The ciliospinal reflex in pentobarbital coma

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Object. This study was conducted to delineate the ciliospinal reflex (CSR), which is defined as pupillary dilation caused by a noxious stimulus to the face or head. The authors anecdotally observed that patients in a pentobarbital coma have a CSR that can mimic pathological conditions. A pentobarbital coma obscures the results of the neurological examination in patients with potentially life-threatening cerebral edema; pupil size and reactivity are the only readily monitored signs. Any condition that incorrectly suggests evolving intracranial pathological processes can lead to unnecessary clinical actions.

Methods. The authors evaluated six consecutive patients in the neurointensive care unit in whom a pentobarbital coma had been induced, documenting the presence and duration of the CSR. The CSR was always bilateral and symmetrical, manifesting as enlarged (6–8 mm), seemingly nonreactive pupils continuing from 1 to 6 minutes and was usually seen after routine nursing maneuvers. The pupils appeared nonreactive to short flashes of direct light but did react if longer flashes were used.

Conclusions. Recognition of the CSR can potentially lead to reduction of unnecessary transportation and complicating medical interventions in critically neurologically ill patients in whom a pentobarbital coma has been induced.

Key Words • ciliospinal reflex • pentobarbital coma • herniation

Noxious stimuli to the face, neck, or upper trunk can cause a self-limited pupillary reflex dilation (1–2 mm from baseline) known as the ciliospinal reflex (CSR). The afferent limb is mediated by trigeminal or cervical pain fibers (lateral spinothalamic tract) and the efferent limb is mediated by sympathetic fibers from the lower cervical and upper thoracic spinal cord (Fig. 1).

The exact location within the spinal cord of the reflex synapse is not known. This reflex can be seen in normal awake and sleeping individuals and comatose patients, and it is apparent in dim light. The CSR is not useful in determining brainstem integrity because the afferent and efferent fibers form a synapse in the lower cervical spinal cord. Lesions involving the cervical sympathetic fibers abolish the reflex. Physicians should recognize this reflex and understand that it does not signify impending neurological deterioration.

Patients at risk for life-threatening brain edema or hemorrhage are usually monitored using serial neurological examinations to guide the timing of appropriate medical or surgical therapy. Pentobarbital coma is occasionally used to protect patients from brain edema, but it simultaneously obscures the early clinical signs of neurological deterioration. While patients remain in a pentobarbital coma, pupillary size and reactivity, heart rate, blood pressure, and intracranial pressure (ICP) are the only readily monitored parameters that reflect the evolving pathological condition in the brain. Changes in these parameters may reflect progressive brain pathological processes that require neuroimaging studies to delineate structural changes. Transport of patients in a pentobarbital coma for neuroimaging can be treacherous, underscoring the importance of interpreting the parameter changes in those patients who may be exposed to the transport risk.

We had anecdotally found that patients in whom a pentobarbital coma had been induced demonstrated a CSR with a subsequently attenuated pupillary reaction to light that mimicked pathological conditions that cause dilated and unreactive pupils. This CSR was often provoked by routine nursing or medical care (for example, turning or suctioning). Our observation that such self-limited pupillary changes led to unnecessary transport of this high-risk group, thus monopolizing personnel and other resources, led us to systematically study and describe the.
Clinical Material and Methods

Six consecutive patients admitted to our neurointensive care unit after undergoing induction of a pentobarbital coma were prospectively evaluated for the characteristics and presence of the CSR. Four of the six were admitted immediately postoperatively after undergoing resection of a large arteriovenous malformation (AVM). Of the remaining two patients, a pentobarbital coma was induced in one for treatment of status epilepticus and the other for perioperative brain swelling (Table 1). The target depth of the pentobarbital coma was burst suppression on electroencephalographic (EEG) monitoring at three to six bursts per minute by means of a continuous pentobarbital drip, with titration performed at least every hour. All patients received continuous hemodynamic, EEG, ICP, and pulse oximetry monitoring. The one exception was in the patient in Case 3, who was in status epilepticus and did not require ICP monitoring. No changes in the ventilator rates were made 1 hour prior to each trial. All assessments of pupillary size and reactivity were performed by the same examiner who used the same illumination and a standard pupil card. Every 12 hours, after receiving no stimulation for 10 minutes, pupil size and reactivity were evaluated and recorded. A standard painful stimulation was applied, consisting of pinching the neck midway between the mandible and clavicle until the CSR appeared or 10 seconds elapsed. The presence of the CSR along with the maximum pupil size attained during the reflex were recorded. The pupil size was measured every minute for 6 minutes. The heart rate, blood pressure, and ICP were measured concurrently and recorded. The EEG burst frequency and pentobarbital level were noted before each trial. All measurements were made within 6 hours of the last assessment of the pentobarbital level. All observations were part of the patients’ routine care and did not necessitate institutional review board approval.

Results

Table 2 contains observations made for all six patients during their pentobarbital coma. The CSR was present and self-limited in all patients studied and usually lasted from 1 to 6 minutes. The duration exceeded 6 minutes in some, but in all patients the pupillary size returned to baseline within 10 minutes. The CSR was always bilateral and symmetrical unless prior pupillary asymmetries existed. During activation of the CSR, the pupils appeared unreactive to light when a short duration of exposure to direct light was used, which is typical of the standard technique to discern pupillary reactivity. However, if longer durations (30–45 seconds) of direct light were applied, delayed

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs)</th>
<th>Diagnosis</th>
<th>Neurological Exam Before PC</th>
<th>Indication for PC</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>35</td>
<td>rt PICA aneurysm, SAH, cerebellar infarction, vasospasm</td>
<td>placed in PC during surgery</td>
<td>prevention of cerebral edema</td>
</tr>
<tr>
<td>2</td>
<td>56</td>
<td>lt parietal AVM resection</td>
<td>obtunded; CN II–XII normal; localized w/ all extremities</td>
<td>prevention of normal perfusion pressure breakthrough</td>
</tr>
<tr>
<td>3</td>
<td>48</td>
<td>generalized tonic–clonic status epilepticus after anoxic event</td>
<td>coma w/ recurrent refractory generalized major motor seizures</td>
<td>treatment of status epilepticus</td>
</tr>
<tr>
<td>4</td>
<td>52</td>
<td>lt temporal AVM resection</td>
<td>normal except for transcortical aphasia</td>
<td>prevention of normal perfusion pressure breakthrough</td>
</tr>
<tr>
<td>5</td>
<td>?</td>
<td>lt paraspinal AVM resection</td>
<td>normal except for mild expressive aphasia</td>
<td>prevention of normal perfusion pressure breakthrough</td>
</tr>
<tr>
<td>6</td>
<td>29</td>
<td>rt frontal AVM resection</td>
<td>normal except for decreased spatial orientation</td>
<td>prevention of normal perfusion pressure breakthrough</td>
</tr>
</tbody>
</table>

* CN = cranial nerve; PC = pentobarbital coma; PICA = posterior inferior cerebellar artery; SAH = subarachnoid hemorrhage; ? = unknown.
The CSR during a pentobarbital coma may mimic bilateral third cranial nerve palsies (dilated and unreactive pupils) or midbrain compression (mid-position and unreactive pupils). These pupillary changes can easily lead to misperception of an evolving pathological condition in the brain and a reactive plan including unnecessary transport for neurological scanning and therapeutic intervention.

Patients in whom pentobarbital coma has been induced have a CSR that is more easily elicited. Identification of its presence should be part of the routine examination, so that it can be anticipated after nursing maneuvers. When pupillary dilation does occur in patients in pentobarbital coma, prolonged pupillary light stimulation should constrict the pupils if the dilation is caused by the CSR. Unreactive pupils in the presence of a CSR (as in Case 1) should always be considered evidence of a pathological process until proven otherwise. Pupillary dilation from bilateral third nerve palsies and midbrain dysfunction cannot be overcome with prolonged light stimulation. Pupillary changes accompanied by ICP, blood pressure elevations, and/or anatomical changes should always be suspected as a neurosurgical emergency; but the 1 minute required to determine if it is the benign phenomenon of CSR can potentially allow us to avoid unnecessary transport and complicating medical interventions.

**Discussion**

The CSR reflex, or the pentobarbital level and duration of the reflex. However, these two methods could not take into account the individual differences between the patients.

**References**


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