Post-traumatic amnesia (PTA) is common early after traumatic brain injury (TBI). Its pathophysiology is poorly understood. The hippocampus is central to memory processing, and normally
shows strong functional connectivity to nodes within a large-scale intrinsic connectivity network, the default mode network (DMN). Using structural and functional MRI, we investigated the neural basis of PTA in a series of patients with varying cognitive impairment acutely after TBI, as well as controls. One patient had profound PTA two days after TBI, and had small haemorrhages bilaterally within the hippocampi. Functional connectivity was abnormally low between the hippocampi and the posterior cingulate cortex, the central node of the DMN. This was associated with abnormalities in measures of memory and information processing speed. The patient was followed up five months later. At this stage her memory function had largely normalised, the hippocampal lesions had almost resolved, and functional connectivity between the hippocampus and the posterior cingulate cortex had normalised. The results suggest that hippocampal pathology is involved in the causation of PTA, and that transient functional disconnection between brain regions involved in memory formation, including nodes within the DMN, may underlie the profound cognitive impairments seen in PTA.