Supplementary Table 1. Definitions of various etiologies of early neurological deterioration according to our center's predefined registry

Etiology of early neurologic Deterioration (END)	Definition
Reversible	
Infectious	END in the presence of a diagnosed infection (e.g., of the urinary or respiratory tracts, cerebral ventricles or meninges, skin or soft tissue, blood vessels or heart valves)* with identified pathogen on microbiology or consolidation on chest imaging with clinical symptoms, in the case of pneumonia
Metabolic derangement	END in the presence of new laboratory derangement based on our center's laboratory standards (e.g., uremia, hyper/hypocal cemia, hyper/hypoglycemia, hyper/hyponatremia, or hyperammonemia)
Hemodynamic	END in the presence of a significant rise or fall in blood pressure during the 24 hours prior to documentation of END as de termined clinically, new-onset arrhythmia as determined via electrocardiography or telemetry, or cardiovascular event (in cluding myocardial infarction) as determined via electrocardiographic, telemetric, and/or laboratory methods (for instance, elevated serum troponin I level†) that may have impaired cerebral perfusion but did not cause a new imaging-confirmed infarction
Edema	END when focal cerebral or cerebellar mass effect was identified on follow-up computed tomographic or magnetic reso nance imaging in the 24-hour window surrounding the date of END
Fluctuation	END in the absence of an identifiable cause should the patient return to prior (day before END) National Institutes of Health Stroke Scale score within 24 hours of the episode of decline
Toxicity	END co-occurrence with the administration of medication with known sedative effects
Seizure	END in the presence of electroencephalogram-confirmed epileptiform activity or periodic rhythm or clinical observation of seizure-like activity during the 24-hour window surrounding documentation of END
Nonreversible	
New stroke	END in the presence of new ischemic findings on CT or MRI (detected within the 24-hour window surrounding documenta tion of END)
Progressive stroke	END due to extension of ischemic findings as detected on computed tomographic or magnetic resonance imaging by the at tending physician or staff radiologist within the distribution of the initial vessel occlusion (during the 24-hour window sur rounding documentation of END), and no clear focal cerebral edema identified on neuroimaging
Hemorrhagic conversion	END due to new or progressive hemorrhage of parenchymal hematoma type 1 or 2 grade was identified on 24-hour follow-up imaging study
Cardiopulmonary arrest	END following cardiopulmonary arrest
Unknown etiology	END without a clear etiologic identification by the attending physician, and none of the aforementioned findings were docu mented in the progress notes

*Bacteremia was defined growth of bacterium on culture from a venous blood sample (excluding common contaminants). Urinary tract infection was defined as > 10,000 colony forming units per millimeter of urine in a clean-catch specimen (excluding contaminants) or suggestive findings on urinalysis. Pneumonia was defined as an infiltrate on chest radiography with appropriate clinical correlates. Other infection types were diagnosed clinically or via laboratory/imaging findings. Clinical symptoms producing END may have occurred hours or days prior to the diagnosis of END due to infection, depending on the manner in which the infection was diagnosed. For example, pneumonia diagnosed via chest radiography in the presence of clinical symptoms may only take one hour for the imaging order to be processed, performed and interpreted, whereas confirmation of bacterial specimen in blood culture may have taken up to 5 days according to our laboratory standards; †Elevated serum troponin I is defined as troponin I level > 0.015 mg/dL, according to our laboratory standards.