THE EPIDEMIOLOGY OF VEGETATIVE STATE

Ettore Beghi, MD, Department of Neurology, University of Milano-Bicocca, Monza, Italy
Laboratory of Neurological Disorders, Institute for Pharmacological Research "Mario Negri", Milano, Italy.

According to the Multisociety Task Force on PVS, the vegetative state (VS) is " ... a clinical condition of complete unawareness of the self and environment, accompanied by sleep-wake cycles with either complete or partial preservation of hypothalamic and brain stem autonomic functions." (1). The condition may be transient or permanent and occur in the context of several clinical conditions, including acute or CNS progressive injuries, metabolic disorders and developmental malformations. The VS is lack of awareness at the presence of wakefulness. Although the VS can be diagnosed according to well-defined criteria (1), the terminological confusion behind the definition of VS and related neurological conditions (coma, brain death, locked-in syndrome, akinetic mutism, neocortical death, apallic state, minimally conscious state) may explain in part the wide variability in the estimates of the frequency of this condition. According to clinical series, the prevalence of VS varies in different countries from 60 to 140 per million population (about one third in children)(2). The mean annual incidence varies from 14 to 67 per million population at six months (3). The commonest causes of the VS are head trauma and anoxic encephalopathy. Persistent VS develops after prolonged traumatic coma in 1-14% of cases and in 12% of those with non-traumatic coma. Although there are no well-defined predictors of vegetative outcome, advanced age, pupillary abnormalities, poor motor responses, ventilatory dysfunction, decorticate posturing, and trauma outside the nervous system soon after the insult have been found to predict post-traumatic VS (4,5). As well, altered eye opening abnormal oculocephalic or motor responses, and inability to obey commands at two weeks may predict VS in non-traumatic coma (6).

The prognosis of VS depends on the underlying etiology. Duration of VS and age are other significant prognostic predictors. In a series of 434 patients in a VS for one month after traumatic brain injury, 67% had died or remained in VS at three months (7). Recovery had occurred in 33% of cases at three months, 46% at six months, 52% at 12 months, and 59% after 12 months. In the same series, functional recovery at 12 months, measured by the Glasgow Outcome Scale, was as follows: death 33%; persistent VS 15%; severe disability 28%; moderate disability 17%; good recovery 7%. After traumatic brain injury, patients older than 40 years had a poorer outcome compared to those who were younger (5). Other adverse prognostic indicators include ventilatory dysfunction, lack of early motor response, late-onset seizures, and hydrocephalus (4,8). By contrast, non-traumatic brain injury has a poorer prognosis, with about 85% dying or remaining in a VS within the first month. Recovery of consciousness is present in only 11% of cases at three

months. Recovery of function in patients regaining consciousness is almost nil (7). The recovery of consciousness in children with traumatic brain injury is slightly better while recovery of function is similar to that of adults (7). By contrast, in children the prognosis of non-traumatic injury is slightly better for recovery of function but comparable to adults for recovery of consciousness (7). Only few patients with traumatic VS persisting for 12+ months or with non-traumatic VS persisting for 3+ months may later present recovery of function. VS caused by degenerative or developmental disorders are unlikely to present recovery.

In patients with VS average life expectancy is reduced to about 2-5 years. At 12 months, 33% of adults with traumatic brain injury and 53% of those with non-traumatic injury have died (children 9 and 22%)(7). A large population-based study of 847 children and adults in a persistent VS reported a significantly shorter survival in children aged less than one year compared to older children and adults (2). Reported causes of death include age, infection, generalized systemic failure, and recurrency of underlying disorder (8-10).

References:

1. The Multisociety Task force on PVS. Medical aspects of the persistent vegetative state – First of two parts. N Engl J Med 1994;330:1499-1508.
2. Ashwal S, Eyman RK, Call TL. Life expectancy of children in a persistent vegetative state. Pediatr Neurol 1994;10:27-33.
3. Jennett B. The vegetative state. J Neurol Neurosurg Psychiatry 2002;73:355-357.
4. Sazbon L, Fuchs C, Costeff H. Prognosis for recovery from prolonged posttraumatic unawareness: logistic analysis. J Neurol Neurosurg Psychiatry 1991;54:149-152.
5. Braakman R, Jennett WB, Minderhoud JM. Prognosis of the posttraumatic vegetative state. Acta Neurochir (Wien) 1988;95:49-52.
6. Levy DE, Caronna JJ, Singer RH, et al. Predicting outcome from hypoxic-ischemic coma. JAMA 1985;253:1420-1426.
7. The Multisociety Task force on PVS. Medical aspects of the persistent vegetative state – Second of two parts. N Engl J Med 1994;330:1572-1579.
8. Sazbon L, Groswasser Z. Medical complications and mortality in patients in the postcomatose unawareness (PC-U) state. Acta Neurochir (Wien) 1991:112:110-112.
9. Dougherty JH Jr, Rawlinson DG, Levy DE, Plum F. H. Hypoxic-ischemic brain injury and the vegetative state: clinical and neuropathologic correlation. Neurology 1981;31:991-997.

10. Higashi K, Sakata Y, Hatano M, et al. Epidemiological studies on patients with a persistent vegetative state. J Neurol Neurosurg Psychiatry 1977;40:876-885.