Der Radiologe

Review

Radiologe 2020 · 60 (Suppl 1):S17–S25
https://doi.org/10.1007/s00117-020-00674-6
Published online: 7 April 2020



**A. Haussmann · U. Yilmaz**

Department of Clinical and Interventional Neuroradiology, Saarland University Medical Center, Homburg/Saar, Germany

# Brain death confirmation

## Definition

The confirmation of brain death is defined as clinical and instrument-based proof of the irreversible loss of the entire human brain functions, called brain death. From the medical and scientific point of view, brain death itself is the total loss of function of the cerebrum, the cerebellum and the brainstem while the cardiovascular and respiratory systems are maintained guaranteeing gas exchange in the lungs [1, 2]. These clinical and instrument-based methods are necessary for estimation of the prognosis of the disease following therapeutic approaches, independent of being a possible donor of organs or tissue. The determination of brain death is completely defined by guidelines, which are used for patients at all ages (adults and children). In cases of children under the age of 2 years specific rules have to be followed. In cases of premature babies born earlier than the 37th week of pregnancy and babies with anencephaly the whole process of brain death confirmation cannot be used.

## Causes

The causes of the irreversible failure of the human brain are diverse and characterized by an increase of the intracranial pressure over the mean arterial pressure causing cessation of cerebral perfusion. A distinction is made between primary and secondary causes and concerning the primary between supratentorial and infratentorial reasons.

Primary cerebral causes:

- Severe traumatic brain injury
- Malignant stroke
- Intracranial hemorrhage
- Brain tumors, metastasis
- Acute obstructive hydrocephalus

Examples for secondary reasons for brain injury:

- Cerebral hypoxia

The detailed differentiation between primary supratentorial and primary infratentorial lesions is important for the subsequent procedure of determination of brain death because in cases of infratentorial lesions an additional instrument-based diagnostic is absolutely necessary [2].

## History

The term *coma dépassé*, an irreversible coma, was first used by the French neurologists Mollaret and Goulon 1959 and 9 years later, in 1968 the ad hoc committee of Harvard Medical School defined brain death as a total loss of movement and respiration, of brainstem reflexes and unconsciousness. Mohandas and Chou [3] published that injuries of the brainstem are important aspects of severe brain damages. Because of that brain death was defined by the Conference of Medical Royal Colleges as a complete and irreversible loss of function of the brainstem 1976 [3]. This statement found its way into the guidelines and therefore the examinations of brainstem reflexes and the testing of apnea are required in the confirmation [3].

## Symptoms

The following symptoms should be seen in patients before starting the cascade of the brain death confirmation:

1. Unconsciousness (coma)
2. Unresponsive bilateral middle to maximum wide pupils without application of a mydriatic medication
3. Bilateral loss of oculocephalic and vestibulo-ocular reflexes
4. Bilateral loss of corneal reflex
5. No reaction on pain in the innervation area of the trigeminal nerve bilaterally and loss of cerebral reactions to pain outside this area of innervation
6. No pharyngeal or tracheal reflexes
7. Apnea

The numbers 2–7 represent the function of the brainstem and can be summarized under brainstem reflexes. The clinical examination must be strictly performed by two physicians, of which one should be a consultant in neurology or neurosurgery combined with several years of experience in intensive care medicine. In cases of children up to the age of 14 years one of the physicians should be a consultant in pediatrics. These physicians are not allowed to be involved in the process of a possible tissue or organ transplantation after confirmation of brain death. The exclusion of possible reversible causes of the loss of brain functions, such as intoxication, sedative medications, hypothermia, brainstem diseases and coma caused by metabolic and endocrine or inflammatory diseases is necessary. For central sedative medications like sufentanyl and propofol no limit range is known. Walter et al. recommend a blood serum concentration of 0.4 µg/ml for propofol and 0.2 ng/ml for sufentanyl. Under this required limit there is no possibility of induced disturbance of consciousness [4]. The complete process of brain death confirmation is regulated by strict guidelines according to the 4th consensus of the scientific advisory board of the German Medical Association from 2015 [2].

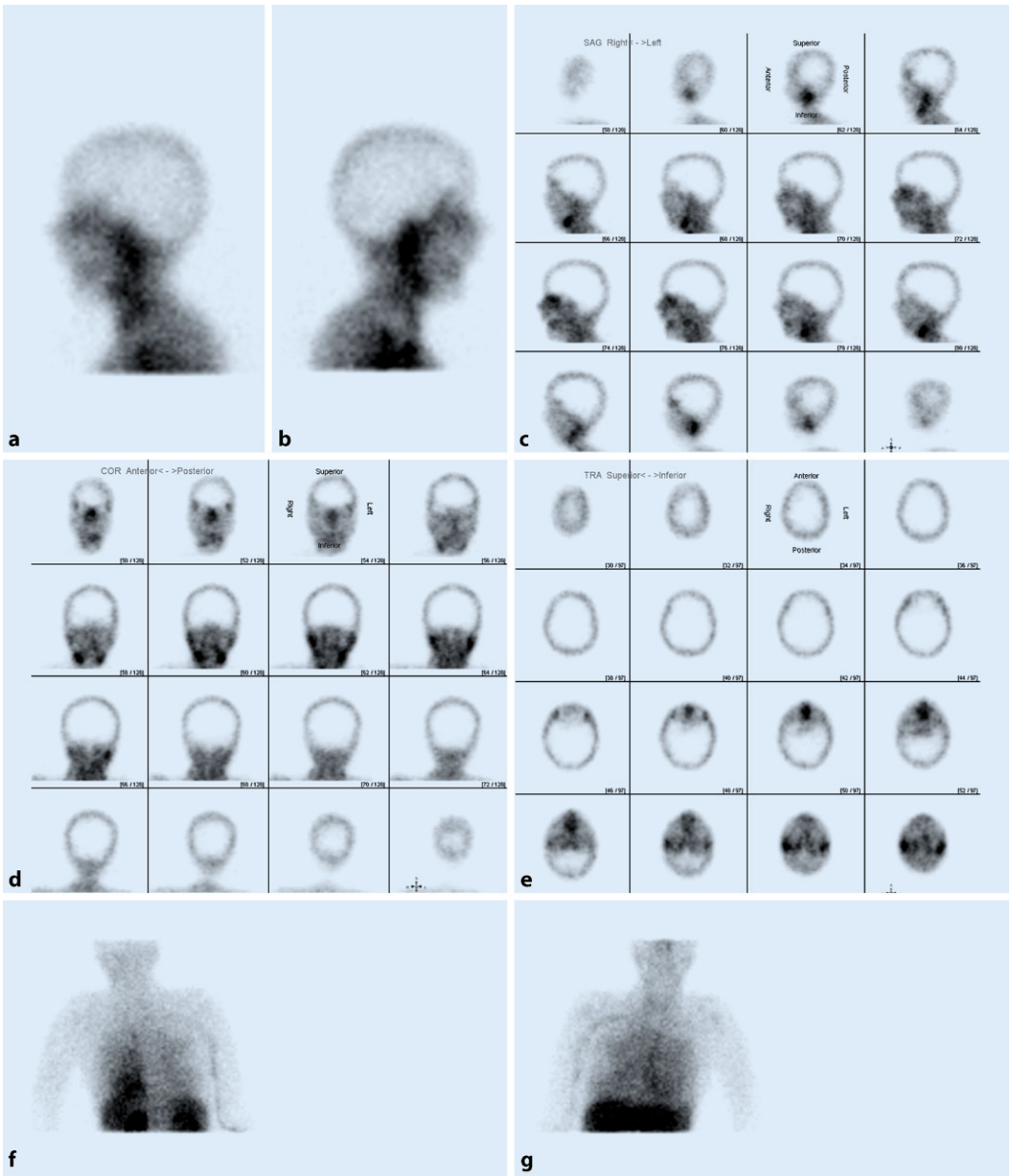


**Fig. 1** ◄ Perfusion scintigraphy of a 40-year-old woman with a ruptured aneurysm of the basilar artery and a severe subarachnoid hemorrhage (SAH). Static images of head and neck (**a, b**) and a single photon emission computed tomography (SPECT) in each section (**c** in sagittal, **d** in coronar, **e** in transversal view) showing the cessation of cerebral perfusion. As a tracer $^{99m}$Tc-HMPAO is used. In each case an in vivo control of the thorax (**f**) and abdomen (**g**) is done demonstrating the correct administration of the tracer

## Clinical diagnostics

The singular assessment of the irreversible loss of cerebrum, cerebellum and brainstem functions is insufficient. Depending on the brain injuries—primary versus secondary—the following clinical examination tests should be reconfirmed and documented by two qualified physicians after a determined time.

1. Degree of unconsciousness:
   Unconsciousness without opening of the patient's eyes or other cerebral reactions on repeated stimulations.
2. Extinguished vegetative and motor cerebral reactions on stimulations of the nasal mucosa or the supraorbital nerve exit points (supraorbital nerve, terminal branch of the trigeminus nerve)
3. Loss of brainstem reflexes:
   - Oculocephalic reflex (doll's eye reflex):
     No eye movement to the contralateral side after brisk turning or tipping of the patients head.
   - Vestibulo-ocular reflex:
     No eye movement seen after the injection of cold water in the external auditory canal under a pause of 5 min between both sides.
   - Corneal reflex:
     Absent blink reflex after contact of the cornea with a cotton swab
   - Pharyngeal reflex (gag reflex):
     Absent gag reflex after irritation of the pharynx with a wooden spatula
   - Tracheal reflex (cough reflex):
     No automatic coughing reflex after irritation of the trachea with a catheter through the tracheal tube.
   - Apnea:
     Before starting the apnea testing the partial pressure of carbon dioxide ($p_aCO_2$) should be between 30–45 mm Hg under a preoxy-

genation of 100%. A cessation of spontaneous breathing is reached at the limit value of $p_aCO_2$ of 60 mm Hg while a continuous measuring of the arterial mid pressure and pulse oxymetry. In children an additional body temperature of more than 35 °C has to be measured.

The blood levels of anticonvulsive, analgesic and sedative medications have to be checked and have to be below a measurable reference range in infants under the age of 1 year before starting the whole procedure of testing. The actual neurological status of the patient has to be re-evaluated after waiting 12 h in cases of primary injury and 72 h in secondary brain lesions. Alternatively, additional instrument-based methods can be used avoiding the waiting time in primary supratentorial and secondary brain injuries. In infants under the age of 2 years the re-evaluation time depends on the age: in neonates aged between 0 and 28 days a neurological re-check-up is carried out after 72 h and in babies older than 28 days after 12 h. The brain death confirmation consists of additional examinations, such as electroencephalography (EEG), auditory brainstem evoked potentials (AEBP) or the detection of cessation of cerebral perfusion in all cases.

## Ancillary tests

The ancillary tests in brain death confirmation are used when clinical symptoms in compliance of the required waiting hours are not unambigously and in every case of a child.

These tests can be divided into functional and image-based methods.

a) Functional methods
   - Electroencephalography (EEG)
   - Auditory brainstem evoked potentials (ABEP)
   - Somatosensory evoked potentials (SEP)

b) Imaging-based methods
   - Doppler duplex sonography
   - Perfusion scintigraphy
   - Cranial computed tomography (CCT), CT angiography and CT perfusion
   - Magnetic resonance imaging (MRI), MR angiography and MR perfusion
   - Digital subtraction angiography (DSA)

### (a) Functional methods

**EEG.** An isoelectric EEG has to be discharged over a minimum time of 30 min under the use of the 10–20 system (a special method adhering the electrodes on the surface of the scull for detecting the cortical electric [5]) activity with silver/silver chloride, platinum or steel needles and repetitive pain stimulus in the face. Specific electric currents, voltages, the upper and lower cut-off frequencies, sensitivities, setting of the amplifier and a fixed amount of EEG canals have to be considered.

**ABEP.** In this procedure auditory brainstem evoked potentials of the cochlear nerve are bilaterally provoked in primary supratentorial and secondary brain damage.

These kinds of potentials are triggered with 100 µs sustained over a whole period of time of 20 ms and a frequency range of 10–15 Hz.

For the leading of the ABEP needle electrodes or adhesive electrodes are used and placed on the vertex. Reference electrodes are positioned on the ipsilateral ear lobe or mastoid.

All in all 5 waves exist and each of them represents a response of a particular part of the auditory pathway.

- Wave I—peripheral part of the vestibulocochlear nerve
- Wave II—central part of the vestibulocochlear nerve
- Wave III—cochlear nuclei
- Wave IV—nuclei complex of superior olivary nuclei and lateral lemniscus
- Wave V—lateral lemniscus and inferior colliculi

The irreversible loss of the whole brain function is considered by seeing a bilateral progressive loss of all waves; an isolated appearance of wave I, II or III or the progressive loss of waves with a unilateral or bilateral residual presence of wave I or I and II.

**SEP.** In 1947 Dawson was the first to provoke the somatosensory provoked potentials at the vertex [6].

Somatic provoked potentials are measured after stimulation of the median nerve on both sides with impulses lasting 0.1–0.2 ms, a frequency of 3–5 Hz and an analysis time of 40–50 ms. Needle or adhesive electrodes are positioned at Erb's



Radiologe 2020 · 60 (Suppl 1):S17–S25
https://doi.org/10.1007/s00117-020-00674-6


A. Haussmann · U. Yilmaz

## Brain death confirmation

**Abstract**

The number of patients waiting for a new organ has continuously decreased in recent years. Brain death confirmation plays an important role in the clinical routine concerning a possible organ transplantation. In many countries a strictly defined protocol prescribes the required neurological examination and ancillary test criteria. Therefore, many years of experience and expertise is absolutely necessary for neurologists and neuroradiologists. Pitfalls can sometimes be very challenging for the treating physicians.

**Keywords**

Organ transplant · Prognosis · Neurological examination · Ancillary tests · Pitfalls

## Bestätigung des Hirntods

**Zusammenfassung**

Die Zahl der Patienten, die auf ein neues Organ warten, ist im letzten Jahr kontinuierlich zurückgegangen. Die Bestätigung des Hirntods spielt eine wichtige Rolle in der klinischen Routine bezüglich einer möglichen Organtransplantation. In vielen Ländern schreibt ein streng definiertes Protokoll die erforderlichen neurologischen Untersuchungs- und Hilfstestkriterien vor. Daher ist langjährige Erfahrung und Expertise für Neurologen und Neuroradiologen absolut notwendig. Die Fallstricke können für die behandelnden Ärzte manchmal eine große Herausforderung sein.

**Schlüsselwörter**

Organtransplantation · Prognose · Neurologische Untersuchung · Zusatztest · Fallstricke


**Fig. 2** ◀ Perfusion scintigraphy of an 81-year-old man with a severe spontaneous intraparenchymal bleeding and a midline shift of 1.5 cm in the native CT scan. A single photon emission computed tomography (SPECT) in each section (**a** in transversal, **b** in coronar, **c** in sagittal view) showing the cessation of the cerebral perfusion. As a tracer $^{99m}$Tc-HMPAO is used. **d**, **e** are dynamic examples showing the flow in the cerebral vessels from a ventral position. In each case an in vivo control of the thorax and abdomen is done demonstrating the correct administration of the tracer (**f**)

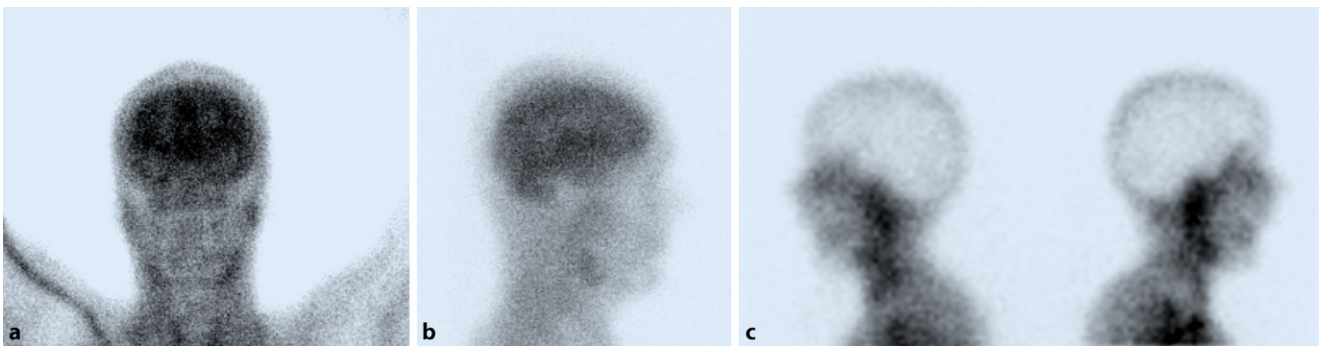


**Fig. 3** ▲ Static images of head and neck a healthy 45-year-old patient (**a**, **b**) compared with those of a brain death patient (**c**)

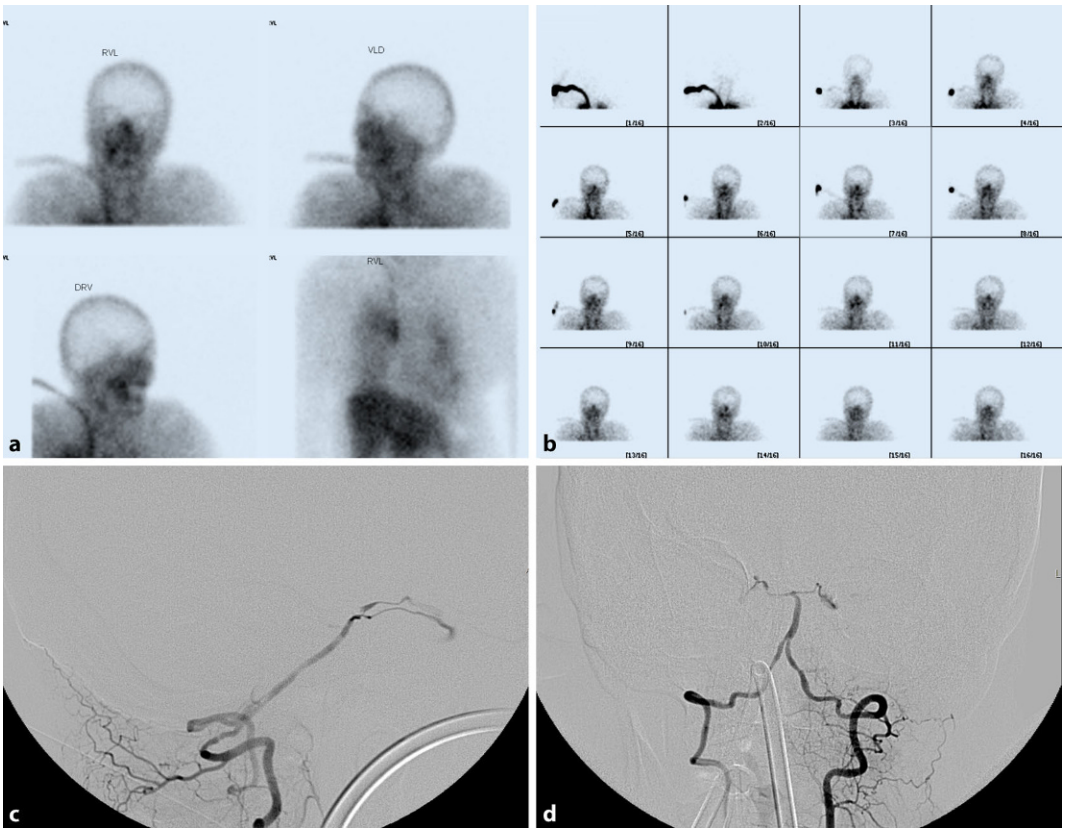


**Fig. 4** ◄ A 43-year-old woman with clinical signs of loss of brain function. She had hypoxic edema of the brain status after cardiopulmonary resuscitation (CPR). The cause for the cardiopulmonary arrest was unknown. In the perfusion scintigraphy the criteria for brain death are fulfilled (**a, b**). In digital subtraction angiography the V4-segments of the vertebral arteries, the basilar artery and both the posterior cerebral arteries are filled with contrast agent. In this case cerebral perfusion of the brainstem is possible and cannot be excluded (**c, d**)

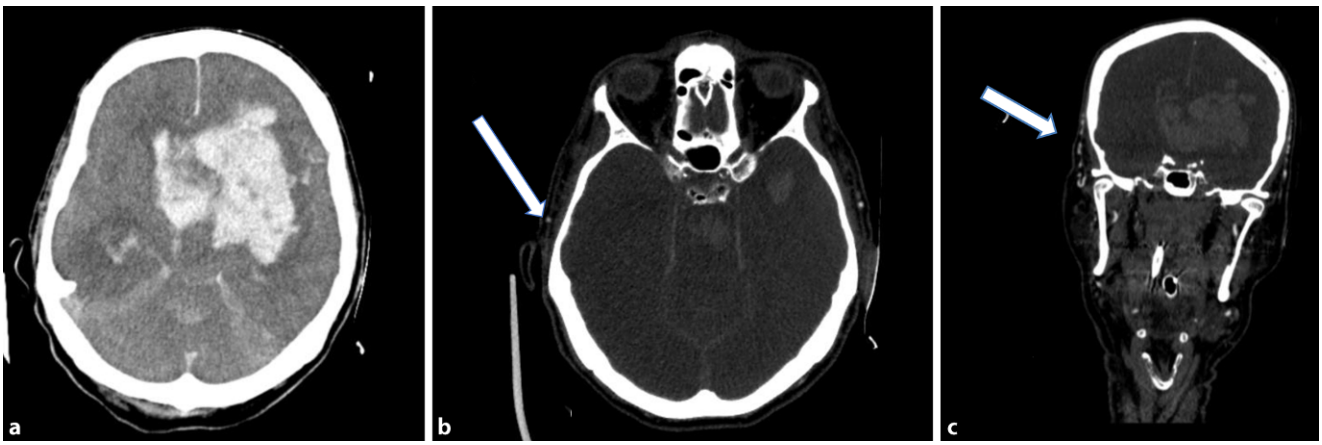


**Fig. 5** ▲ A 50-year-old man with a SAH and intraparenchymal bleeding ruptured into the ventricular system because of a ruptured middle cerebral artery (MCA) aneurysm (**a**). The CT angiography shows the contrast-filled superficial temporal artery as the in vivo quality control of correct contrast administration (*arrow*) (**c**). The criteria of brain death confirmation are fulfilled because no intracranial cerebral arteries are filled with contrast agent (**c**). *SAH* subarachnoid hemorrhage

point, at the spinous process of cervical vertebrae 2 and 7, such as cortical (C3' and C4') representing the sensitive cerebral cortex.

## (b) Image-based diagnostic methods

With these kinds of diagnostic options the cessation of cerebral perfusion can be detected but not the cut-off of brain functions.

**Doppler/duplex sonography.** In the year 1991 the doppler/duplex sonography was accepted as being part of possible additional diagnostic methods in the brain death confirmation by the scientific advisory board in the second revised version [3].

The cessation of cerebral perfusion is proven by no visualization of the middle cerebral arteries, the internal carotid arteries and the extracranial vertebral arteries in Doppler sonography.

In duplex sonography the M1 segments of the middle cerebral arteries, the internal carotid arteries, the V4 segments of the vertebral arteries and the basilar artery being part of the posterior

**I. MANDATORY CLINICAL CRITERIA**

| Vital Signs including Temperature, Blood Pressure, and Oxygen Saturation must be recorded in the chart at time of each declaration | First MD Exam | | Second MD Exam | | Comments (required for all "NO" responses |
|---|---|---|---|---|---|
| **Initial Evaluation Absence of Reversible Causes** | Yes | No | Yes | NO | |
| Injury Mechanism/Diagnosis consistent with Brain Death (*List in comments) | ☐ | ☐ | ☐ | ☐ | *1st Physician:<br>*2nd Physician: |
| Primary Hypothermia excluded as the cause of coma **(core temp must be greater than 36.5°)** | ☐ | ☐ | ☐ | ☐ | |
| Other causes of death excluded, for example:<br>(*Toxins/drugs (No contributory abnormalities)<br>Metabolic parameters (No contributory abnormalities) | ☐ | ☐ | ☐ | ☐ | *Urine toxicology results: |
| **Clinical Neurologic Examination** | | | | | |
| Coma | ☐ | ☐ | ☐ | ☐ | |
| Pupils fixed and dilated | ☐ | ☐ | ☐ | ☐ | |
| Corneal reflex absent | ☐ | ☐ | ☐ | ☐ | |
| Oculocephalic reflex absent (patient not in C-Spine precautions) | ☐ | ☐ | ☐ | ☐ | |
| Oculovestibular reflex absent (pt in C-Spine precautions) | ☐ | ☐ | ☐ | ☐ | |
| Motor response to noxious central pain stimulation absent | ☐ | ☐ | ☐ | ☐ | |
| Gag / Cough reflex absent | ☐ | ☐ | ☐ | ☐ | |
| Absence of Spontaneous Respirations | ☐ | ☐ | ☐ | ☐ | |

**II. OPTIONAL CONFIRMATORY TESTING:** After all clinical criteria are met; either an apnea or other confirmatory test can be used as an adjunct to the confirmation of clinical brain death. The addition of any confirmatory studies is at the discretion of the physician.

| **Apnea Test (confirming absence of spontaneous respirations when):** | | | | | |
|---|---|---|---|---|---|
| pH lesser than 7.30 **AND EITHER** | ☐ | ☐ | ☐ | ☐ | |
| PCO2 greater than 60 mm Hg **OR** greater than or equal to 20 mm Hg over baseline | ☐ | ☐ | ☐ | ☐ | |
| **Other Confirmatory Tests** | | | | | |
| EEG | ☐ | ☐ | ☐ | ☐ | |
| Nuclear scan | ☐ | ☐ | ☐ | ☐ | |
| Other: | ☐ | ☐ | ☐ | ☐ | |
| | ☐ | ☐ | ☐ | ☐ | |

I certify that the above tests have been performed and that according to hospital policy ADM-XX-XXX this patient is brain dead.

| 1st Physician Name (Print) | Physician Signature | Date | Time |
|---|---|---|---|
| 2nd Physician Name (Print) | Physician Signature | Date | Time |

**Fig. 6** ▲ Checklist for brain death confirmation [13, 14]. *CNS* central nervous system, *C-spine* cervical spine, *PEEP* Peak end-expiatory pressure, *CPAP* continuous positive airway pressure, $FiO_2$ Fraction of inspired oxygen, *HMPAO* hexamethylpropyleneamine oxime, *SPECT* single photon emission computed tomography, *EEG* electroencephalography, *TCD* transcranial doppler sonography

circulation should either show a biphasic flow signal or an early systolic peak lower than 50 m/s and under 200 ms. A mean arterial pressure of 60 mm Hg and a re-evaluation of the sonography after 30 min are absolutely recquired.

## Perfusion scintigraphy

Perfusion scintigraphy was included as an additional procedure in the guidelines of the German Medical Association in the third update in 1998 [7]. This is a static and dynamic nuclear medicine diagnostic method, in which the perfusion of the human brain is detected after the injection of a lipophilic and minimally radioactive pharmaceutical, such as technetium-99m ethylene cysteine dimer (Tc-99m-ECD) or $^{99m}$Tc-hexamethylpropyleneamine oxime (HMPAO). Directly after the administration the flow in the cerebral vessels is documented in a ventral position and after that the perfusion in a static mode. The posterior circulation should be evaluated using lateral projections. Only a consultant of nuclear medicine is allowed to analyze this perfusion scintigraphy. The irreversible loss of cerebral perfusion in a patient is confirmed in the absence of vessel contrast, the perfusion of the brain and the coincidental enhancement of the radioactive pharmaceutical in the human brain [8].

As an in vivo control, the detection of the physiological distribution of this substance in the patient's thorax and abdomen is necessary ([2, 7, 8]; **Figs. 1, 2, 3 and 4**).

**Table 1** Types of non-heart beating donation according to the Maastricht criteria (modification of Madrid 2011 [16])

| | | |
|---|---|---|
| Non-controlled | Type I | Death outside hospital. Includes sudden death (traumatic or otherwise) occurring outside hospital and which for obvious reasons is not subjected to resuscitation |
| | Type II | Unsuccessful resuscitation. Includes patients suffering cardiac arrest and subjected to resuscitation maneuvers that prove unsuccessful. This category comprises two sub-categories: |
| | | II.a. Out-of-hospital<br>Cardiac arrest occurs in the out-of-hospital setting, being attended by the out-of-hospital emergency service, which transfers the patient to hospital with cardiac compression and ventilatory support |
| | | II.b. In-hospital<br>Cardiac arrest occurs in the in-hospital setting, being witnessed by the healthcare personnel, with the immediate start of resuscitation maneuvering |
| Controlled | Type III | In wait of cardiac arrest. Includes patients subjected to limitation of life support[a] after agreement within the health-care team and between the latter and the relatives or representatives of the patient |
| | Type IV | Cardiac arrest under conditions of brain death. Includes patients suffering cardiac arrest while brain death is being diagnosed, or after brain death has been diagnosed, but before transfer to the operating room. It is probable that restoration of cardiac activity is attempted first, but if this is not achieved, the process can be switched to non-heart beating donation |

[a]Includes withdrawal of any type of ventricular or circulatory support (including extracorporeal membrane oxygenation, ECMO)

## CT angiography

The CT angiography plays an increasing role in the clinical routine as a further instrument-based and image-based diagnostic method, next to the important clinical diagnostics. It was used in the setting of a brain death confirmation for the first time in 1998 [9]. Before the CT angiography a non-contrasting CCT scan is needed: helical mode from the skull base to the vertex with a 5 mm axial reconstruction, the gantry is parallel to the orbitomeatal line, with 120 kV and 170 mA. The CT angiography is planned from the 6th cervical vertebra to the vertex and a bolus of 65 ml of a non-ionic contrast agent is injected intravenously with a flow of 3.5 ml/s followed by 30 ml isotonic sodium chloride solution. The bolus is tracked and after detection of a minimum contrast of 150 HU (hounsfield units) in the common carotid artery the angiography starts automatically 5 s afterwards. The table incrementation is 4 cm/s, the voltage 120 kV and the amperage 200 mA. The axial reconstructions are performed in a layer thickness of 2 mm. The following criteria are seen in the stagnation of cerebral perfusion: no contrast in the M1 segments of the middle cerebral arteries and in the A1-segments of the anterior cerebral arteries in the anterior circulation or the P1 segments of the posterior cerebral arteries and the basilar artery in the posterior cerebral circulation with a documented minimum arterial mean pressure of 60 mm Hg. The contrast-enhanced common arteries and the superficial temporal arteries are used as a perfect in vivo quality control of a correct contrast administration (**Fig. 5**).

Only a consultant of radiology with expertise in neuroradiology is allowed to assess the non-contrasting CCT scan and CT angiography. This image-based method is not allowed to be used in children because of the difficult evaluation and subsequent possible misdiagnosis [10]. A CT perfusion can also be used as an additional possibility for detecting brain death, under a renewed administration of contrast agent after SAMW (Schweizer Akademie der Medizinischen Wissenschaften) guidelines (Switzerland).

## MR angiography and MR perfusion

Regarding the SAMW guidelines (Switzerland) an MRI combined with a MR angiography and MR perfusion play a minor role in the clinical routine.

## Digital subtraction angiography

Angiography was performed for the first time in the setting of detection of a standstill of cerebral perfusion in 1964 by Heiskanen and later on by Bergquist and Bergstrom [11]. All cerebral vessels of the anterior and posterior cerebral circulation have to be assessed by a consultant of radiology during a mean arterial pressure of 80 mm Hg (**Fig. 4**).

For a long time DSA was the gold standard but with the increase in noninvasive methods and together with the clinical methods it is becoming less significant. A check list for the complete required clinical neurological and imaged-based criteria is showed in **Fig. 6** [12].

## Organ transplantation

The whole process of brain death confirmation play an important role in organ transplant. According to Eurotransplant 13,985 people were listed as waiting for a new organ in Europe in 2019 [15]. In Germany organ transplant is strictly regulated by a Transplantation Act. The removal of organs from brain dead patients is only allowed with an appropriate advance directive of the patient or with the consent of the family representative. The brain death has to be determined according to a detailed protocol. Because of the decreasing number of patients on the transplantation waiting list some countries, such as Switzerland, the USA, Belgium, Spain, Austria and the Netherlands developed ideas to expand the pool of possible organ donors. The explantation of the patient's organs is allowed after a cardiac arrest of 10 min and a documented flat line electrocardiography together with a rejection of life support. A brain death confirmation is not needed [16]. These so-called non-heart beating donors are classified based

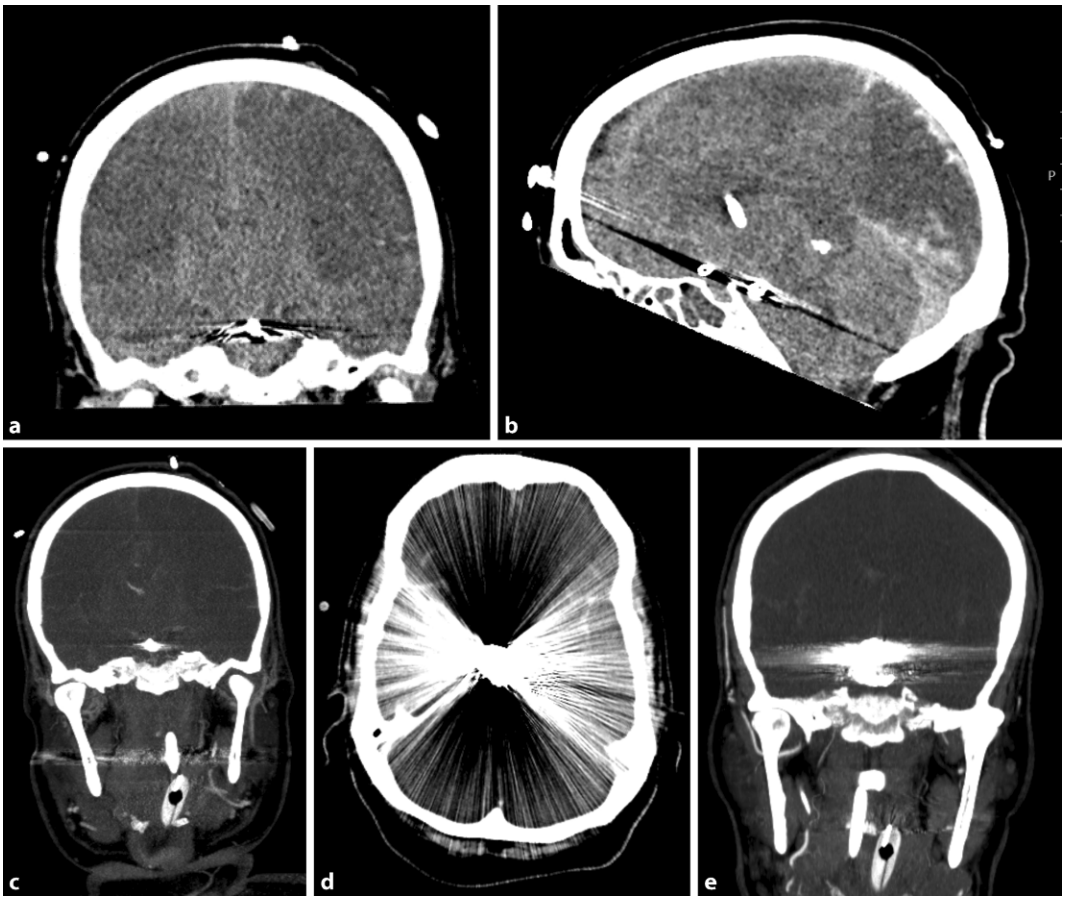


**Fig. 7** ◀ 60-year-old woman with a severe SAH and a coiled aneurysm of the basilar artery in the emergency setting (**a**–**c**). The native CT scan shows multiple infarctions in each vessel territory because of vasospasms in coronar (**a**) and sagittal (**b**) reconstruction. The CT angiography shows the difficulty of seeing a possible less contrast filling of the basilar artery in axial (**c**) because of the coils and the konsecutive metal artefacts. The native CT scan and the CT angiography also demonstrates an extreme example of metal artefacts in axial (**d**) and and coronar reconstruction (**e**)

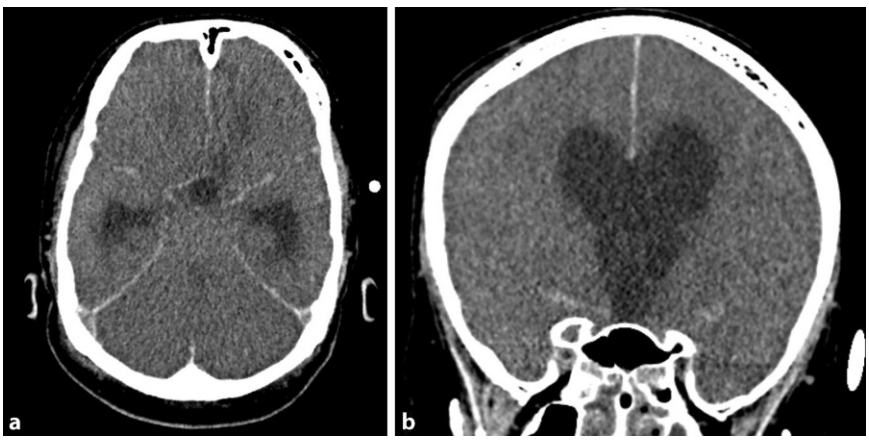


**Fig. 8** ▲ 25-year-old woman with meningoencephalitis and an obstructive hydrocephalus. The native axial (**a**) and coronal CT scans (**b**) show a pseudo SAH with hyperdense basal cisterns

on the Maastricht protocol of 1995 [16, 17] into 4 categories:

I. Death outside hospital
II. Unsuccessful resuscitation
   A. Outside hospital
   B. Inside hospital
III. In wait of cardiac arrest
IV. Cardiac arrest under brain death

The detailed Maastricht criteria for non-heart beating donors are listed in **Table 1**.

## Pitfalls of brain death confirmation

Although confirmation of brain death follows strictly and well-defined guidelines, there are some cases in which the reporting radiologist is faced with enormous challenges. In the following some examples of difficult pitfalls are listed and explained.

### Pitfall 1

Patients with a severe malignant stroke or an intracranial hemorrhage and clinical and CT-graphical signs of elevated brain pressure need a decompressive craniectomy, especially in cases of a nonpalliative treatment approach. The whole procedure of structured brain death confirmation starts when an aggravation of symptoms and the loss of brainstem reflexes are detected in the daily routine examination. Especially the report of the CT angiography can be very challenging for the radiologist because of the following reasons: in cases of the decompressive craniectomy the brain can prolapse over the skull level and the intracranial pressure can drop. Because of that the vessels of the anterior and posterior intracranial circulation can

be filled with contrast agent while performing a CT angiography. The criteria for the cessation of cerebral perfusion are not fulfilled and the radiologist is not allowed to confirm brain death of this patient. Another CT angiography performed several hours later can also show the same situation. Because of the craniectomy the continuously elevating compression on the intracranial vessels through the swelling brain parenchyma can be absent.

### Pitfall 2

Another example for difficulties is patients with a subarachnoid hemorrhage (SAH) in the basal cisterns in the context of a ruptured aneurysm. Because of the high density in the basal cisterns and directly around the blood vessels an interpretation of a possible contrast filling of the intradural part of the internal carotid artery, the middle cerebral artery and anterior cerebral artery, especially in the anterior part of the circle of Willis (circulus arteriosus) are very difficult. Especially patients with a SAH because of a ruptured aneurysm and being coiled in the emergency setting show more or less artefacts in CCT and CT angiography depending on the amount of coils which were used and this can be very difficult for the radiologist in the evaluation (**Fig. 7**).

In this context the occurrence of a so-called pseudosubarachnoid hemorrhage (pseudo SAH) should be mentioned. This phenomenon occurs in cases of severe bacterial meningitis: the blood-brain barrier is very fragile and a high concentration of protein material is able to flow into the subarachnoid spaces. This protein material shows high density and imitates a real SAH. Other reasons for a pseudo SAH are malignant stroke, bilateral subdural hemorrhage, hypoxic brain edema, sinus venous thrombosis or the intrathecal administration of contrast agent (**Fig. 8**).

### Pitfall 3

The next pitfall concerns patients with moyamoya disease. This is a rare cerebrovascular disease characterized by a hyperplasia of the intima of smooth muscle cells in the cerebral vessels followed by progressive stenosis and occlusions of the circle of Willis. Because of the slow progression of the stenosis multiple collaterals are developed, called puffs of smoke. During their lifetime the patients suffer recurrent strokes and need an extracranial (EC)-intracranial (IC) bypass maintaining the cerebral perfusion. It is a neurosurgical operation method in which the superficial temporal artery is stitched to a peripheral branch of the middle cerebral artery [12]. This is the crux of the matter: the contrast filling of the superficial temporal artery is an in vivo quality control of the confirmation of brain death. In patients with moyamoya syndrome and an EC-IC bypass the use of CT angiography to detect the cessation of cerebral perfusion should be discussed.

## Conclusion

The confirmation of brain death is defined in a structured way through well-defined and complex guidelines and in Germany this is described in the 4th consensus of the scientific advisory board of the German Medical Association 2015 [2].

Deviations from the standardized procedure like status after hemicraniectomy, status after coiling and a known moyamoya disease can be very struggling for the neuroradiologist in brain death confirmation.


## Corresponding address

**A. Haussmann**
Department of Clinical and Interventional Neuroradiology, Saarland University Medical Center
Kirrberger Str. 1, 66424 Homburg/Saar, Germany
alena.haussmann@uks.eu


## Compliance with ethical guidelines

**Conflict of interest.** A. Haussmann and U. Yilmaz declare that they have no competing interests.

For this article no studies with human participants or animals were performed by any of the authors. All studies performed were in accordance with the ethical standards indicated in each case.


The supplement containing this article is not sponsored by industry.


## References


1. Goila A, Pawar M (2009) The diagnosis of brain death. Indian J Crit Care Med 13(1):7–11
2. Wissenschaftlicher Beirat der Bundesärztekammer (2015) Richtlinien zur Feststellung des Hirntodes. Dtsch Arztebl. (4. Fortbeschreibung). https://doi.org/10.3238/artzbl.2015.rl_hinfunktionsausfall_01
3. Wijdicks EF (2001) The diagnosis of brain death. N Engl Med 344(16):1215–1221
4. Walter U, Brüderlein U, Gloger M et al (2015) Hirntoddiagnostik nach Gabe von Propofol oder Sufentanil. Empfehlungen zum Umgang mi toxikologischer Analytik. Med Klin Intensivmed Notfallmed 110:145–149
5. Jasper H (1958) The ten-twenty electrode system of the International Federation. Electroencephal Clin Neurophysiol 10:371–375. https://doi.org/10.1016/0013-469(58)90053-1
6. Bloom KK, Goldberg G (1989) Tibial nerve somatosensory evoked potentials in spinal cord hemisection. Am J Phys Med Rehabil 68(2):59–65. https://doi.org/10.1097/00002060-198904000-00003
7. Wissenschaftlicher Beirat der Bundesärztekammer (1998) Richtlinien zur Feststellung des Hirntodes. Dtsch Arztebl 95(30):A-1861–1868
8. Kranert T, Menzel C, Bartenstein P et al (2013) DGN Handlungsempfehlung (S1-Leitlinie) Hirnperfusions-SPECT mit Tc-99m-Radiopharmaka; Stand: 07/2013-AWMF Registernummer: 031-016: S. 1–10
9. Dupas B, Gayet-Delacroix M, Villers D et al (1998) Diagnosis of brain death using two-phase spiral CT. AJNR Am J Neuroradiol 19:641–647
10. Henderson N, McDonald M (2017) Ancillary studies in evaluating pediatric brain death. J Pediatr Intensive Care 6:234–239
11. Tan W, Wilbur A, Jafar J, Spigos D, Abejo R (1987) Brain death: use of dynamic CT and intravenous digital subtraction angiography. AJNR Am J Neuoradiol 8(1):123–125
12. Koizumi A, Kobayashi H et al (2016) A new horizon of moyamoya disease and associated health risks explored through RNF213. Environ Health Prev Med 21(2):55–70
13. Wijdicks FM et al (2010) Evidence-based guideline update: determining brain death in adults: report of the quality standards subcommittee of the American Academy of Neurology. Neurology 74(23):1911–1918
14. Spinello I (2015) Brain death determination. J Intens Care Med 30(6):326–337. https://doi.org/10.1177/0885066613511053
15. www.eurotransplant.org. Accessed 01.04.2020
16. Minambres E et al (2015) Experience of a Maastrich type II non heart beating donor programm in a small city: preliminary results. Med Intensiva 39:433–441
17. Westerterp K et al (1995) The Maastrich protocol for the measurement of body composition and energy expenditure with labeled water. Obes Res 3(Suppl 1):49–57
18. Wissenschaftlicher Beirat der Bundesärztekammer (1991) Richtlinien zur Feststellung des Hirntodes. Dtsch Arztebl 88(49):A4396–4407