São Paulo (Brazil) July 24, 2023

To: The Honorable Samuel Thumma & All Members of the Determination of Death Act Committees & Style/Drafting Committees; Uniform Law Commission; 111 N. Wabash Avenue, Suite 1010; Chicago, IL 60602; E-mail: info@uniformlaws.org; Phone: (312) 450-6600

I am a Brazilian neurologist (Associate Professor of Neurology and Neuroscience at the federal University of São Paulo, Brazil since 1997) who had an additional training in pediatric neurology (Fellowship) in 1984 and 1985 at the Jackson Memorial Hospital (University of Miami). Further training on the pathophysiology of brain ischemia at the University of Lund (Sweden) in 1992 and 1993.

About irreversibility of damage to the whole brain.

The so-called "ischemic penumbra" is a phenomenon demonstrated in the brain of stroke patients since the early nineties and accepted worldwide as a scientific fact. The term ischemic penumbra applies to the state of reversible absence of neurological functions achieved when the brain tissue is supplied with reduced levels of blood flow (approximately lower than 50% and higher than 20% of the normal brain blood flow). In these circumstances the brain tissue remains viable, and normal neural function can be restored, provided that normal brain blood flow is timely achieved.

As published in 1999 (DOI: 10.1590/s0100-879x1999001200005), when the brain tissue is expanding within the rigid osseous cranial vault following, e.g., a transient ischemic state (lack of blood flow, like in cardiac arrest followed by reanimation) or a head trauma, the brain blood vessels are progressively compressed as intracranial pressure increases. Inevitably, at some point, the blood flow to the whole brain will be within the range of ischemic penumbra, when current criteria for brain death will be fulfilled. After the diagnosis of brain death, residual hypothalamic function has been demonstrated by low or even normal levels of hypophyseal hormones in the systemic circulation, which would not be possible if the blood flow to the hypothalamus were absent. In spite of that, currently "confirmatory" image tests (like cerebral angiography) do not detect the residual brain blood flow that is unquestionably present. This is a clear indication that the levels of blood flow proper of "ischemic penumbra" are not detected by those image tests.

In addition to that, the apnea test (done before the confirmatory image tests), as published by others, causes hypotension (thereby further reducing brain circulation and aggravating brain damage). The apnea test may even collapse brain circulation,

Dr. Cicero Galli Coimbra
CRM-SP 55.714
Clínica Médica - Neurologia
Professor Associado Livre Docente
Universidade Federal de São Paulo

thereby causing irreversible brain damage that supposedly would only demonstrate (please watch: https://www.youtube.com/watch?v=jAUKSP0OmUM&t=430s).

Moreover, impaired TSH (Thyroid Stimulating Hormone) synthesis caused by low levels of blood flow (proper of ischemic penumbra) to the hypothalamus may cause brain myxedema. In other words, patients who are candidates to the apnea test may have their brains in ischemic penumbra precisely because they are not receiving a life-saving replacement of thyroid hormones. As demonstrated in hypothyroid newborns and hypothyroid animals, the human brain may become edematous due to the lack of thyroid hormones. Conceivably, in victims of head trauma or transient cardiac arrest, low levels of thyroid hormones (hypothyroidism secondary to hypothalamic dysfunction) turn brain edema (initially caused by the traumatic or ischemic insult) into brain myxedema, giving rise to a vicious cycle that could only be interrupted by replacing thyroid hormones. If thyroid hormone replacement is not provided this vicious cycle is not resolved: brain myxedema sustains increased intracranial pressure which, in turn, sustains the state of ischemic penumbra and the progression of brain myxedema, thereby preventing the recovery of normal brain blood flow and resolution of coma.

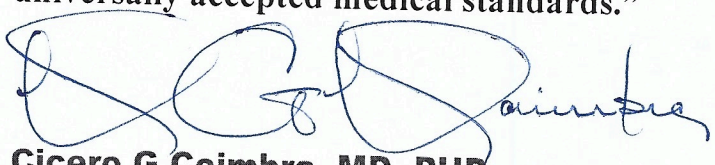
In 2011 I was asked to evaluate a 39-year-old female patient who had been submitted to an urgent abdominal second surgery four days earlier due to septic shock (secondary to rupture of intestinal sutures and leak of intestinal content into the abdominal cavity). She did not resume consciousness after the general anesthesia, and two days later the staff neurologist told the family that she was clinically brain dead, completely dependent on mechanical ventilation. A laboratory profile of her thyroid status revealed that her TSH and serum thyroid hormones were low, but detectable four days after the urgent operation associated with septic shock.

I prescribed 100 mcg of the thyroid hormone T4 twice a day. Eight days after she was breathing spontaneously. She was extubated and submitted to a tracheostomy. About one month later she was able to communicate with her parents and her sister by lip reading. THS levels rose progressively, but after 3 months she died due to pulmonary embolism. Conceivably, if thyroid hormone replacement would have started immediately after the initial brain insult, recovery to normal daily life could have occurred.

The Law ought to protect the person from being declared dead when still alive. Full and complete information about the apnea test and any tests used to declare "brain death" (BD) must be provided with freedom, at any time, by patients, surrogates, physicians, and other health care providers, to decline or cease the apnea test, exams, and protocols, for the determination and declaration of BD.

Dr. Cicero Galli Coimbra
CRM-SP 55.714
Clínica Médica - Neurologia
Professor Associado Livre Docente
Universidade Federal de São Paulo

Model Statute with minimum criteria required before death is:
"No one shall be declared dead unless respiratory and circulatory systems and the entire brain have been destroyed. Such destruction shall be in accord with universally accepted medical standards."



Dr. Cicero Galli Coimbra
CRM-SP 55.714
Clínica Médica - Neurologia
Professor Associado Livre Docente
Universidade Federal de São Paulo

Cicero G Coimbra, MD, PHD

Cicero G Coimbra, MD, PHD
Internal Medicine and Neurology
Associate Professor of Neurology and Neuroscience
Department of Neurology and Neurosurgery
Federal University of São Paulo - UNIFESP

Disciplina de Neurociência
Escola Paulista de Medicina - Universidade Federal de São Paulo
Rua Pedro de Toledo, 669 - 2 andar
Vila Clementino, São Paulo / SP
CEP 04039-032