

**ABSTRACT**

Central nervous system (CNS) damage is often associated with robust body temperature changes, such as hypothermia and delayed hyperthermia. Hypothermia is one of the most common body temperature changes to CNS insults in rodents and is often associated with improved outcome. Although the acute hypothermia seen in rodents is rarely observed in humans, clinical methods to induce hypothermia have shown promise for the protection against CNS damage. The hypothermic response to CNS injury is thought to provide protection against the insult by reducing metabolic demands and the generation of harmful reactive oxygen species. The most common body temperature response observed in clinical studies of CNS damage is delayed hyperthermia, which is often referred to as “fever”. This elevation in body temperature may persist for several days or be recurrent and episodic in nature, but regardless of its periodicity it is typically associated with poor prognosis to a variety of CNS injuries. The tendency to refer to this response as a fever is somewhat misleading since the mechanism(s) mediating this elevation in body temperature remain poorly understood. Interestingly, hyperglycemia is another pathophysiological response associated with CNS damage that aggravates morbidity and mortality. It is thought that elevated body temperature and hyperglycemia may have synergistic actions that are deleterious for recovery, although the mechanisms responsible for these effects remain to be elucidated. This review discusses the incidence of hypothermia and delayed hyperthermia during recovery from a variety of CNS insults and proposes putative mechanisms that mediate these responses as well as the potentially deleterious interaction of high body temperature with hyperglycemia.

**Key words:** delayed hyperthermia; fever; hypothermia; CNS injury; hyperglycemia