The Relationship of the Alpha and Gamma Motor Systems to the Efficacy of the Surgical Therapy of Parkinsonism*

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Recent studies have demonstrated that stimulation of the ventrolateral nucleus of the thalamus (V.L.), globus pallidus, or substantia nigra evokes augmentation of the so-called alpha motor system.19 Stimulation of these loci also produces a reciprocal inhibition of the gamma motor system.1,18 Similarly, chlorpromazine facilitates alpha and depresses gamma activity.3,17,18 Excessive chlorpromazine can evoke a parkinsonian symptom-complex of tremor and rigidity.2,18 In patients undergoing thalamotomy, stimulation of V.L. inhibits the contralateral knee jerk22 and induces short-latency alpha activation.20

Calma and Kidd3 postulated the concept of linked coordinated activity between the alpha and gamma systems. Regulation is achieved by a mechanism of excitation and inhibition for each of the two systems. Gamma facilitation and alpha inhibition accompany stimulation of paravermian anterior cerebellum. Stimulation of the vermis, on the other hand, will inhibit gamma and augment alpha activity.

From the data assembled in the laboratory and the operating room, we feel that the following inferences may be drawn:

1) The rigidity and bradykinesia of Parkinson’s disease are associated with an altered alpha-gamma balance.

2) This imbalance is characterized by depressed gamma activity and augmented alpha activity.

3) Reduction of this poorly modulated alpha drive by ablation of subcortical loci (V.L., G.P., S.N.) with restoration of a more nearly normal alpha-gamma balance is a mechanism in the efficacy of the surgical therapy of Parkinson’s disease (Fig. 1).

While Byrnes1 was the first to implicate the muscle spindle in the pathology of paralysis agitans, it was Hassler7 who postulated that paralysis of the gamma efferent system played a major role in parkinsonian rigidity. Some observers believe that the rigidity of Parkinson’s disease is based on gamma hyperactivity.2,15,16 This opinion is based on the relief of rigidity (not tremor) by procainization of the rigid muscles21 or by dorsal-root section.14 Presumably, the effect of procaine results from selective block of the peripheral gamma efferent fibers.12 The dorsal-root section is believed effective because of interruption of the allegedly hyperactive gamma loop.

Opposed to this concept are the following arguments:

1) Increased gamma activity would mean that all patients with parkinsonism would have hyperreflexia. This clearly is not the case.6

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FIG. 1. Linked coordinated regulation of alpha-gamma activity. Alteration of normal alpha-gamma balance characterized by alpha facilitation and gamma inhibition is associated with rigidity.
2) In electromyographic studies of parkinsonian rigidity without tremor, Hoefer found a constant influx of alpha activity into both agonist and antagonist muscles. It is a function of the spindle afferent discharge to inhibit the antagonist muscle. This failure of normal reciprocal innervation means either that the spindle afferent discharge is insufficient or that there is malfunction of an interneuron that might mediate the inhibitory effect. In either case, there is uncontrolled tonic alpha input to agonist and antagonist groups.

3) One of the points upon which Hassler based his postulate that rigidity was a manifestation of gamma paralysis was the failure to achieve reinforcement of the deep tendon reflex by Jendrassik’s maneuver (clenching hands) in the limb affected by Parkinson’s disease. Jendrassik’s maneuver is assumed to be effective in reinforcing deep tendon reflexes by increasing gamma efferent discharge.

It may be of interest to note that when we began our investigation of the alpha and gamma systems, it was our initial working hypothesis that the mechanism of parkinsonian rigidity was gamma hyperactivity. As our knowledge of the supraspinal modulation of the alpha and gamma motor systems has increased, it has become apparent, increasingly, that this hypothesis is not consistent with the current body of experimental and clinical observations. All of the data are consistent with the hypothesis that parkinsonian symptoms are associated with diminished gamma and augmented alpha activity except for the observations relating to the consequences of dorsal-root section or chemical blockade of peripheral nerve. These latter observations indicate that either dorsal-root section or differential block of peripheral nerve reduce or abolish parkinsonian rigidity. If one assumes, however, that a residual spindle afferent input is necessary to provide a facilitatory background for the augmented alpha drive generated at more cephalic levels, these observations then become consistent with the hypothesis proposed.

The mechanisms that have been discussed do not account easily for the tremor that accompanies the bradykinesia and rigidity of parkinsonism. Since tremor is said to persist after dorsal-root section (although its rate and rhythmicity may be changed), it would be reasonable to assume that the oscillating motor movement is the result of rhythmic activation of anterior-horn cells. It has been shown that direct electrical activation of portions of the mesencephalic reticular formation in monkeys evokes a rhythmic movement which has similarities to parkinsonian tremor. Thus increased activity in these brain-stem circuits may be postulated to be reflected by increased alpha drive and consequent tremor. The major source of the activation of these circuits in patients with parkinsonism presumably is the same constellation of subcortical nuclei responsible for tonic alpha drive, i.e., V.L. and G.P. This formulation would be consistent with the common observation that electrical stimulation of V.L. often augments tremor in patients with parkinsonism.

It is clear that our current knowledge regarding the role of feedback loops in the modulation of motor activity is still at a primitive stage and it is probable that a hierarchy of servo systems exists at all levels of the central nervous system. The gamma loop is only one of these and little is known of the central systems of control which must be organized in a complex fashion to produce the exquisitely fine motor activity that characterizes human motor performance. However, at the present time we feel that the correlation between the rigidity and bradykinesia of parkinsonism and altered alpha-gamma balance with diminished gamma activity and increased alpha drive in the neuraxis has only gained strength as we have added to our knowledge of the physiology of motor controls. We believe further that stereotactic surgery, as it is performed today, is effective so far as it helps restore a more nearly normal alpha-gamma balance.

**Summary**

1) The rigidity and akinesia of parkin-