Table 1: Generalized symptoms of peripheral and central origin. Labyrinthine/VIIIth nerve Sudden, memorable onset Typically true vertigo at onset Paroxysmal spontaneous events <24 hours Head movement provoked symptoms <2 minutes Vestibular crisis: sudden onset vertigo slowly improving from continuous to head movement provoked symptoms in days More likely to have auditory involvement Central vestibular or nonvestibular symptoms Sudden onset of vertigo, lightheadedness/imbalance with one of the Ds Slow-onset imbalance standing and walking Vague symptoms of any character Slow subjective vertigo (spinning within the patient's head) lasting 24/7 Table 2: Generalized signs for peripheral and central vestibular lesions. Labyrinthine/VIIIth nerve Direction-fixed, dominantly horizontal nystagmus Abnormal vestibulo-ocular reflex, via head thrust or caloric testing Nystagmus more likely to be seen with fixation removed Nystagmus more likely to be exacerbated when gazing in the direction of the fast component of the jerk nystagmus (Alexander's law) Nystagmus more likely to be exacerbated post horizontal headshake - horizontal nystagmus Pursuit tracking and saccade performance normal (or age dependent) If sudden onset, can stand and walk with assistance Central vestibular or nonvestibular signs Direction-changing nystagmus Nystagmus more likely enhanced with fixation present Nystagmus more likely to be pure vertical or pure torsional Nystagmus post-headshake vertical Likely to have abnormal performance on pursuit and/or saccades If sudden onset, likely not to be able to stand and walk even with assistance Table 3: Characteristics of gaze-evoked nystagmus of peripheral origin. Acute lesion: In a peripheral lesion, nystagmus is usually only visible with fixation present when the lesion is acute in nature. Direction fixed: Nystagmus with fixation present or absent should be direction fixed in nature. It may have both horizontal and vertical components, but it must have a horizontal component to be considered of peripheral origin (i.e., pure vertical nystagmus is taken as central until proven otherwise; see text for explanation). Alexander's law: The horizontal nystagmus should follow Alexander's law (i.e., the nystagmus increases in its intensity as the patient gazes further in the direction of the fast component of the nystagmus); this applies to horizontal nystagmus component only. Enhanced with fixation removed: This is the primary determiner of the periphery being the source of the nystagmus. When fixation is removed, nystagmus is brought forth when absent with fixation, or nystagmus intensity increases if seen with fixation present. Nystagmus enhanced with headshake test: If ongoing, direction-fixed nystagmus of peripheral origin is present it can usually be enhanced with headshake testing. Linear slow component: On the tracing of the nystagmus, the slow component is a linear trace (straight line). Table 4: Characteristics of gaze-evoked nystagmus of central origin. Acute or chronic: When nystagmus is seen with fixation, it can be from an acute or chronic (beyond 12 weeks) lesion. The nystagmus persists following the lesion onset without any significant diminution in intensity with time. Direction fixed or changing: While the nystagmus could be direction fixed in nature, such as pure up or down beat, it is likely to be direction changing based on the direction of gaze (i.e., right beat with right gaze, left beat with left gaze, etc.). This also applies to a form of nystagmus called “rebound” nystagmus. With rebound nystagmus, the direction of the beat is always in the last direction that the eye moved. Also, pure vertical or pure torsional nystagmus even though direction fixed is taken as indicative of central involvement until proven otherwise. Rarely in primary: It is rare to have horizontal nystagmus persist in the primary (straight ahead) gaze position (it can be there for a brief interval when rebound is present and returning from eccentric gaze). Pure vertical or pure torsional nystagmus can persist in the primary gaze position with central involvement. Enhanced with fixation present: Typically, nystagmus is increased in its intensity with fixation present and no change or a reduction in the nystagmus is seen when fixation is removed. Vertical nystagmus post headshake test: It would be unusual to see horizontal nystagmus enhanced with horizontal headshake when the nystagmus is of central origin only. It is possible that following either a horizontal or vertical headshake test the nystagmus produced is that of pure vertical when a central lesion is the source of the nystagmus. Decreasing speed of slow component: The horizontal nystagmus trace many times will show a slow component that is nonlinear, implying a slowing in the speed of the eye as it moves from the lateral toward the primary gaze position.