Diagnosis of Brain Death in adults.

Guidelines

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On behalf of Intensive Care Society of Ireland (ICSI)
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These Guidelines are based on our interpretation of the existing legal situation and ‘custom and practice’ in Ireland, on previously published Guidelines in Ireland and internationally, and on published literature in this area.

Introduction

The concept of ‘Brain Death’ is ubiquitous in medical and lay literature. It is a recognition that some brain injured patients maintained on artificial ventilation have complete and irreversible loss of brain function. Brain Death is now accepted as the legal equivalent of circulatory death, the usual criterion for certification that death has occurred. The historical evolution of the concept of Brain Death is outlined in Appendix 1.

The ability to certify Brain Death when there is irreversible cessation of brainstem function enables specialists working in Intensive Care to withdraw futile treatment (e.g. mechanical ventilation), on humanitarian, ethical and (coincidentally) utilitarian grounds.

There is no statutory law on this area in the Republic of Ireland. Pending enactment of legislation, compliance with the recommendations of these ICSI Guidelines, the 1988 Irish Memorandum on Brain Death (1) and the 2016 ICSI Guidelines (2), (which are all in accordance with each other), has the legal status of ‘custom and practice’ in the Republic of Ireland. There is a widespread consensus on the concept and diagnostic criteria for Brain Death (3-8). These Irish Guidelines are aligned with international practice.

There are differences in terminology between the United Kingdom (which specifies brainstem death as the criterion for determining death) and the United States, Australia and other countries which require ‘death of the whole brain’. In practice the clinical tests to ascertain death are the same. The concept of “Death by neurological criteria” has emerged, which encompasses both approaches. For this review we will use the term Brain Death which is more universally recognised.

There is no documented case of an adult who has fulfilled the preconditions and the clinical criteria for Brain Death subsequently regaining consciousness (6,8).

Clinical Diagnosis of Brain Death

Establishing a diagnosis of Brain Death in a comatose patient should proceed with certain principles in mind; (A) establish that coma is due to a condition which can cause irreversible brain damage, (B) exclude reversible factors, (C) demonstrate the loss of all cranial nerve and brainstem responses which can be tested in coma.

A diagnosis of Brain Death based on clinical tests should not be made unless the criteria in A) and B) have first been fulfilled.

(A) Diagnosis of a condition which can cause irreversible brain damage.
A number of conditions which can cause the loss of brainstem reflexes are reversible e.g. Guillain Barré syndrome, brainstem encephalitis, poisoning etc and these must be excluded. If there is any doubt about the primary diagnosis, Brain Death cannot be diagnosed clinically.
The underlying neurological condition which caused severe brain injury must be diagnosed definitively and this must be clearly documented. There must be evidence of severe structural brain damage from CT scan, angiography or MRI scan. However radiological evidence of severe brain damage does not obviate the need to search for confounders and to test brainstem reflexes.

(B) **Exclusion of reversible causes of coma**

(i) **Sedative drugs:**

If sedative drugs have been used, adequate time must be allowed for residual effects to have worn off. If sedatives have been used for a prolonged period or in large doses (e.g. with head injury) it may be difficult to decide whether these are still contributing to the coma. Particular difficulty arises with highly lipid-soluble drugs like thiopentone but there are also concerns with other drugs like midazolam or morphine if these are used in large doses for prolonged periods. If these drugs have been administered for a number of days, the plasma half-lives are likely to be considerably extended (i.e. the concept of context-sensitive half-life). In addition these drugs produce metabolites with sedative effects.

The presence of sedative drugs may be excluded by history, examination of the medication sheet and a drug screen. Consideration should be given to:

- The doses and the duration of infusion of sedative agents,
- Altered pharmacokinetics with very high doses,
- The possibility of drug metabolites with sedative effects
- The effects of hypothermia. Therapeutic hypothermia significantly reduces sedative drug clearance and these effects persist after rewarming (9-11)
- The effects of renal or liver dysfunction on elimination of these agents.

If administration of the antidotes naloxone or flumazenil led to a change in neurological examination, this would exclude a diagnosis of Brain Death. However lack of response to the administration of antidotes does not exclude an effect from sedative agents. If neuromuscular blocking agents have been used, a nerve stimulator will confirm full recovery.

Measurement of plasma concentrations of drugs should reveal drug concentrations below the therapeutic range. The median concentration of thiopentone permitting motor response is 12 mg/L (12) but there is considerable individual variation. ANZICS guidelines recommend thiopentone levels should be < 10mg/L before brainstem tests (6).

The exclusion of sedative agents as a contributing cause of coma requires considerable knowledge and experience. Ultimately clinicians must use their knowledge and judgement to decide. It may be helpful to get advice from an experienced Consultant in Intensive Care Medicine. If there is any uncertainty regarding a possible confounding effect of a sedative drug then the preconditions are not met and a diagnosis of Brain Death cannot be made by clinical tests alone.

(ii) **Poisoning and drug overdose:**

Toxins, poisons and drug overdose (e.g. barbiturates, baclofen, amitriptyline, etc) may cause coma; the possibility of this should be apparent from the history. Obtain a toxicology screen where appropriate (although this may be positive for opiates and benzodiazepines if these have been administered for therapeutic reasons). The blood alcohol level should be below the legal limit for driving before testing for Brain Death.

(iii) **Hypothermia:** core body temperature should be more than 35°C when clinical assessment of brain stem responses is carried out.
Therapeutic hypothermia can lead to delayed neurological recovery and prolonged metabolism of sedative agents. Targeted temperature management aiming for a temperature of 36°C after cardiac arrest should not delay brainstem testing unless temperature has been < 35°C for more than 6 hours; this requires a delay of 24 hours after return to normothermia before clinical tests for Brain Death (6).

If therapeutic hypothermia to 32-34°C has been used, there is no clear guidance on how long brain stem tests should be delayed after the return of normothermia. Return of motor responses may be delayed for up to 5 days after therapeutic hypothermia to 32-34°C (9-11) which suggests that brain stem testing should be delayed for 5 days also, especially if large doses of sedative agents have been used. Multimodal assessment with clinical examination, EEG and MRI may allow earlier brainstem testing or demonstration of absence of cerebral blood flow may be used to support a diagnosis of Brain Death by clinical testing (see ‘Ancillary tests ‘below).

(iv) Metabolic or endocrine causes that may contribute to coma must be excluded. Hypothyroidism, panhypopituitarism, adrenal dysfunction, renal failure and hepatic failure can lead to a profound decrease in the level of consciousness.

The commonest metabolic abnormality in brain dead patients is hypernatraemia, often related to diabetes insipidus. Serum sodium should not be grossly abnormal; we recommend 130-155 mmol/L as the acceptable limits for brain stem testing. Similarly blood glucose, phosphate and magnesium levels should not be grossly deranged.

(v) Severe hypotension precludes testing of brain stem reflexes. Blood pressure should be greater than 90mmHg systolic (MAP > 60mmHg) for brain stem tests. Infusion of fluid and vasoactive drugs may be needed to maintain blood pressure.

The diagnosis of Brain Death sometimes requires complex clinical judgement. In complicated cases and especially if a practitioner does not undertake Brain Death testing often, we suggest getting advice from a Consultant in Intensive Care Medicine who undertakes the diagnosis of Brain Death on a regular basis.

(C) Formal clinical testing for Brainstem reflexes

Observation period.
The first formal examination can be undertaken when the patient fulfils the pre-conditions in A) and B) and cranial nerve and brainstem responses have been absent for at least 4 hours (i.e. pupils unreactive, no cough and no apparent respiratory effort). If the cause of coma is cardio-respiratory arrest, brainstem testing should not be undertaken until at least 24 hours after the return of circulation.

Neurological responses

(1) Absent motor responses Cranial Nerves V, VII. Look for a motor response of face or limbs in response to painful stimulation within the trigeminal nerve distribution. Absence of response to a painful stimulus applied peripherally could be the result of a high cervical injury, thus stimulation must always be performed within the distribution of the trigeminal nerve. Stimulation can be applied by pressure on the supraorbital notch or at the level of the tempero-mandibular joint.

(2) No pupillary response to light. Cranial nerves II, III. A history of pre-existing abnormalities of the pupil or previous surgery to the eye (e.g. iridectomy) may influence interpretation of this test. Examine each eye with dimmed light in the room and use a strong light. The normal response is brisk constriction of the pupil. Round, oval or irregularly shaped pupils are compatible with Brain Death. In most brain dead patients pupils are in the mid position (4-6mm).
(3) **Corneal reflex.** Cranial nerve V, VII. Touch the cornea with a wisp of cotton wool and look for blinking. If this fails to elicit a response a stronger stimulus is applied with for example, a sterile throat swab and firm direct pressure. Blinking of the eyelids is the normal response and both eyelids must be observed.

(4) **Oculovestibular reflex (caloric testing).** Cranial nerves VIII, VI, III. Before testing this reflex, both ears are inspected using an auroscope to confirm that the tympanic membrane is intact and the external auditory canal is not obstructed by wax or other material. A fractured base of skull resulting in blood, CSF or brain tissue in the external auditory canal is a contra-indication to performing the test on that ear. The patient’s head is placed in the midline and elevated 30° from the supine position. This ensures that the lateral semi-circular canal is vertical, maximising the response. A soft catheter is introduced into the external auditory canal for gentle, slow irrigation with at least 50ml of iced water while the eyes are held open by an assistant. The eyes should be observed for a minute after irrigation is completed.

In an unconscious patient, an intact oculovestibular reflex causes slow deviation of the eyes towards the irrigated ear. Any movement of one or both eyes, whether conjugate or not, excludes a diagnosis of Brain Death. When the reflex is absent the eyes remain fixed.

(5) **Oculocephalic reflex (Doll’s eye phenomenon).** Cranial nerves VIII, VI, III. The examiner holds the patient’s eyes open and the head is turned suddenly from the middle position to the side. When the reflex is intact the eyes turn contrary to the side of head movement as if lagging behind. The reflex is absent when the eyes move with the head and do not move within the orbit.

If Test (4) above can be performed this test may be omitted as both tests assess the same neurological pathway. This test must not be performed in patients with an unstable cervical spine injury.

(6) **Pharyngeal (gag) reflex.** Cranial nerves IX, X. A tongue depressor is used to stimulate each side of the oropharynx and the patient observed with a torch for any pharyngeal or palatal movement.

(7) **Laryngeal (cough) reflex.** Cranial nerves IX, X. A suction catheter is introduced into the endotracheal or tracheostomy tube far enough to stimulate the carina. The patient is observed for any cough response or movement of the chest or diaphragm.

(8) **Apnoea testing.** This test is an essential part of the diagnosis of Brain Death. It should be undertaken when all other brain-stem reflexes are found to be absent.

The three components of the test are:

- Disconnection from mechanical ventilation for long enough to allow arterial CO₂ tension to reach a critical point (with a resultant respiratory acidaemia).
- Prevention of hypoxaemia during this period.
- Absence of spontaneous respiratory efforts during this period.

Ventilate the patient with 100% oxygen before the test to ensure maximal oxygenation. Check the patient’s arterial blood gases. If the PaCO₂ is outside normal limits, adjust the ventilator until a normal PaCO₂ for that patient (normally 4.8-5.8 kPa) is achieved. Disconnect the ventilator (as connection to the ventilator can lead to artefactual detection of breathing by the ventilator due to the cardiac impulse). Deliver oxygen at 7l/min via C-circuit with the valve fully open (a Valsalva effect may occur if the expiratory valve is fully closed). An appropriate CPAP valve can be attached to keep the airways open and optimise oxygenation. A CPAP valve may also prevent atelectasis and improve the condition of the lungs for subsequent transplantation.

Delivering oxygen via a narrow suction catheter inserted into the tracheal tube is no longer favoured due to the loss of positive airways pressure associated with this technique and conversely the risk of barotrauma or Valsalva effect if airway pressures are too high (13).
Inspection of the reservoir bag of the breathing circuit will enable monitoring of respiration; visual inspection for chest and abdominal movement is also required. Visual inspection of the chest and abdomen may show minimal movement synchronous with heart beat. Arterial blood gases are checked until the PaCO2 increases to 8.0 kPa or higher and associated acidaemia has developed (pH<7.30). If the patient has a history of chronic respiratory disease, allowance must be made for decreased sensitivity to a high PaCO2. Occasionally it takes more than 10 minutes for the required changes in PaCO2 and pH.

The apnoea test may lead to instability in the patient’s condition and cardiac arrests have occurred during this procedure. Maintain oxygen saturation levels within the normal range with application of CPAP or delivery of 1 - 2 breaths if patient desaturates. If hypotension occurs, vasopressors may need to be increased. If, despite hypercarbia and acidaemia, there is no attempt at spontaneous respiration, the test is consistent with Brain Death.

It may not be possible to undertake apnoea testing in patients with a high cervical cord injury or in patients with poor ‘gas exchange’. In such patients, determination of Brain Death by clinical tests alone will not be possible and additional ancillary tests (e.g. cerebral angiography) may be needed to confirm Brain Death. (see item (D) below).

More detailed descriptions of the clinical tests of brainstem function are available if required (6)

**Observations Compatible with a Diagnosis of Brain Death:**

- Spinal reflexes. (14-16)
  These movements result from spinal cord activity which can persist despite Brain Death. Spinal reflexes have been consistently documented in patients who fulfil clinical criteria for Brain Death confirmed by absent intracranial blood flow.
  Movements may be spontaneous or elicited by stimulation and include the following:
  - extension or flexion of the arms,
  - leg movements,
  - head rotation
  - movement of the body to sit up to 45° (Lazarus sign).
- Sweating
- Blushing
- Tachycardia

**Observations incompatible with a Diagnosis of Brain Death:**

- Decerebrate or decorticate posturing
- Facial movement
- Seizures

**Declaration of death.**

It is standard practice for a second clinician to repeat the entire process described in (A), (B) and (C) above. A ‘reasonable’ period of time should intervene between the two sets of tests (although no specific minimum time period has been recommended).

The apnoea test is the final test to be done. At the end of the second apnoea test, if there is no ventilatory response and the preconditions for a clinical diagnosis of Brain Death have been satisfied, the patient is declared dead and the time and date noted. This is the official time of death.

Many ICUs use a checklist to be completed by the two clinicians undertaking tests. An example of such a list is appended (Appendix 2).
Who should do the clinical tests?

Two sets of tests should be undertaken; one by a doctor who is a Consultant, the other by a doctor who has been fully registered for at least 5 years. Both should be engaged in acute hospital care and have expertise in testing for Brain Death. If organ donation is being considered, the doctors certifying Brain Death should not be involved in the transplantation procedure.

Other staff may accompany those doing the tests for training purposes and to gain an insight into the concept of Brain Death.

Documentation

The findings of each of the formal sets of tests should be fully documented, ideally in a checklist form and summarised in the medical records. If both sets of tests show no evidence of brain-stem function and the preconditions for a clinical diagnosis of Brain Death are satisfied, Brain Death is diagnosed and documented.

When a diagnosis of Brain Death is not possible using clinical criteria alone:

The internationally accepted standard for diagnosis of Brain Death is by clinical testing. Despite the existence of formal Guidelines for tests for Brain Death, issues requiring clinical judgement often arise. Clinicians must use their clinical judgement in conjunction with the formal Guidelines to ensure the pre-conditions are fulfilled and that sufficient brainstem tests have been performed to diagnose Brain Death clinically.

Severe head and facial injuries can make it impossible to test all the brainstem reflexes. Periorbital swelling or direct eye injury may affect the ability to test pupillary and corneal reflexes. Likewise CSF otorrhoea or occlusion of the external auditory canal may preclude caloric testing of the oculovestibular reflex. Lesions of the cervical cord, severe respiratory disease and other conditions may preclude apnoea testing.

There is no reported consensus on the minimum number of brainstem reflexes that must be tested (although apnoea testing is essential). The ANZICS Guidelines contain a useful rule of thumb that you should be able to test ‘at least one eye and at least one ear’ (6)

If the clinician is not fully confident that clinical tests allow the diagnosis to be made definitively, then Brain Death cannot be diagnosed through clinical tests alone. However the diagnosis of Brain Death is still possible if ancillary tests are used to supplement the findings of the clinical tests that could be performed.

Ancillary tests for diagnosis of Brain Death

Tests to support the findings of clinical tests for Brain Death rely on demonstrating the absence of parenchymal blood flow in the brain.

1. Four vessel cerebral angiography that demonstrates the absence of intracranial blood flow is the ‘gold-standard’ ancillary test to diagnose Brain Death (6-8). There is a detailed description in the ANZICS guidelines (6).

   This aid to diagnosis of Brain Death is only possible in specialist centres where cerebral angiography is available.

2. CT angiography (CTA) is more widely available although not as universally accepted as 4-vessel angiography. See the US and ANZICS Guidelines for details (6, 8, 17). Despite these reservations, CTA is widely used internationally to confirm the absence of cerebral blood flow. Formal protocols for the application of CTA as an ancillary test are useful and have stood the test of time (e.g. in France since 1998) (18-19). Clinicians relying on CTA must be personally satisfied with its reliability as an ancillary test and ensure there is verification from an experienced radiologist that the CTA study confirms the absence of blood flow in the brain.

   EEG, MRI and Transcranial Doppler are not currently recommended as reliable tests to confirm a diagnosis of Brain Death.
International guidelines agree that ancillary tests should be used in combination with clinical tests (rather than as stand-alone tests) to diagnose Brain Death. When ancillary tests are required, death cannot be diagnosed until (i) the clinical tests have shown no responses and (ii) the ancillary radiological test has demonstrated the absence of cerebral blood flow as assessed by the radiologist. At this point, one of the clinicians who undertook clinical tests can confirm that the findings of two sets of clinical tests and of the ancillary test are diagnostic of Brain Death; this is the official time of death.

**Maternal Brain Death**

The approach to the diagnosis of Brain Death in pregnancy is the same as in any other adult. Concerns may be expressed about the impact of apnoea testing on the fetus. Changes in maternal and fetal acid-base relationship during apnoea testing have not been subject to clinical trials. However, there are numerous reviews and reports of mechanical ventilation in ARDS and acute severe asthma in pregnancy, suggesting that provided hypoxaemia is avoided, maternal hypercapnia can be tolerated by the fetus with ultimate good outcomes (20-21). International Intensive Care guidelines adopt the same approach to the diagnosis of Brain Death in pregnancy as are outlined in this ICSI Guideline for all other adult patients.

There have been a number of documented cases of maternal Brain Death in the second and third trimester of pregnancy in Ireland (22-24). One such case with a second trimester pregnancy, became the subject of a high profile High Court judgement (24) The focus of this case was entirely on whether the fetus could be brought to a point of maturity to have the potential to survive. The potential for significant disability, although recognized by the court, was not considered to be a determining factor.

The international literature continues to identify cases of maternal Brain Death which, with extended somatic support, resulted in viable fetal outcomes including a case where the fetus was ≤ 17 weeks gestation. There are two central critical decisions to be made before continuing such support: a) Can such support be successfully sustained? b) When is fetal viability?

With regard to (a), this largely falls within the expertise of Intensive Care Medicine and will be approached case-by-case given the inter-individual variability of aetiology and physiologic response for the mother. With regard to (b), the threshold for fetal viability is now considered to be 23 weeks gestation. However the question of viability should be referred to obstetricians and neonatologists on a case-by-case basis and informed by changes in practice and outcomes.

The High Court has set the context, the Constitutional basis and the relevant questions to be asked (24). It does not direct that all such cases need referral to the High Court.

**Communication, organ donation and other considerations**

Family should be made aware that tests are being undertaken to diagnose death. An outline of the process including the need for two separate sets of tests and two separate doctors should be explained in advance. The findings should be communicated after each set of tests including the definitive diagnosis of death after the second set.

The diagnosis of Brain Death provides the opportunity for families to agree to the donation of organs for transplantation. After the second set of clinical tests result in the diagnosis of Brain Death the family should be sensitively informed that this opportunity arises. Sometimes families ask about organ donation at an earlier stage and it is reasonable to convey that if Brain Death is diagnosed, organ donation will be fully discussed with them.

For many patients in whom Brain Death is diagnosed, the circumstances make it mandatory to report the death to the Coroner (as per Coroner’s Guidelines). If the case is reportable to the Coroner, his/her permission is required for organ retrieval for transplantation. The Coroner normally grants permission for this; however he/she may place limitations on transplant procedures. Families should be advised of the role of the Coroner in these cases.
Currently Clinical Leads in Organ Donation and the Organ Donor Nurse Managers are available in hospital groups to discuss and facilitate organ donation as required. They are available to answer questions and provide support to families and give further information that may be required by medical or nursing staff.

The National Organ Procurement Service (NOPS) operating through Organ Donation Transplant Ireland (ODTI; https://www.hse.ie/eng/about/who/organdonation/) have specialist organ donor coordinators on call to accept referrals. NOPS provides a 24-hour service, planning and arranging transplantation, liaising with medical teams and supporting the patient’s family

If organ donation is not to take place, then a time should be set to withdraw ventilation. Most families prefer to be in attendance when the final cessation of circulation occurs; they should be advised that it may be 10 - 20 minutes after the ventilator is stopped before this happens. Privacy and religious or other ceremonies should be facilitated as much as possible during this time.

The Joint faculty of Intensive Care Medicine of Ireland runs an annual one-day Irish Donor Awareness Programme (IDAP) as part of the required training of Intensive Care Medicine trainees and as a Continuing Professional Development opportunity for Consultants or others. This Programme includes training in the diagnosis of Brain Death.
References


Appendix 1.

Historical development of the concept and criteria for Brain Death:

1959: Coma depassé et necroses nerveuses centrales massives. This was the reference publication proposing a name (‘Coma depassé’) for the ‘death of the nervous system’, the clinical, electrophysiologic and angiographic features of which had been described in the French and Scandinavian literature between 1956-1959.

1968: The concept of brain death as death was proposed by an Ad Hoc Committee of Harvard Medical School

1976: UK Royal Medical Colleges defined brain death as complete irreversible loss of brainstem function and specified clinical criteria to certify brain death.

1981: USA Presidents Commission published Guidelines. Recommended confirmatory tests to reduce the required period of observation. Recommended a period of 24 hours observation for patients with anoxic brain damage.


1993: First Australia New Zealand Intensive Care Society (ANZICS) guidelines

2006: Canadian Council for Donation and Transplantation published National Guidelines for determination of brain death
Appendix 2:
Checklist for diagnosis of Brain Death (ICSI 2020)

| Name _______________________________________ | Cause of coma: ........................................... |
| Address _____________________________________ | Adequate observation period; Y/N |
| Date of birth _________________ MRN__________ | Is cause of coma irreversible? Y/N |

Preconditions: could apnoeic coma be due to any of the following?

<table>
<thead>
<tr>
<th>Assessment A</th>
<th>Yes</th>
<th>No</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sedative drugs</td>
<td>q</td>
<td>q</td>
</tr>
<tr>
<td>Neuromuscular blocking drugs</td>
<td>q</td>
<td>q</td>
</tr>
<tr>
<td>Hypothermia</td>
<td>q</td>
<td>q</td>
</tr>
<tr>
<td>Metabolic causes</td>
<td>q</td>
<td>q</td>
</tr>
<tr>
<td>Endocrine disturbance</td>
<td>q</td>
<td>q</td>
</tr>
<tr>
<td>Other ‘confounder’</td>
<td>q</td>
<td>q</td>
</tr>
</tbody>
</table>

If preconditions are not fulfilled, an ancillary test which demonstrates absence of intracranial blood flow is required (in addition to the clinical tests possible), to allow a diagnosis of brain death.

In all cases there must be a diagnosis of a condition which has caused irreversible brain damage

<table>
<thead>
<tr>
<th>CLINICAL TESTS OF BRAIN STEM FUNCTION:</th>
<th>Assessment A</th>
<th>Yes</th>
<th>No</th>
<th>Assessment B</th>
<th>Yes</th>
<th>No</th>
</tr>
</thead>
<tbody>
<tr>
<td>Motor response to painful stimulus in cranial nerve distribution?</td>
<td>q</td>
<td>q</td>
<td>q</td>
<td>q</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Do the pupils react to light?</td>
<td>q</td>
<td>q</td>
<td>q</td>
<td>q</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Are corneal reflexes present?</td>
<td>q</td>
<td>q</td>
<td>q</td>
<td>q</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Do the eyes move on caloric or dolls eye testing?</td>
<td>q</td>
<td>q</td>
<td>q</td>
<td>q</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Is there a gag reflex?</td>
<td>q</td>
<td>q</td>
<td>q</td>
<td>q</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Is there a cough reflex?</td>
<td>q</td>
<td>q</td>
<td>q</td>
<td>q</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Apnoea test; PaCO2 pre- and post- pH; pre- and post- Pre-____ Post-____ Pre-____ Post-____

Were respiratory movements detected? q q q q

Assessor A | Assessor B
Name, IMC no | Name, IMC no
Grade | Grade
Signature | Signature

DETERMINATION OF BRAIN DEATH

Do the above clinical tests alone confirm brain death? Yes q No q
If preconditions not met, was an ancillary test undertaken? Yes q No q
Did ancillary test demonstrate absent intracranial blood flow? Yes q No q N/A q

Date / time of death _____________________________

Name _____________________________ IMC no _________ Signature _____________________________

Outcome of tests should also be documented in the clinical notes, with time/date of confirmation of death.
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