Recovery patterns from focal traumatic lesions have not been well described . The clinical natural history should be defined by: location, size, bilaterality, and concomitant pathological factors. There is an acute phase, when problems may appear more widespread an less localizable, reflecting in large part the extent of localized reactive pathophysiological processes and diffuse pathologies. There is a subacute phase when localizing effects may be more distinct, and a chronic phase with relatively stable deficits, which are direct consequences of a fixed lesion. The point of stability may be reached in months. Of course, even after reaching a pathophysiological endpoint, new learning, proceduralization of compensatory skills, and overall plasticity of brain functions will allow functional change beyond that point. (Finger S and Stein D, 1982)

When the lesion is diffuse the pattern of recovery is qualitatively similar across the range of from most mild to severe. Loss of consciousness occurs immediately and emergence from unconsciousness is followed by a proportionally longer period of confusion and amnesia; when confusion and amnesia clear, a yet longer period of residual impairment and restoration of function occurs. The duration of each of these periods is unequivocally related to severity of diffuse axonal injury.

There is increasing evidence that central nervous system neurons are capable of dendritic and axonal sprouting following an injury in a variety of animal species. This represents a potential mechanism for reestablishment of neural connections over time that could contribute to functional recovery. It is possible that there is a role for experience and functional training in promoting fiber sprouting or in selecting and perpetuating new “useful” connections.