The neuropsychological sequelae of attempted hanging

Alice A Medalia, Arnold E Merriam, John H Ehrenreich

Abstract

Only one report on the neuropsychological sequelae of attempted hanging exists in the English language. Two cases of attempted hanging with subsequent isolated memory deficits are reported. Possible mechanisms for induction of this amnesia are discussed. In these two cases it is most likely that circulatory disturbance produced by the ligatures caused ischaemic hippocampal damage, which in turn led to amnesia.

Every year approximately 4000 people in the United States commit suicide by hanging.1 While no precise figures are available, it is safe to assume that additional thousands unsuccessfully attempt suicide by this method. Nevertheless, relatively little is known about the neuropsychological sequelae of failed hanging. In the only English language report on the subject,2 attempted hanging was described as the cause of a persistent anterograde amnestic syndrome resembling that seen in Korsakoff’s syndrome. We describe two cases of attempted hanging with subsequent anterograde memory deficits.

Case 1

A 26 year old man with a history of polysubstance abuse (which from the report did not include IV drug administration) had attempted suicide three times over the three years preceding our assessment of him. Information about the first two attempts, which occurred three days apart, is scant. First he slashed his throat and then he stabbed himself with a fork. Apparently the wounds were superficial; there was no substantial blood loss or ventilatory obstruction. Psychometric testing seven weeks later revealed no signs of higher cortical dysfunction and specifically no memory impairment, although it did suggest the presence of psychosis. His intelligence was found to be in the average range (Performance IQ = 94; Verbal IQ = 95; Full Scale IQ = 94).

The third suicide attempt, a year later, was by hanging. The patient was cut down unconscious and incontinent of urine. Detailed information about the immediate medical consequences of this incident, including the duration of unconsciousness before and subsequent to discovery is not available. He was admitted to a general hospital for medical treatment and within a week was admitted to a psychiatric hospital for treatment of a paranoid psychotic condition that was marked by paranoid delusions, thought blocking and suspiciousness. At that time he had a gross impairment in recent memory. Psychological testing carried out three weeks after the hanging found severe memory deficits on the recall portion of the Bender Gestalt Test, a measure of nonverbal memory. IQ testing revealed a sharp drop in intelligence from the prehanging level (Performance IQ = 82; Verbal IQ = 80; Full Scale IQ = 80). A neurological examination three months after the hanging was unremarkable except for the memory impairment. Contrast CT was normal. Seventeen months after the hanging his psychosis had remitted and mental status was remarkable only for persistent memory deficits.

Neuropsychological testing performed two years after the hanging when he was nonpsychotic, revealed a return to his premorbid intellectual level which was in the average range (Performance IQ = 104; Verbal IQ = 93; Full Scale IQ = 97). His Performance IQ was substantially higher than the prehanging assessment which was performed when he was psychotic. This was interpreted as compatible with the improvement in task performance that can be seen when psychosis remits. Language, somatosensory, visual-spatial, constructional, abstracting and grapho-motor abilities were intact. He did not show the expected right hand dominance on a test of fine motor coordination; whether this finding predates or is a consequence of the hanging cannot be determined.

The one abnormal feature in his otherwise unremarkable performance on neuropsychological tests was his dense anterograde and retrograde amnesia. He did not recall the hanging incident, the subsequent admission to hospital or previous psychological assessments. He remembered events in his life up to three years before the hanging, but did not recall the details of his life from that time forward. On formal testing he showed a marked verbal and nonverbal memory deficit. He was unable to learn more than seven of a list of 15 words after five trials and was very sensitive to proactive interference. Attentional capacity was normal; his digit span was better than average. Scores on psychological tests are presented in the table.

Case 2

A 28 year old man with a history of polysubstance abuse had attempted to hang himself three years before our assessment of him. He was reported to have been anoxic for eight minutes before a tracheotomy was performed.
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IQ = Wechsler Adult Intelligence Scale- Revised; WMS = Wechsler Memory Scale-Revised; Ravens Progressive Matrices is a test of visual-spatial and reasoning skills; Spreen Benton Token Test is a language test; Block Design from the IQ test is a test of constructional skills.

He was comatose for several days afterwards and remained in a general care hospital until his transfer to a psychiatric facility two months later because of intermittent agitation and persistent memory deficits.

He was not maintained on psychotropic medication. Neurological examination three years post-hanging was normal except for mild bradykinesia and tremor. CT and MRI done at that time were normal. Neuropsychological testing indicated borderline normal intelligence. Although this was entirely compatible with his premorbid abilities as estimated from educational and vocational history, a mild degree of generalised cognitive impairment resulting from the hanging cannot be excluded. Arithmetic, abstraction, visual-spatial perception, constructional praxis and language were intact. Memory, however, was severely impaired. A retrograde memory deficit was evident in his amnesia for the hanging and the events surrounding that time. There were significant verbal and nonverbal anterograde memory deficits; retrieval was more impaired than storage. List learning was limited to six of 15 words after five trials and he was very sensitive to proactive interference. Memory for newly learned information declined significantly after a delay of 30 minutes. His attention and concentration were intact. Results of IQ and memory testing are presented in the table.

Discussion

This report describes the second and third cases of amnesia secondary to attempted hanging that have been reported in the English language literature. Both cases are remarkable for the severity of anterograde amnesia in the context of relative preservation of other cognitive capacities. What was the mechanism by which the amnesia was induced?

Prominent amnestic syndromes have occurred after hypoxia, after ischaemic insults due to mechanical posterior circulation interference or posterior circulation thrombotic events, and in the setting of delayed post-anoxic encephalopathy (see below). Each of these mechanisms may be set in motion by attempted hanging. The two principal means by which hanging induces brain damage are cerebral ischaemia resulting from ligature-induced obstruction of cerebral blood flow and, to a lesser extent, cerebral anoxia resulting from asphyxia due to mechanical airway obstruction. Reports of successful hanging in individuals with tracheotomies at a level lower than the ligature, suggest that asphyxia alone does not play a lethal role, although upward displacement of the tongue and glottis are in some cases sufficient to occlude the airway. The relative contributions to the morbidity and mortality of interruptions to the anterior and posterior cerebral circulation induced by hanging have not been well delineated. There is evidence that flow ceases in both circulations after a 16 kg pull on a noose, far less than is created when an individual's weight is truly suspended by the ligature. The survivor of an attempted hanging might therefore theoretically suffer from the effects of cerebral circulatory insufficiency and/or the effects of asphyxiation. Additional mechanisms by which near-hanging has the capacity to produce neurological deficits in survivors are by mechanical interruption to flow in the vertebral arteries as a result of extension, rotation, and traction of the neck, by the instigation of arterial thromboses with resulting cerebral infarction, and by the induction of a delayed post-anoxic encephalopathic syndrome.

In most cases the dual insults of arterial hypotension and arterial hypoxia coexist to some degree, hence the term "cardiopulmonary arrest". Amnesia is a well-recognised sequel of cardiopulmonary arrest, and neuropathological correlation in such a case suggests that the amnesia is mediated by bilateral hippocampal injury. Neuropathological findings in this case consisted of atrophy, neuronal loss, and glial proliferation. In circumstances other than combined cardiac and pulmonary arrest a relatively pure hypotensive or hypoxic state may occur; perhaps the best examples are cardiac arrest while intubated and ventilated during general anaesthesia (pure ischaemia) and carbon monoxide poisoning without circulatory collapse (pure hypoxia). Prominent amnestic syndromes have been reported in both of these settings. While pure circulatory collapse can evoke a profound amnestic syndrome as an immediate sequel, carbon monoxide poisoning does so only after a latency of a month, as a manifestation of the delayed encephalopathy characteristic of this disorder. The state of delayed carbon monoxide encephalopathy with multiple cognitive defects has also been reported to resolve, but for the presence of persistent memory deficits.

As for the issue of vertebral artery insufficiency provoked by mechanical neck twisting, extension, and traction, persistent memory defects may be caused by posterior circulation insults initiated by this mechanism during vigorous chiropractic manipulation. In this case, however, the patient additionally suffered a bilateral homonymous hemianopia due to ischaemia of both occipital lobes. In a case of death induced by cervical manipulation, necropsy examination revealed...
the presence of arterial thromboses presumed to be secondary to vascular damage. The relative contributions of thrombotic and temporary mechanical interruption to flow are likely to be similar.

One of the two patients we have presented exhibited no neurological findings other than amnesia, while the other exhibited only mild bradykinesia and tremor in addition to the memory defect. The onset of the neurological and neuropsychological symptoms in both cases was simultaneous to the hanging episode, obviating the diagnosis of a delayed postanoxic syndrome. Neither patient required ventilatory assistance after rescue. This weight of evidence is that both cases of amnesia occurred primarily on the basis of circulatory interruption produced by the ligatures, rather than on the basis of ventilatory impairment. The circulatory disturbance may have caused ischaemic hippocampal damage which in turn led to amnesia. In the absence of radiological data confirming the presence of such lesions, the anatomical basis of the amnesia remains speculative. These patients, however, did have temporarily limited retrograde amnesia and profound anterograde amnesia, an amnestic profile often seen with hippocampal lesions.

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