Section 1

Burt Kushner  “Does Form Denote Function”

1. Form often denotes function as in some cases of 4th nerve palsy in which the SO muscle is atrophic, in cases of overacting muscles due to myotoxicity in which there is hypertrophy.

2. The paradigm that overacting muscles are always hypertrophied and paretic muscles are atrophic is inconsistent with many clinical observations.

3. Changes in innervation can cause normal sized muscles to overact or underact, as can changes in muscle fiber type.

4. Muscles may appear to overact or underact when the cause of the motility abnormality may have nothing to do with the suspected muscle.

Section 2

Stephen Christiansen  “CNS Perturbations, Plasticity, and Secondary EOM Plasticity in Strabismus”

1. Primary forms of strabismus (infantile esotropia, infantile exotropia, intermittent exotropia, and accommodative forms of esotropia) can be characterized as ocular motor dystonias.

2. Eso- or exo- biases are usually neutralized by neural gain control mechanisms, presumably at the level of the neural integrator.

3. Failure of neural gain control allows a manifest strabismus.

4. CNS plasticity adapts to the altered state, re-establishing a steady state, albeit a pathologic one.

5. This new steady state results in long-term alterations in innervational tone to the EOMs which, in turn, result in secondary changes in EOM sarcomere number, EOM resting length, innervational density, and myosin heavy chain distribution.
Section 3

Linda McCloon “Extraocular Muscle Adaptations and Fiber Remodeling”

1. Extraocular muscles are extremely complex anatomically.

2. Normal extraocular muscles are continually remodeling.

3. Extraocular muscles rapidly adapt to chemical and surgical perturbations.

4. After a drug or surgical treatment of a single extraocular muscle, similar adaptations are seen in the yoked muscle on the contralateral side.

5. After a drug or surgical treatment of a single extraocular muscle, the antagonist muscle also adapts, but in a reciprocal manner.

6. Examination of surgical waste specimens show that a diagnosis of eso- or exotropia does not predict whether a muscle has decreased or increased density of innervation or myofiber cross-sectional areas.

Section 4

Vallabh Das “Neural Properties of Oculomotor Structures in Monkeys with Strabismus”

1. Animal models for strabismus replicate many of the properties of human strabismus such as A/V patterns, DVD, DHD, alternating fixation, latent nystagmus etc.

2. Neurophysiological investigation in animal models of strabismus reveals disruption in oculomotor neural processes that lead to the generation and maintenance of strabismus.

3. Our data suggest that when strabismus is of sensory origin, moment-by-moment neural drive from motoneurons to EOM accounts for strabismus properties including eye misalignment, A/V patterns, DVD and DHD. It is likely that muscle-specific factors play only minor roles.

4. Although frequently-used terms such as under-action or over-action might have relevance when targeting EOM for surgical therapy, these same terms can be misleading when attempting to identify mechanisms underlying the strabismic state.