

paresis may appear to have full eye movements despite a complaint of horizontal diplopia upon looking to the left. In this situation, the cover test provides a more sensitive method for demonstrating the ocular misalignment. It should be conducted in primary gaze and then with the head turned and tilted in each direction while the patient fixates a central, distant target. In the above example, a cover test with the head turned to the right bringing the eyes into left gaze will maximize the fixation shift evoked by the cover test.

Occasionally, a cover test performed in an asymptomatic patient during a routine examination will reveal an ocular deviation. If the eye movements are full and the ocular misalignment is equal in all directions of gaze (comitant deviation), the diagnosis is strabismus. In this condition, which affects about 1% of the population, fusion is disrupted in infancy or early childhood. To avoid diplopia, retinal input from the nonfixating eye may be partially suppressed. In some children, this leads to impaired vision (amblyopia, or “lazy” eye) in the deviated eye.

Binocular diplopia results from a wide range of processes: infectious, neoplastic, metabolic, degenerative, inflammatory, and vascular. One must decide whether the diplopia is neurogenic in origin or is due to restriction of globe rotation by local disease in the orbit. Orbital pseudotumor, myositis, infection, tumor, thyroid disease, and muscle entrapment (e.g., from a blowout fracture) cause restrictive diplopia. The diagnosis of restriction is usually made by recognizing other associated signs and symptoms of local orbital disease. Dedicated, high-resolution orbital imaging is helpful when the cause of diplopia is not evident.

Myasthenia Gravis (See also Chap. 448) This is a major cause of painless diplopia. The diplopia is often intermittent, variable, and not confined to any single ocular motor nerve distribution. The pupils are always normal. Serial observation of a fatigable ptosis, often accompanied by diplopia from fluctuating ocular misalignment, establishes the diagnosis. Many patients have a purely ocular form of the disease, with no evidence of systemic muscular weakness. Classically, the diagnosis was confirmed by an IV edrophonium injection, which produces a transient reversal of eyelid or eye muscle