

## Reverse ocular dipping

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In 1961, C.M. Fisher described ocular bobbing as one of the abnormal vertical eye movements seen in comatose patients.<sup>1</sup> This is an intermittent, usually conjugate, rapid downward movement of the eyes followed by a slower return to midposition, in patients with pontine dysfunction.<sup>1-3</sup> Reverse ocular bobbing, in which the eyes jerk upward with a fast component and slowly return to midposition, has been described in association with metabolic encephalopathy.<sup>4</sup> A slow conjugate downward movement of the eyes, followed by a rapid return to midposition, has been termed "ocular dipping," and has occurred following anoxic coma and after prolonged status epilepticus.<sup>5-6</sup> We now report a related abnormality of vertical eye movements consisting of a slow upward deviation of the eyes followed by a rapid return to midposition, which descriptively is the reverse of ocular dipping.

**Case report.** A 42-year-old man with a history of IV drug abuse, AIDS, and persistent cryptococcal meningitis was admitted to the hospital for placement of an Ommaya reservoir and intrathecal amphotericin. This therapeutic regimen resulted in modest clinical improvement coincident with a drop in CSF cryptococcal antigen titers. However, his mental status remained significantly impaired. He could respond to two-step lateralized motor commands and answer questions using simple phrases. A right homonymous hemianopia, moderate right-sided facial, limb weakness, and hyperactive deep tendon reflexes were present. There were no abnormal brainstem or cerebellar signs.

He had two brief seizures during the course of his illness, one generalized and the other right focal motor. These were treated with phenytoin (300 mg/d). Phenytoin levels were consistently in the low therapeutic range. The interictal EEG showed diffuse background slowing; CT demonstrated diffuse cerebral atrophy with focal hypodense areas in the left parieto-occipital and right paravermian regions. CSF pressure at the time of Ommaya reservoir placement was normal.

During the period of modest clinical improvement, abnormal eye movements were first noted. These consisted of gradual conjugate upward movement of the eyes over 2 to 4 seconds, so that the pupils were completely covered by the upper lids. The globes then stayed in this position for 2 to 10 seconds followed by a rapid downward movement to midposition. The cycle was repeated at irregular intervals of 5 to 15 seconds. These ocular movements were dampened by voluntary effort and exacerbated by noxious stimuli. Eye movements were normal as assessed on bedside clinical examination. He had intact horizontal and vertical saccadic and pursuit movements and optokinetic nystagmus in both the horizontal and vertical planes. Vertical and horizontal oculocephalic and caloric responses were also preserved. Pupils were midposition and equal and minimally reactive to light and accommodation. The patient was awake and responsive throughout these ocular movements and showed no adventitious movements or EEG concomitants.

**Discussion.** This patient demonstrates an abnormal vertical eye movement that cannot be categorized under the existing nomen-

clature that has evolved since the initial description of ocular bobbing by C.M. Fisher. The abnormal eye movements consisted of slow upward deviation of the eyes, a brief tonic phase, and then a rapid return to midposition. Our patient was awake and partially responsive, whereas all reported cases with ocular dipping and most cases of ocular bobbing were associated with coma.<sup>1,5,6</sup> He also had intact oculocephalic and caloric responses, which are absent in "typical" ocular bobbing but may be present in atypical forms. There was an absence of roving eye movements, which were present in the patients with ocular dipping.<sup>5</sup>

Recognition of the phenomenon of reverse ocular dipping serves to complete the clinical tetrad of non-nystagmoid biphasic vertical oscillations under the general heading of ocular bobbing and variants.

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## Hyperhidrosis and hypothermia responsive to oxybutynin

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Severe sweating can be a major disability that generally is resistant to most forms of therapy short of surgical ablation of sympathetic ganglia. This disorder presents in its most extreme form in the syndrome of episodic hyperhidrosis with hypothermia (EHH), in which profuse sweating can last for hours, accompanied by major drops in body temperature with resultant impairment of neurologic function. Although its pathophysiology is undetermined, EHH usually has been associated with agenesis of the corpus callosum and so is likely of central (possibly hypothalamic) origin.<sup>1,2</sup>

A report<sup>3</sup> in this journal in 1983 reviewed clinical and pharmacologic experience in 16 cases of EHH. Although certain patients achieved some degree of control from anticonvulsants or from a variety of other medications, consistently effective treatment for this disorder has not been determined. Among the cases reported in 1983, phenobarbital and cyproheptadine gave some degree of control of sweating and hypothermia in one case (patient 1), but lost effect after continuous use for several years. Three months ago, this patient began receiving oxybutynin (Ditropan), 5 mg twice a day, to control urinary urgency. Within a few hours, the episodes of severe sweating (previously occurring many times daily) were abolished and did not recur since. The body temperature, which prior to the oxybutynin had been maintained generally at less than 35°C, returned to consistent control in the range of 35.4 to 35.6°C. In addition, hyperhidrosis has not occurred since, although with exertion there has been an appropriate amount of physiologically "normal" sweating. The continuing relief of disabling sweating, as well as improv-

Table. Clinical spectrum of ocular bobbing

	Vertical excursions			
	Initial		Return	
	Direction	Phase	Direction	Phase
Ocular bobbing	Down	Fast	Up	Slow
Reverse ocular bobbing	Up	Fast	Down	Slow
Ocular dipping	Down	Slow	Up	Fast
Reverse ocular dipping	Up	Slow	Down	Fast

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## Hemidys

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ation and cognition with the near-normal body temperature, resulted in a remarkable recovery from a previously major disability.

Oxybutynin, a muscarinic blocking compound with anti-spasmodic effects on smooth muscle, differs from other anticholinergic drugs used for urological practice by its local anesthetic action.<sup>1</sup> This drug does not possess antinicotinic effects at autonomic ganglia, nor does it have additional properties that explain its serendipitous efficacy in this case of EHH (which had been unresponsive to the anticholinergic trihexyphenidyl and numerous other medications).<sup>2</sup>

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## Hemidystonia due to subdural hematoma

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Late-onset hemidystonia is usually due to a destructive lesion of the basal ganglia or thalamus.<sup>1</sup> The lesion is usually ischemic or traumatic, but hemidystonia due to tumor infiltrating the basal ganglia has been described.<sup>2</sup> This case developed a hemidystonia from a subdural hematoma.

**Case report.** A 56-year-old man crashed his bicycle during a race. He was wearing a helmet, and was not known to be unconscious, although he has no memory of the crash. Mild confusion cleared a few minutes after the accident. He did sustain a fractured clavicle, fractured ribs, and 20% pneumothorax, and later developed pneumonia. These problems cleared in 3 or 4 weeks, and he resumed bicycle racing.

Ten weeks after the crash he noticed that he would bump into things on either side while walking. He also had difficulty writing because of impaired control of the right hand. He had no difficulty with word finding or comprehension but did notice that his memory was not quite as good as it had been. One day after the onset of these symptoms, he first developed mild, fleeting, generalized headaches.

He was examined 2 days after the onset of neurologic symptoms. There was no external evidence of head trauma. He was alert and oriented; he remembered objects and performed calculations well. Language function was normal. He was able to name only four different makes of bicycle. Cranial nerves were normal.

While he was sitting and talking, his right hand appeared to have a life of its own. It would spontaneously drift off his lap with slow spontaneous movements of his fingers, which were flexed at the MP and extended at the IP joints. There were also slow flexion and extension movements as well as twisting movements of the wrist. These movements also waxed and waned, but were always present to some degree. The patient was unaware of these movements, and seemed somewhat surprised when they were called to his attention. All spontaneous gestures were done with the left hand. Although he was right-handed, he used only the left hand to take off his shoes.

There were also slow contractions of the right quadriceps muscle

without movement of the leg. When he walked, movement of his right leg was slow with some inversion and circumduction, but without the springiness of a spastic gait. The right arm did not swing; he held it abducted at his shoulder and extended at the elbow, with a slow twisting movement of the right hand that had the index and middle finger extended, the ring and little fingers flexed, and the thumb flexed across the palm.

There was no arm drift and no weakness. Fine and rapid alternating movements were clumsy on the right, and he had mild passive rigidity in the right arm and leg. Deep tendon reflexes were equal; plantar responses were flexor on the left, abnormal but not definitely extensor on the right. Sensation was entirely normal.

Cranial CT showed a large subdural hematoma high on the left hemisphere, with a 4-mm shift from left to right. The hematoma was evacuated the next day. The day after surgery the patient no longer had any dystonic posturing, nor in fact any abnormality of movement or motor function. A follow-up MRI 6 weeks after surgery showed a small amount of blood in the subdural space without displacement or mass effect. There were no visible lesions in the basal ganglia or thalamus.

He was last seen 3 months after surgery, at which time his neurologic examination was entirely normal.

**Discussion.** The usual motor manifestations of subdural hematoma are weakness or clumsiness, associated with pyramidal signs. Subdural hematomas have rarely been reported to produce parkinsonism,<sup>3</sup> chorea,<sup>4</sup> or choreoathetosis.<sup>5</sup> To my knowledge, there have been no previous reports of dystonia from subdural hematoma, and a literature search failed to yield any reports of this association.

Transtentorial herniation may produce focal areas of ischemia by vascular compression.<sup>6</sup> The anterior choroidal artery may be compressed against the tentorium, producing ischemia of the medial putamen and lateral globus pallidus.<sup>6</sup> The vascular supply of the thalamus may also be compressed during herniation.<sup>7</sup> Our patient apparently didn't have an infarction since he recovered so rapidly, but he could well have had ischemia of either the medial basal ganglia or the thalamus, causing dystonia that was relieved by removing the subdural.

Whatever the mechanism, the association of hemidystonia with a subdural hematoma emphasizes the need to perform a neuroimaging study in the evaluation of patients with a recent onset of hemidystonia.

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