Contralateral Conjugate Eye Deviation in Acute Supratentorial Lesions

C.C. Tijssen, MD, PhD

Background Conjugate eye deviation (CED) in patients with acute supratentorial lesions is generally directed ipsilateral to the lesioned hemisphere. Incidentally, CED occurs to the contralateral side. We report five new cases and review previously published reports to elucidate the lesion responsible for and the mechanism underlying this phenomenon.

Case Descriptions In a prospective study of 133 consecutive patients with CED caused by an acute supratentorial lesion, 5 patients showed contralateral CED. This was caused by an intracerebral hemorrhage located thalamic (n=2), frontoparietal (n=1), and frontoparietotemporal (n=1). In 1 patient the cause was a subdural hematoma, an association that has not been reported earlier. Four of the 5 patients died. All patients had clinical signs of rostral brain stem dysfunction and a shift of midline structures on computed tomographic scan or at autopsy.

Conclusions Contralateral CED is always associated with hemorrhagic lesions, most commonly in the thalamus. The prognosis of patients with this sign is generally poor. Involvement of descending oculomotor pathways from the contralateral hemisphere at midbrain level is the most probable explanation for this phenomenon. (Stroke. 1994;25:1516-1519.)

Key Words • cerebrovascular disorders • eye abnormalities • hematoma

Conjugate eye deviation (CED) occurs rather frequently in patients with supratentorial stroke. This can result from lesions at different sites within the hemisphere and is seen more frequently after right-sided damage. Although CED is generally directed ipsilateral to the lesioned hemisphere, rare cases have been reported with contralateral CED, also designated as "wrong-way eyes." The explanation for this phenomenon is uncertain.

We describe 5 patients with contralateral CED, taken from a prospective study of 133 patients with CED caused by an acute supratentorial lesion.

Case Reports

The relevant data of the 5 patients are summarized in the Table.

Case 1

This 69-year-old hypertensive woman suddenly developed unconsciousness and right-sided weakness. At examination she had impaired consciousness with a Glasgow coma score of 1-5-2. The pupils were equal, with positive reactions to light. There was right face, arm, and leg hemiparesis. She exhibited CED to the right, which persisted until her death 3 days after admission. Caloric stimulation with cold water revealed a tonic deviation of the eyes on both sides without contralateral quick phases. Computed tomographic (CT) scan showed a hemorrhage in the left frontoparietotemporal subcortical region, with a shift of midline structures to the right and blood in the subarachnoid space and also around the brain stem. The patient died 3 days after admission of respiratory and cardiac failure. Autopsy revealed a large amount of blood in the subarachnoid space with an intraparenchymal left hemispheric hemorrhage adjacent to the Sylvian fissure. No abnormalities were seen in the right hemisphere, thalamus, or brain stem.

Case 2

An 80-year-old woman with known diabetes was admitted to the hospital with the sudden onset of a speech disturbance and right-sided weakness. At examination she was unconscious, with a Glasgow coma score of 1-4-1. The pupils were equal and reactive to light. CED to the right was observed and persisted for 2 days. There was right face, arm, and leg hemiparesis. The electroencephalogram (EEG) at the time of CED showed diffuse slow-wave delta activity of the left hemisphere with a maximum in the frontotemporal region and paroxysmal bilateral slow-wave disturbances; no epileptiform activity was noted. The patient deteriorated with Cheyne-Stokes respirations and died 5 days after admission. Autopsy demonstrated an intraparenchymal hemorrhage in the left frontoparietal subcortical region and the lentiform nucleus. The right cerebral hemisphere and brain stem showed no abnormalities.

Case 3

A 61-year-old man presented with the sudden onset of headaches, vomiting, slurred speech, and a left-sided weakness. Medical history was notable for hypertension and the use of anticoagulants because of a myocardial infarction. At examination he was somnolent, with a Glasgow coma score of 2-5-2. The pupils were equal and only minimally reactive to light. There was left face, arm, and leg hemiparesis. The eyes showed conjugate deviation to the left, which lasted for 1 day. Optokinetic nystagmus could not be evoked to either side using a hand-held rotating drum. Caloric stimulation with cold water resulted in a tonic deviation without contralateral
Relevant Data of Five Patients With Conjugate Eye Deviation Contralateral to a Supratentorial Lesion

<table>
<thead>
<tr>
<th>Pt/Sex/ Age, y</th>
<th>CED</th>
<th>Coma Score</th>
<th>CT Scan</th>
<th>Midline Shift</th>
<th>Course</th>
<th>Autopsy</th>
</tr>
</thead>
<tbody>
<tr>
<td>1/F/69</td>
<td>R</td>
<td>1-5-2</td>
<td>Subcortical hematoma L frontotemporal, subarachnoid hemorrhage, blood around brain stem</td>
<td>+</td>
<td>Died after 3 days</td>
<td>Blood subarachnoid space, L perisylvian hemorrhage</td>
</tr>
<tr>
<td>2/F/80</td>
<td>R</td>
<td>1-4-1</td>
<td>ND</td>
<td>+</td>
<td>Died after 5 days</td>
<td>Subcortical hemorrhage L frontoparietal, lentiform nucleus</td>
</tr>
<tr>
<td>3/M/61</td>
<td>L</td>
<td>2-5-2</td>
<td>Hemorrhage R thalamus, extension in ventricles</td>
<td>+</td>
<td>Died after 9 days</td>
<td>Hemorrhage R thalamus, demyelination medial part R cerebral peduncle at mesencephalic level</td>
</tr>
<tr>
<td>4/F/76</td>
<td>L</td>
<td>3-4-1</td>
<td>Hemorrhage R thalamus, extension in ventricles</td>
<td>+</td>
<td>Died after 4 days</td>
<td>Hemorrhage R thalamus</td>
</tr>
<tr>
<td>5/M/56</td>
<td>R</td>
<td>2-5-1</td>
<td>Subdural hematoma frontotemporoparietal</td>
<td>+</td>
<td>Operation, survived</td>
<td>...</td>
</tr>
</tbody>
</table>

Pt indicates patient; CED, conjugate eye deviation; CT, computed tomographic; R, right; L, left; and ND, not done.

Discussion

Contralateral CED as reported in the literature is usually associated with thalamic hemorrhage (12 cases), with a perisylvian bleed secondary to a ruptured aneurysm and a traumatic frontal lobe hematoma accounting for two other cases. It is of interest that the cases in the literature have all been associated with hematomas and not with infarction. Similarly, contralateral CED was associated with supratentorial hemorrhage in all 5 patients in the present series. In patients with ipsilateral CED the responsible lesions are predominantly infarctions that in the right hemisphere are located in the subcortical (frontal)parietal and the internal capsule; the lesions in the left hemisphere are usually larger, covering the entire frontotemporoparietal area. In a study of 64 patients with thalamic stroke horizontal gaze palsy was seen in 20% of 23 patients who had an intrathalamic hemorrhage and in 47% of 21 patients who had a thalamic hemorrhage with ventricular extension. This included only 2 patients with wrong-way eyes, both due to a thalamic hemorrhage with ventricular extension. There appears to be no clear preference for the right or left hemisphere in relation to this sign, in contrast to the fact that a right-sided ipsilateral gaze preference is more common than left-sided gaze preference after hemispheric lesions. The association of a subdural hematoma with contralateral CED, as in our case 5, has not been reported earlier.

The prognosis of patients with contralateral CED after hemispheric lesions is generally poor, possibly related to the size and the mass effect of the hematoma. Four of our 5 patients died, as did 13 of 14 reported in
Computed tomographic scan of patient 5, showing large left subdural hematoma in frontotemporalparietal area with compression of the ventricles and a clear shift of midline structures. L indicates left.

One of the 2 surviving contralateral CED patients had a traumatic frontal lobe hematoma, and patient 5 in the present series had a subdural hematoma. At least one of these lesions is surgically treatable, which may help to account for this patient's survival.

Three mechanisms underlying this phenomenon have been postulated. First, a possible epileptic origin of the contralateral CED has been proposed. This possibility seems unlikely because no patient in the present or reported cases has demonstrated other clinical or EEG evidence of epileptic activity.

Second, a smooth eye movement imbalance between the two hemispheres due to an ipsilateral defect of horizontal smooth pursuit has been suggested. This theory is based on the fact that ipsilateral smooth pursuit asymmetry is known to occur after parieto-occipital as well as frontal lobe lesions. The descending pursuit pathway from the parieto-occipital area runs through the posterior limb of the internal capsule, which lies near the thalamus. However, lesions in these areas leading to ipsilateral pursuit asymmetry occur rather frequently in stroke patients, whereas the phenomenon of contralateral eye deviation occurs only very rarely. This theory was postulated based on the case report of Sharpe et al, whose patient sustained a traumatic frontal hematoma. Saccadic eye movements in this case also showed hypometria of ipsilateral saccades and prolonged latencies of both ipsilateral and contralateral saccades, which is in contrast to an ipsilateral hemispheric lesion. Perhaps another injury due to the head trauma might have been concomitantly present in this patient, although there were no clinical, EEG, or CT signs of left cerebral damage according to the authors.

The third explanation is that a lesion of the descending horizontal oculomotor pathway of the contralateral hemisphere, either before or after decussation, accounts for contralateral eye deviation. The cerebral oculomotor projections subserving contralateral saccadic eye movements first traverse the anterior limb of the internal capsule, then diverge in the rostral diencephalon and follow a pathway through the medial-most part of the posterior limb of the internal capsule and midbrain cerebral peduncle. They project to the rostral paramedian pontine reticular formation after a partial decussation in the midbrain tegmentum. Additionally, there is a transthalamic projection to mesencephalic preoculomotor structures. There are several arguments in favor of this hypothesis. First, in all cases contralateral CED is caused by a hemorrhage, with no reports of infarctions available. These bleedings have an obvious mass effect, often with surrounding edema and a shift of midline structures to the other side. Our patient with a subdural hematoma had such a clear midline shift, which can lead to compression of the aforementioned pathways. Second, in most cases there is extension of the hemorrhage in the ventricles, particularly the third. Third, all cases have a disturbance of consciousness, suggesting a midbrain dysfunction. Often these patients had other clinical signs of brain stem dysfunction, which may result from the transtentorial herniation syndrome, such as small pupils with decreased or absent pupillary reflexes, hyperthermia, irregular respiratory patterns like Cheyne-Stokes respiration, and ipsilateral CED at ice-water caloric tests. Fourth, in one of our patients, who died 9 days after hemorrhage, at microscopic examination a demyelination of the medial part of the cerebral peduncle was found at the mesencephalic level ipsilateral to the thalamic hemorrhage. Ipsilateral dissection of blood downward into the midbrain and pons was also seen in two of the three patients examined by Fisher. Although the exact mechanism for contralateral CED remains unclear, factors in addition to hemispheric mass effect, including deep midline shifts, ventricular extension of thalamic hemorrhage, and dissection of blood around the midbrain, probably influence the phenomenon in some way.
In conclusion, the data from 5 present cases and 14 cases from the literature indicate that contralateral CED has always been associated with hemorrhagic lesions, most commonly in the thalamus. The hypothesis that CED is caused by an impairment of the contralateral descending oculomotor pathways is advocated.

References
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