Double lucid interval in patients with extradural hematoma of the posterior fossa

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The classic clinical sequence in posterior fossa extradural hematoma is head injury, lucid interval, deterioration; the deterioration is usually terminated by death or surgical intervention. This report emphasizes a syndrome characterized by a double lucid interval in which the phase of deterioration terminates spontaneously with improvement. This is actually only a second lucid interval and can lead surgeons into a false sense of security since it is followed by deterioration, as the clot continues to enlarge. Four such cases are described in detail. The characteristics of the initial phase of deterioration are described and contrasted with those of the deterioration following the lucid interval in the classic supratentorial extradural hematoma.

Key Words: extradural hematoma · posterior fossa · double lucid interval

To neurosurgeons, the term “lucid interval” refers to a period of relative well-being after a head injury which implies that any subsequent deterioration is probably due to an accumulating clot, often extradural. In the glossary prepared by the Congress of Neurological Surgeons1 for head injuries, the term “lucid interval” is not defined. We suggest a convenient measure would be the quality of the patient’s response to the spoken word. With death as the baseline and the pre-accident state as the starting point, the ability to respond to voice characteristically occupies a level somewhere in between (Fig. 1). During the lucid interval the patient is never quite his normal self if only to the extent that he has some headache and a desire to be less active. The lucid interval may last a few seconds or a few days but is usually a few hours, and is terminated by a progressively rapid decline in the level of consciousness. Timely surgical removal of the clot results in an equally rapid restoration of the pre-accident state of well-being. Death is the alternative (Fig. 1). Should spontaneous recovery occur after a sequence of head injury-lucid interval deterioration, then it would be quite natural to suspect some other cause for the deterioration, such

![Fig. 1. Diagram of clinical course of a typical supratentorial extradural hematoma. With death as a baseline and a patient's pre-accident state of well-being as a normal level, the quality of the patient's response to voice is arbitrarily indicated as lying midway in the intervening levels of consciousness. Time is indicated in hours. Surgical intervention is indicated at Point S, and the continuing dotted line indicates inevitable death if surgery is delayed.](https://example.com/fig1.png)
Double lucid interval in extradural hematoma

as shock, drug intake, or fat embolism. We have recently observed a case and found three others in the literature that proved to be an exception in that the patients had a "double lucid" interval.

Case Report

The patient was an 8-year-old boy who fell while swinging from an overhead bar and struck the back of his head on the left.

First Lucid Interval. The patient was pleasant and cooperative when examined, and complained of a left frontooccipital headache. He had a tender swelling over the left occiput, was hyporeflexic, slightly ataxic in gait, and had normal skull films. During the next 36 hours no significant deterioration was noted.

First Deterioration. The patient then began to vomit and became progressively drowsy. At 38 hours he ceased to respond to voice, and at 40 hours his only response to pain was sluggish withdrawal. A left carotid angiogram at that time revealed no evidence of a supratentorial mass. The sweep of the anterior cerebral was considered to be slightly widened; the lateral sinus was not obviously separated from the bone.

Second Lucid Interval. The patient then stabilized and began to improve. Eight hours later he was up and about the ward eagerly feeding himself and answering questions appropriately. He had thus passed through the sequence of head injury-lucid interval deterioration and recovered spontaneously to a second lucid phase (Fig. 2). He continued on in this state of relative well-being day after day. He acknowledged some headache and was hyporeflexic and minimally ataxic. He was being considered for discharge on the 7th day.

Second Deterioration. On the 7th day, the patient developed diplopia due to a left lateral rectus paresis. On the 10th day bilateral papilledema with fresh hemorrhages was noted along with some nuchal rigidity. On the 16th day the patient became again progressively drowsy. A ventriculogram was followed by removal of a predominantly left-sided posterior fossa extradural hematoma. He made a complete and uneventful recovery.

Comparable Cases

In the various reports since McKenzie's original clinical description, attention has been drawn to the high mortality rate from unrecognized lesions and to the wide variation in the length and character of the lucid interval, but in none of the reports could we find recognition of a "double lucid interval" sequence. However, reviewing these reports we found three cases which actually did follow this same general pattern.

All three cases started with occipital trauma. Two had linear occipital fractures. The initial lucid interval was associated with some headache and cerebellar signs, and lasted from 8 to 40 hours. The initial period of deterioration and subsequent recovery was gradual over a period of many hours or days, and led into a second lucid interval which again was associated with some headache and minimal cerebellar signs and went on unchanging day after day. In one case a posterior fossa extradural hematoma was removed on the 11th day. In the second case papilledema and lateral rectus paresis developed on the 12th day and a posterior fossa extradural hematoma was removed on the 16th day. Both of these cases made complete and uneventful recoveries. The third patient was sent home during the second lucid interval and on the 15th day died at home. A posterior fossa extradural hematoma was removed at autopsy.

Discussion

In the classic supratentorial extradural hematoma the decline in level of consciousness is associated with one or more phenomena.
such as irritability, convulsion, stertorous respirations, widening pulse pressure, slowing pulse rate, hemiparesis, and dilating pupil. Not one of these was present in our case nor evident in the three cases from the literature.

Our patient remained flaccid with near normal pulse, respiration, and blood pressure until he became deeply comatose and both pulse and respiration became irregular. The pupils remained small and equal. On the whole it was a quiet descent into coma more suggestive of fat embolism or drug overdose than that of clot accumulation. Although the angiogram had given us a clue, once the patient began to recover our concern with an accumulating clot became progressively remote.

The mechanism remains obscure. The clot in our case was old and of uniform consistency, with nothing to suggest two separate hemorrhages 16 days apart or continuing bleeding. We can only speculate as to periods of compensation and subsequent decompensation.

Two features warrant emphasis. First, a surgeon who finds himself observing a period of apparently spontaneous recovery after deterioration must not be lulled into a false sense of security that might lead to his sending the patient home, as we nearly did. Second, there really should never be a second lucid interval. The sequence of occipital trauma, lucid interval associated with some cerebellar signs, and nuchal rigidity in turn followed by the quiet development of coma is typical for some cases of posterior fossa extradural hematoma and should be recognized for what it is, an indication for immediate surgical treatment of the clot.

References
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