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Brain Death Scintigraphy: Do Not Blow the Flow

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Brain death denotes the loss of function in both the cerebrum and the brain stem, leading to coma, absence of spontaneous respiration in the setting of adequate stimulus, and the cessation of all brain stem reflexes. Although spinal reflexes such as deep tendon, plantar flexion, and withdrawal reflexes may persist, recovery is not possible. The cessation of brain function qualifies as death because of its central role in coordinating vital bodily functions. Although brain death is largely determined by a clinical and neurologic examination, confounding variables may necessitate ancillary testing such as cerebral brain perfusion imaging.

Key Words: quality assurance; brain; DTPA; death; HMPAO; scintigraphy

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Brain death, also referred to as death by neurologic criteria, denotes the irreversible cessation of all brain activity after a catastrophic brain injury. In 1995, the American Academy of Neurology established formal guidelines for determining brain death in adults. This guideline was revised in 2010. Additionally, the pediatric guideline, established by the American Academy of Pediatrics, the Child Neurology Society, and the Society of Critical Care Medicine, was updated in 2011 (*1*).

In October 2023, a new consensus practice guideline was published in the medical journal of the American Academy of Neurology, *Neurology*. Developed through collaboration between the American Academy of Neurology, the American Academy of Pediatrics, the Child Neurology Society, and the Society of Critical Care Medicine, this 2023 guideline integrates guidance for adults and children into a single comprehensive framework (*1*). It provides a practical approach for clinicians to evaluate patients who have sustained catastrophic brain injuries to determine whether they meet brain death criteria. Given that brain death is based on clinical assessment, ancillary testing is required only when the clinical assessment demonstrates uncertain neurologic examination results, when the assessment cannot be safely or fully completed, when confounding variables render the clinical examination unreliable, to shorten the duration of the observation period, or to encourage family member cooperation (*1*).

CLINICAL PREREQUISITES

Diagnosing brain death typically occurs with a clinical and neurologic assessment, necessitating specific conditions within the clinical setting and evidence indicating the absence of brain function on neurologic examination. Before brain death assessment is initiated, specific clinical prerequisites must be met as described in Table 1 (*1*,*2*). The apnea test will be performed once all other clinical and neurologic criteria have been met. Spontaneous breathing, absence of coma, intact brain stem reflexes, or motor activity beyond spinally mediated reflexes all indicate brain function and are inconsistent with brain death (*1*).

ANCILLARY TESTING

Although brain death determination relies primarily on clinical and neurologic examinations, certain clinical scenarios may arise in which a component of the neurologic assessment or the apnea test cannot be completed or the findings cannot be adequately interpreted. In such cases, ancillary testing may be necessary to ensure accurate brain death determination. It is important to note that ancillary testing does not replace careful clinical assessment but serves as a supplemental tool (*1*,*2*). Examples of situations in which ancillary testing may be warranted include fractures or trauma hindering cranial nerve assessment, the presence of neuromuscular paralysis or heavy sedation, an invalid apnea test, difficulties in interpreting the neurologic evaluation, or uncorrected metabolic disturbances (*1*,*2*). For infants less than 1 y old, and particularly those less than 2 mo old, 2 positive ancillary tests are typically required (*1*). The indications for ancillary tests and the selection of appropriate tests may be governed by hospital or state policies (*1*).

Several diagnostic imaging ancillary tests may be used as described in Table 2; however, this article will elaborate on

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TABLE 1
Clinical Prerequisites and Findings (*2*)

Parameter	Finding
Clinical prerequisites	Clinical or neurologic evidence of acute central nervous system catastrophe compatible with diagnosis of brain death, with known or proximate cause of brain death identified
	Exclusion of confounding variables such as severe electrolyte, metabolic, endocrine, or circulatory disturbance
	No presence of drug intoxication or poisoning that could confound clinical assessment, including any sedative drugs administered by clinicians
	Core temperature maintained above 36°C (97°F), as hypothermia may suppress brain function, potentially leading to inaccurate clinical determination
	Systolic blood pressure maintained above 100 mg Hg and mean arterial pressure of at least 75 mm Hg for adults; in children, systolic blood pressure and mean arterial pressure should be at or above fifth percentile for age; administration of vasopressors may be needed to achieve this level
Clinical findings	Coma
	Absence of brain-originating motor response, including response to pain stimulus above neck or other brain-originating movements
	Absence of reflexes, including pupillary light, corneal, gag, oculocephalic (doll's eyes), and oculovestibular reflexes (caloric responses)
	Absence of jaw jerk
	Absence of cough with tracheal suctioning
	Absence of sucking or rooting reflexes (in neonates)
	Apnea as demonstrated by apnea test
Observation period	Minimum of 6 h; longer periods recommended in children and for certain conditions such as after cardiopulmonary resuscitation or other severe acute brain injuries

only radionuclide brain perfusion scintigraphy, to include indications, contraindications and precautions, radiopharmaceutical dose and method of administration, imaging parameters, image interpretation, and image artifacts and sources of error.

RADIONUCLIDE BRAIN PERFUSION SCINTIGRAPHY

Indications

Brain death scintigraphy is recommended for assessing cerebral blood flow in patients suspected of brain death. This diagnostic tool becomes particularly valuable for clinical and neurologic assessments and may be less reliable if there are confounding variables such as severe hypothermia, coma induced by barbiturates, electrolyte or acid–base imbalances, endocrine or metabolic disturbances, drug intoxication, poisoning, or neuromuscular blockade. Moreover, this study often serves as a critical and decisive step for the patient's family to provide their consent to withdraw care and harvest organs (*3*–*5*). Various events, such as head trauma, anoxia, cerebrovascular accidents, and edema, can cause fluid accumulation in the confined space of the calvarium. The resulting increased intracranial pressure causes cessation of cerebral blood flow, thus increasing the specificity of brain death scintigraphy (*4*,*6*).

Contraindications and Precautions

Brain death scintigraphy does not require the withdrawal of medical therapy, but specific precautions must be considered. Although some hospitals may possess a mobile γ-camera, brain death scintigraphy is typically conducted in the nuclear medicine department. Given the patient's acute condition, collaboration with personnel responsible for monitoring and ensuring safe patient transport and imaging is essential (*3*,*5*). The patient should have stable blood pressure and be otherwise systemically stable. A halo with metal components, a breathing apparatus, or patient positioning may interfere with the examination protocol (*5*).

Radiopharmaceutical Dose and Method of Administration

Several ^{99m}Tc-labeled radiopharmaceuticals may be used, including ^{99m}Tc-bicisate (^{99m}Tc-ECD; ^{99m}Tc-ethyl cysteinate dimer), ^{99m}Tc-exametazime (^{99m}Tc-HMPAO; ^{99m}Tc-hexamethylpropylene amine oxime), and ^{99m}Tc-pentetate (^{99m}Tc-DTPA; ^{99m}Tc-diethylenetriaminepentaacetic acid) (*3*,*5*,*6*). ^{99m}Tc-DTPA is a nondiffusible, non–brain-specific radiopharmaceutical that does not cross the intact blood–brain barrier and therefore provides information only on low-resolution vascular flow. Some hospitals prefer to use diffusible, lipophilic, brain-specific radiopharmaceuticals such as ^{99m}Tc-HMPAO and ^{99m}Tc-ECD because image interpretation is less dependent on the quality of the bolus and relies more on the assessment of parenchymal uptake on delayed static imaging for determining the presence or absence of cerebral blood flow (*3*–*7*). Further brain-specific radiopharmaceuticals allow for the evaluation of regional brain tissue perfusion and permit the use of SPECT, which makes image interpretation even more straightforward (*3*,*7*). It is important to note that SPECT acquisition may not be feasible in unstable patients on life support equipment (*3*).

In adults, the bolus injection can be up to 1,110 MBq (30 mCi) administered in a peripheral vein as close to the access point as possible (*3*,*5*–*7*). Pediatric doses should be based on body weight and kept as low as reasonably

TABLE 2
Diagnostic Imaging Used to Confirm Brain Death (*1*,*2*,*7*)

Test	Diagnostic criterion	Advantage	Disadvantage
Four-vessel conventional cerebral angiography	Absence of blood flow at or beyond carotid bifurcation or circle of Willis	Traditional gold standard among cerebral blood flow tests	Requirement for transportation to imaging department
			Invasive
			Contrast precautions for kidneys
			Contrast stasis or delayed filling in intracranial arteries
Radionuclide perfusion scintigraphy	Absence of blood flow at or beyond carotid bifurcation or circle of Willis	Sensitive for confirming brain death in presence of confounding variables	Requirement for transportation to imaging department
	Hollow skull if using brain-specific radiopharmaceutical		Potential for limitation of brain stem evaluation via planar imaging
			Requirement for coordination with multiple hospital personnel and nuclear pharmacy
			Possibility that on-call nuclear medicine staffing is not supported
			Restricted off-hours availability of radiopharmaceuticals
Transcranial Doppler	Presence of small systolic peaks without diastolic flow or reverberating flow pattern, indicating high vascular resistance and supporting diagnosis of brain death	Noninvasive	Requirement for technical experience
		Can be done at bedside	Evaluation precluded by 10%–25% prevalence of temporal bone thickening
		No contrast medium used	Reports of both false-positive and false-negative results (compared with cerebral angiography or other standard)
MRI of arterial blood flow	Absence of arterial blood flow	Demonstration of variable degrees of cerebral edema and mass effect	Requirement for transportation to imaging department
			Requirement for gadolinium contrast agent for improved sensitivity
			Requirement for patients to lie flat
			Possibility of short periods in which clinical monitoring is impossible
CT angiography	Absence of cerebral circulation perfusion	Widespread availability	Uncertain clinical utility of CT angiography
			Requirement for iodine contrast injection

achievable for diagnostic image quality. Typically, children receive a dose of 11.1 MBq/kg (0.3 mCi/kg), with a minimum dose of 185 MBq (5 mCi) for brain-specific radiopharmaceuticals (*3*,*7*).

Tables 3, 4, and 5 describe radiation dosimetry in adults, children (5 years old; normal renal function), and the pregnant or potentially pregnant patient, respectively.

Imaging Parameters

Brain death scintigraphy routinely involves a cerebral angiogram, which evaluates dynamic blood flow in the brain's vessels, and static planar images. Non–brain-specific radiopharmaceuticals such as ^{99m}Tc-DTPA are charged hydrophilic compounds that cannot normally localize in the brain parenchyma because of the intact blood–brain barrier (*4*,*6*). Instead, they are found in the overlying scalp soft tissues, calvarium, subarachnoid spaces outlining the cerebral hemispheres, and larger blood pools such as the sagittal and transverse sinuses (*7*). Non–brain-specific radiopharmaceuticals are able to enter the brain when the blood–brain barrier has been disrupted by some pathologic process (*5*,*6*). In contrast, ^{99m}Tc-HMPAO and ^{99m}Tc-ECD are lipophilic, allowing them to cross the intact blood–brain barrier and be retained in the brain parenchyma in proportion to regional cerebral blood

TABLE 3
Radiation Dosimetry: Adults (*8*)

	Administered activity (intravenous)		Largest radiation dose			Effective dose	
Radiopharmaceutical	MBq	mCi	Organ	mGy/MBq	rad/mCi	mSv/MBq	rem/mCi
^{99m}Tc-DTPA	555–1,110	15–30	Urinary bladder wall	0.065	0.24	0.0063	0.023
^{99m}Tc-HMPAO	370–1,110	10–30	Kidneys	0.034	0.0126	0.0093	0.034
^{99m}Tc-ECD	370–1,110	10–30	Urinary bladder wall	0.05	0.18	0.0077	0.028

flow. Because non–brain-specific radiopharmaceuticals such as ^{99m}Tc-DTPA do not show brain parenchymal uptake, dynamic blood flow images are crucial for interpretation. Conversely, with brain-specific radiopharmaceuticals, the absence of blood flow in dynamic images confirms brain death when delayed static images fail to show brain visualization (*3*–*6*).

The dynamic blood flow images are acquired at the time of radiopharmaceutical injection, with image acquisition commencing immediately before or immediately after injection (*3*–*6*). Since these patients are often receiving multiple medications, it is essential to determine the optimal venous access point and inject as close to the body as feasible. Injection technique with a high-quality bolus injection (0.5–1 mL) is more critical for non–brain-specific radiopharmaceuticals than for brain-specific radiopharmaceuticals, which rely more on the assessment of parenchymal uptake on static images for determining brain death (*6*). During blood flow imaging of the brain, a series of 1- to 3-s-per-frame anterior (or anterior and posterior) images of the head are acquired for 1–2 min (*3*,*5*,*6*). Static anterior or anterior/posterior and lateral blood pool images are acquired immediately after the dynamic images for non–brain-specific radiopharmaceuticals and at 20 min after injection for brain-specific radiopharmaceuticals (*5*). The static images should be acquired for long enough to permit 500,000–1,000,000 counts per view (*3*). Delayed static images may also be acquired at 1–4 h after injection (*5*).

The top 10 professional tips for performing radionuclide brain perfusion scintigraphy are listed below:

1. Coordinate with nursing, patient transport, respiratory therapy, and the nuclear pharmacy to ensure your dose arrives on time and there is a camera available when the patient arrives.
2. Do not blow the flow. Ensure a quality bolus injection and initiate imaging promptly. It is better to start too early than too late. If necessary, set the image acquisition to 120 s to ensure that imaging begins before the bolus reaches the carotid arteries and continues well after the venous phase.
3. Position the patient supine and head-in, with the camera anterior or anterior/posterior. Provided there is no trauma to the skull and there are no fixating devices, head-in positioning allows for the use of a head holder and strap, which help ensure that the head is positioned straight to allow for comparison of right and left carotid flow as well as visualization of the anterior and middle cerebral arteries. Additionally, head-in positioning facilitates the securement of all infusion lines and medical devices out of the field of view and outside the camera rotation range. Moreover, this positioning facilitates closer detector proximity for lateral statics and SPECT (if applicable).
4. Ensure proper patient positioning to visualize the carotid arteries and skull vertex in the field of view and to allow assessment of the symmetry of blood flow to both sides of the head and superior sagittal sinus activity. Prompt visualization of the carotid arteries serves as an assessment for a quality bolus injection with no infiltration.
5. Double-check the camera position, patient information, and that the correct image acquisition is being performed before moving the patient to the imaging table.
6. Ensure that the image acquisition can be initiated from the persistence scope or hand controller.
7. For the dynamic blood flow, consider placing a tourniquet or elastic band under the posterior protuberance of the skull, over the ears, and just above the orbits to minimize scalp circulation, which may be confused with cerebral perfusion. Avoid this step if the patient has head trauma.
8. Obtain at least one blood pool image in the same view as the dynamic blood flow images. Consider obtaining optional static images, including posterior and vertex.

TABLE 4
Radiation Dosimetry: Children (5 Years Old; Normal Renal Function) (*8*)

	Administered activity (intravenous)		Minimum dose		Maximum dose		Largest radiation dose			Effective dose	
Radiopharmaceutical	MBq/kg	mCi/kg	MBq	mCi	MBq	mCi	Organ	mGy/MBq	rad/mCi	mSv/MBq	rem/mCi
^{99m}Tc-DTPA	7.4	0.2	370	10	740	20	Urinary bladder wall	0.17	0.63	0.017	0.063
^{99m}Tc-HMPAO	11.1	0.3	185	5	740	20	Thyroid	0.14	0.52	0.027	0.099
^{99m}Tc-ECD	11.1	0.3	185	5	740	20	Urinary bladder wall	0.11	0.41	0.022	0.081

TABLE 5
Radiation Dosimetry in Pregnant or Potentially Pregnant Patient (*8*)

	Fetal dose			
Stage of gestation	mGy/MBq	rad/mCi	mGy	rad
^{99m}Tc-HMPAO				
Early	0.0087	0.032	3.2–9.7	0.32–0.97
3 mo	0.0067	0.025	2.5–7.4	0.25–0.74
6 mo	0.0048	0.018	1.8–5.3	0.18–0.53
9 mo	0.0036	0.013	1.3–4.0	0.13–0.40
^{99m}Tc-DTPA				
Early	0.012	0.044	6.7–8.9	0.67–0.89
3 mo	0.0087	0.032	4.8–6.4	0.48–0.64
6 mo	0.0041	0.015	2.3–3.0	0.23–0.30
9 mo	0.0047	0.017	2.6–3.5	0.26–0.35

9. Perform SPECT or SPECT/CT if using ^{99m}Tc-HMPAO or ^{99m}Tc-ECD to enhance visualization of perfusion to the posterior fossa and brain stem structures. However, SPECT acquisition may not be possible for unstable patients reliant on life support equipment.
10. Use zoom or magnification techniques if imaging pediatric patients.

Table 6 describes the benefits and drawbacks of radiopharmaceuticals, and Table 7 describes the image acquisition parameters.

Regarding the processing and display of images, scale static and blood flow images to provide the best visualization of the areas of interest and display in gray scale (*3*). SPECT and SPECT/CT images should be processed as per the manufacturer's specifications and the interpreting physician's requests (*3*,*5*). SPECT data should be filtered in 3 dimensions and reconstructed at the highest pixel resolution, 1 pixel thick. Three-dimensional filtering can be accomplished either by 2-dimensionally prefiltering the projection data or by applying a 3-dimensional postprocessing filter to the reconstructed data (*5*). The entire brain should be reconstructed to include the vertex and cerebellum, and images should be displayed in transverse, sagittal, and coronal slices (*5*).

Image Interpretation

The cerebral angiogram illustrates uptake in the arterial, capillary, and venous phases. After the injection of the radiopharmaceutical into a peripheral vein, prompt and symmetric visualization should occur in the subclavian, carotid, and cerebral arteries (*5*). Visualization of the anterior and middle cerebral arteries forms a trident appearance and indicates cerebral perfusion (Fig. 1). During the capillary phase, symmetric and diffuse activity is observed in both cerebral hemispheres (*5*). The venous phase reveals visualization of the sagittal sinus and jugular veins (*5*). If a patient is suspected of being brain-dead on the basis of clinical and neurologic evaluation and there is no evidence of cerebral perfusion on the cerebral angiogram, the diagnosis is confirmed (*3*).

Interpretation of brain death when using non–brain-specific radiopharmaceuticals relies on the dynamic flow study because these radiopharmaceuticals do not cross the blood–brain barrier (*6*). In contrast, brain-specific radiopharmaceuticals can evaluate both dynamic flow and parenchymal uptake in the brain as they are lipophilic and diffuse across the blood–brain barrier. Diffuse radiopharmaceutical uptake in the brain parenchyma will accumulate over time if the brain perfusion is conserved (Fig. 2) (*6*).

When brain death occurs, blood flow to the internal carotid artery ceases because of increased intracranial pressure or clotting (*6*). During the cerebral angiogram, blood flow halts at the level of the internal carotids and base of the skull, with no blush of activity observed in the anterior and middle cerebral arteries or pooling in the sagittal sinus.

TABLE 6
Choose Wisely: Benefits and Drawbacks of Radiopharmaceuticals (*3*,*5*–*8*)

Nondiffusible, non–brain-specific radiopharmaceutical		Diffusible, brain-specific radiopharmaceutical	
Benefit	Drawback	Benefit	Drawback
Rapid renal excretion facilitates repeat examinations if necessary	Primarily planar imaging is performed	Planar imaging and SPECT or SPECT/CT can be performed if patient condition allows and is needed	Repeat examination on same day is often not possible because of parenchymal retention
	There is greater dependency on injection technique	No significant redistribution occurs for several hours, making it easy to perform and interpret imaging	High radiochemical stability and purity are essential to prevent false-positive interpretation
	Delayed images may show superior sagittal sinus activity even in presence of brain death in as many as 50% of patients.	Procedure is more technically forgiving—dynamic imaging is noncritical step in image acquisition	
	Superficial scalp blood flow interferes	Parenchymal trapping appears preserved even in presence of metabolic disturbances	

TABLE 7
Image Acquisition Parameters (*3*,*5*)

Dynamic flow	Static	SPECT
Anterior	Anterior for blood pool images; additional static images include posterior and laterals	Only if using ^{99m}Tc-HMPAO or ^{99m}Tc-ECD and only as needed
1–3 s/frame for 1–2 min	500,000–1,000,000 counts per image	Time per stop and counts acquired dependent on radiopharmaceutical dose administered (e.g., 64 projections, 20–40 s/stop [200,000 counts]); use of institutional guideline for other brain SPECT studies
15%–20% window around 140-keV photopeak	15%–20% window around 140-keV photopeak	15%–20% window around 140-keV photopeak
128 × 128 matrix	128 × 128 matrix	At least 128 × 128 matrix
LEAP, LEHR collimators	LEAP, LEHR collimators	LEAP, LEHR, or UHR collimators; fanbeam or other focused collimator is needed for increased resolution and higher counting rates
Zooming or magnification optional	Zooming or magnification optional	Zoom set to produce pixel size of 3.5 mm or less
		Circular or noncircular orbit with smallest radius possible
		Continuous or step-and-shoot

LEAP = low-energy all-purpose; LEHR = low-energy high-resolution; UHR = ultrahigh resolution.

Subsequent to the cerebral angiogram, blood pool images will reveal soft-tissue uptake in facial structures, but no uptake will be evident in the sagittal or transverse sinuses (*5*). If using a brain-specific radiopharmaceutical, there will be an absence of uptake in the brain parenchyma that results in a light-bulb or hollow-skull phenomenon indicating a lack of brain perfusion and supporting the diagnosis of brain death (Fig. 3) (*2*). Blockage of the internal carotid arteries may redirect blood to the maxillary branch of the external carotid arteries, potentially resulting in increased accumulation of activity in the nasopharynx and the appearance of a hot nose (*3*,*5*–*7*). The hot nose sign does not specifically indicate brain death but may be used as a secondary sign when intracerebral perfusion is absent.

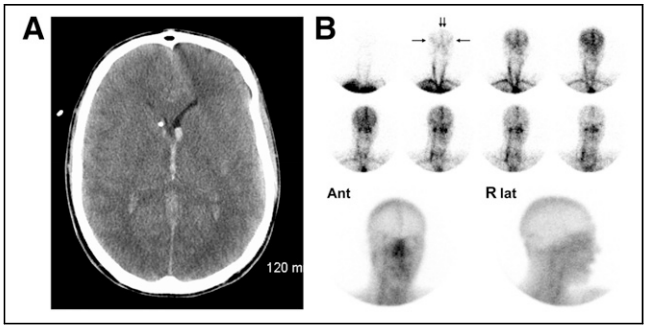

FIGURE 1. Findings of persistence of blood flow on blood flow study using nonlipophilic radiopharmaceutical in 22-y-old man with ruptured aneurysm of left posterior inferior cerebral artery. (A) CT scan demonstrates intraventricular and subarachnoid hemorrhage. (B) Two-second flow images (top rows) after injection of 905 MBq of ^{99m}Tc-DTPA demonstrate excellent visualization of anterior (vertical arrows) and middle (horizontal arrows) cerebral arteries, forming trident appearance indicating presence of perfusion, which progresses into visualization of intracranial venous sinuses. On immediate anterior (Ant) and right lateral (R lat) static images (bottom row), radiopharmaceutical does not cross blood–brain barrier; however, activity is noted in venous sinuses. (Reprinted from (*8*).)

Image Artifacts and Sources of Error

Ensuring venous patency and using proper injection technique are critical for accurate image interpretation (*3*,*5*). The occurrence of radiopharmaceutical infiltration or prolonged infusion poses a risk to the assessment of cerebral

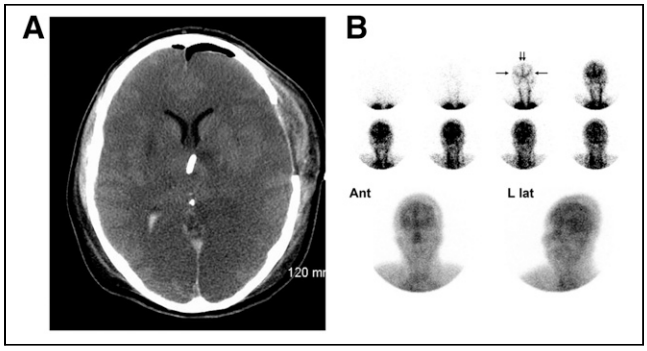

FIGURE 2. Findings of persistence of blood flow on blood flow study using lipophilic radiopharmaceutical in 18-y-old man after motor vehicle accident followed by left-sided craniotomy to evacuate subdural hematoma. (A) CT indicates loss of white matter–to–gray matter differentiation in left posterior cerebral artery territory and right posterior parasagittal cortex territory. There is diffuse cerebral edema and transtentorial herniation with compression of basilar cisterns. (B) Two-second flow images (top rows) demonstrate excellent visualization of anterior (vertical arrows) and middle (horizontal arrows) cerebral arteries, resembling trident, with retention of activity within brain parenchyma, thereby indicating presence of blood flow. Anterior (Ant) and left lateral (L lat) parenchymal phase images (bottom row) demonstrate somewhat inhomogeneous though extensive brain perfusion. (Reprinted from (*8*).)

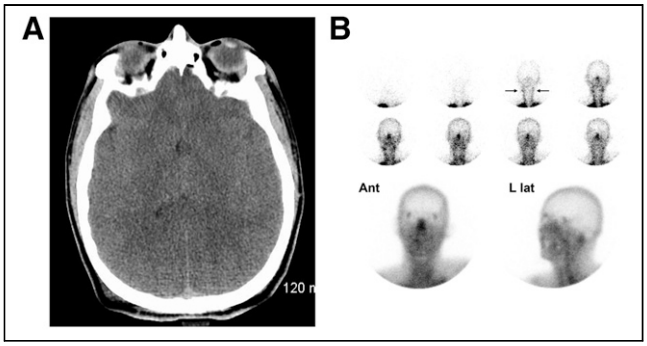

FIGURE 3. Findings of absence of blood flow on blood flow study using lipophilic radiopharmaceutical in 33-y-old man after hanging. (A) CT scan demonstrates diffuse cerebral edema with narrowing of lateral ventricles and bilateral infarction of lentiform nuclei. (B) Two-second flow images (top rows) after injection of 799 MBq of ^{99m}Tc-HMPAO demonstrate excellent visualization of common carotid arteries (arrows) but absence of flow into calvarium, consistent with brain death. Anterior (Ant) and left lateral (L lat) parenchymal phase images (bottom row) demonstrate complete lack of perfusion within boney skull (light-bulb or hollow-skull sign), including absence in posterior fossa and superior sagittal sinus. More technically demanding SPECT imaging is generally not performed in these critically ill patients. (Reprinted from (*8*).)

angiogram results. Failure to promptly visualize the radiopharmaceutical in the carotid arteries may indicate complete dose infiltration (*3*,*5*). In such instances, obtaining an image of the injection site is advisable to confirm infiltration. Additionally, the presence of metal plates or life-support equipment such as respirators can cause attenuation, potentially compromising the clinical usefulness of planar imaging and SPECT procedures (*3*,*5*). Documenting head trauma, cerebrospinal fluid shunts, and intracranial pressure transducers is essential because of their potential to induce hyperemic blood flow, which could consequently produce false-negative cerebral angiography results (*3*,*5*,*7*).

CONCLUSION

Conducting brain death scintigraphy presents technical challenges, yet many complications can be preempted through careful planning and effective communication with the nuclear pharmacy, support staff, and interpreting physician. It is crucial to minimize distractions and meticulously configure the camera acquisition, ensuring that the persistence scope or hand controller is enabled to initiate the acquisition accurately. Patient positioning may be facilitated with a head-holder and strap. Additionally, when non–brain-specific radiopharmaceuticals such as ^{99m}Tc-DTPA are used, it is advisable to extend the blood flow dynamic image acquisition time and begin the image acquisition immediately before the injection to prevent blowing the flow.

DISCLOSURE

No potential conflict of interest relevant to this article was reported.

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