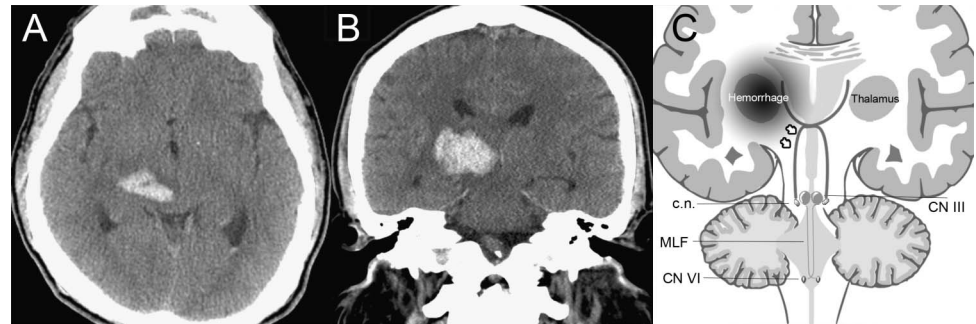


# Teaching NeuroImages: Spontaneous thalamic hemorrhage causing convergence excess

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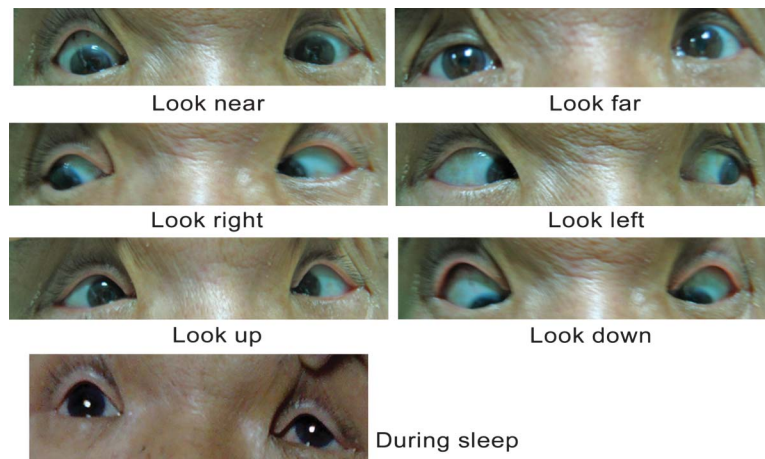
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**Figure 1** Axial and coronal CT with structure illustration shows impact on the convergence system by the thalamic hematoma



(A, B) Right thalamic hemorrhage with midbrain extension and 2-mm third ventricle midline shift. (C) Convergence neurons (c.n.) are dorsolateral to oculomotor nuclei and controlled by descending cortical pathways decussating at the subthalamic region. Unilateral thalamic lesion disrupts ipsilateral and decussated contralateral fibers (arrows), which causes bilateral convergence excess with prominence on the contralateral eye. MLF = medial longitudinal fasciculus.

**Figure 2** The patient's eye movements show bilateral convergence excess and upward gaze limitation



Convergence excess was more prominent in the left eye (contralateral to the thalamic lesion) and disappeared during sleep. Upward gaze limitation was secondary to dorsal midbrain involvement. The phenomenon resolved at 3-month follow-up.

A 61-year-old man had acute stupor and left-sided weakness. Head CT showed right thalamic hemorrhage (figure 1, A and B). Regaining consciousness the next day, he had bilateral convergence excess and upward gaze limitation (figure 2) with preserved light and oculocephalic reflexes. The phenomenon disappeared in sleep.

Convergence excess is rarely caused by thalamic lesion.<sup>1</sup> Convergence neurons, dorsolateral to oculomotor nuclei, are controlled by the descending cortical pathways.<sup>2</sup> As pathways decussate in the subthalamic region, the unilateral thalamic lesion may disrupt ipsilateral and crossed contralateral

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fibers (figure 1C). The concurrence with sobriety suggests its connection with the nearby reticular formation.

#### **AUTHOR CONTRIBUTIONS**

Dr. Tai was responsible for the concept and the drafting of the manuscript. Dr. Tang was responsible for the revising and final approval of the manuscript. Dr. Jeng was responsible for the critical revision of the manuscript.

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#### **DISCLOSURE**

Y. Tai served as a neurologist (residency) in the Department of Neurology at National Taiwan University Hospital and clinical fellow at the E-Da Hospital and reports no disclosures. S. Tang served as a neurologist (attending) in the Department of Neurology at National Taiwan University Hospital and reports no disclosures. J. Jeng served as a neurologist (attending) in the Department of Neurology at National Taiwan University Hospital and reports no disclosures. Go to [Neurology.org](http://Neurology.org) for full disclosures.

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