The relationship between physical trauma and onset or relapse of multiple sclerosis (MS) has been a controversial issue and a topic to be debated in the neurological literature (Poser, 1994, 2000; Kurland, 1994; Cook, 2000). Although the probability that physical trauma might be associated with the causation or exacerbation of MS dates back to the time of Charcot, the first controlled study on this issue was reported by McAlpine and Compston in 1952, which favored such a relationship. However, as there were major concerns regarding the design of this retrospective case-controlled study, the evidence presented was rather weak. The following studies regarding the association between trauma and MS gave controversial results but all had methodological limitations until the work of Sibley et al in 1991. The results of their prospective cohort study clearly showed no association between physical trauma and MS. The population based Mayo study that was published two years later (Siva et al, 1993), and The Gusev et al. study in 1996, both despite some limitations also provided good Class II evidence (Evidence provided by one or more well-designed clinical studies such as case control and cohort studies) against an association between head trauma and MS.

Based on these and all other related works, in a report of the Therapeutics and Technology Assessment Subcommittee of the American Academy of Neurology, it was concluded that there was no association between physical trauma and either MS onset or MS exacerbation (Goodin et al. 1999).

Recently in a large record linkage study, Goldacre et al also reached the conclusion that injuries to the head were not associated with either the aetiological initiation or the clinical precipitation of onset of multiple sclerosis (2006).

The belief that trauma may precede or exacerbate MS as summarised has come primarily from anecdotal reports and limited case series that provide no rates and no basis for critical comparison. The proponents of the hypothesis that physical trauma may cause or exacerbate multiple sclerosis, have postulated that physical trauma and mostly head and neck trauma causes an increase in the permeability of the blood-brain barrier that in turn is responsible for the formation of the MS plaque through facilitating an immune-mediated demyelination in these patients (Poser, 1986 & 1994). Although such a speculation is conceptually plausible, it is not at all clear whether trauma-related abnormalities in the BBB always occur and to what extent. Furthermore, when there are such changes, it is also not known that they may result in immune-mediated responses. There is no doubt that the change in the BBB permeability is a well known fact in the pathogenesis of MS. However, current evidence favor that this change is not the initial event but rather a consequence of the ongoing immune reaction. Recent evidence from MRI spectroscopy and MRI diffusion studies also indicates that abnormalities can occur in normal-appearing white matter before gadolinium enhancement is detected, suggesting that brain inflammation may cause a secondary change in the BBB rather than the opposite (Brassat et al 1999).

In conclusion, currently the available scientific data fail to show any association between MS onset or MS exacerbation.