was not found in the presence of SVV tilt in patients with (posterior) insular stroke.

In primary position, both skew deviation and trochlear nerve palsy present with hypertropia. Unilateral trochlear nerve palsy causes weakness of ipsilateral superior oblique muscle and less-opposed supracyotation, resulting in hypertropia and inability of the affected eye to incyclotort. Because of that, compensatory contralateral head tilt position is assumed to minimize or eliminate vertical diplopia. In skew deviation, instead of being compensatory (hypertropia may or may not be affected with tilt [2**]), head tilt reflects underlying pathophysiology, as a patient’s SVV is tilted toward the side of the head tilt [4].

For example, a patient with right hypertropia and head tilt to the left may either have right trochlear nerve palsy or skew deviation. If the same patient has excyclotorted right eye and incyclotorted left eye, then the head tilt is compensatory and right trochlear nerve palsy is the most likely explanation. Whereas if the patient has incyclotorted right eye and excyclotorted left eye, then skew deviation is the most likely diagnosis. These show that one cannot rely on vertical deviation to clinically diagnose skew deviation. Nonetheless, skew deviation can be differentiated from trochlear nerve palsy by the direction of the ocular torsion [5]: in trochlear nerve palsy, the hypertropic eye is excyclotorted, whereas in skew deviation, the hypertropic eye is incyclotorted.

As mentioned before, one cannot rely on vertical deviation to clinically diagnose skew deviation. However, a new clinical test was proposed by Wong [2**] – the upright-supine test – to differentiate skew deviation from trochlear nerve palsy on the basis of vertical deviation. In their study, the degree of vertical misalignment was stable in either upright and supine head position in the case of unilateral trochlear nerve palsy. Conversely, the amount of vertical misalignment was improved at least 50% in skew deviation by changing the upright to supine head position. The test reportedly had a sensitivity and specificity of 76 and 100%, respectively [2**].

Cause and localization
Skew deviation is commonly caused by ischemia of brainstem or cerebellum. Among many causes are basilar artery occlusion. The basilar artery is formed by the joining of the vertebral arteries. It gives of pairs of branches – in caudo-rostral direction – posterior inferior cerebellar arteries, anterior inferior cerebellar arteries, and superior cerebellar arteries. Herpes simplex virus vasculitis, which appeared as an MRI enhancement of the involved vessel, was associated with stenosis of the basilar artery and posterior paramedian pontine stroke [6]. To be more specific, unilateral stroke anterior to the aqueduct of Sylvius [7–10,11*], and/or the medial aspect of the ipsilateral thalamus [9,10,12*], was reported with skew deviation and vertical gaze palsy. As the medial longitudinal fasciculus is involved in horizontal gaze, although the rostral aspect of the medial longitudinal fasciculus – the rostral interstitial nucleus (rMLF) – is involved in vertical sacades, horizontal or vertical gaze palsy, including internuclear ophthalmoplegia [8], may coexist with skew deviation. Nonetheless, vertical strabismus may be found in up to 57% isolated unilateral abduction deficit [13]. In this scenario, the vertical deviation is more likely because of mechanical cause or to nerve-muscle tone imbalance rather than skew deviation.

Apart from pontine stroke, unilateral involvement of posterior inferior cerebellar arteries or anterior inferior cerebellar arteries may cause unilateral cerebellar infraction with skew deviation in addition to acute vestibular syndrome (AVS) [14]. However, if the AVS was caused by unilateral infarction restricted to the vestibular nuclei (medial and inferior) [15], or restricted to unilateral flocculus or tonsil, skew deviation was absent [15]. However, if the lesion is restricted to unilateral flocculus or tonsil [16], skew deviation may be absent [16]. As the utricular input from the semicircular canals primarily terminate in the lateral vestibular nucleus of Deiters [17], this may explain the absence of skew deviation in the restricted involvement of the other vestibular nuclei.

Other pathophysiology may also produce skew deviation. It occurred in 13.5% patient with tumor [18], multiple sclerosis [19,20], infection [21], hemorrhage [22], vestibular neuritis [23], and increased intracranial pressure [24]. In multiple sclerosis with skew deviation, the demyelinating plaque was found unilaterally at the ‘middle cerebellar peduncle with satellite lesion probably extending to pontine 8th nerve fascicle’ [20]. Yet again, involvement of the vestibular nerve fascicle is not always followed by skew deviation. In a case reported by Veros et al. [25] with a demyelinating plaque