I. MYASTHENIA AND OCULAR MYOPATHIES
   A. These disorders produce ocular motor dysfunction due to involvement of the extraocular muscles and the neuromuscular junction
   B. Some of these entities may simulate isolated or combined ocular motor cranial nerve palsies

II. MYASTHENIA GRAVIS
   A. Disease characterized clinically by muscle weakness and fatigue
   B. It is the most common disorder affecting the neuromuscular junction
   C. Myasthenia involves skeletal and not visceral musculature; therefore, the pupil and ciliary muscle are clinically unaffected. Major ophthalmologic complaints are ptosis and diplopia
   D. Ocular involvement eventually occurs in 90% of myasthenics and accounts for the initial complaint in 75%. Approximately 80% of patients with ocular onset progress to involvement of other muscle groups (usually within 2 years), while 20% have only ocular complaints
   E. Impaired neuromuscular transmission of myasthenia is due to the presence of antibodies to acetylcholine receptors in the motor endplate of striated muscles. This leads to a reduction in the number of acetylcholine receptors
   F. Clinical characteristics of ocular myasthenia:
      1. Variability of muscle function within minutes, hours, days, or weeks
      2. Remissions and exacerbations (often triggered by infection, increased body temperature, and trauma)
      3. Onset at any age
      4. Ptosis (unilateral or bilateral) worse at end of day, may “shift” from eye to eye
      5. Extraocular muscle involvement follows no set pattern; any ocular movement pattern may develop and thus mimic any ocular motor cranial nerve palsy or central gaze disturbance (e.g., gaze palsy, internuclear ophthalmoplegia, gaze-evoked nystagmus)