in a stable cardiovascular and respiratory state.

3.2. Adjust ventilator to maintain Pa CO₂ at or around 40 mmHg.*

3.3. Pre-oxygenate with 100% O₂ for 10 minutes.

3.4. Disconnect from ventilator.

3.5. Deliver 100% O₂ via tracheal catheter at 6L/min.

3.6. Monitor O₂ saturation with pulse oximetry.

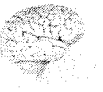
3.7. Measure Pa CO₂ after 5 minutes and again after approximately 8 minutes if Pa CO₂ has not exceeded 60 mmHg.

- 3.8. Re-connect to ventilator after test.
- 3.9. The disconnection of the ventilator shall not exceed 10 minutes at any one time.
- 3.10 The apnoea test is positive when there is no respiratory effort with a Pa CO₂ of \geq 60 mmHg.
- 3.11 If during apnoea testing, there is significant hypotension, marked desaturation or cardiac arrhythmias, immediately draw an arterial blood sample, re-connect to ventilator and analyse arterial blood gas results. Should the PaCO₂ not exceed 60 mmHg, the result is indeterminate. It is left to the discretion of the physician to decide whether to repeat the test or to depend on an ancillary test to finalise the clinical diagnosis of brain death.³³

** For patients with COPD, the baseline PaCO₂ may already be above 40 mmHg. The apnoea test is then considered positive if there is no respiratory effort at a PaCO₂ of 20 mmHg above the baseline PaCO₂.*

Pitfalls:

"Lazarus sign" - Spontaneous motor responses of the spinal origin may be seen.



Appendix II

Guidelines for children

There are some medical issues unique to children in determining brain death.³²⁻³⁴ It is generally assumed that the young child's brain may be more resilient to certain forms of injury, although this issue is controversial. The newborn is difficult to evaluate after perinatal insults. This relates to many factors including difficulties of clinical examination, determination of approximate cause of coma, and certainty of the validity of laboratory tests. These problems are accentuated in the premature newborn. However, after a period, currently suggested as the first seven days after the insult in a term newborn, the extent and reversibility of the injury can be determined by the history, physical examination and ancillary tests.

Hence, the guidelines are modified in two areas - the interval between two examinations is lengthened depending on the age of the child, and an ancillary test (EEG) is recommended for those less than one year old.

No recommendations are made for newborns or preterm infants.

Table 1. Age dependent observation period and ancillary tests

Age	Hours between 2 examinations	Recommended no. of EEGS
7 days - 2 months	48	2
2 months - 1 year	24	2*
> 1 year**	12	not needed

* *A repeat examination and EEG are not necessary if a concomitant cerebral radionuclide study demonstrates no visualisation of cerebral arteries.*

** *If hypoxic ischaemic encephalopathy is present, observation for at least 24 hours is recommended. This interval may be reduced if an EEG shows electrocerebral silence or a radionuclide study is negative for cerebral blood flow.*

Appendix III

Pitfalls in Diagnosis

- Severe facial trauma
- Pre-existing pupillary abnormalities
- Toxic levels of any sedative drugs, aminoglycosides, tricyclic antidepressants, anticonvulsants, chemotherapeutic drugs, neuromuscular blocking agents.
- Sleep apnoea or severe pulmonary disease resulting in chronic retention of carbon dioxide.

1. DRUGS

Presence of certain drugs, metabolites or poisons may confound the clinical determination of brain death.³⁶ Drug screens are useful, but not all drugs can be quantified. Furthermore, even if a drug has been identified, the critical threshold is often not known:

A reasonable approach is:

- if screening tests reveal traces of a drug below the therapeutic range, brain death can be declared.
- when the drug or poison cannot be quantified but is certain, the recommendation is to observe the patients for at least 4 times the excretion half-life, provided elimination of the drug or toxin is not interfered with, by other drugs or organ dysfunction.
- when the drug is not known but suspicion of its presence is high, observe the patients for 48 hours for a change in brainstem reflexes and motor response; if none are observed, perform a confirmatory/ ancillary test for brain death.



Common drugs that may confound the neurological examination in brain death

Drugs	Elimination T 1/2(hrs)	Therapeutic range
Lorazepam	10 - 20	0.1 - 0.3ug/ml
Midazolam	2 - 5	50 - 150ng/ml
Diazepam	40	0.2 - 0.8ug/ml
Carbamazepine	10 - 60	2 - 10ug/ml
Phenobarbitone	100	20 - 40ug/ml
Pentobarbitone**	10	1 - 5ug/ml
Thiopentone	10	6 - 35ug/ml
Morphine	2 - 3	70 - 450ng/ml
Amitriptyline	10 - 24	75 - 200ng/ml
Alcohol*	10ml/h	800 - 1,500mg/L

* Plasma t 1/2 may easily change as a result of interacting drugs and organ failure. Use legal alcohol limit for determination of brain death.

** When high dose barbiturates are used, depressed neuronal as well as decreased cerebral blood flow may occur. A confirmatory test using angiogram may be erroneously positive for absent flow. The clinical diagnosis for brain death should only be allowed, if serum pentobarbitone levels have decreased to levels in which it is highly improbable that brainstem function is depressed (In most laboratories usually less than 5ug/ml)



2. DRUG-INDUCED ACID BASE ABNORMALITIES

Profound changes in acid-base balance may point to certain intoxication, and may also indicate a potentially reversible medical illness or endocrine crisis. Determination of brain death should be deferred in the presence of severe acidosis or alkalosis.

Drugs that may induce acid-base abnormalities are as below:

- Metabolic acidosis:

- Acetaminophen

- Ethanol, methanol, ethylene glycol

- Salicylates

- Isoniazid

- Cyanide, cocaine, toluene

- Respiratory acidosis:

- Opiates

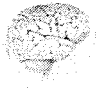
- Ethanol, other alcohols

- Barbiturates

- Anaesthetics

3. SPONTANEOUS AND REFLEX MOVEMENTS IN BRAIN DEATH

Spontaneous and reflex movements have been observed in patients with brain death. The most common are finger jerks. The undulating toe flexion sign (snapping the big toe leads to an undulating movement of the other toes), triple flexion response, persistent Babinski response, Lazarus sign, pronation - extension reflex, and facial myokymia can also occur. These movements are spinal in origin and generally occur during the apnoea test or are triggered during forceful flexion of the neck, rotation of the body, or by the ventilator, synchronous with pulmonary insufflation. They do not occur spontaneously. They do not preclude the diagnosis of brain death.



Appendix IV

Ancillary investigations in brain death

1. CONVENTIONAL ANGIOGRAPHY

- Absence of intracerebral filling of the intracranial arteries at the entry into the skull.³⁵⁻³⁸

2. TRANSCRANIAL DOPPLER ULTRASONOGRAPHY

- Absence of Doppler signals is not a diagnostic feature and may be due to poor insonation windows.⁴¹
- Small systolic peaks in early systole with retrograde (reverberating, oscillating) or absent flow during diastole.^{35,37,39-41}

3. NUCLEAR IMAGING - Technetium-99m hexamethylpropyleneamineoxime (HMPAO) brain scan or single photon emission computed tomography (SPECT) scan

- Absence of cerebral perfusion - "empty skull sign".^{35,37,42-44}

4. ELECTROENCEPHALOGRAPHY (EEG)

- No brain electrical activity during at least 30 minutes of recording.^{35,37,45,46}
- The EEG recording must adhere to technical specifications for brain death.

Brain Death Certification

1.0 IDENTIFICATION

NAME: _____ AGE: _____ SEX: _____
 NRIC: _____ RN: _____ RACE: _____
 DATE OF ADMISSION: _____ WARD: _____

2.0 DIAGNOSIS (INCLUDING CAUSE OF IRREMIABLE BRAIN DAMAGE):

3.0 PAST MEDICAL HISTORY (LIVER, RENAL, ENDOCRINE, ETC): _____

4.0 CT SCAN BRAIN DONE: YES NO

5.0 DATE AND TIME PATIENT PUT ON VENTILATOR: DATE: _____ DD/MM/YY
 TIME: _____

6.0 FIRST TEST

DATE: _____
 TIME: _____

SECOND TEST

DATE: _____
 TIME: _____

	DOCTOR A		DOCTOR B		DOCTOR A/C		DOCTOR B/D	
	YES	NO	YES	NO	YES	NO	YES	NO
7.0 ALL PRECONDITIONS HAVE BEEN FULFILLED	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
8.0 ALL EXCLUSIONS HAVE BEEN MADE	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
9.0 THE FOLLOWING SIGNS ARE ABSENT								
9.0.1 PUPILLARY LIGHT REFLEX	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
9.0.2 OCULO-CEPHALIC REFLEX	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
9.0.3 MOTOR RESPONSE IN CRANIAL NERVE DISTRIBUTION	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
9.0.4 CORNEAL REFLEX	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
9.0.5 VESTIBULO-OCCULAR REFLEX	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
9.0.6 ORO-PHARYNGEAL REFLEX	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
9.0.7 TRACHEO-BRONCHIAL REFLEX	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
10.0 APNOEA CONFIRMED BY APNOEA TEST	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
11.0 POSSIBILITY OF RECOVERY OF BRAIN FUNCTION EXCLUDED	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
12.0 LOSS OF BRAIN FUNCTION AS A WHOLE PERSISTS AFTER AN APPROPRIATE PERIOD OF OBSERVATION OF TRIAL OF THERAPY	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>

	DOCTOR A	DOCTOR B	DOCTOR A/C	DOCTOR B/D
NAME:	_____	_____	_____	_____
POSITION:	_____	_____	_____	_____
SIGNATURE:	_____	_____	_____	_____

BRAIN DEATH DECLARED:

DATE: _____ TIME: _____

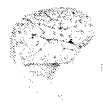
CONSULTANT IN CHARGE: _____



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