Brain death is clinically diagnosed by verification of the absence of brain stem reflexes, motor responses, and the ventilatory drive. In some circumstances, ancillary tests are necessary to confirm the diagnosis of brain death, especially when there is uncertainty about the reliability of parts of the neurologic examination or when an apnea test cannot be performed. Ancillary tests are also used to reduce the observation period.

In clinical practice, electroencephalography (EEG), cerebral angiography, nuclear scanning, transcranial Doppler sonography, computed tomographic angiography, and other imaging techniques may be used to support the diagnosis of brain death.
brain stem reflexes. Refractory intracranial hypertension bilateral mydriasis with nonreactive pupils and absence of 36°C. On initial physical examination, the patient had 70 beats per minute, a mean arterial blood pressure that mechanically ventilated. Vital signs revealed a heart rate of stem hematoma. The patient was intubated, sedated, and unit (ICU) after surgical evacuation of a hypertensive brain arterial hypertension was admitted to the intensive care sonography was used for this purpose.

death; consequently, transcranial color-coded duplex
findings were also evident in a third transcranial color-
doppler (Figure 1B). At that moment, the bilateral distal portion of the internal carotid artery was interrogated and clearly revealed a reverberating flow pattern, confirming the diagnosis of cerebral circulatory arrest (Figure 1C). These findings were also evident in a third transcranial color-coded duplex sonographic examination performed 30 minutes later. With the compatible neurologic signs and the persistent sonographic findings, the diagnosis of brain death was confirmed, and standardized organ retrieval procedures were performed.

For transcranial Doppler diagnosis of cerebral circulatory arrest to support the diagnosis of brain death, insonation of both the middle cerebral artery and basilar artery is the method suggested. In difficult cases, the submandibular view may show a cerebral circulatory arrest flow pattern by interrogation of the distal portion of the internal

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phy, and magnetic resonance angiography are ancillary tests currently used in adults. In general, non–transcranial Doppler tests have little practical applicability compared with transcranial Doppler sonography; they are limited by their high costs, the need to transport the patient, and the inability to perform immediate assessments. The American Academy of Neurology’s practice parameters for determining brain death in adults consider transcranial Doppler sonography a confirmatory test for brain death along with clinical testing and other allied tests.

Transcranial Doppler sonography is performed to assess the circulation of basal cerebral arteries and to confirm cerebral circulatory arrest. However, its use requires an experienced operator and interpreter. Additionally, 10% to 20% of patients have insufficient bone windows for ultrasound transmission. Transcranial Doppler sonography is considered useful only if a reliable signal is found. It has been validated as an ancillary test for brain death, with specificity of 98% to 100% and sensitivity ranging from 88% to 99%.

Although optional, transcranial Doppler sonography is of special value when the therapeutic use of sedative drugs renders EEG unreliable. The main limitation of the use of ECG in these patients is the presence of central nervous system depressants.

Nowadays, the blind transcranial Doppler technique is increasingly being replaced by transcranial color-coded duplex sonography, since it is an easier, more informative, and more practical modality. A real advantage of transcranial color-coded duplex sonography over the blind transcranial Doppler technique is that the former is performed with the same transducers and equipment used for the most common sonographic examinations in critical care departments.

I present the case of a patient who was a potential organ donor, so there was a need to promptly confirm the diagnosis of brain death; an apnea test could not be performed, and the EEG result was inconclusive. There was no other diagnostic method available to confirm brain death; consequently, transcranial color-coded duplex sonography was used for this purpose.

A 60-year-old man with history of long-standing arterial hypertension was admitted to the intensive care unit (ICU) after surgical evacuation of a hypertensive brain stem hematoma. The patient was intubated, sedated, and mechanically ventilated. Vital signs revealed a heart rate of 70 beats per minute, a mean arterial blood pressure that varied between 70 and 90 mm Hg and a core temperature of 36°C. On initial physical examination, the patient had bilateral mydriasis with nonreactive pupils and absence of brain stem reflexes. Refractory intracranial hypertension (up to 40 mm Hg) was recorded on intracranial pressure monitoring. An intravenous norepinephrine infusion was necessary to reach optimal arterial blood pressure levels and an adequate cerebral perfusion pressure. The blood oxygen arterial pressure, oxygen saturation, and arterial carbon dioxide levels were maintained within the recommended values. No electrolytic abnormalities were noted on the blood sample. Twelve hours after admission, neurologic signs showed no modifications, and the sedative medications were interrupted. Because the patient had clinical signs compatible with the diagnosis of brain death, and he was considered a potential organ donor, confirmation of brain death was promptly required. Accordingly, the local organ procurement organization team was immediately notified. The apnea test was not performed because central nervous system depressant drugs were not absent, and hemodynamic instability was evident. An inconclusive EEG result for the diagnosis of brain death was obtained by the local organ procurement organization team 18 hours after admission to the ICU.

No other ancillary tests for confirmation of brain death were available; therefore, a bedside transcranial color-coded duplex sonographic examination was performed. The first sonographic examination was performed by an intensivist 24 hours after admission to the ICU and revealed a high-pulsatility flow spectrum in both middle cerebral arteries, which was nondiagnostic for cerebral circulatory arrest (Figure 1A). Doppler signals in the basilar artery could not be obtained. In a second transcranial color-coded duplex sonographic examination, performed 6 hours later by the same operator, obtaining both color and spectral Doppler signals in the intracranial arteries was a challenging procedure. However, a reverberating flow pattern was detected in the right middle cerebral artery (Figure 1B). At that moment, the bilateral distal portion of the internal carotid artery was interrogated and clearly revealed a reverberating or oscillating flow pattern, confirming the diagnosis of cerebral circulatory arrest (Figure 1C).
carotid artery. The latter is considered a specific but late finding for the diagnosis of cerebral circulatory arrest.8

Serial transcranial color-coded duplex sonographic examinations are also helpful to improve the sensibility of transcranial Doppler sonography for detection of cerebral circulatory arrest.2,4

A study of the orbital window (carotid siphon interrogation) should only be performed when a sonogram cannot be obtained through the temporal window. As for the occipital window, if there is no flow in the basilar artery, vertebral arteries can be insonated through the same window, at a lower depth. If both vertebral arteries show cerebral circulatory arrest patterns, it can be assumed that the flow in the basilar artery is equal or worse.2

The registration of each artery should be maintained for at least 30 seconds, recording the bilateral middle cerebral arteries and the basilar artery or the vertebral arteries.2 The study should be repeated after 30 minutes.2–4

Some prerequisites are necessary to avoid false results for the diagnosis of cerebral circulatory arrest as a confirmation of brain death2,3: (1) there should be evidence of cerebral injury; and (2) the patient must have normal hemodynamic findings, a core body temperature higher than 32°C, arterial PCO2 between 35 and 45 mm Hg, and absence of important metabolic abnormalities.3

The recommendation to perform 2 different transcranial Doppler explorations separated by 30 minutes is sustained by the fact that similar patterns of cerebral circulatory arrest can appear in transitory elevations of intracranial pressure (plateau waves), rebleeding after subarachnoid hemorrhage, and cardiac arrest. A period of 30 minutes is considered enough to confirm the irreversibility of cerebral circulatory arrest.2,3

The increase in intracranial pressure and subsequent decrease in cerebral perfusion pressure that occur in devastating brain injuries result in progressive changes in the Doppler spectral waveform. When the intracranial pressure equals the diastolic blood pressure, the brain is only perfused in systole. In this stage, the end-diastolic velocity in the Doppler spectra is 0. When the intracranial pressure equals the mean arterial pressure, brain perfusion ceases, and the Doppler spectra typically show an oscillating or reverberating flow pattern.2,4,9 When the intracranial pressure reaches the systolic blood pressure, sharp systolic spikes (systolic peak <50 cm/s and duration <200 milliseconds) are present on transcranial Doppler imaging.3 With time, the acoustic signal disappears. The existence of any anterograde diastolic flow does not correspond to a cerebral circulatory arrest pattern.5,3
The following 3 patterns are accepted as evidence of cerebral circulatory arrest: reverberant flow, systolic spikes, and disappearance of previously registered flow.\textsuperscript{2–4,9} Despite a clinical diagnosis of brain death, persistent antegrade diastolic flow could be present if the skull has lost hermeticity\textsuperscript{2,3,10} (ventriculostomy, decompressive craniectomy, opening of the calvarium, or skull base fracture), there is a traumatic arteriovenous fistula or anoxic-ischemic encephalopathy, or intra-aortic balloon counterpulsation is used.\textsuperscript{2} In other cases, such as severe brain stem injury and clinical diagnosis of brain death, middle cerebral artery Doppler findings can show a normal spectral pattern.\textsuperscript{3}

In conclusion, brain death is usually diagnosed in patients with severe neurologic injuries and artificially supported vital functions. At least 1 intensivist is usually involved in certifying brain death on most occasions. A matter of extreme importance, diagnosis of brain death is imperative when a potential organ donor is present. As such, transcranial color-coded duplex sonography may aid the rapid diagnosis of cerebral circulatory arrest to support the diagnosis of brain death. False-positive and false-negative results can occur, and these situations must be suspected when there is discordance between the clinical and transcranial Doppler findings. To ensure that the transcranial Doppler diagnosis of cerebral circulatory arrest is done to support the legal diagnosis of brain death, intensive care physicians must be properly trained. Each country or region may certify these physicians differently; a consensus group recommends a minimum of 50 transcranial Doppler studies in neurocritical patients and 20 diagnoses of cerebral circulatory arrest.\textsuperscript{2}

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