Brainstem Auditory Evoked Potential in a Case of Severe Carbon Monoxide Intoxication

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A 36-year-old man was brought unconscious to the emergency room; he suffered anoxic brain damage due to carbon monoxide (CO) intoxication, and had decerebrate rigidity clinically and died 1 month later after the acute insult.

Computed tomography with contrast enhancement failed to show a brainstem lesion, but the brainstem auditory evoked potential (BAEP) demonstrated the brainstem involvement.

The BAEP can be used as an objective diagnostic aid for evaluating brainstem lesions in CO intoxication.

Key Words: Brainstem auditory evoked potential, CO intoxication.

Anoxia is believed by some to be main pathological factor in acute carbon monoxide (CO) intoxication and the human brainstem is considered relatively invulnerable to anoxia, only occasionally being involved in cerebral ischemia in man. Changes in the brain after acute CO intoxication have mainly been investigated at necropsy. Although assessing brainstem function using laboratory tests is difficult, brainstem auditory evoked potential (BAEP) recently has been used as an electrophysiological means of localizing the brainstem lesion in neurological diseases. We report a case of severe CO intoxication in which the brainstem lesion was demonstrated by BAEP.

REPORT OF A CASE

A 36-year-old man was found unconscious in a closed room at 8:30 AM on April 30, 1983. He had been seen last at approximately 11:30 PM the night. At first, he was brought to the emergency room of a rural hospital. On admission, the patient was unconscious. Temperature was 37.2°C; pulse rate 90 beats per minute; respiration, 24/min, and blood pressure, 130/80 mmHg.

Examination of the head and neck disclosed no evidence of trauma. Pupils were equal and reacted normally. There was no papilledema. The neck supple and without adenopathy. The carotids were normal and no jugular venous distention was present. The chest was clear; the heart and abdominal examination were unremarkable. The patient exhibited decerebrate rigidity and response to deep pain was elicited. Eye movement was elicited by doll's head maneuver. The muscle stretch reflexes were normal. There was ankle clonus and Babinski's signs bilaterally.

Received September 8, 1983
The hemoglobin level was 16.9 g% and the leukocyte count was 13,800/mm³; the serum electrolyte levels were normal. The serum SGOT & SGPT were 78 and 182 IU/1. The COHg concentration was 54%. Blood drawn while the patient was breathing room air demonstrated a pH of 7.42; PO2 was 60 mmHg; PCO2 41 mmHg; O2 saturation, 95%; base excess, 2.5. Chest and skull X-rays were normal.

He was immediately treated with hyperbaric oxygen therapy in the emergency room under the diagnosis of CO intoxication. After admission, he was managed in the intensive care unit, but there was no improvement.

The patient was transferred to Severance hospital, Seoul at 3 PM on May 11, 1983.

On admission, the patient was unconscious. Vital signs were stable. He exhibited decerebrate rigidity, and response to deep pain was elicited. Bilateral ankle clonus and Babinski’s signs were elicited. Others were normal.

Complete blood cell count, urinalysis, 12-factor blood chemistry studies and serum electrolytes were normal. ECG showed ST-T changes on all leads. EEG revealed diffuse delta ranged slow waves without normal background activity.

C-T scan with contrast enhancement 21 days after anoxia showed decreased densities on both basal ganglia and the white matter of both cerebral hemispheres, and the visualized brain stem was unremarkable (Fig. 1).

On May 22, the ipsilateral and contralateral BAEPS (using the Nicolet CA 1000) were obtained after monoaural stimulation; no potentials in the left BAEP, and normal latency to wave 1 (1.92 msec) but prolongation of latency to waves II, III and V (3.20, 4.76 and 6.96 msec) and prolongation of II-III and II-V interwave latency
Fig. 2. BAEPs showed prolongation of latency to waves II, III and V, and prolongation of II-III and II-V interwave latency in right side, and no potentials on the left side.

(1.56 and 3.76 msec) in ipsilateral and contralateral right BAEP (Fig. 2).

After admission, he was managed with conservative treatment. On May 31, vomiting suddenly developed and respiratory failure followed. Chest X-rays taken at that time disclosed no abnormality.

He died on June 1, 1983.

Autopsy could not be permitted.

**COMMENT**

CO reversibly interact with hemoglobin and cause tissue anoxia. There are at least 2 topographical patterns of anoxic ischemic alterations in the central nervous system; a rostrocaudal pattern of decreasing vulnerability and a pattern of brainstem and thalamic damage (Leech and Alvord, 1977). In the human adult, there is the well recognized rostrocaudal pattern of decreasing vulnerability with the cerebral cortex, the hippocampus and cerebellar cortex being most sensitive and the brainstem least sensitive. The brainstem involvement in the adult is occasionally seen and is usually restricted to the substantia nigra, inferior colliculi and inferior olives, but brainstem damage is common in infants and children with anoxic accidents (Brierley et al, 1973; Leech and Alvord, 1977).

When the patient exhibited decerebrate rigidity, brainstem involvement was suspected
clinically (Plum and Posner, 1980), but assessing the brainstem function using laboratory tests was difficult.

C-T scan can establish the lesions of the basal ganglia and cerebral hemispheres in CO intoxication, but the brainstem lesion is usually missed (Sawada et al, 1980; Choi, 1982).

BAEP is thought to be the far-field recording of sequential electrophysiologic events at successively higher levels of the brainstem auditory pathways in animals, the primary generators of waves I-V are the acoustic nerve, the cochlear nuclei, the superior olivary complex, the lateral lemniscus, and the inferior colliculi, respectively. In humans, a similar location of BAEPs was reported (Brierley et al, 1973; Stockard and Rossiter, 1977). Thus, BAEPs have been used as an electrophysiologic means of localizing the brainstem lesions. According to Starr and Achor (1975), when coma was due to toxic or anoxic factors, BAEPs were usually normal with regard to latency and amplitude of all of the components.

In our case, the patient had clinically decerebrate rigidity with prolonged coma which suggested brainstem involvement. C-T scan showed the lesions in the basal ganglia & cerebral hemispheres, but failed to show the brainstem abnormality.

BAEPs revealed the abnormal findings; prolongation of latency to wave II, III & V on right side that suggested involvement of pons and midbrain, and no potential on the left side that might be the damage to the cochlear nerve due to CO intoxication (Choi, 1982). So we think the BAEP can be used as an objective diagnostic aid for evaluating brainstem function in CO intoxication.

REFERENCES


Stockard JJ, Rossiter VS: Clinical and pathological correlates of brain stem auditory response abnormalities. Neurology (Minneapolis) 27:316-325, 1977