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THE PROBLEM OF THE PERIPHERAL CONTROL OF SKILLED MOVEMENTS†

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This article deals with the problem of the role of peripheral stimuli originating during the performance of motor acts, in controlling those acts. The thesis is presented that the Purkinje cells play the role of a filter by which information about passive stretching of muscles is suppressed and cannot reach the brain; whereas information about active contraction is transmitted. Both experimental support for and consequences of this hypothesis are discussed.

I

What I shall try to do in this article is to collect some bits of evidence clarifying the problem of what is the role of peripheral stimuli originating during the performance of motor acts in controlling these acts.

The first question to be raised is that of whether afferentation of the limb is at all necessary to produce a movement; in other words, whether the somatic sensation of a limb and of its posture is indispensable for its performance. Intuitively one can guess that when one does not 'know' about his limb because of its complete deafferentation, one cannot move it unless he has another source of information such as vision. This belief was indeed held by many neurologists and physiologists, but it has appeared to be wrong. Numerous experiments performed on rats, cats, dogs and monkeys have shown that after deafferentation of a given limb the *instrumental* movements of that limb are preserved, thus showing that efferent 'orders' arising in the brain can easily reach the limb even if the brain is completely unaware of the posture, and even the existence, of that limb (Knapp, Taub, Berman, 1958; Jankowska, 1959; Gorska, Jankowska, 1961; Taub, Berman, 1968).

If so, one can ask what *is* the role of proprioception in the performance of a movement. The answer is clear. The purposeful movements which have a behavioral meaning are mostly complex and should have a certain precision. Accordingly, if we require the animal to perform a very simple or very well trained movement, this movement does not need any feedback and therefore proprioception is not necessary for its performance. If, however, the movement must take a definite form as far as its character and amplitude are concerned, then the proprioceptive control is necessary. The same is true if we are dealing with a chain of movements, such that the accomplishment of one of them is a stimulus for performing the next one.

The next question to be asked is this. Most of our movements, as well as those of higher animals, are *learned*; that is, they are elaborated with the help of some training. When a child performs an instrumental movement serving some purpose, he does it at first very slowly and awkwardly, and only gradually does the movement acquire smoothness and rapidity. And so we can ask in which way this learning occurs.

The general answer to this question is that the training of skilled movement occurs by way of integration of its elements into a unitary motor act. We have much evidence to believe that this integration occurs in the premotor cortex (area 6), since, in humans, lesions in this area produce a dramatic disintegration of skilled movements (so called apraxia). In fact, a patient with a lesion in the premotor area of the dominant hemisphere is not able to reproduce the patterns of movements well-trained during his life, although he is neither paralytic nor even paretic. On the other hand, if a lesion is sustained in the postsensory cortex, behind the projective somatosensory area, the patient has all trained patterns of movements fully preserved.

† Written originally for the Festschrift in Honor of Professor R. W. Gerard.

in spite of the fact that his sense of position of the limbs is severely impaired or abolished.

According to these data we should discern two separate proprioceptive systems: one of them, which may be called the somesthetic proprioceptive system, is represented in the somatosensory and postsensory areas of the cortex and is responsible for perception of *posture*; the other one, which may be called the kinesthetic proprioceptive system, is represented in the motor and premotor areas and is responsible for perception of *movement*.

We would not like to enter here into the intriguing problem of what is the cortical representation of the unitary skilled movements; in particular whether they are represented as 'cell assemblies', according to Hebb's theory (Hebb, 1949), or by individual gnostic units, as was postulated in my recent monograph (Konorski, 1967). Suffice it to say that in the premotor area there is a representation of skilled movements which are controlled by the nerve cells of this very area. Accordingly, the performance of skilled movements is accomplished in such a way that the particular groups of nerve cells in the premotor cortex send messages (through the motor cortex) to the particular groups of muscles causing their contractions. The strength of these contractions and their duration depend on the task to be performed, a task whose fulfillment is signalled to the cortex by proprioceptors, and partially exteroceptors, of the limb involved in the movement.

In order to avoid any misunderstanding, let me briefly analyze some typical movements performed by the upper limb in man.

1) Static movement occurring when we carry an object (for instance holding a suitcase by the handle). The muscles which fulfill this task are the flexors of the fingers. They are persistently contracted against resistance, because the weight of the case tends to extend them. The main feedback is here provided by the articular sensation of the posture of the fingers. If the suitcase is heavy and we feel that our fingers get passively extended, we immediately increase the muscle effort so that the fingers remain flexed. The best illustration of the decisive role of the joint feedback in regulating this movement is the fact that one of the first complaints of a patient with a tumor in the lateral part of the parietal lobe was that she would fail to notice the loss of a parcel she had in hand. The examination of this patient showed that her sensation of the position of her fingers was completely lost.

However, the sensation telling about the position of finger joints is not the only feedback involved in holding a load. If the load is augmented, and therefore the effort of flexion of our fingers is increased, we feel that effort very precisely quite apart from the pressure of the handle on the palm. Therefore the sensation of the strength of muscular contraction is another feedback of the movement concerned.

2) Suppose now that we perform some dynamic movement against resistance; for instance, we lift a heavy load by flexing the elbow, or we push a load, by extending the arm, or turn a knob by a movement of the wrist, etc. Here again our information about the execution of the movement comes from the joints. However, estimation of the muscular effort is also in operation. If this effort appears to be ineffective because the load is too heavy to be lifted or pushed, or the knob does not yield, its estimation is still there, although no change in the position of the limb occurs.

3) Let us take into account now those movements which are also directed towards the external world, but in which besides an effort exerted upon some external objects, a great precision of performance is needed. Here belong professional movements of some craftsmen (for instance, watchmakers), surgeons, sculptors, etc. The great precision of movements is accomplished by simultaneous contraction of antagonistic muscles, securing the maximal stability of the limb. In such a state, even minute movements may be performed by a very small increase of contraction of one group of muscles and relaxation of the antagonistic muscles. Accordingly, in this case, too, the movements are executed again resistance, but whereas in the preceding cases this resistance was caused by the manipulated object, here it is produced mainly by contraction of antagonistic muscles. In these actions feedback arising from the muscular efforts probably plays a greater role than feedback coming from the joints, because of the very small changes in the position of the limb concerned.

4) Finally, there are movements which follow some definite, even very complex, patterns, but do not require any effort because they are not directed toward objects of the external world. Here belong the movements of the mouth involved in speaking (in contrast to those involved in gnawing). The resistance against which these movements are executed is minimal and therefore the contractions of the corresponding muscles are mostly isotonic.

To sum up, we may notice that, whereas in

physiological experiments on muscle contractions we deal mainly with either isotonic or isometric contractions, utilizing corresponding types of myographs, in normal life the contractions are neither of the two because both the tonus and the length of the muscle change considerably in every motor act.

The above considerations seem to indicate that, against the view of some physiologists who claim that the only feedback generated by movements comes from the joints (Mountcastle and Powell, 1959), a no less essential feedback comes from the muscles themselves. It seems that at least the three following facts speak in favor of this thesis:

1) As noted before, the contraction of muscles may be purely isometric, and therefore, when they are performed *in situ* they do not involve any change in the position of a limb. Nevertheless, the strength of the contraction—the effort exerted by the subject when he is performing such an isometric movement— is precisely estimated by him.

2) We clearly distinguish between active displacement of our limbs due to contractions of the muscles, and passive displacement when the limb is flaccid and someone else flexes or extends it. In both these cases exactly the same changes in joints occur. Nevertheless the sensation of the active movement and that of its passive displacement are quite different. The same difference between these two phenomena is also manifested in experimentation on animals; it has been shown that, whereas active movements performed by the animal can be easily transformed into instrumental responses by their proper reinforcement, the purely passive displacement of the limb as a rule cannot become an instrumental response. This fact shows that not the somesthetic, but the kinesthetic, analyzer is involved in instrumental conditioning (Konorski, 1967).

3) We have often observed that patients with parietal lesions, who have totally lost the sense of position of a limb contralateral to the lesion, are able to perform the movements required without errors. For instance, we take a patient's hand and flex or extend his fingers; he is completely unaware of their position. Now, we ask him to flex or extend his fingers actively. He always performs this task correctly, without hesitation. When the fingers are extended and we ask him to flex them he does so very quickly; but if we ask him to extend the extended fingers he only contracts extensors, not changing the position of the fingers. The same is true when we ask the patient to flex the fingers which are already in flexion. The patient simply increases insignificantly the flexion, being unaware whether the position of the fingers did or did not change. Experiments of this type clearly show that, although the position of the joints is completely unknown to the patient, his appreciation of the muscular contraction is quite normal. Analogous data in animals have been also obtained (Kenard and Kessler, 1940).

II

Accepting the existence of the sensation of movements ('Muskelsinn' of the German physiologists of the 19th century) as a phenomenon quite different from the sensation of position, we must now elucidate the problem of the pathways which convey the information about this sensation to the cerebral cortex. Since the pathways from the muscle and tendon receptors, in contradiction to those from joint receptors, run mainly to the cerebellum, and from there continue their course to the motor area of the cortex, it seems most reasonable to accept that these are the main routes conveying information from muscles and tendons to the cortex (Figure 1). Accordingly, we may expect that if this pathway is destroyed in one of its links, the feedback from movement will be conveyed only through the lemniscal system, informing the cortex of the changes in joints. Hence we should expect that if both systems are destroyed, then the peripheral control of the motor acts should be completely abolished.

These experiments have been performed recently by Jaworska and Slowik in our laboratory (unpublished). It was decided to remove those links of each of these systems which are most easily accessible surgically so that the ablations should be virtually exact. Accordingly, the muscular afferent system was injured by removal of either the total cerebellum, or only its paravermal part; the articular afferent system was injured by removal of the somatosensory cortex from the central dimple to the ansate sulcus. Sometimes both S_I and S_{II} were removed. All the operations were bilateral.

The following were the main results of these experiments:

1) The total removal of the cerebellum in cats produces a very strong motor incapacitation. In the first postoperative period the animals lie helplessly on their sides with strong opisthotonus and extension of the forelegs. After a few months

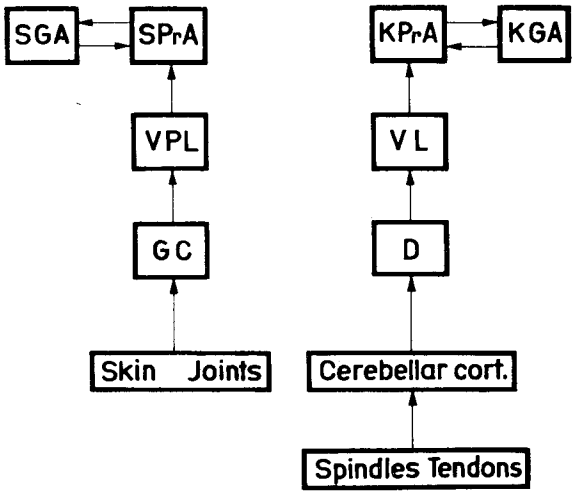

FIGURE 1 Block models of somesthetic and kinesthetic afferent systems (simplified).
(left) Somesthetic afferent system. GC, nuclei gracilis and cuneatus; VPL, ventral postero-lateral thalmic nucleus; SPrA, somesthetic projective area; SGA, somesthetic gnostic area.
(right) Kinesthetic afferent system. D, dentate and interpositus cerebellar nuclei; VL, ventrolateral thalamic nucleus; KPrA, kinesthetic projective area; KGA, kinesthetic gnostic area (nucleus ruber is omitted for the sake of simplicity).
Note the clear symmetry between the two systems except for the peripheral segment of the kinesthetic afferent system. (Konorski, 1967.)

they begin to walk, using first only the forelegs and dragging the hindlegs; thereafter the hindlegs are also used. While the forelegs are strongly extended during walking, the hindlegs are flexed. After a year, the cats are able to walk slowly with the legs widely spread apart. The food intake, which was, after operation, strongly impaired, also becomes better after a year or so. When only partial bilateral lesions are sustained, which are limited to the paravermal parts of the cerebellum, the defect is much less clearly expressed and consists mainly of difficulty in walking. At the beginning, the cats walk with the forelegs widely spread apart, but after several months there is practically full compensation of function.

2) When bilateral lesions in the sensory cortex or in the motor cortex are sustained, the motor defects are much smaller and are usually compensated in a few weeks. It is interesting to note the following striking difference between the effects of cerebellar lesions and somatosensory cortical lesions. The cats with cerebellar lesions sustained long ago, although severely incapacitated in their motor activity, assume completely normal posture when they are in a lying position. On the other hand, the cats with somatosensory lesions, although quite skillful in their motor activity, assume bizarre and abnormal positions of the legs when lying. They correct themselves only when they start to move. This shows that after cerebellar lesions the postural patterns are not impaired while the motor patterns are strongly impaired; after somatosensory lesions, on the other hand, the motor patterns are fully preserved while the postural patterns are impaired.

3) When a long time (about one year) after a total cerebellar ablation we remove the somatosensory cortex, sparing the motor cortex, the whole cerebellar syndrome, except tremor, returns. The animal is not able to walk. He only crouches with all four legs extended, and there are no signs of improvement, even after many months.

The effect of the second operation is even more conspicuous when the cerebellar lesion is limited to the paravermal part, since the compensation after this operation is almost complete. In such a cat the ablation of the somatosensory cortex has a most dramatic effect, because the animal returns to the state observed immediately after the cerebellar operation, except that he has no tremor.

4) On the contrary, if the cerebellar lesion is followed by ablation of the motor cortex, the condition of the cat does not change considerably, and after a few weeks he returns to his previous condition.

5) In some experiments the sequence of the operations was the reverse: first, the lesion was sustained in the somatosensory or in the motor cortex, and after a few months the cerebellar lesion (either total or only paravermal) was made. The results were exactly the same as in previous experiments: the animals with somatosensory and cerebellar lesions were severely incapacitated and their condition failed to improve in the following months, whereas the animals with motor and cerebellar lesions did not differ from those with cerebellar lesions only.

Explanation of all these results from our point of view does not present any difficulty. If we admit that motor activity is controlled by the joint operation of articular proprioception, depending on the lemniscal system, and of muscular proprioception, depending on the spinocerebellar system, then it is clear that both these sources of information may mutually substitute for each other. Accordingly, the compensation of the motor

impairment after a cerebellar lesion may occur by substituting the information from joint receptors for the information from muscular receptors. If then the somatosensory cortex representing joint proprioception is removed, the animal loses all its proprioceptive information. On the other hand, if the cerebellar lesion is combined with a lesion in the motor cortex, then, although information from the muscular system is abolished, information from the joint receptors is fully preserved.

III

The results of these experiments clearly show that the cerebellar control of motor behavior is well-documented. Therefore the next question we have to answer concerns the mechanism of this control.

In order to approach this question we should turn now to the electrophysiological experiments to see what information is conveyed to the individual Purkinje cells from peripheral receptors. It is amazing, that in spite of the great amount of electrophysiological work performed on the cerebellum, this very problem has been hardly touched. Although there were a few studies in which cerebellar responses were recorded by using either macroelectrodes or microelectrodes, no authors except Thach (1968) tried to record the responses of the Purkinje cells to natural stimuli.

In the experiments performed recently by Tarnecki and myself on cats (1970), we have found that very regular and constant responses from Purkinje cells are obtained when at least one of the animal's legs is placed in a definite position. In this respect Purkinje cells may be divided into two groups. The cells of one group are completely, or almost completely, silent when the legs are in a semi-extended position; this being the background position in our experiments. However, when at least one of the legs, in most cases ipsilateral to the side of the cerebellar electrode, is flexed in a definite joint (for instance, in the wrist, or in the elbow, or in the ankle, or in the knee, etc.), a strong discharge of impulses occurs, which lasts as long as the given position is sustained (Figure 2). The response is quite regular and it consists of

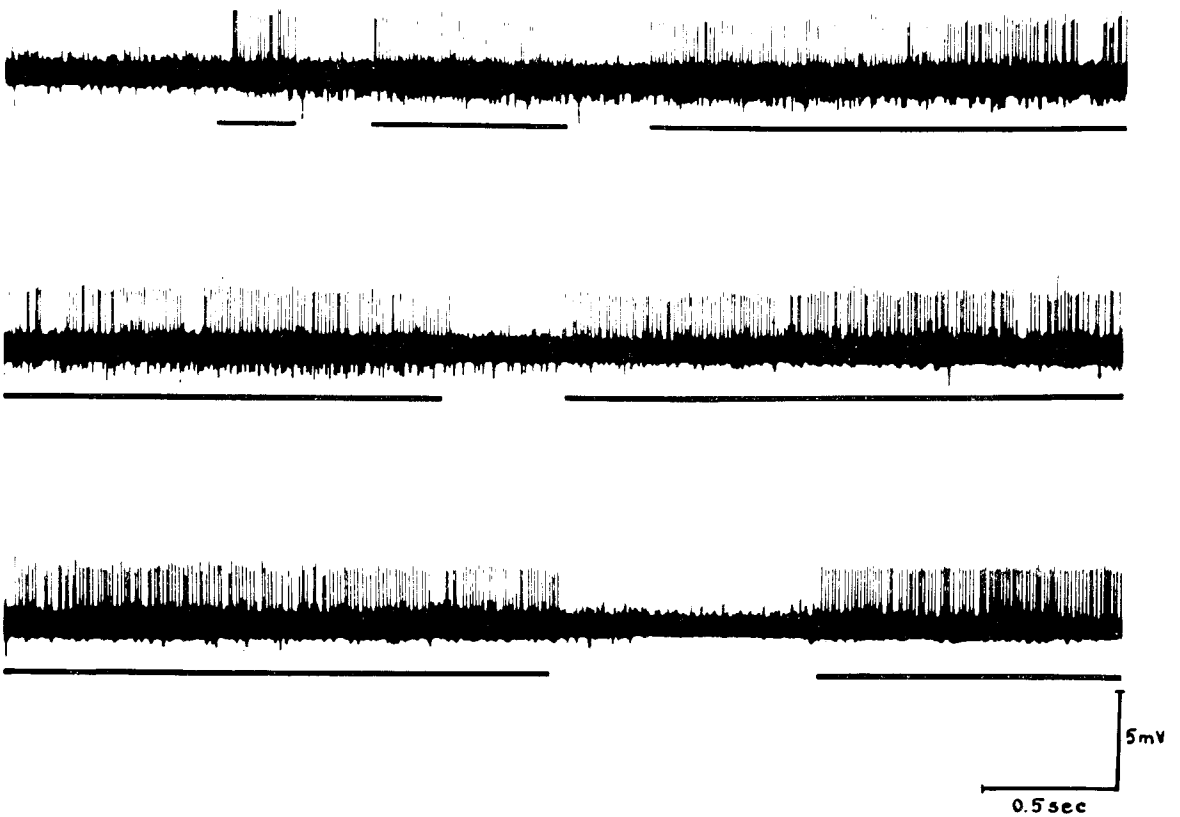

FIGURE 2 Discharges of Purkinje cell in response to passive flexion of the ipsilateral knee. Passive flexions are denoted by horizontal lines beneath the record. (Tarnecki and Konorski, 1970.)

discharges with constant rate, usually from 40 to 80 impulses per second. The cells of the other group are 'spontaneously' active with exactly the same rate of discharge, but flexion of a definite leg caused their complete silencing which again lasts during the whole period of flexion. The cells renew their activity immediately when the previous semi-extended position is restored (Figure 3). There are also cells which, although 'spontaneously' active, cannot be silenced by any of our manipulations, and also cells which are active or silent alternately, independent of our manipulations. The all-or-nothing character of discharging—full silence or constant activity—is preserved in the majority of cases.

Some cells react occasionally to squeezing of the distal part of a given leg, but then the response is rather phasic and not regular. Tactile stimuli, such as stroking the leg or touching it, only occasionally produce responses in Purkinje cells, and this happens only in very reactive preparations.

As follows from our experiments, the postures of limbs provide natural stimulation for the Purkinje cells, which react to these postures with great regularity: some of the cells react to the flexed position of the legs, either in one or more joints; the other cells, on the contrary, react when the given limb is extended and stop reacting when it is flexed. To sum up, the activity of the Purkinje cells reflects in some way the posture taken by the animal at a given moment.

At first glance these results appear to be completely unintelligible because they seem to indicate that the Purkinje cells are mainly involved in definite postural patterns; that is, they would completely imitate the cortical cells situated in the somatosensory cortex. However, this similarity is only apparent, because in all probability Purkinje

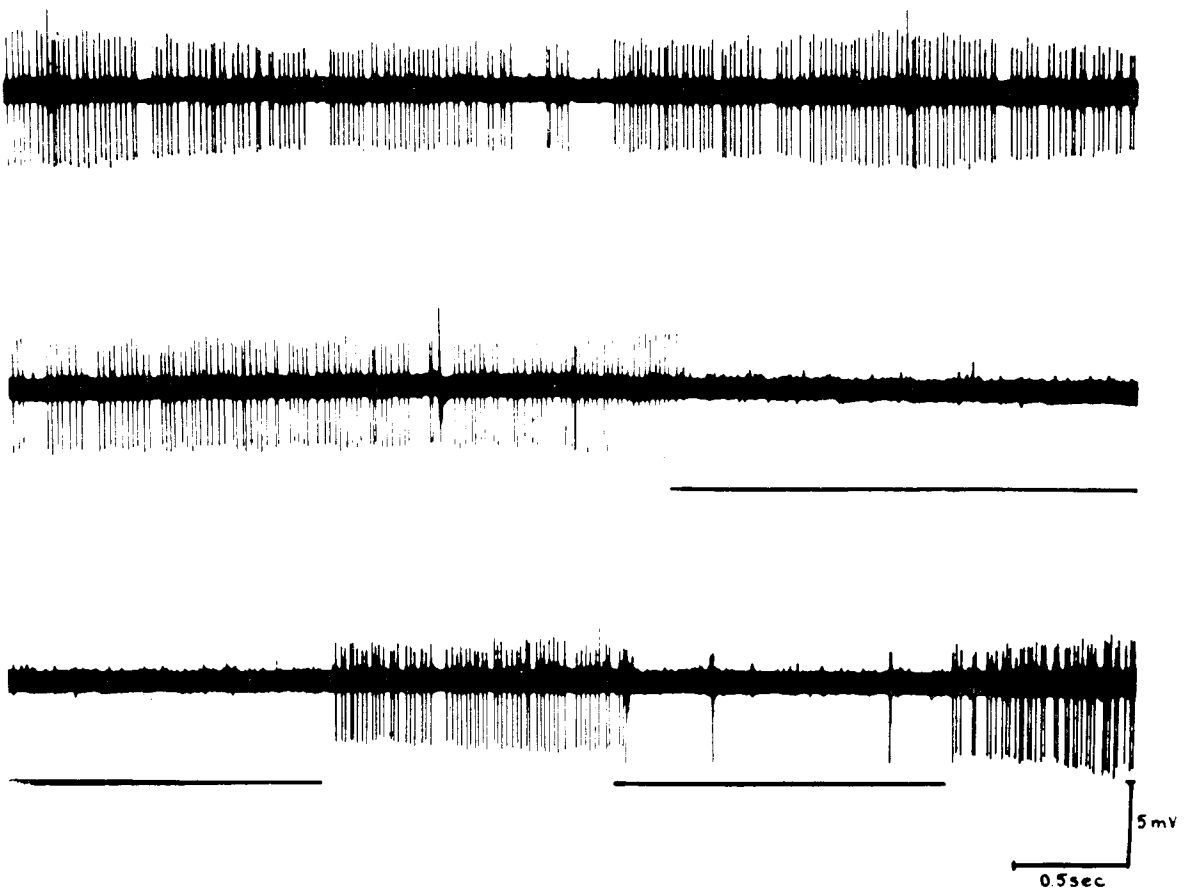

FIGURE 3 Silencing of spontaneously discharging Purkinje cell by passive elbow flexion of the ipsilateral foreleg. The lines below the record denote the periods of flexion. (Tarnecki and Konorski, 1970.)

cells do not react to the activity of joint receptors, but only to stretching of the muscles. In other words, when they react to a leg's being in flexion, this means that the extended extensors produce the reaction; conversely, when the Purkinje cell reacts to a leg's being extended, this means that it reacts to the stretch of flexor muscles.

The problem then arises as to which stretch receptors, if not all, are responsible for activation of Purkinje cells. As is well known (cf. Matthews, 1964), muscle spindles are supplied with primary endings and secondary endings Figure 4). Primary

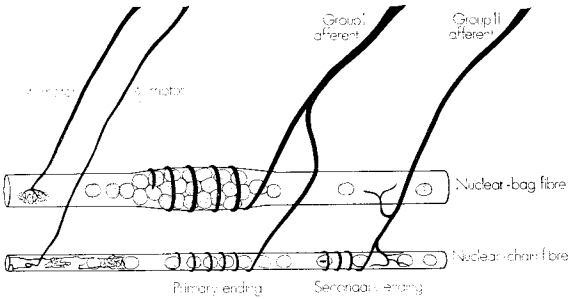

FIGURE 4 Greatly simplified diagram of the central region of the muscle spindle. (Matthews, 1964.)

endings have mainly a dynamic character reacting primarily to a change of the length of the muscle; secondary endings have mainly a static character, reacting to every stretch, even if it lasts for a long time. The afferents from primary endings belong to Group I; they have large diameter and high velocity of conduction. The afferents from secondary endings belong to Group II fibers; their diameter is smaller and their conduction slower. The receptors in tendons, the so-called Golgi organs, are innervated by afferents belonging to Group I fibers, conducting impulses with almost the same velocity as those from primary spindle endings. Which of these three types of receptors activate the Purkinje cells will be discussed further in the text.

Now we should recall that if the muscle is contracted without any resistance—the ideal case of this is when the tendon is cut—then stretch receptors are hardly activated. If, however, the muscle contracts against resistance, as is the case in most skilled movements, then the situation is more complex: the tendon organs are strongly activated, this activation being positively correlated with the strength of resistance and the strength of muscular contraction. On the other hand, spindle receptors are subjected to two opposite influences: for one thing, if the gamma system is not in operation, and therefore the intrafusal muscle fibers are relaxed by shortening of the muscle, the spindle receptors are completely silenced; for another thing, when the gamma system *is* in operation and intrafusal muscle fibers are contracted, then both the primary and secondary endings are activated.

This being so, one can be puzzled by the problem of how information about movements is conveyed to the brain, taking into account that practically nothing about muscle contractions is transmitted by either tendon organs or spindles taken separately.

However, if we take into consideration our own experimental data, and compare them with some other facts which have been obtained recently, by other authors, it seems that the solution of the problem of the function of the cerebellum is not as hopeless as it appears. A hypothesis suggesting this solution has been recently advanced by Konorski and Tarnecki (1970). Here we present its main points.

IV

To begin with, let us pay attention to the important fact found recently by Ito *et al.* (1964), to the effect that Purkinje cells convey to the intracerebellar nuclei not excitatory messages, as one could expect, but, on the contrary, strong inhibitory messages. Accordingly, the problem arose as to from what sources the main influx of excitatory impulses to those nuclei does originate. It has been postulated that this influx comes from many sources, and, among others, from the peripheral receptors along the spinocerebellar tracts. In other words, it was assumed that some fibers arriving at the cerebellum from the periphery do not reach granule cells, which send their axons to the Purkinje cells, but go straight to the intracerebellar nuclei (Eccles, Ito, Szentagothai, 1967).

It has been found recently that Purkinje cells are activated only by stimulation of Group II afferents originating in secondary spindle endings, whereas Group I afferents originating either in primary spindle afferents or in tendon organs do not reach these cells (Eccles and coworkers, 1969). The immediate conclusion which may be inferred from this fact is that these very afferents go straight to the intracerebellar nuclei. This conclusion is supported by Tarnecki's new data (unpublished) which show that the volleys originating in Group I afferents of the hind legs reach the units of the intracerebellar nuclei after a latency of only 8 milliseconds.

According to all these data put together we may come to the conclusion that the input to the cerebellum of the messages originating in muscles and tendons is quite complex. The messages arising in tendon organs and primary spindle endings, conveyed by Group I afferents, go straight to the intracerebellar nuclei and *activate* their units; on the other hand, the messages arising in secondary spindle endings, conveyed by Group II afferents, go through granule cells to the Purkinje cells and hence to the intracerebellar nuclei, *inhibiting* their units (Figure 5).

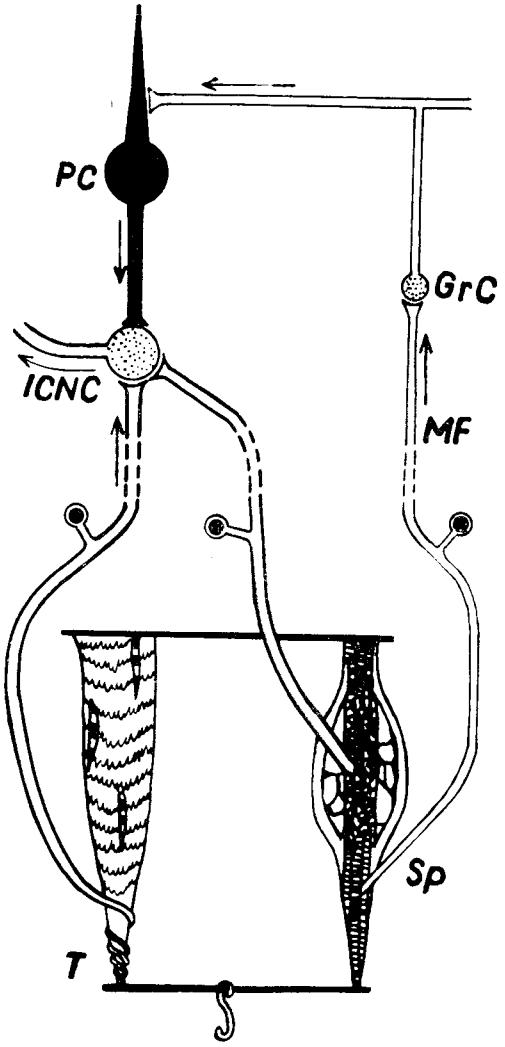

FIGURE 5 Supposed mechanism of the function of the cerebellum. PC, Purkinje cell; GrC, granule cell; MF, mossy fibers; ICNC, intracerebellar nuclear cell; T, Golgi tendon organ; Sp, muscle spindle with primary and secondary endings. Inhibitory neuron drawn in black, excitatory neurons drawn in white. Explanations in text. The upper part of the Figure is modified from Eccles *et al.* (1967).

What is the functional significance of all this arrangement?

Imagine that a muscle is passively stretched either in an artificial way, by being pulled, or in a natural condition, when the antagonistic muscle is contracted. In that case, impulses arising in tendon organs and primary spindle endings directly reach the units of the intracerebellar nuclei, producing their excitation. However, impulses arising in secondary spindle endings activate Purkinje cells, which send inhibitory impulses to the units of these nuclei. In effect the intracerebellar units are inhibited, because evidently the inhibitory effect from Purkinje cells overcomes the excitatory effect from tendon organs.

The direct experimental proof of these relations was, in fact, obtained recently by Tarnecki (unpublished). He stimulated the muscle nerves of the hind leg in cats and recorded responses of individual units of the interposite intracerebellar nucleus. If the current was just suprathreshold, activating only Group I afferents, it produced a short excitatory response following each shock. However, if the current was increased, then the excitatory response was followed by the inhibitory response whose duration was the greater, the stronger the shock applied (Figure 6).

Let us now analyze the situation when the given muscle is contracted against some resistance. In that case, the tendons are strongly stretched; as a consequence, the Golgi organs are maximally activated and send their messages to the intracerebellar nuclei. In contrast, the secondary spindle receptors are silenced (the influence of the gamma system being for a moment neglected) and do not send excitatory impulses to the Purkinje cells. In effect, these cells fail to inhibit intracerebellar nuclei, which are now strongly excited.

And so the role of the cerebellar cortex in conveying the information from muscles and tendons to the intracerebellar nuclei and hence to the cerebral cortex seems to consist in withholding information about the passive displacements of the limbs by activation of the inhibitory relay located in the Purkinje cells, and in passing through that information which is generated when the muscles are contracted against resistance. If the contraction is purely isotonic with no resistance at all, then according to our concept the information about it is hardly conveyed to the brain, simply because tendon organs are not activated. It is, however,

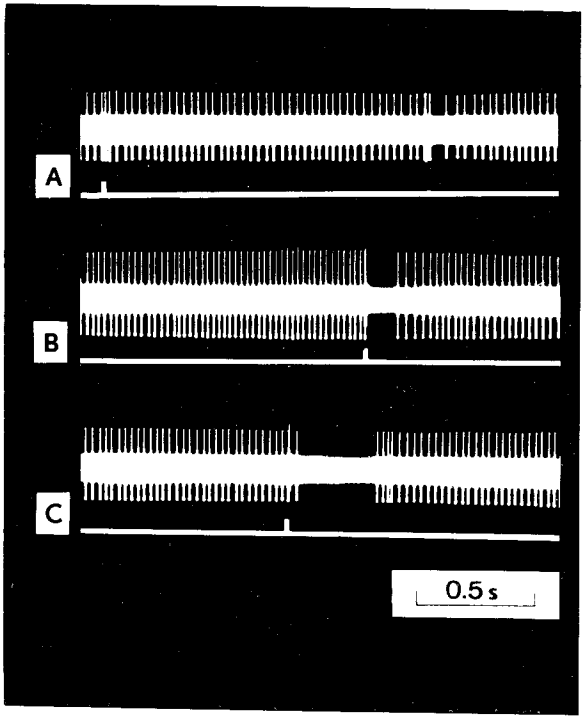

FIGURE 6 Response of intracerebellar cell to stimulation of the biceps femoris nerve. A, threshold stimulation; B, stimulation with twice threshold current; C, stimulation with seven times threshold current. (Tarnecki, unpublished.)

possible that the primary spindle endings activated just at the beginning of contraction send the information about it to the intracerebellar nuclei.

Now we should consider the question of what may be the role of gamma efferents in the above described mechanisms. It is well known that if the gamma efferents are activated in addition to the alpha efferents, both the primary endings and the secondary endings are no longer silenced, but they generate impulses which certainly reach the cerebellum (Figure 7). Whereas Group I afferents convey excitatory impulses to the intracerebellar units, Group II afferents activate Purkinje cells and thus inhibit these units. However, it must be taken into account that when the muscle is contracted under strong resistance, the tension of the tendon region is certainly exceedingly high, and thus the impulses sent to the intracerebellar nuclei are very copious. The impulses from primary spindle endings, which are also quite numerous, especially when the contraction is strong (see Figure 7), still add to the increase of excitation of these nuclei. Therefore, if even the secondary endings are excited by the gamma system, their inhibitory influence is certainly counterbalanced by very strong excitation of these nuclei.

If this reasoning is correct, then the functional significance of the gamma system, as far as cerebellar function is concerned, is comprehensible. In fact, it would consist in damping the excitation of the intracerebellar nuclei and defending them from overexcitation, which might occur if this system were inoperative. In this sense, the role of the gamma system would be similar to that of constriction of the pupils when the light is too strong, or constriction of the tensor tympani when a sound is too loud.

On the other hand, as indicated by Granit (1955), when the tendon is stretched, the gamma motoneurons are inhibited, and therefore during the contraction of the muscle against resistance the

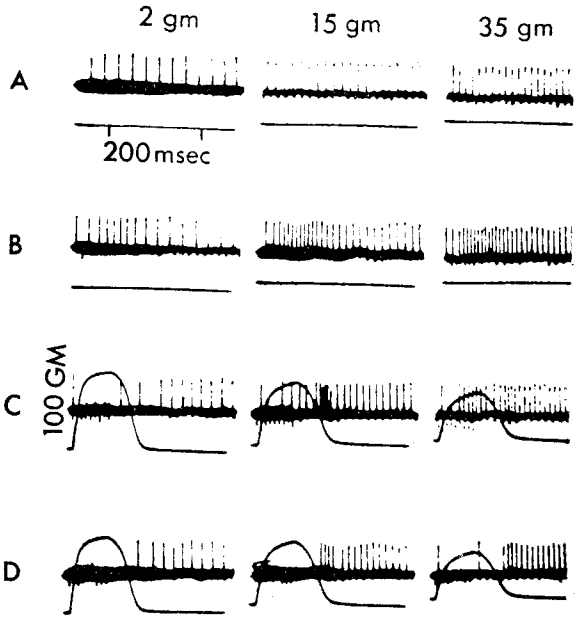

FIGURE 7 Effect of muscle contraction on discharges from single spindle receptor from flexor digitorum longus. Initial tensions of 2, 15 and 35 g. Second beam indicates strain gauge response. A, baseline discharge. B, stimulation of isolated gamma fiber (9 stimuli at 10-msec intervals at beginning of sweep). Note that effect on afferent discharge increases as muscle tension is raised. No muscle contraction results. D, similar stimulation of a portion of the ventral root containing no gamma fibers. Cessation of discharge during contraction. C, simultaneous stimulation of gamma fiber as in B and large alpha fibers as in D. At 2 g tension there is a pause in the discharge, while at 15 and 35 g gamma stimulation becomes increasingly effective. Potentials, 0.2 mV. Maximal tetanus tension, 140 g.
(Hunt and Kuffler, 1951a, *J. Physiol.* **113:** 283.)

afferents from the spindles are, in fact, completely silent (Figure 8). This may be considered to be the very mechanism allowing for good detection of muscular contractions by the intracerebellar neurons.

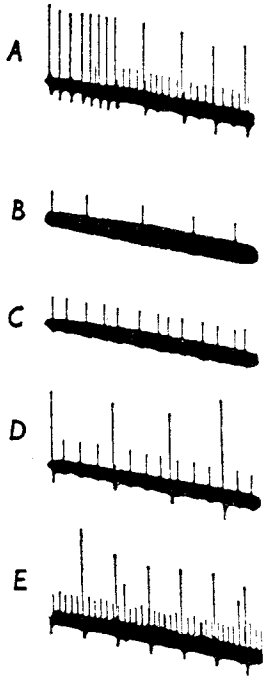

FIGURE 8 The effect of twitch versus passive extension of ankle extensor muscle *in situ*. Records from spindle (small spikes) and Golgi tendon organ (large spikes). In A, sudden contraction of muscle by efferent volley on the left side of the record: intense discharges from the tendon organs and complete silencing of spindle discharges; the end of contraction is marked by increase of spindle discharges and decrease of tendon discharges. In B, baseline discharges with weak firing of tendon organ only. In C–E, slow passive flexion of the ankle causing increase of discharges both from tendon organs and spindles. (Granit, 1955.)

In summary, we do not yet have enough information on the role of the gamma system in kinesthesis, but whatever this role is, it does not affect the essential features of the mechanism we have proposed.

Let us see now how this whole mechanism works under normal conditions. Suppose that we flex the arm when lifting and holding a load in our hand. In that situation the flexor muscles of the arm are contracted against resistance, and consequently their tendons are strongly stretched. As a result, the units of the interposite (or dentate?) nucleus in the cerebellum, representing the flexor muscles of the arm, are strongly excited. On the other hand, the secondary spindle endings are much less excited, if at all, and as a result the messages about the contraction of the flexor muscles reach the nucleus concerned, from which they are conveyed to the cerebral cortex.

Quite different is the situation of the extensor muscles. These muscles are, of course, passively stretched. In consequence, the Purkinje cells representing the stretch of extensor muscles are strongly activated and send inhibitory impulses to the intracerebellar units representing stretching of tendons of extensors. As a result, the message about the stretch of extensors will not be passed through by these units.

The same reasoning may be applied when the extensor muscles are contracted against resistance —the situation which occurs, for instance, when we push a heavy object by hand. Extensors are then strongly contracted, and in consequence, secondary spindle endings are silenced while tendon organs are activated; accordingly, the messages from these, delivered to the interposite nucleus, are not inhibited by Purkinje cells and are passed through to the cerebral cortex. On the other hand, the flexor muscles are simply passively stretched, and as explained above, the messages about this stretching cannot pass to the brain.

We should add that, as earlier indicated, in the normal condition, the greatest part of skilled movements is executed by joint operation of flexors and extensors, because in this way very precise and subtle motor acts can be accomplished. Accordingly we are dealing here with the precise coordination of both flexors and extensors, which insures that the complex information concerning their contractions will be conveyed to the intracerebellar nuclei and transferred to the cortex.

Thus we see that, with the aid of Purkinje cells, all the passive stretches of muscles involving no elements of their contractions are, so to speak, sifted out; whereas all stretches of muscles accompanied by their contractions are passed through. Accordingly, all the active movements are, so to speak, caught by the intracerebellar nuclei and information about them is passed to the cortex.

Of course one can speculate about further integration of the information concerning movements, which takes place in the higher levels of the nervous system. As we have pointed out before, whereas information about movements is conveyed to the motor cortex (which preferably should be

called kinesthetic area) through the cerebellum, information about position of limbs, as well as information about passive tensions of muscles (Oscarsson, 1966), is conveyed through the lemniscal system to the somesthetic cortex. All this information put together can give the cortex a clear picture of the situation existing at each moment in proprioceptive receptors. Thus the cortex receives a precise feedback of everything which is going on in proprioceptors before, during, and after the skilled movement is performed.

SUMMARY

1) After deafferentation of a limb, the animal is still able to perform instrumental responses with this limb, if these responses are either very simple or well trained. On the other hand, if the response requires precision, it cannot be performed by the deafferented limb. Probably training of skilled movements in the young also requires afferent input from the limbs concerned.

2) The greatest part of purposeful motor acts in normal life is executed against resistance, which is provided either by the manipulated objects, or by antagonistic muscles if the movement requires precision. This means that these movements produce additional stretch of tendons.

3) The feedback controlling skilled movements is generated either by articular receptors (somesthetic proprioception), or by stretch receptors in muscles and tendons (kinesthetic proprioception), or by both. Articular receptors inform the brain about positions of the limbs and their changes, while muscular-tendon receptors inform the brain about movements. The former information is conveyed through the lemniscal afferent system; the latter through the cerebellum.

4) It is supposed that the somesthetic and kinesthetic proprioceptive systems can to some extent substitute for one another: accordingly, if both are destroyed, the animal is strongly incapacitated in its motor behavior. This incapacitation was produced in our experiments by serial removals of the cerebellum (or its paravermal part) and the somatosensory area of the cerebral cortex.

5) The cerebellum is considered in this paper as the convertor of messages concerning stretching of tendons and muscles into messages concerning motor acts. This convertor seems to operate in the following way: The impulses from tendon organs are supposed to be conveyed directly to the intracerebellar nuclei bypassing the Purkinje cells. On the other hand, the impulses from spindles—and specifically from secondary endings—are conveyed to Purkinje cells, which send inhibitory impulses to the intracerebellar nuclei. As a result, when a muscle is passively stretched, the impulses run both from the tendon organs to the intracerebellar cells, and from spindles to the Purkinje cells; since the Purkinje cells inhibit the intracerebellar cells, no information about the passive stretching is passed through the intracerebellar nuclei to the cerebral cortex. On the other hand, if the muscle is contracted against resistance, the intracerebellar cells are excited by stretching of the tendons, but corresponding Purkinje cells remain silent; therefore, the intracerebellar cells are excited and convey this excitation to the cerebral cortex. Accordingly, the Purkinje cells play the role of a filter by which information about passive stretching of muscles is suppressed and cannot reach the brain, whereas information about active contraction is passed through.

6) The experimental findings supporting this hypothesis are presented and its further consequences are discussed.

REFERENCES

Eccles, J. C., Faber, D. S., Murphy, J. T., Sabah, N. H., and Taborikova, H., 1969, Firing patterns of Purkinje cells in response to volleys from limb nerves, *Brain Research* **14:** 222–226.

Eccles, J. C., Ito, M., and Szentagothai, J., 1967, The cerebellum as a neuronal machine. Berlin, Heidelberg, New York: Springer Verlag.

Gorska, T., and Jankowska, E., 1961, The effect of deafferentation on instrumental (Type II) conditioned reflexes in dogs, *Acta Biol. Exp. Vars.* **21:** 2; 9–34.

Granit, R., 1955, Receptors and sensory perception. New Haven, Conn.: Yale University Press.

Hebb, D. C., 1949, The organization of behavior: Neuropsychological Theory. New York: Willey.

Ito, M., Yoshida, M., and Obato, K., 1964, Monosynaptic inhibition of the intracerebellar nuclei induced from the cerebellar cortex, *Experientia* **20:** 575–576.

Jankowska, E., 1959, Instrumental scratch reflex of the deafferentated limb in cats and rats, *Acta Biol. Exp. Vars.* **29:** 233–247.

Kenard, M. A., II, and Kessler, M. M., 1940, Studies of motor performance after parietal ablations in monkeys. *J. Neurophysiol.* **3:** 248–257.

Knapp, H. D., Taub, E., and Berman, A. J., 1958, Effect of deafferentation on a conditioned avoidance response. *Science* **128:** 842–843.

Konorski, J., 1967, Integrative activity of the brain: An interdisciplinary approach, Chapter II. Chicago and London: The University of Chicago Press.

Konorski, J., 1967, Integrative activity of the brain: An interdisciplinary approach, Chapter XI. Chicago and London: The University of Chicago Press.

Konorski, J., and Tarnecki, R., 1970, Purkinje cells in cerebellum: their responses to postural stimuli in cats, *Proc. Nat. Acad. Sci.* **65:** 892–897

Matthews, P. B. C., 1964, Muscle spindles and their motor control, *Physiol. Rev.* **44:** 219–288.

Mountcastle, V. B., and Powell, T. P. S., 1959, Central nervous mechanisms subserving position sense and kinesthesis, *Bull. Johns Hopkins Hosp.* **105:** 173–200.

Oscarsson, O., 1966, The projection of Group I muscle afferents to the cat cerebral cortex. In: *Muscular Afferents and Motor Control*, ed. R. P. Granit, pp. 308–316. New York: Wiley.

Tarnecki, R., and Konorski, J., 1970, Patterns of responses of Purkinje cells in cats to passive displacements of limbs, squeezing and touching, *Acta Neurobiol. Exp.* **30:** 95–119.

Taub, E., and Berman, A. J., 1968, Movement and learning in the absence of sensory feedback. In: *Neuropsychology of Spacially Oriented Behavior*, ed. S. J. Freedman. Homewood, Ill.: Dorsey Press.

Thach, W. T., 1968, Discharge of Purkinje and cerebellar nuclear neurons during rapidly alternating arm movements in the monkey, *J. Neurophysiol.* **31:** 785–797.