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Linkage of Alpha and Gamma Motoneurones in Voluntary Movement

THE special intrafusal musculature of the muscle spindles is innervated by gamma motoneurones whose axons conduct at a slower average rate than those of a muscle's alpha motoneurones. The two types of motoneurone have mostly been found coactivated in movement by a mechanism linking their pathways at some point within the nervous system[1]. To consider this further it is necessary to use a simple model of the organization of the alpha-gamma linkage. It is assumed that, at some common point, the highly complex pacemaker ordering a movement divides into two branches (Fig. 1), one for the alpha, the other for the gamma motoneurone[2]. Any voluntary order from the pacemaker will thus strike the alpha and gamma path with equal force and at the same time.

The two types of motoneurones are, however, not inevitably compelled to be coactivated. If this had been the chief aim of evolution, then it could be said to have been reached at the level of the amphibians in which the alpha axon divides within the muscle to innervate its spindles also. The means of independent action is preserved in the model by facultative inhibitory fibres to both types of neurones. These fibres give the required independence without complicating the model to a degree that—as is so often the case with neural modelling—it becomes more rewarding to work with the living thing than with its hypothesized imitation.

One target of both alpha and gamma pathways is the alpha motoneurone which is influenced by the gamma system indirectly across the loop to the spindles. Their powerful excitatory fibres mono and polysynaptically depolarize the motoneurones of their own and synergic muscles. These fibres originate in the primary endings of the spindle.

What is the contribution of the coactivated gamma loop, in terms of frequency of discharge of the primary endings, to the activation of the alpha motoneurones in different degrees and types of contraction, especially those of a voluntary character?

Some basic facts need to be summarized briefly before the chief questions are approached. (1) The spindles are in parallel with the principal muscle and are thus unloaded when the muscle shortens in contraction. (2) Intracellular work has shown that the alpha motoneurones differ with respect to gain as in the diagram of Fig. 2, in which the firing rate of the moto-

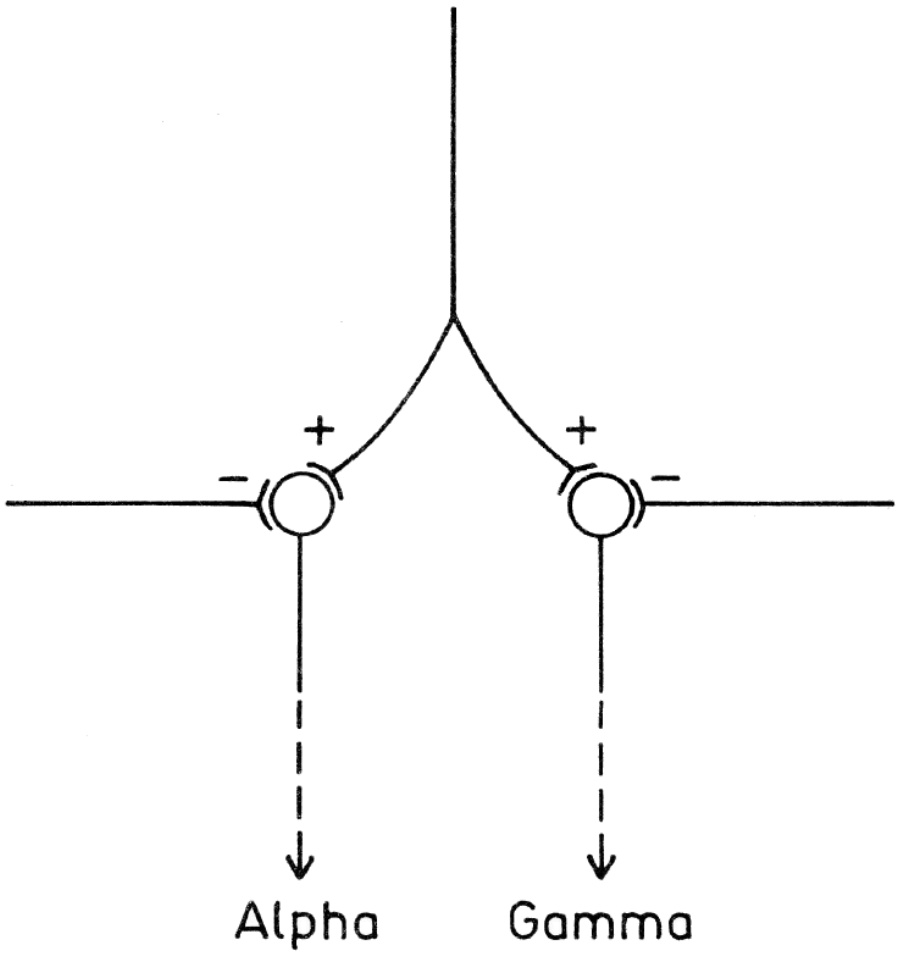

Fig. 1 Diagrammatic representation of a simple model for the organization of the alpha-gamma linkage.

neurone is depicted in relation to depolarization of its cell membrane. The large motoneurones, governing phasic motor units, have high gain and respond with a considerable increase of discharge rate to depolarizations that would have produced a much smaller alteration in the firing rate of the small, tonic alpha motoneurones. (3) Within a large range of the firing rates seen in reflex and voluntary movements there is algebraic addition of excitation (depolarization) and inhibition (repolarization) at the cell membrane of motoneurones. (4) Muscles and their motoneurones are adjusted to one another so that slowly contracting motor units are managed by small motoneurones of low gain and large phasic ones by those of high gain. Tonic adjustments tend to be automatic. (For references to all these statements, see ref. 1.)

It has been found possible to record from spindle afferents in man by microelectrodes[3,4], and, with recent improvements in technique, it has been shown that single spindle afferents from primary endings have already begun to discharge during the rising phase of a voluntary isometric contraction[5,6]. So far

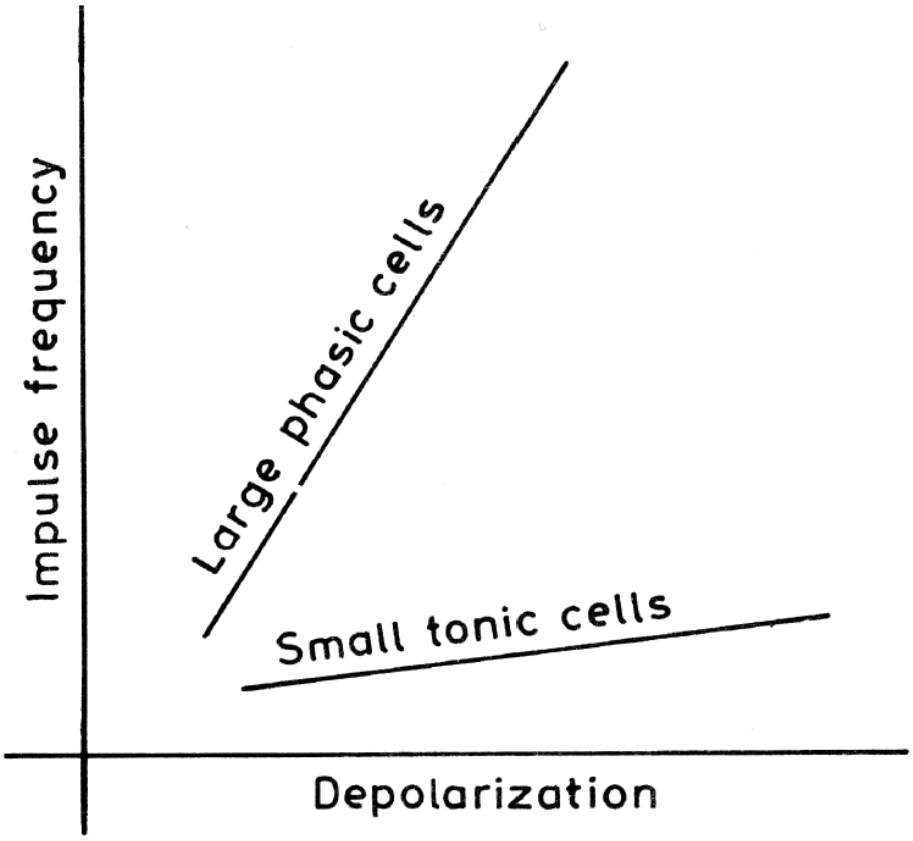

Fig. 2 Relative gain of different types of motoneurone.

the records refer only to hand muscles which have a fairly homogeneous set of fast motor units. There are no corresponding records from slow ones. Much experience with animals suggests that the contribution of the gamma loop to tonic contractions is essential for performance[1].

Starting with isometric voluntary movements of different strengths and thus without contractile shortening of a muscle beyond what is caused by its series elasticity, the model of Fig. 1 requires that the firing rate of the spindles should increase with an increase of the voluntary effort to contract. This is what actually has been found[5] but the investigated range of variation of muscle power has been narrow because the electrodes have to be inserted through skin and contracting musculature and are therefore easily dislodged. To extend the range of measurement it will probably in the end be necessary to study the alpha–gamma linkage in cortically-elicited contractions in animals. The important consequence of what so far has been observed is that the stronger the contraction, the greater the contribution of the spindles to the depolarization of the motoneurones. The linkage is of such a nature (see Fig. 1) as to force a high firing rate upon the motoneurones across the gamma loop. Clearly this will also happen whenever an increasing load compels the muscle to oppose shortening. The most significant effect of

coactivation will be on the rate of rise of the contraction which is particularly sensitive to firing rate.

In isotonic contractions which permit shortening of the muscle, the unloading of the spindles will counteract the effect of their coexcitation. As shortening will take place with some delay, the initial phase can still benefit from the automatic facilitation of the rate of rise of the contraction across the gamma loop, the more so the greater the voluntary effort spent on it. It is in later phases of the contraction that the difference between isometric and isotonic movement becomes apparent, dependent of course upon the amount of shortening that takes place. If, for example, unloading and coactivation of the spindles are in balance, there will be algebraical addition of depolarization across the loop at all muscle lengths.

The number of spindles per g of muscle plays a role in this connexion. In general this figure tends to be large in muscles which do not shorten much in normal action. To this category belong the muscles of neck, pronators, supinators and fingers, all of which also need to be precisely controlled[1].

Most skilled rapid movements are held to be ballistic[7–9]. This means that the fast contraction only generates the kinetic energy that in the actual movement emerges as momentum of the stroke. This itself is executed without opposition from antagonist muscles during relaxation of the agonist. The nearest approach to ballistic studies involving spindle records in voluntary movement is a number of brief isometric "twitch-like" contractions in Vallbo's work[6]. These exhibited coactivation by alpha-gamma linkage. His movements were of course not truly ballistic but fast enough to imitate one aspect of such movements. Skilled movements are often heavily preprogrammed by training and it would be interesting to know whether this might have led to suppression of the gamma component. One used to believe that the indirect route across the gamma loop would be too slow for fast movements. This is not the case. Finger tapping at the maximum rate of 8 to 10 s^{-1} leaves a margin of 125 to 100 ms, which should be ample for loop action. Absence of coactivation should therefore—on the model—indicate true inhibition.

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