basis of field size. LUNDBERG and OSCARSSON [88] observed that 50% of their units in this class did not respond to touch, but gave a few spikes upon pressure or pinch, a finding not confirmed by MANN [95]. All cutaneous and deep DSCT units studied in the absence of nerve dissection and stimulation were found to be activated by touch and/or movement of single hairs.

Volleys in cutaneous and high-threshold muscle afferents (the FRA) produced EPSPs in some of the cells studied by HONGO and OKADA [53]; however, they were few enough for the authors to suggest that the cell bodies of FRA-DSCT units may not be located in CC [54, 71, 78]. A similar suggestion also has been made for cutaneous DSCT somata [OSCARSSON, personal commun.].

Some preliminary data are available that speak to this question. The spinal cord level at which fibers enter Clarke's nucleus can be determined by electrophysiological means. LLOYD and MCINTYRE [84] showed that the conduction velocity of group I dorsal column fibers drops markedly at the L_3 level, implying that collaterals are being put down into the nucleus beginning at that level. This picture of group I is too simplified, because Ib fibers to CC come out of the dorsal columns below the level of the nucleus, while Ia and also group II fibers drop down later [104]. This finding is in agreement with SZENTÁGOTHAI's [123] demonstration that some primary afferents proceed a long distance in the cord before ending on a Clarke cell, and also with the data of HOGG [50] demonstrating three types of primary afferents in the nucleus – two of which send collaterals elsewhere as well [112]. HONGO *et al.* [54] located the somata of DSCT cells and showed that while there is considerable overlap in the distributions, the peak for FRA cells is 10 mm caudal to that for group I cells. KUNO *et al.* marked, with fast-green dye, the locations of cells that responded to cutaneous volleys and to FRA volleys. Histological examination revealed that cutaneous DSCT somata were within CC, while those of FRA DSCT cells were lateral to the column [KUNO, MUNOZ-MARTINEZ and RANDIC, personal commun.]. On the other hand, cells that respond antidromically to stimulation of the inferior brachium of the cerebellum and also to natural stimulation of skin have been isolated in a similar manner in the very lateral part of lamina IV or V of the dorsal horn in the lower part of the fourth or upper part of the fifth lumbar segments of the spinal cord [MANN, TAPPER and BROWN, unpublished observations]. No activity was evoked in these cells by muscle stimulation; i.e. they were not FRA cells.

B. Cortical Influences on Clarke Cells

90% of group I DSCT cell discharges are strongly inhibited by stimulation of the contralateral sensorimotor cortex, whether the unit is excited by natural or electrical stimuli. The remaining 10% were first excited, then inhibited [54, 105]. An IPSP occurs following cortical stimulation most often in cells that also show IPSPs following FRA stimulation. Furthermore, IPSPs are observed more often in cells with excitatory convergence from more than one muscle group than in cells with no such convergence. Single stimuli applied to the cerebral cortex produce no effect in Clarke cells, but a brief tetanus produces an IPSP that has a longer time course than those evoked from the periphery. The latency of this response is characteristic of conduction through two interneurons [53]. In view of the longer time course of the IPSP, it seems likely that at least one of these interneurons fires in bursts. Properly timed volleys from cutaneous nerves and the cortex lead to summation of IPSPs [53]. HONGO and OKADA [53] demonstrated an increase in the excitability of group Ib terminals but not Ia during trains of cortical stimuli. The relative importance of pre- and postsynaptic inhibitions from the cortex have not as yet been determined.

FRA DSCT cells are strongly excited by the same cortical stimulation; the effect is mediated by the pyramidal tract and the lateral corticospinal pathway. HONGO *et al.* [54] suggest that the activity from the cortex acts to eliminate background discharges, due to static activity in muscle afferents or to interneuronal impingement, without influencing transmission of more synchronous impulses. It may also serve to prevent firing of the DSCT cell to the wrong afferent input, thus preserving the distinction between Ia and Ib within the tract. On the other hand, facilitation of cutaneous (or FRA) DSCT cells may play a role in producing the very high synaptic efficiency of these cells.

C. Discharge Characteristics of Clarke cells

The discharge characteristics, i.e. thresholds, spikes per discharge and frequency of discharge, of DSCT axons are quite different from those observed in motoneurons for stimulation of the same afferent fibers. We may surmise that the form of the postsynaptic potential and the spike may be somewhat different in these cells, and intracellular recordings show this to be the case.

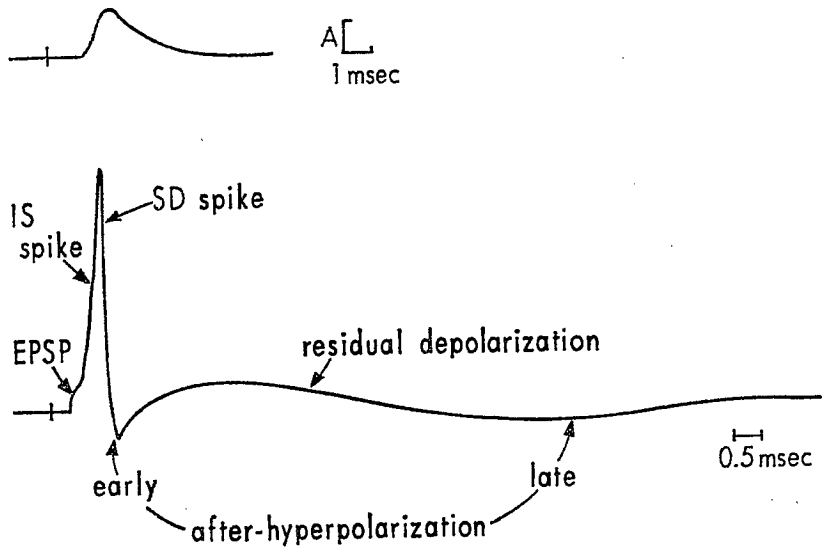

Fig. 7. Diagrammatic potentials illustrating typical time course of EPSPs (upper trace) and action potentials (lower trace) in cells of CC. The indicator A in the upper trace has a value of 25–30 mV for group I stimulation, and 25–65 mV for dorsal root stimulation. Initial segment-somadendritic spike separation is slightly exaggerated for the purpose of illustration.

The spike potential evoked orthodromically, antidromically or by current injection in Clarke cells is characteristically of short duration, on the order of 0.50–0.65 msec [24, 53, 74], and followed by a brief hyperpolarization, a depolarization (residual depolarization), that reaches 4 mV in amplitude and 10 msec in duration, and a hyperpolarization of 2 mV, lasting 30–120 msec [37] (fig. 7). The durations of the action potentials are reminiscent of those reported by HUNT and KUNO [57] for cells of the dorsal gray matter.

There is a clear separation between initial segment and somadendritic spikes in the response, suggesting that the soma has a higher threshold than does the initial segment and that the spike is initiated at the initial segment. The two components persist at high frequencies of stimulation (>50/sec), implying a high safety factor in conduction of the spike to the soma [24, 37, 73]; it is nearly impossible to cause the somadendritic spike to fail. Such a high safety factor also is characteristic of the type C neuron of the spinal intermediate nucleus [26].

1. Repetitive Firing

ECCLES *et al.* [31] suggested that the residual depolarization was the rebuilding of the EPSP as the result of a prolonged transmitter action, a phenomenon originally proposed by MCINTYRE and MARK [91] to explain repetitive firing in DSCT neurons. Two lines of evidence have been used to argue against this interpretation: (1) unitary EPSPs are too short to be caused by long lasting transmitter action and (2) the residual depolarization is abolished by passage of hyperpolarizing current [37]. KUNO and MIYAHARA [74] calculated that the transmitter action does not exceed 2 msec, a time too short to account for the residual depolarization. This rebuilding of the EPSP could be the apparent result of the shortness and early initiation of the spike potential on the EPSP so that the EPSP outlasts the spike. Another possible explanation is that the EPSP on the proximal dendrite facilitates the antidromic invasion of the dendrites by the action potential, giving a delayed depolarization. This explanation, suggested by GRANIT *et al.* [42], receives support from the observation that the residual depolarization is greater after orthodromic spikes than after antidromic spikes. Whichever is the case, the explanation depends upon the properties of the somadendritic membrane [37].

As noted above, many DSCT axons discharge repetitively to electrical nerve stimulation. MCINTYRE and MARK [91] also proposed that it could be the result of a repetitive presynaptic bombardment of the Clarke cell. KUNO and MIYAHARA [74] also noted this tendency to discharge in multiple fashion, but they demonstrated that in every Clarke cell the strength of afferent stimulation always could be adjusted to produce only one spike. This observation makes it clear that the tendency toward multiple discharge is not a property of the postsynaptic membrane but depends upon the input. Transmitter action is too short to account for this phenomenon, but the residual depolarization may play a role in the initiation of multiple spikes. KUNO and MIYAHARA [74] showed that EPSPs have three or more phases in cells that respond in this fashion, implying that repetitive firing is due to temporal dispersion of presynaptic volleys. As mentioned earlier, such a dispersion has been noted in the dorsal columns for cutaneous impulses [84]; DSCT axons activated by cutaneous stimulation most often show such behavior [88, 91].

2. High-Frequency Discharges

The DSCT is specialized to carry impulses at very high frequencies for prolonged periods in contrast with motoneurons, which can dis-

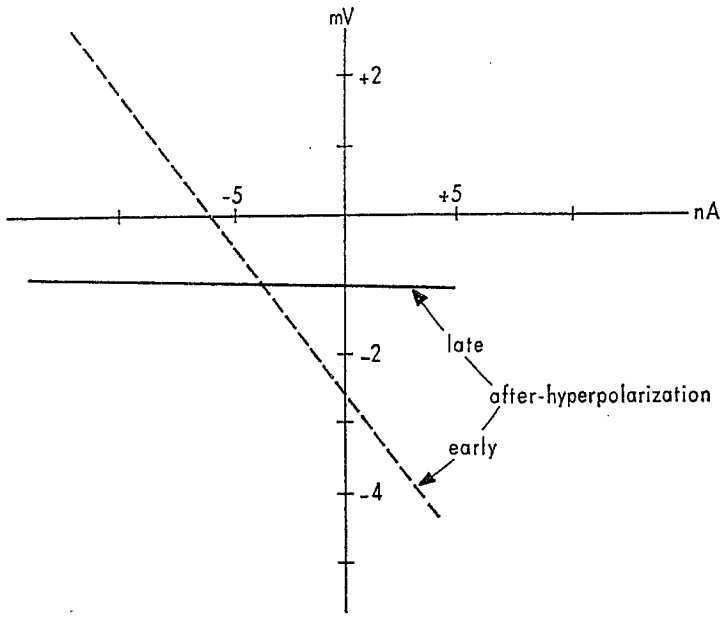

Fig. 8. Changes in peak voltages (ordinate) of the early and late after-hyperpolarization in CC cell due to alterations in the membrane potential. The potential can be altered by injection of current through a microelectrode in amounts indicated along the abscissa. Values taken from KUNO *et al.* [75].

charge only at low frequencies. There are several factors in the discharge of DSCT cells that allow the high discharge frequencies.

The early after-hyperpolarization in Clarke cells is small and of short duration, and so produces only little depression of excitability in the neuron. It varies in amplitude with changes in membrane potential and its polarity is reversed by hyperpolarizing the cell membrane [75], as shown in the graph of figure 8. It behaves like the after-hyperpolarization in squid axons [49].

The excitability of the neuron is decreased during the late after-hyperpolarization that follows the action potential in DSCT neurons, but the after-hyperpolarization lacks the reversal point characteristic of those in motoneurons [22] (fig. 8). Accompanying the hyperpolarization is a change in membrane conductance of only 10%, which is small in contrast with the 40% observed in motoneurons [59]. EIDE *et al.* [37] suggest that it is unlikely that the hyperpolarization is caused by in-

creased permeability to potassium ions because of the small change in conductance. They suggest that the membrane potential in Clarke cells may be more sodium-dependent than in motoneurons. According to EIDE *et al.* [37], the after-hyperpolarization might then be the result of a slow increase in outward sodium conductance (possibly in conjunction with a decline in potassium conductance) after an inactivation by the spike. Such a system could operate without appreciable conductance changes during the after-hyperpolarization; however, that such a unique membrane actually exists awaits further evidence. Because the conductance change in Clarke cells is only one fourth of that in motoneurons does not necessarily imply that K^+ plays no role. The after-hyperpolarization is, after all, smaller in Clarke cells.

An alternative explanation was supported by KUNO *et al.* [75] who suggested that the increase in external K^+ and internal Na^+ concentrations trigger an electrogenic pump, producing the hyperpolarization. This explanation is supported by their observations that the hyperpolarization is temperature-dependent, its half-decay time is increased by hyperpolarizing the cell near the K^+ equilibrium potential (thus reducing the K^+ efflux), and its amplitude exceeds the equilibrium potential of K^+ estimated from the early after-hyperpolarization of the action potential. Whatever the mechanism for this hyperpolarization, it does not prevent most DSCT cells from firing at rates of several hundred per second in response to peripheral stimulation [51]. This is also in spite of a reduction in the size of EPSPs (at least for Ia and Ib volleys) at stimulation frequencies above 1/sec [31].

A supramaximal antidromic volley, initiated during a repetitive train of spikes evoked either orthodromically or by injection of depolarizing current, usually evokes a discharge of the Clarke cell that briefly interrupts the train by collision. If the volley fails to evoke a spike discharge, the train of spikes in the Clarke cell is unaltered, implying that there is not feedback inhibition [53, 74]. The lack of feedback inhibition, the short duration of the spike potential and the relatively little after-hyperpolarization provide favorable conditions for the high-frequency discharges which may be evoked by orthodromic stimulation or by current injection [74]. Frequencies up to 1,000/sec have been observed by many investigators. KOSTYUK [73] has recorded action potentials evoked monosynaptically by stimulation of peripheral nerves and by stimulation of the DLC above the recording electrode in cells of Clarke's nucleus that do not project into the DLC. He suggests that these cells are the border

cells of RETHELYI [112] or the small cells of CC that remain unchromatolyzed following section of the DLC [82], and that they are interneurons which have a facilitatory effect on cells that do project into the DSCT. They are clearly not inhibitory, since no disynaptic IPSPs were recorded in DSCT somata following DLC stimulation, but rather, an increased depolarization was observed. KOSTYUK [73] attributed to this facilitation a role in maintaining the excitability of the membranes of DSCT cells that remains fairly constant during repeated stimulation over long periods. High-frequency orthodromic firing is probably also aided by potentiation of the amplitude of the EPSP that was observed by ECCLES *et al.* [31] for group Ib volleys at repetitive rates between 30 and 100/sec.

3. Sensitivity of Clarke Cells

As mentioned earlier, the DSCT has a very steeply rising input-output function as measured from the mass potential recorded from the dissected dorsolateral funiculus. Mechanisms that may produce this potency of transmission have been elucidated by intracellular recordings.

A maximal group I volley produces a monosynaptic EPSP in Clarke cells of 25–30 mV, lasting about 10 msec, the duration of the EPSP in motoneurons [36]. A typical time course of EPSPs in Clarke cells is shown in figure 7. KUNO and MIYAHARA [74] observed monosynaptic EPSPs up to 65 mV amplitude following stimulation of the dorsal roots, contrasting with the 30 mV maximum observed for the group I stimulation. The notion that the larger potentials are due to activation of cutaneous fibers receives support from the observation of MCINTYRE and MARK [91] that cutaneous units in the DLC have longer discharge trains than group I units. The decay of the EPSP was observed by ECCLES *et al.* [31] to have two phases, suggesting a prolonged transmitter action; however, most investigators [37] agree that the decay has only one phase unless the cell is badly damaged. The single phase exponential decay is consistent with the passive properties of the membrane and a shorter transmitter action.

u-EPSPs, observed during muscle stretch, range from <1 mV to as much as 5 mV in amplitude. The u-EPSPs have rise times of 0.2–1.0 msec and half-decay times of 1–3 msec [31, 36, 38, 74], which are the same as for large composite EPSPs [74]. The large u-EPSPs (those with amplitudes between 1 and 5 mV) contrast strikingly with those observed in motoneurons, which never exceed 1 mV in amplitude [15].

The distribution of sizes of u-EPSPs indicates that many quanta of transmitter are released by a single presynaptic impulse. EIDE *et al.* [36] computed that an impulse in a single group I afferent releases 20–50 quanta of transmitter. This large number may be related to the extensive and multiple contacts of the primary afferents on the DSCT cells [124]. A quantum of transmitter arriving at the postsynaptic membrane would produce, on the average, 20–40 μV of depolarization in a Clarke cell [36], not very different from the 20–30 μV computed for motoneurons [99]. The very large size of u-EPSPs in Clarke cells must be the result of the large number of quanta released at a synapse, as well as the extensive convergence on the cells and the high membrane input resistance (see below) [74].

Using the values of the amplitude of the maximum EPSP observed and the average amplitude of the u-EPSPs, EIDE *et al.* [38] computed that 12–16 primary afferent fibers converge onto each Clarke neuron. This convergence is on the order of one fourth to one third of the primary afferents of one of the ankle muscles and contrasts with the 94% reported by MENDELL and HENNEMAN [99] for group Ia afferents on homonymous motoneurons. It must be remembered that these results were obtained by different methods and thus may not be strictly comparable. From consideration of the tactile pad receptor and its connections with type I cutaneous afferent fibers, MANN [95] concluded that activity from as many as 37 type I afferent fibers converges onto a single cutaneous DSCT cell. Similar estimates are unavailable for other types of cutaneous afferent fibers.

The average membrane input resistance for DSCT cells has been measured as 3.6 MΩ and the time constant of the membrane at 4.4 msec [74]. This value of input resistance is surprisingly high in comparison with that observed for motoneurons by COOMBS *et al.* [21], on the order of three times higher.

JANSEN *et al.* [62] suggested that the variability in size of EPSPs recorded from the soma of a Clarke cell was due to differences in electrotonic attenuation, the result of location of synapses at different distances from the soma [124]. That location differences are the only explanation seems unlikely, since the time course of u-EPSPs is less variable than for motoneurons [15] and furthermore, the rise times and half-decay times are about the same for large and small composite EPSPs [74]. Differences in the number of quanta released at different synapses are probably more important. This notion is supported by the demonstration by

KUNO and MIYAHARA [74] that activation of different single primary afferent fibers evokes EPSPs of different sizes.

The level of depolarization on the EPSP at which the spike potential is initiated is about 8 mV [38, 74]. Knowing this and the average amplitude of the u-EPSPs, it is clear that as few as 2 or 3 presynaptic impulses may bring the Clarke neuron to threshold, causing a discharge. Circumstantial evidence from knowledge of the firing characteristics of type I cutaneous afferent fibers [13] suggests that cutaneous DSCT cells may require fewer afferent impulses and, in fact, may fire in response to a single action potential stimulus [95]. Whether this observation will hold for all cutaneous DSCT cells remains unknown. A similar argument has already been made for such efficient transmission from the same afferent fibers to cells of the dorsal gray matter of the L_7-S_1 segments of the spinal cord [125, 126].

D. Information Conveyed in Clarke Cell Discharges

What one would like to know about any fiber tract is what information it carries and how that information is coded. Unfortunately, we know little about coding in cutaneous DSCT cells and nothing at all about FRA DSCT cells.

Discharge patterns of muscle-activated DSCT neurons have been studied in a series of experiments by JANSEN *et al.* [62–66], JANSEN and RUDJORD [67], and JANSEN and WALLØE [68, 69]. Some properties they described for group Ia-, Ib- and group II-activated neurons have been reported above. If maintained currents are injected into a Clarke cell, maintained firing is evoked. There is a linear relationship between mean frequency of discharge, in DSCT neurons activated by group Ia afferents, and muscle length [67]. For those units responding to muscle spindle afferents, there is an irregularity of discharge in trains evoked by muscle stretch that is not characteristic of the primary afferents themselves. This irregularity is not the result of random variations, because there is a strong negative serial correlation (the correlation between the length of given interval and the interval before it) between the durations of neighboring interspike intervals [62, 63]. The correlation is less strong for group II DSCT cells than for group Ia DSCT cells. The simplest interpretation of this correlation, that two impulses occurring close together have their subnormal periods summed, producing a depression

that is more pronounced and longer lasting than after a single impulse, is supported by the finding that the refractory period is longer after two spikes occurring close together than after a single spike [62]. A related observation is the summation of after-hyperpolarization from two antidromic spikes [37, 75].

The significance of the pattern of spike discharges in group Ia DSCT cells is perhaps indicated by the work of WALLØE [133]. The 'reading time' required to determine the frequency of primary afferent fiber discharge, and thus the muscle length, from the DSCT cell discharge was significantly shorter when the intervals were read in the order of occurrence than when the same intervals were read in random order. Apparently, the ordering of the intervals is of critical importance in transmission of information. It must be borne in mind that as many as 15 primary afferent fibers converge onto a single DSCT cell, making a direct 'reading' impossible. Perhaps this is a compromise of simplicity of information transmission for a smaller number of cerebellopetal fibers.

DSCT units in all classes discharge spontaneously [88], but not all groups of cells are equally active (table I). Group Ia DSCT cells tend to discharge at about 10/sec, while Ib DSCT fibers discharge at about 20/sec [67]. On the other hand, cutaneous cells discharge spontaneously at a mean rate 8/sec, while FRA cells discharge at 12/sec [95]. There are conflicting results as to whether this spontaneous activity remains following section of all of the dorsal roots. HOLMQVIST *et al.* [51] showed that section of all lumbosacral dorsal roots does not abolish spontaneous activity. They speculate that it is maintained by excitatory connections with interneurons that already have been described in the pathways to some DSCT cells. PYATIGORSKY [111; see 73] showed that some DSCT cells became silent when the ipsilateral hindlimb was completely denervated, and he concluded that background firing was determined primarily by background rhythmic impulses arriving through low-threshold muscle afferents. On the other hand, cutaneous DSCT axons discharge spontaneously [95], even though cutaneous primary afferent fibers are not spontaneously active [12, 14]. Spontaneous activity, whatever its sources, may serve the function of maintaining the level of excitability, permitting the discharge in primary afferents with monosynaptic connections to be transmitted during adequate activation. In engineering language, it may control the operating point of the system.

The spontaneous discharge of the proprioceptive DSCT neuron is highly regular compared to the response evoked by stretching the mus-

cle. STEIN [121] and JANSEN and WALLØE [69] have shown that in models of neuronal systems like the DSCT, the standard deviation of the interval distribution is inversely proportional to the square root of the number of quanta required to reach threshold. The high degree of regularity of background firing suggests that the amount of depolarization caused by a single interneuron impulse is small. This may be related to the small size of interneuron synapses shown by anatomists [113, 124]. Also suggested by these findings is the possibility that the EPSPs at group II synapses are appreciably smaller than at Ia synapses, since they produce a more regular discharge [63]. These notions await further data.

Conduction velocities of DSCT axons were measured in two studies from the antidromic latency and conduction distance [88, 95]. Using HURSH's [58] constant, axons of the DSCT were computed to be 3–20 μm in diameter, corresponding to velocities of 20–120 m/sec. The observed range of velocities was approximately the same for each of the classes of DSCT cells (table I) so that on the average, activity reaching the nucleus at a given time, regardless of its source, will arrive at the cerebellar cortex at the same time. That is, the DSCT preserves any differences in timing introduced by differences in conduction velocities of the primary afferent fibers. Thus, if the cerebellum is acting as a clock for precise timing of peripheral events as proposed by some investigators [8, 40], the extraction of such information from simultaneously evoked peripheral signals arriving at the central nervous system at different times occurs in the cerebellar cortex, not in the DSCT [95].

E. Cerebellar Cortical Activity Due to DSCT

Up to the end of 1971, there have been no studies of responses of single cerebellar interneurons evoked by DSCT activity alone. It is exceedingly difficult to design and time-consuming to perform an experiment that isolates DSCT activity within the cortex, since the DSCT is mixed with many other fiber tracts at both the spinal cord [95] and brain stem levels [16]. Usually the pathway taken by impulses in reaching the cerebellum is inferred from the primary afferent fibers stimulated and the known composition of the various pathways. With the reservation in mind that this is not necessarily a safe procedure nor is there necessarily a unique pathway, we can draw certain conclusions from some work that has been done.

Upon entering the cerebellum, the conduction velocities of the DSCT fibers are reduced, probably as a result of narrowing of the fibers [115]. It is unlikely that this slowing is much the result of branching, since LUNDBERG and OSCARSSON [88] showed that the terminations of single DSCT fibers are very restricted and there is only one termination site for each axon. Once into the cortex, the DSCT fibers appear to do what all other mossy fibers do (with the possible exception of the reticulocerebellar mossy fibers [3]) namely, excite first the deep Golgi cells and then, about 0.5 msec later, the granule cells [34]. Spatial and temporal summation appear to be necessary to bring single granule cells to discharge except when the excitation is produced by volleys in some low-threshold cutaneous afferent fibers. The mechanism for this summation is partly provided by the convergence of four to five mossy fibers onto each granule cell [30]. As a result of this convergence, granule cells typically respond to stimulation of a larger array of peripheral nerves than do DSCT axons [27].

The mossy fiber excitation of deep Golgi cells produces inhibition of the mossy fiber-to-granule cell transmission that may be strong enough to completely suppress impulse discharges in weakly excited granule cells [34]. ECCLES *et al.* [34] have speculated that this may serve to sharpen the focus of mossy fiber action. The granule cells that do reach firing level as a result of a mossy fiber volley strongly excite the superficial Golgi cells, which in turn inhibit the mossy fiber-to-granule cell transmission. In addition, the granule cell axons, the parallel fibers, influence Purkinje cells directly by excitation (fig. 3) and indirectly, probably through basket and stellate cells, by inhibition. That basket cells produce the inhibition is inferred from the similarity in the timing of basket cell discharges and the Purkinje cell inhibition.

Mossy fiber inputs to Purkinje cells are often predominantly inhibitory, which may seem surprising when it is recalled that nearly 200,000 granule cells contribute excitatory synapses to each Purkinje cell [30]. However, in order to be excited by a mossy fiber volley, a given Purkinje cell must be on the beam of parallel fibers excited by that volley. The cell need not be on the beam of parallel fibers to receive inhibitory effects produced through other interneurons. Strong excitatory effects on Purkinje cells from muscle afferent volleys are rare except in unanesthetized decerebrate preparations. A single volley usually has a small effect, but double and triple volleys may be very effective by comparison [28]. A few Purkinje cells are dominated by group I muscle afferent activity

while others respond predominantly to group II activity. These cells show any combination of excitatory and inhibitory actions by converging mossy fibers, apparently regardless of the physiological action of the muscles involved [29].

Most Purkinje cells, on the other hand, respond predominantly to stimulation of cutaneous nerves; especially effective are those fibers of lowest threshold. Natural stimulation of the skin has been employed in one study of mossy fiber effects in the cerebellum [33]. Some Purkinje cells respond to pressure on the footpads of the hindlimb, and they do so with increased frequency to increased pressure. The same large stimuli will inhibit other Purkinje cells, also in a graded manner in relation to the amount of pressure applied. Most Purkinje cells give only phasic responses whether they are inhibitory or excitatory, but others may give a mixture of phasic and tonic responses depending upon the site of stimulation. Excitation of the hairy skin with air puffs (that excite predominantly hair-associated afferent fibers) often results in mossy fiber-evoked Purkinje cell activity which resembles that from stimulation of adjacent foot- or toepads. Such behavior has been noted for different DSCT axons as well [95]. While it cannot be stated with certainty that these particular Purkinje cells responses were mediated by the DSCT, it does seem likely!

Many of these same Purkinje cells that respond to hindlimb mossy fiber volleys also respond to climbing fiber volleys, especially in a parasagittal strip in the extreme lateral zone of the vermis of lobule V [29]. There is some difficulty in interpreting the generality of this convergence of mossy fiber and climbing fiber activity, since both inputs are differentially sensitive to the anesthetic state of the animal [28]. Forelimb mossy fiber activity is found convergent with hindlimb activity in Purkinje cells that lie near the CCT-DSCT projection borders [43].

The question of whether surface-evoked potentials or even unit responses can be translated into a coherent map of the body surface is still open. ADRIAN [1], using evoked potentials, demonstrated an organization of the cortex into transverse zones with the hindlimb represented anterior to lobule V and the forelimb caudal to lobule IV. Within the hindlimb zone, parts of the limb were represented, in rostral to caudal order: hindfoot, ankle, knee and hip. A similar, but less precise, somatotopic organization was demonstrated by SNIDER and STOWELL [118]. These studies are in good agreement with the CCT and DSCT terminal degeneration studies of GRANT [43] and VOOGD *et al.* [131].

MORIN and HADDAD [100] showed that evoked potentials could be recorded from muscle, joint and cutaneous stimulation, even when only the DLC was intact. Flechsig's fasciculus carries superficial and deep activity from hindlimbs to the same area of the anterior lobe, so that the area that shows a maximum voltage deflection for stimulation of one nerve of the leg also shows a maximum for stimulation of other nerves of the same leg. This finding does not necessarily preclude somatotopy at the cortex, because some peripheral nerves have overlapping receptive fields.

Using single Purkinje cell recordings, ECCLES *et al.* [29] were unable to find any basis for other than a patchy distribution of afferent activity to the cortex. Closely situated Purkinje cells showed quite different responses to stimulation of an array of peripheral nerves, but unfortunately this was true even for Purkinje cells located on the same beam of parallel fibers. Perhaps the responses chosen for relating cells were not adequate; certainly, cells connected to the same afferent fibers should show similar responses. There is, in addition, a methodological problem. Oblique penetrations of the cerebellar cortex by a microelectrode cut across a number of folia and in any given penetration only one or two cells are recorded in each folium. This certainly is not an adequate sample for determining the sort of relationship that these investigators are seeking. Numerous *closely spaced* penetrations of single folia should yield the answer to this question!

F. Comparative Studies

Among the mammals, only the cat, rabbit and phalanger have received any attention from physiologists. MAGNI and OSCARSSON [93, 94] compared the responses in Flechsig's fasciculus in these three species and found them very similar. In the rabbit, there is a greater tendency for units to discharge in multiple fashion than has been described already for the cat. This tendency is even more exaggerated in the phalanger, which often shows several peaks in discharges recorded from the dissected dorsolateral column [93]. In the dissected column, the monosynaptic response due to cutaneous stimulation is only ipsilateral in all three species, but it is very much smaller in the rabbit and phalanger than in the cat [94]. On the basis of these observations, we may draw the tentative conclusion that there are some differences between species

within the placental mammals, as well as between placentals and marsupials, but the similarities are even more striking.

WHITLOCK [135] has carried out a unique coordinated histological and electrophysiological study of some avian species for which the anatomy has been reviewed. The DSCT, which originates in the lower lumbar spinal cord, is activated by stimulation of various peripheral nerves and by movement of joints and deflection of feathers ipsilaterally. There is no response in the dissected DLC of the duck due to stimulation of low-threshold muscle afferent fibers. This observation led OSCARSSON *et al.* [107] to the conclusion that either there is no DSCT in the duck, or it is not activated by group I muscle afferents as it is in the cat, rabbit and phalanger. In the duck, the low-threshold muscle activity is found in the ventrolateral column, presumably in the VSCT [107]. Further work is required to select between these alternatives and to describe the innervation of the DSCT, if it exists in the duck.

IV. Conclusions

The DSCT has received a good deal of attention, perhaps because of its accessibility and presumed simplicity. The accessibility of the pathway is real, but its simplicity may be brought into question by a quick glance at figure 9, which summarizes what is 'known' about it to date. Despite its complexity, this pathway is still one of the simplest to be found in the nervous system.

In view of the recent evidence of the origin of cutaneous DSCT cells, it appears that the notion of the DSCT as made up only of Clarke cell axons is not tenable. A clear separation of cutaneous and muscle DSCT cells is no more possible at the somata than in the DLC, and perhaps even at the cortex. Thus, it seems useful to think of the DSCT as a mixed tract rather than to invent new terminology for the cutaneous subdivision, confusing an already somewhat muddy taxonomic picture.

The DSCT is a heterogeneous pathway carrying exteroceptive as well as proprioceptive information directly from the spinal cord to the cerebellar cortex. En route it gives off collaterals that provide excitatory input to the cerebellar nuclei, and perhaps also to the lateral cervical nucleus. Since fibers originating in the lateral cervical nucleus send collaterals to the inferior olive [102], the same activity could reach cerebellar

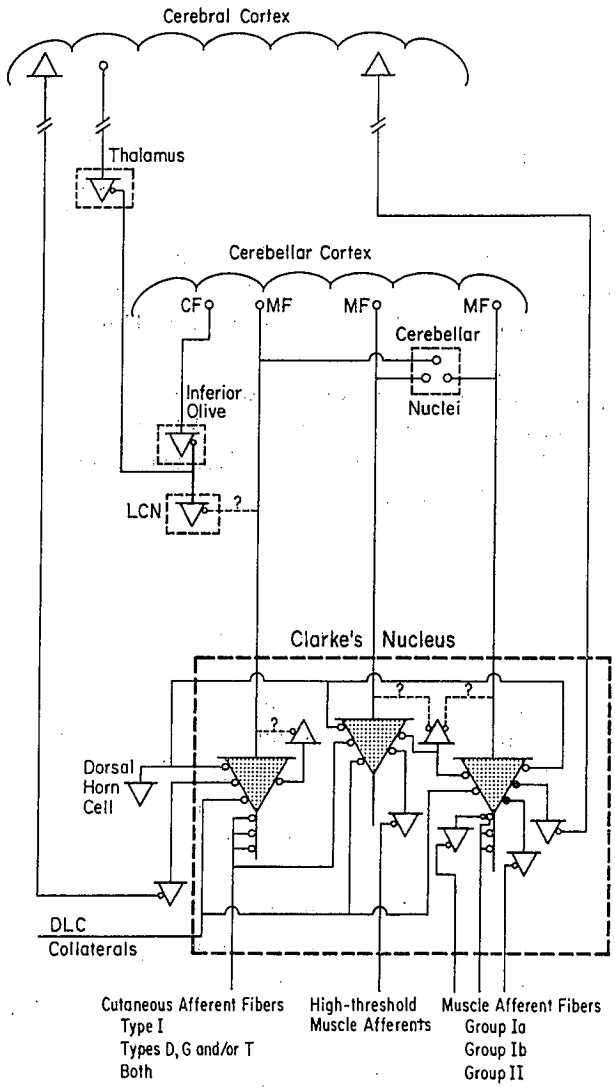

Fig. 9. A summary diagram of what is currently 'known' about the dorsal spinocerebellar system. Stippled cells are those that give rise to the DSCT. Open terminals indicate excitatory connections, while filled terminals indicate inhibitory connections. Dashed connecting lines indicate connections that are suggested, but for which no firm 'proof' exists.

Purkinje cells by way of mossy fibers and then, somewhat later, by way of climbing fibers. The significance of this dual activation has yet to be explored. If such a pathway through the lateral cervical nucleus exists, it is probable, in the light of unit recordings in the nucleus [41, 101, 109] that only cutaneous (perhaps FRA) DSCT cells send collaterals into the nucleus. Little muscle activity has been reported in lateral cervical nucleus recordings. MANN *et al.* [96] have recorded evoked activity in cerebral cortex following stimulation of single type I cutaneous afferent fibers. Such evoked activity disappeared following section of the DLC, but not the dorsal column. Since type I activity has not been found in the SCT [11], but is in the DSCT [95, 97], it is tempting to argue that it is being relayed to cortex via DSCT and lateral cervical nucleus. It must be borne in mind that non-DSCT, type I activity has also been found in the DLC; however, its destination is unknown [97]. In addition, attempts to activate lateral cervical nucleus neurons from the cerebellar cortex by way of antidromic conduction in DSCT axons have been unsuccessful, implying that DSCT to lateral cervical nucleus connections are infrequent, if they exist, or that there is a blockage of conduction at the bifurcation of the DSCT axons [55].

Upon reaching the cerebellar cortex, the DSCT terminates as mossy fibers that excite granule cells which, in turn, excite Purkinje cells [30]. There is too little indication in the literature of what discharge patterns are observed in any of these cerebellar elements upon natural stimulation of receptors [33, 127], but they have been partly worked out for the DSCT. Coded in the discharge of group Ia and group II DSCT cells is the length of a muscle under stretch, and the coded information has been altered little by the imposition of the synapse [66]. Group Ib DSCT fibers similarly code the tension developed in the muscle by stretch or contraction. It is well documented, however, that receptors of more than one muscle group can cause a given DSCT cell to discharge [86, 95], leading one to question how well the length information for one muscle can be isolated from the DSCT discharge when another muscle is stretched simultaneously. This is the likely situation when the animal is actually moving around, and it is this discharge that the cerebellum receives.

The information coded in cutaneous DSCT cell discharges has not been studied in detail, but it seems clear that the system can distinguish tonic from phasic receptor stimulation by the characteristics of the discharge in cells that respond to both, and also by the activity in those

cells that respond only to stimulation of either tonic or phasic receptors [95]. A preliminary look (fig. 6C) shows that information about the amount and duration of skin displacements is maintained in the discharges of DSCT cells that are connected only to a receptor that encodes such information, the touch corpuscle [95]. Although this receptor is also capable of signalling the frequency of repeated stimulation, the ability of DSCT cells to follow or transform such a signal needs to be studied.

The efficiency of synapses on DSCT cells makes them well suited for transmission of signals from primary afferent fibers. In fact, their efficiency makes them appear almost obligatory, but there is a transformation of the primary afferent signal that probably results from integration of signals in several primary fibers, and from regulation by cerebral cortex and perhaps other supraspinal centers [52]. Such regulation usually has the effect of enhancing any cutaneous activity and depressing muscle activity, thus emphasizing what already has a bigger effect in the cerebellar cortex as judged from evoked potentials [29, 32, 72]. If the DSCT is responsible for a substantial part of these evoked potentials, it is even more remarkable that the minority of fibers (19% of DSCT was found to be cutaneous by MANN [95]) can have a larger effect than the majority. This is further evidence of the remarkable extent of cutaneous activity in the cerebellum.

The question of function is always asked with regard to any pathway in the central nervous system, but seldom is a pathway known well enough to give support to speculations. The DSCT is well studied; however, its role in the operation of cerebellar cortex is yet a mystery. The tract is capable of transmitting information about muscle length, or muscles' lengths and tensions. It has been suggested that all the mossy fiber pathways except DSCT and CCT signal activity in interneuronal pools and that only these two pathways, which are remarkably similar, carry information about peripheral events coordinated with the evolving movement [20]. The DSCT signals different types of skin stimuli with good spatial discrimination, especially good around the foot and toes [95]. Pad DSCT cells probably give information about pressure on the foot- and toepads, while FRA cells perhaps signal the state of the flexor reflex networks or reflex patterns. OSCARSSON [105] has suggested that the FRA cells may serve as a feedback system to tell the cerebellum about the effectiveness of its regulation of interneuronal activity at a given level. It must be remembered that afferents that take part in the

flexor reflex probably participate in other activities as well, activities that may not be related to reflex activities.

At the cerebellar cortex, the DSCT axons terminate in restricted areas (often <2 mm^2) in contrast to VSCT and other tract axons, which end over wider areas. In comparison to these other tracts, DSCT activity must affect few cortical units by way of a narrow beam of parallel fibers [30]. The information encoded in this activity may play a part in the coordination of movement and posture in the ipsilateral limbs [106], the function ascribed to the intermediate cortex by CHAMBERS and SPRAGUE [17]. Whether the DSCT takes part in the cerebellar regulation of sensory transmission or in any other functions of the cerebellum is unknown, since we have only begun to see what the cerebellum does.

Although it is apparent from the brief review of studies in nonfeline species (a review not much more brief than the amount of available data) that there are species differences in the DSCT, there is no indication of the significance of these differences. Many species have received no attention, and those that have, have received too little. We have, for example, no idea of how old the DSCT is nor what were the pressures that made its development necessary for the survival of organisms. Far more comparative work is required before we can even speculate about the evolutionary significance of the DSCT.

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