Evoked cortical potentials in experimental spinal cord trauma

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The somatosensory cortical evoked response following tibial nerve stimulation was recorded for 4 hours in cats sustaining 100, 300, and 500 gm-cm impact injuries to the posterior thoracic, posterior cervical, and anterior cervical spinal cord. The return of the evoked response could be correlated with the severity of pathological damage to the spinal cord. The evoked potential returned almost immediately in animals sustaining only gray matter petechial hemorrhages, was delayed until 1 hour in animals with perigray white matter hemorrhages, and did not return at all in animals with central cavitation and posterior column fragmentation.

Key Words: spinal cord injury • cerebral cortex • peripheral nerve stimulation • evoked potential

Materials and Methods
Twenty-seven laboratory cats weighing between 3 to 3.5 kg were anesthetized with nitrous oxide and halothane using a Harvard small animal respirator after muscle paralysis with intravenous succinyl choline. After placing the animal in a stereotaxic frame, a midline scalp incision was made and the coronal suture identified. Screw electrodes were placed in the right and left frontal skull so as to be over the primary somatosensory area. Location of the recording electrodes was verified by opening the skull at the completion of the experiment. Reference electrodes were placed in the frontal sinuses.

The left tibial and left sciatic nerves were exposed. Bipolar stimulating electrodes were placed on the tibial nerves and recording electrodes on the sciatic. The exposed nerves were kept moist with a mineral oil-vaseline mixture. Depending on the group studied a one-level laminectomy was performed in the
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midthoracic region or at C-5. In the cats undergoing anterior cervical injuries, the body of C-5 was drilled away through an anterior incision. The dura was not opened. After the spinal cord was exposed the halothane was discontinued and the animals carried on 75% nitrous oxide. The left tibial nerve was stimulated with a 0.1 msec pulse of 1 V/sec. The nerve stimulus and cortical activity were monitored on a Grass model 6 electroencephalograph and Tetronix type 3474 oscilloscope. Sixty-four successive cortical transients were channelled to a Biomac 1000 computer for analysis. The analysis time was 0.160 sec. Cortical evoked responses were averaged before and after surgical exposure of the cord and served as controls.

With the technique of Albin and White,1 tapered weights weighing 5, 15, or 25 gm were dropped from a height of 20 cm onto the posterior thoracic, posterior cervical, or anterior cervical spinal cord to produce a 100, 300, and 500 gm-cm injury. The cortical evoked response was averaged immediately, and at 5-min intervals up to 1 hr following trauma. After 1 hr, responses were averaged at 30-min intervals up to 4 hrs following trauma. At 4 hrs the animals were heparinized and sacrificed with intravenous barbiturates. The vascular system was flushed with saline and a five-segment cord specimen was taken for pathological examination.

All animals received maintenance intravenous saline. Arterial blood pressure and end expiratory pCO₂ were monitored throughout the study. Rectal temperature was maintained at 38° to 39°C with a shielded Gorman-Rupp Therm X Change blanket.

Results

In all animals the cortical evoked response could not be recorded in the 64-sec period following injury with 100, 300, or 500 gm-cm. During this same period the arterial systolic blood pressure dropped slightly and then rose to 20 to 30 mm Hg above control levels. The blood pressure returned to pretrauma levels in 3 to 5 min.

Posterior Thoracic Injury

Except for the initial brief absence of the cortical evoked response, animals sustaining a 100 gm-cm dorsal thoracic injury had a return of the evoked response at 5 min post injury. There was no significant change in the latency or amplitude of the evoked response throughout the 4 hrs of recording (Table 1). The microscopic sections at 4 hrs after injury revealed small petechial hemorrhages in the central gray matter and minimal edema. Both 300 and 500 gm-cm injuries to the posterior thoracic cord were associated with an immediate loss of the evoked cortical response which had not returned at 4 hrs post trauma. The pathological specimens from the 300 gm-cm lesions showed vast numbers of petechial and flame hemorrhages in the central gray matter with marked edema of the white matter. The 500 gm-cm specimens revealed central cavitation, hematoma formation, and white matter hemorrhages.

Posterior Cervical Injuries

Like the thoracic injuries, there was essentially no change in the cortical evoked response with 100 gm-cm injuries to the posterior C-5 segment (Table 1). Microscopically there were minimal petechial hemorrhages at the site of impact (Fig. 1). With 300 gm-cm damage the evoked response could not be recorded until 45 min posttrauma in two animals and 1 hr in a third. The response did not return to pretrauma amplitude until 2 to 2 1/2 hrs post injury. Microscopic sections showed central gray flame hemorrhages, edema, and hemorrhages at the base of the posterior columns (Fig. 1).

There was no return of the cortical evoked response following 500 gm-cm injuries to the posterior cervical cord. Microscopically there was central cavitation and

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<tr>
<th>Site of Injury</th>
<th>100</th>
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<th>500</th>
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<tr>
<td>posterior thoracic</td>
<td>same</td>
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* "Same" indicates a cortical evoked response of pretrauma amplitude; "absent" indicates no cortical evoked response.

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hematoma formation, but not as severe as in the thoracic group.

**Anterior Cervical Injuries**

Unlike the posterior cervical and thoracic injuries, trauma to the anterior C-5 segment produced brisk bleeding from the large extradural venous plexus. This bleeding was easily controlled with small strips of Gelfoam.

The electrical records and pathological changes of the anterior cervical group were essentially the same as those in the posterior cervical group (Table 1). There was no loss of the evoked response with 100 gm-cm lesions; the response returned at 1 hr with 300 gm-cm lesions; there was no return of the evoked response with 500 gm-cm lesions (Fig. 2). One pathological difference was the increased amount of anterior epidural and subdural blood with anterior lesions. Blood could be seen along the anterior median fissure in the animals with 300 and 500 gm-cm lesions.

**Discussion**

With the stimulus intensity used in this experiment the evoked cortical response is probably due to impulses conducted in the ipsilateral posterior column and spino-cervical tract. The mechanism inhibiting conduction following the spinal cord impact injuries is essentially unknown. At the site of injury conduction may be inhibited by mechanical distortion or disruption of fibers, ischemia, hypoxia, or neurochemical depression.

After 100 gm-cm injuries there were small hemorrhages scattered throughout the gray matter with no acute demonstrable changes in the white matter. Because the white matter escaped significant damage the evoked response returned shortly after the impact and remained unchanged throughout the duration of the recordings. When the evoked response did not return by 4 hrs after injury that is, in the 300 and 500 gm-cm thoracic and 500 gm-cm cervical groups, the cord specimen showed not only severe central cord damage but also hemorrhages, edema, and axonal swelling in the white matter. In some cases there was fragmentation of the posterior columns.

The results of the 300 gm-cm cervical injuries are more difficult to explain. The larger central gray hemorrhages and minimal petechial hemorrhages in the perigrey white matter were not associated with permanent loss of the evoked response. Since the evoked response returned within 1 hr after injury the white matter must remain intact. Tract compression secondary to hematoma or edema formation could not have been the cause of inhibition since at 1 hr after trauma these processes should be waxing, not waning. Likewise the neurochemical depressant, norepinephrine, should be increasing rather than decreasing at this time.

The greater susceptibility of the thoracic cord to 300 gm-cm injury when compared to
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The response evoked in the cerebral cortex by peripheral nerve stimulation was recorded for a 4-hr period following controlled impact injuries to the posterior thoracic, posterior cervical, and anterior cervical cord in cats. The return or disappearance of the evoked response following impact could be correlated with the severity of the spinal cord damage, specifically, posterior column damage. The evoked response, therefore, may have clinical application in predicting the extent of traumatic spinal cord damage.

However, before the method can be used in patients the evoked response will have to be studied in traumatized animals undergoing long-term recordings and, of course, in patients in whom the pathological findings can be verified. Furthermore, it should be stressed that the technique as currently employed only determines the integrity of the posterior column, and does not identify the factors that initiate the pathological spiral of changes following impact injuries to the spinal cord.

References

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