A Rectifying Electrotonic Synapse in the Central Nervous System of a Vertebrate

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ABSTRACT The adductor muscles of the pectoral fins of the hatchetfish Gasteropelecus are innervated by bilateral pools of about 40 motoneurons which lie primarily in the first spinal segment. A pair of giant fibers on each side of the medulla send processes ventroposteriorly to the motoneuron pools. Electrophysiological evidence indicates that giant fibers are presynaptic to ipsilateral motoneurons, but not to contralateral ones. Transmission across the giant fiber, motoneuron synapse is electrically mediated as is indicated by direct measurement of electrotonic spread in either direction across the synapse, and by the extremely short latency of the giant fiber postsynaptic potentials (PSP's) in the motoneuron. The coupling resistance across the synapse was calculated from measurements of input and transfer resistance. The coupling resistance rectifies in such a way as to facilitate spread of depolarization from giant fiber to motoneuron, and to oppose transmission in the opposite direction. As a consequence of rectification, the giant fiber PSP in a motoneuron is augmented by hyperpolarization of the motoneuron. The coupling resistance calculated on the basis of this effect is in good agreement with calculations from input and transfer resistance data. Rectification at the electrotonic synapses may permit the motoneurons to act in small swimming movements as well as to fire synchronously in an extremely fast escape reflex mediated by Mauthner and giant fibers.

INTRODUCTION

Recent studies of neural control of electric organs and sonic muscles have shown that neurons which fire highly synchronously are likely to be electrotonically coupled (2–6). The functional significance of electrotonic transmission in these systems appears to be that it allows more rapid mutual excitation between cells than can be provided by reciprocal excitatory synapses.
that transmit chemically. The control of the pectoral fins in the hatchetfish was investigated as an extension of these studies of synchronized effector organs to a fast locomotor system. It was found that the giant fibers activating the

motoneurons were electrotonically coupled to them, and that some of the motoneurons probably were coupled to each other by an additional pathway. An unexpected finding was that the electrotonic synapses between giant fibers and motoneurons rectified in such a way as to facilitate impulse transmission from the giant fibers and oppose transmission in the opposite direction.

Figure 1. Characteristic responses of motoneurons controlling the pectoral fin adductor muscle in Gasteropeleus. Two superimposed sweeps in B, D, and E. A, an antidromic spike evoked by stimulation of the motor nerve innervating the adductor muscle. B, effect of hyperpolarization on the antidromic spike. Hyperpolarizing current (upper trace) could cause a component of the spike to fail at the inflection on the rising phase. C–E, PSP's produced in a motoneuron by repetitive spinal stimulation. The frequency was increased to the point at which excitation of the giant fibers could be expected to fail. C, the maximal PSP in the motoneuron obtained at a low frequency of stimulation. D, at a higher frequency of stimulation, the PSP could show two components somewhat separated, or the later component could fail. Apparently one of the presynaptic giant fibers could be excited somewhat later than the other. E, at a higher frequency of stimulation the PSP could have a single component ascribable to excitation of one presynaptic giant fiber, or this component could fail. F, strong spinal stimuli evoked relatively long lasting depolarizations following the PSP produced by the giant fibers (four superimposed sweeps of different stimulus strengths). Calibrations are identical in C–E.
The morphology of the giant fiber, motoneuron system and the experimental methods used were described in the preceding paper (1). The antidromic volley produced by stimulation of the peripheral nerve was usually monitored by means of a monopolar electrode on the ventral root. The identification of giant fibers was discussed in the preceding paper.

RESULTS

Identification and Properties of the Motoneurons

The motoneurons innervating the pectoral fin adductor muscle were penetrated near the entry of the first ventral root and were identified by their short latency (about 0.3 msec) spikes evoked by stimulation of the peripheral nerve to the muscle (Fig. 1 A). The resting potentials in the motoneurons were about 70 mv inside negative. The spikes were up to 90 mv in amplitude and had a duration of about 1 msec. Their rising phase usually had an inflection typical of antidromic spikes in cell bodies, suggesting delayed invasion at the axon hillock (cf. references 7 and 9). Their falling phase had a characteristic “shoulder” that was not seen in responses of either Mauthner or giant fibers. One spike component, presumably representing firing of the soma, was easily blocked by hyperpolarization. Failure of this component occurred at the inflection on the rising phase of the spikes and the remaining component, about 40 mv in amplitude, is ascribable to activity of the initial segment of the axon (Fig. 1 B).

Spinal stimulation at frequencies less than 2/sec produced a brief PSP in the motoneurons that was all-or-none when the stimulus strength was graded. This PSP was usually about 8 mv in amplitude, and its latency and duration were close to those of the giant fiber spikes (Fig. 1 C). When the frequency of spinal stimulation was increased to 5–10/sec, i.e. to the point at which the Mauthner fibers would have failed to excite the giant fibers (1), the brief PSP in the motoneuron failed. At intermediate frequencies, this PSP could be seen to consist of two all-or-none components. In the two superimposed sweeps of Fig. 1 D, one of the components was present in both sweeps while the second was present in only one sweep in which it was somewhat delayed compared to the first component. In Fig. 1 E the remaining component was present in one of the superimposed sweeps and absent in the other. As shown below, these components were due to electrotonic spread of spikes from the two ipsilateral giant fibers. That the PSP consisted of two components could also be demonstrated when paired spinal stimuli were given separated by an interval such that the second stimulus would have failed to excite one or both ipsilateral giant fibers. When repetitive stimulation caused failure of both components, a slower and much smaller potential often remained. The latency of this smaller potential was about the same as that of the two components noted above; this potential probably resulted from electrotonic spread into the
motoneurons of the PSP's in the giant fibers due to the Mauthner fibers. When the strength of spinal stimulation was increased well above threshold for the Mauthner fibers, PSP's due to the giant fibers were followed by prolonged depolarizations that were apparently PSP's mediated by other fibers (Fig. 1 F). In some experiments, these late PSP's could be large enough to excite the motoneurons. From the recording of PSP's and typical two component spikes and the known position of the motoneuron cell bodies, it can be assumed that the usual site of electrode penetration was the cell soma.

Antidromic stimulation of the nerve to the pectoral fin adductor muscle

Figure 2. Responses in motoneurons produced by graded antidromic stimuli. A, graded depolarizations were evoked in a motoneuron with a relatively high threshold axon by graded antidromic stimulation subthreshold for the cell's axon. The strongest stimulus excited the cell's antidromic spike (several superimposed sweeps). The graded depolarizations were of only slightly longer latency than the antidromic spike suggesting that they were due to electrotonic spread of spikes from neighboring motoneurons with lower threshold axons. B, antidromic spike and PSP due to antidromic invasion of a giant fiber recorded in a motoneuron that showed little coupling to neighboring motoneurons. An antidromic stimulus that was large enough to cause antidromic invasion of a giant fiber, as signalled by the brief PSP, did not produce a longer lasting depolarization underlying the PSP (cf. C-E), nor did weaker stimuli evoke a graded response. A slightly stronger stimulus evoked an antidromic spike (two superimposed sweeps). C-E, antidromic invasion from motoneurons to giant fibers signalled by PSP's in a motoneuron with a higher threshold axon. C, the antidromic depolarization which was graded with weak stimuli had a brief all-or-none component superimposed when the stimulus was sufficiently strong. D, a stronger stimulus evoked a second brief component that was somewhat delayed compared to the first. E, a still stronger stimulus caused both brief components to appear at the shorter latency. The two brief components are ascribable to antidromic invasion across the electrotonic synapses connecting the two giant fibers presynaptic to the motoneurons. Calibrations are identical in C-E.
evoked responses in the motoneurons in addition to antidromic spikes. When stimulation of the peripheral nerve was graded in the range below the threshold of the axon of a penetrated cell, graded depolarizations were evoked in many cells (Fig. 2 A). These potentials were of very short latency, about 0.1 msec (or less) longer than the antidromic spikes. They were unaffected by dorsal root section and increased in proportion to the size of the antidromic volley. These properties suggest that the graded responses resulted from electrotonic spread of activity from neighboring cells that were excited by antidromic stimuli which were subthreshold for the penetrated cell. Similar potentials have been observed in other neuron pools in which electrotonic coupling has been directly demonstrated (2–4).

The graded potentials produced by antidromic stimulation could reach an
amplitude of 4–6 mv in cells with higher threshold axons. Further increase in stimulus strength often resulted in the appearance of one or two brief all-or-none components superimposed on the longer lasting and more smoothly graded antidromic potential (Fig. 2 C–E). As discussed below, these two additional components are ascribable to antidromic excitation of the giant fibers across their electrotonic synapses with the motoneurons. In other motoneurons, the brief potentials signalling antidromic invasion of the giant fibers could be seen, although there was very little graded depolarization (Fig. 2 B). Evidently these motoneurons were at most weakly coupled to other motoneurons.

The input resistance of the motoneurons was measured indirectly as described in the preceding paper (1). A single electrode in a bridge circuit was used to determine the change in antidromic spike height as a function of applied current (Fig. 3 A). This relation gives a measure of input resistance on the assumption that the resistance at the spike peak is low compared to that at rest (8). Measurements were necessarily over a restricted range since the antidromic spikes were easily blocked by hyperpolarization (Fig. 1 B) and small depolarizations excited the cells. In eight experiments, the input resistance had a mean value of 0.84 megohms, and ranged from 0.6 to 1.1 megohms. These measured values were probably somewhat low because of the finite resistance at the peak of the spike (1, 8), and in subsequent calculations, the input resistance of the motoneuron is taken as 0.9 megohm.

Relation between Giant Fibers and Motoneurons

The synaptic relation between giant fibers and motoneurons was established by experiments in which these elements were simultaneously penetrated. In these experiments the giant fibers were penetrated caudal to the region of the cross-branches contacting the contralateral Mauthner fibers, and there was no ambiguity about the side on which the giant fibers ran. Furthermore, the probability of penetrating the cross-branches must be much less than that of penetrating the main longitudinal courses of the fibers. In over 20 experiments a directly evoked spike in a giant fiber produced a PSP in an ipsilateral motoneuron (Figs. 4 A and 5 E). In seven additional experiments both giant fibers on one side were penetrated successively and each produced a PSP in a simultaneously penetrated ipsilateral motoneuron. In six of these experiments a different motoneuron was recorded from during stimulation of each of the two giant fibers. In the remaining experiment the same motoneuron was recorded from while the two giant fibers were successively penetrated.

These results indicate that both ipsilateral giant fibers are presynaptic to most or all the motoneurons on the same side. In at least five experiments, stimulation of a giant fiber produced no effect in a contralateral motoneuron
Figure 4. Synaptic relations between motoneurons, giant fibers, and Mauthner fibers. A, latency of the PSP in a motoneuron. A directly excited giant fiber spike (middle trace; stimulating current on the lower trace) produced a PSP in the motoneuron (upper trace). The latency from the beginning of the spike to the beginning of the PSP was less than 50 μsec. In this and subsequent figures, the labels on the traces, mf, gf, and mn, refer to Mauthner fiber, giant fiber, and motoneuron respectively. B, PSP’s in a motoneuron produced by repetitive stimulation of a giant fiber. A train of three directly excited giant fiber spikes (middle trace, stimulating current on lower trace) produced PSP’s in the motoneuron (upper trace) that were essentially identical in amplitude and shape although the stimuli were separated by intervals of only 8 msec. C, a directly excited spike in a Mauthner fiber (middle trace, current on lower trace) produced a characteristic PSP in the motoneuron (upper trace). In a second (superimposed) sweep, a depolarizing current of 1.3 namp was applied in the motoneuron. The PSP alone was not adequate to excite the motoneuron, but it summated with the applied current to initiate a spike. D, antidromic excitation of a giant fiber by graded stimulation of the ipsilateral motor nerve. As stimulus strength was graded, graded depolarizations were evoked in the ipsilateral giant fiber (upper trace) but not in a contralateral giant fiber (lower trace). These depolarizations became large enough to excite the giant fiber in one of the several superimposed sweeps. The apparent firing level was about 8 mv.

That had been simultaneously penetrated, which indicates that there is no synaptic relation between the contralateral giant fibers and motoneurons. This is supported by the observation above that repetitive spinal stimulation showed only two brief components in the PSP, since there would have been additional components if there were significant contributions from the contralateral giant fibers or other ipsilateral fibers. Moreover, as described
below, antidromic stimulation of the peripheral nerve caused graded depolarizations only in ipsilateral giant fibers (Fig. 4 D).

The amplitudes of the PSP’s produced in one motoneuron by the two ipsilateral giant fibers were often nearly equal but sometimes differed by as much as a factor of two or three. The relative amplitudes could be assessed by repetitive spinal stimulation as in Fig. 1 C–E, by graded antidromic stimulation of the peripheral nerve where invasion of the two ipsilateral giant fibers occurred successively (Fig. 2 C–E), and by comparison of the PSP’s in response to direct stimulation of one giant fiber to the PSP’s generated by spinal stimulation that could be assumed to have activated both giant fibers (Fig. 5 D and E).

Although a directly excited spike in a giant fiber evoked a PSP in a postsynaptic motoneuron, a directly excited spike in the motoneuron produced very little response in the presynaptic giant fiber (Fig. 5 F). This finding would be expected for a chemically transmitting synapse, but is not inconsistent with electrical transmission (cf. reference 2). As will be discussed below, an important factor contributing to this asymmetry of impulse transmission is rectification in the junctional membranes that favors spread of depolarization from giant fiber to motoneuron.

When a large number of motoneurons were activated by antidromic stimulation, appreciable antidromic responses could be recorded in giant fibers as noted in the preceding paper. These potentials were seen only in ipsilateral giant fibers (Fig. 4 D) and had the same properties as the graded antidromic depolarizations in the motoneurons, that is, they were unaffected by dorsal root section, they were of only slightly longer latency than the spikes in the motoneurons, and they increased in size in proportion to the size of the antidromic volley. These properties and the electrotonic coupling demonstrated below indicate that the depolarizations were a result of electrotonic spread of antidromic spikes from motoneurons to giant fibers. When enough motoneurons were excited antidromically, the giant fiber could be excited (Fig. 4 D). Thus potentials typical of giant fiber PSP’s could be evoked in motoneurons by antidromic stimulation of the peripheral nerve (Fig. 2 C–E).

Under the experimental conditions, the PSP’s from the giant fibers did not excite the motoneurons. However, if an otherwise subthreshold depolarizing current was passed during the PSP, a characteristic motoneuron spike could be evoked (Fig. 4 C). Spinal stimulation in an uncurarized animal produced activity in the pectoral muscle at a latency consistent with excitation of the motoneurons by way of the Mauthner and giant fibers. Probably the absence of excitation in the microelectrode experiments was a result of depression due to preparative manipulations or injury by the penetrating microelectrode.
Electrotonic Coupling between Giant Fibers and Motoneurons

The latency of the PSP produced in a motoneuron by a giant fiber spike was less than 0.05 msec (Fig. 4 A). The smallness of this value indicates that transmission between the two cells is electrically mediated, since chemically mediated transmission is characterized by a much longer delay (6, 12). Fur-
thermore, repetitive stimulation had little effect on PSP amplitude which is more characteristic of electrically mediated transmission (Fig. 4 B).

The occurrence of electrically mediated transmission was demonstrated directly by measurement of electrotonic coupling between giant fibers and motoneurons (Fig. 5 A–C). Both hyperpolarizing and depolarizing currents passed through an electrode in one cell produced a potential in the second cell as is shown for hyperpolarizing currents in Fig. 5 A and B. When either recording or current electrodes were withdrawn to a just extracellular position, the potentials were much smaller, showing that most of the recorded potentials were developed across the cell membrane and that voltage drops across the resistance of the extracellular medium were not significant (Fig. 5 C). The smallness of the extracellular potentials may also be seen from the slow rise and fall of the electrotonic potentials in Fig. 5 B. Extracellular potentials are in general much more rapidly rising (cf. reference 2).

The coupling resistance between giant fibers and motoneurons behaved like a rectifier whose resistance was lower when the giant fiber was positive to (that is, more depolarized than) the motoneuron. This orientation is such that depolarization is transmitted more readily from giant fiber to motoneuron than in the opposite direction. The electrical properties of the giant fiber, motoneuron synapse can best be discussed with respect to the equivalent circuit shown in Fig. 6 A. The subscripts, gf and mn, indicate the giant fiber and motoneuron respectively; $R_{gf}$ and $R_{mn}$ are the cell resistances, that is input resistances exclusive of current flow through the junctions between them; $R_e$ is the coupling resistance which is a function of transjunctional potential; $I_{gf}$ and $I_{mn}$ are currents applied in either cell; and $V_{gf}$ and $V_{mn}$ are either the voltages due to applied current or voltages generated by cell activity. Note that the input resistances, $V_{gf}/I_{gf}$ or $V_{mn}/I_{mn}$, are less than the cell resistances, and that membrane capacities are not considered.

This circuit indicates the relation between a single giant fiber and motoneuron and simplifies the actual relations, since giant fibers end on many motoneurons and motoneurons are apparently coupled to each other as well (see below). However, it is a simple theorem of circuit theory that for “three terminal” resistance measurements, any network can be reduced to an equivalent of this type (cf. reference 2). (In these experiments the three terminals were the two microelectrodes and the ground electrode in the bath.) The approximations introduced by the simplified circuit are small and will be discussed later in connection with a more elaborate circuit.

The evidence for rectification in $R_e$ is as follows. When sufficiently large hyperpolarizing currents were applied, the transfer resistances (final or steady-state values of $V_{mn}/I_{gf}$ and $V_{gf}/I_{mn}$) differed in the two directions (Figs. 5 A and B and 6 B and C). Circuit theory requires that for a linear three terminal network the transfer resistances in the two directions should
Figure 6. Transfer and coupling resistances between giant fibers and motoneurons. A, equivalent circuit representing the relation between one giant fiber and one motoneuron. The subscripts, gf and mn, indicate giant fiber and motoneuron respectively. $R_{gf}$ and $R_{mn}$ are the cell resistances, and $R_c$ is the coupling resistance, shown as variable. $I_{gf}$ and $I_{mn}$ are currents applied in each cell. $V_{gf}$ and $V_{mn}$ are the potentials in the cells. B, potential in a motoneuron as a function of current in a giant fiber. C, potential in the same giant fiber as a function of current in the same motoneuron (sample records from this experiment are shown in Fig. 5 A and B). D, coupling resistance as a function of transjunctional potential ($V_{gf} - V_{mn}$). The coupling resistance decreased when the giant fiber was relatively depolarized and increased when the motoneuron was relatively depolarized. This curve was calculated from equations 2 and 4 and the data of Figs. 6 B and C and 3 A and B.
be equal (cf. reference 2). Thus, one or more of the resistances in the equivalent circuit of Fig. 6 A must have been nonlinear; that is, its magnitude was altered during the period that current was applied. When hyperpolarizing currents of increasing amplitude were applied in the giant fiber, the transfer resistance decreased; with increasing hyperpolarization in the motoneuron, the transfer resistance increased. Complete transfer characteristics were obtained in four experiments. In five other experiments, data were obtained for hyperpolarizing currents applied in either the giant fiber or motoneuron, and in each case, there were changes in transfer resistance corresponding to those of Fig. 6 B or C.

It is clear from the equivalent circuit that rectification in $R_e$ could explain the changes in transfer resistance. If $R_e$ increased when the giant fiber was hyperpolarized, the electrotonically spread potential in the motoneuron would be smaller than if $R_e$ remained constant, and the transfer resistance, $V_{mn}/I_{gf}$, would decrease as $I_{gf}$ increased. If $R_e$ decreased with hyperpolarization in the motoneuron, the potential in the giant fiber would be larger than if $R_e$ remained constant, and the transfer resistance, $V_{gf}/I_{mn}$, would increase as $I_{mn}$ increased.

The changes in the transfer resistances may also be understood from their relation to the other resistances in the circuit. Since the transfer resistances in the circuit of Fig. 6 A are given by $R_{mn}R_{gf}/(R_{mn} + R_{gf} + R_e)$ for either site of current application, the observed changes in transfer resistance would occur if $R_e$ were changed in the opposite direction. Moreover, it can be shown that the variation of the transfer resistances cannot be explained by changes in $R_{gf}$ or $R_{mn}$. Over the full range of hyperpolarizing currents applied in the giant fiber, the transfer resistance to the motoneuron decreased by a factor of about two. If this change were to be explained by a change in $R_{gf}$, the input resistance of the giant fiber would have had to decrease by a factor of approximately two, since $R_e$ is much larger than $R_{gf}$. However, the input resistance, $V_{gf}/I_{gf}$, measured by means of two electrodes in six experiments, actually increased 20–40% over the full range of hyperpolarization used in the transfer resistance measurements (Fig. 3 B). As discussed below, an increase of input resistance of this magnitude would be predicted from the increase in resistance of all the junctions that a giant fiber makes with the ipsilateral motoneurons.

Over the full range of hyperpolarizing currents applied in the motoneuron, the transfer resistance to the giant fiber increased by a factor of about two. If this change were to be explained by a change in $R_{mn}$, the input resistance of the motoneuron would also have had to increase about twofold, since $R_e$ is much larger than $R_{mn}$. As noted above, the input resistance of the motoneurons was evaluated by using the relation between change of antidromic spike height and hyperpolarizing current (Fig. 3 A). Over the limited range
that could be studied in this manner, the input resistance was virtually constant. The foregoing considerations indicate that the difference in electrotonic spread in the two directions is due to the rectifying properties of the junctional membrane.

The coupling resistance, $R_c$, was calculated as a function of transjunctional potential, $(V_{gf} - V_{mn})$, from the input and transfer resistances and equivalent circuit. For a hyperpolarizing pulse applied in the giant fiber,

$$V_{mn} = \left( \frac{R_{mn}}{R_{mn} + R_c} \right) V_{gf}$$

or

$$R_c = \left( \frac{V_{gf}}{V_{mn}} - 1 \right) R_{mn}$$

The input resistance of the motoneurons (mean value, about 0.9 megohm) is low compared to $R_c$ and may be used to approximate the cell resistance, $R_{mn}$. Thus, $R_c$ as a function of $(V_{gf} - V_{mn})$ can be calculated by taking $V_{mn}$ from Fig. 6 B and $V_{gf}$ from Fig. 3 B at corresponding currents. The results of this calculation are shown on the left of Fig. 6 D. The coupling resistance, $R_c$, increased as the giant fiber became more negative; i.e., as the motoneuron became relatively more positive. While the data used in these computations are typical values from different experiments, the general shape of the relation between $R_c$ and $(V_{gf} - V_{mn})$ would be unaffected if extreme instead of typical values were used.

For a hyperpolarizing pulse applied in the motoneuron:

$$V_{gf} = \left( \frac{R_{gf}}{R_{gf} + R_c} \right) V_{mn}$$

or

$$R_c = \left( \frac{V_{mn}}{V_{gf}} - 1 \right) R_{gf}$$

Since under these conditions $V_{gf}$ is small and the input resistance of the giant fiber is low compared to $R_c$, the resistance, $R_{gf}$, can be approximated by the (mean) input resistance of the giant fiber for small hyperpolarizations; i.e., 0.5 megohm. If the input resistance of the motoneuron is assumed to be constant at 0.9 megohm, $R_c$ can be calculated by taking $V_{gf}$ from Fig. 6 C and $V_{mn}$ as the input resistance times the corresponding current. The results of this calculation are shown on the right of Fig. 6 D. The coupling resistance, $R_c$, decreased as the motoneuron became more negative; i.e., as the giant
fiber became relatively more positive. Over the entire range of transjunctional potentials, from maximum hyperpolarization applied in the motoneuron to maximum hyperpolarization applied in the giant fiber, the coupling resistance increased by a factor of about six. When both cells were at the resting potential, the coupling resistance had an intermediate value. The calculated degree of change may have been affected by the use of measurements from different experiments, but the shape of the resistance vs. voltage relation would not have been significantly altered.

Membrane capacities affected the potentials electrotonically spread between giant fiber and motoneuron as is indicated by their slowly rising and falling phases (Fig. 5 A and B). These phases could be roughly fitted by exponentials with time constants on the order of 5 msec. Although it was difficult to measure the (effective) time constant of the motoneuron using a single electrode, strength-latency data indicate that it is less than 0.4 msec. The short duration of the falling phase of the giant fiber PSP in the motoneuron confirms that the motoneuron time constant is short. The time course of the potentials in the giant fibers due to rectangular pulses of hyperpolarizing current applied in them could not be fitted by exponentials, there being an early rapid phase followed by a later slow phase (inset, Fig. 3 B). This time course presumably resulted because the fibers were not isopotential (cf. reference 14). The time constants of the later phases were 3–4 msec which in a uniform cable would approach the membrane time constant. Also, the membrane of the giant fiber terminals would be expected to have a longer time constant than the nodal membrane in the region of electrode penetration, because the terminal membrane would be likely to be of higher resistivity but the same specific capacity. It can be concluded that most of the slowing of the electrotonically spread potentials was due to the capacity of the giant fiber and that there was relatively little effect of the motoneuron capacity.

Correspondence of Coupling Measurements and PSP's in the Motoneurons

Transfer resistances for depolarizing currents were not tested over a large range because of complications introduced by the excitability of the cells. However, the degree of transmission of a giant fiber spike to the motoneurons was consistent with the junctional parameters measured using hyperpolarizing currents. If the amplitude of the giant fiber spike at the junction, $V_{gf}$, is taken as 90 mv, and $R_{m}$ is 0.9 megohm, the PSP amplitude, $V_{m}$, predicted from equation 1 and the data in Fig. 6 D is about 5 mv which is in good agreement with the observed value. $V_{gf}$ was chosen as 90 mv because of data given later in this section, and of course, there is uncertainty as to the height of the spike in the giant fiber terminals. For example, the amplitude recorded in the axon trunk could be smaller than that in the terminals.
because of injury by the microelectrode or the amplitude in the terminals could be smaller than that recorded because of loading by the junctional resistances.

Calculated as in the preceding paragraph, a 10 mv PSP in a giant fiber would produce about 0.4 mv depolarization in a motoneuron. During repetitive Mauthner fiber stimulation, just fast enough to cause failure of excitation of the giant fibers, there would be a 10–15 mv PSP in the two giant fibers presynaptic to a given motoneuron (1), and thus about 1 mv of depolarization in the motoneuron. Actually, potentials in the motoneurons under these conditions were usually much less than this value. The discrepancy was probably due to the capacity of the giant fiber membrane. In passive spread down the fiber, the brief PSP would have been decremented more than long lasting potentials produced by polarizing currents. The giant fiber spike, although brief, was probably actively propagated into the terminals, so that it would have been little changed in amplitude in reaching the synapses on the motoneurons.

Further confirmation of rectification in the junctional membrane was provided by the effect on the PSP of hyperpolarizing current applied in the motoneuron (Fig. 7). From Fig. 6 D, the junctional resistance decreases as...
the potential in the motoneuron becomes more negative, so that the PSP produced by a giant fiber spike should correspondingly increase. In agreement, hyperpolarization of a motoneuron always markedly augmented the PSP, as illustrated in Fig. 7. From the data of this experiment and equation 2, the change in $R_e$ was calculated as a function of transjunctional potential. $R_{mn}$ was again taken as 0.9 megohm, $V_{gf}$ was taken as 90 mv, and $V_{mn}$ was the amplitude of the PSP in the motoneuron. The values of $R_e$ computed in this way were quite close to those determined from the transfer and input resistances. In the case of the data of Figs. 6 D and 7, the best agreement between $R_e$ values was obtained when the presynaptic spike height, $V_{gf}$, was taken as 90 mv rather than 80 or 100 mv.

The change in the amplitude of a PSP produced by a giant fiber spike was linear with applied hyperpolarizing current. The apparent reversal potentials obtained by extrapolating the relation between PSP amplitude and hyperpolarization to zero PSP amplitude were about 75 mv above the resting potential, which is close to values obtained at chemically transmitting excitatory synapses (Fig. 7). Although hyperpolarization spread from motoneuron to giant fiber should have augmented the spike and thereby the PSP, the transfer resistance data indicate that less than 5% of the observed change could have had this origin. Augmentation of the presynaptic spike is more pronounced at the septa in the lateral giant axons of the crayfish (16).

The agreement between the observed amplitude of PSP's in the motoneurons and that calculated from the transfer characteristics obtained using longer lasting currents indicates that the junctional rectification occurred very rapidly. If the rectification were appreciably delayed, the observed PSP's would have been much smaller than the PSP's calculated as above. No data have been obtained concerning the time required for the junctional resistance to return to normal when potentials applied across the junctions are terminated.

Electrotonic Coupling between Motoneurons

The graded depolarizing potentials produced in the giant fibers when graded antidromic stimuli were applied to the peripheral nerve can be explained by electrotonic spread of spikes from motoneurons to giant fibers and by variation in the number of motoneurons excited antidromically. Since potentials in the giant fibers can also spread to motoneurons, some electrotonic spread between motoneurons must occur by way of the giant fibers. However, the data from antidromic stimulation indicate that there is an additional pathway coupling the motoneurons to each other. The graded antidromic potentials in the motoneurons often reached amplitudes of 5 or 6 mv before the giant fibers were antidromically invaded (Fig. 2 C–E). As noted above, anti-
dromic excitation of the giant fibers was signalled in the motoneurons by typical giant fiber PSP's. The PSP's were easily distinguished from the graded antidromic depolarizations by their brevity, all-or-none character, and longer latency (Fig. 2 C-E). Since the summed PSP’s from the two giant fibers were about 8 mv in amplitude (Figs. 1 C and 2 E), 5 mv of graded depolarization in the motoneurons would require graded depolarizations in the giant fibers more than half the amplitude of the giant fiber spikes, if coupling of motoneurons were assumed to be solely by way of giant fibers (cf. equation 1). Also, these large graded potentials in the terminals of the giant fibers would have to be below threshold for spike initiation. Calculated from the steady-state measurements, antidromic depolarizations of 50 mv in the giant fibers would be required to produce 5 mv depolarizations in the motoneurons. The graded potentials actually recorded in the giant fibers had a maximum amplitude of about 8 mv before antidromic invasion occurred (Fig. 4 D). Presumably the potentials in the terminals were somewhat greater, but it is very unlikely that they could have been large enough to explain the graded depolarizations in the motoneurons and still have been subthreshold for excitation of the giant fibers. It is probable therefore that there is an additional pathway coupling the motoneurons.

Further support for a second pathway coupling the motoneurons is provided by the time courses of the graded antidromic potentials (Figs. 2 A and 4 D). In the motoneurons these potentials fall smoothly with a time constant of several milliseconds. In the giant fibers they are much briefer and must therefore be transmitted from active motoneurons to the recording site in the giant fiber by way of relatively short time constant pathways. These potentials in the giant fiber have a shorter time course than would be expected from the slow fall of hyperpolarizations electrotonically spread from the motoneurons. A reasonable explanation for the more rapid fall of the antidromic depolarizations is that the depolarizations cause delayed rectification in the nonjunctional membrane of the giant fiber processes and thereby shorten the time constant. Because of the reduced time constant, the graded potentials in the giant fiber would be expected to reach the terminals on unexcited motoneurons with little distortion. Since transmission of impulses from giant fibers to motoneurons appears to involve little prolongation of the PSP compared to the presynaptic spike, the relatively long duration of the graded potentials in the motoneurons makes it unlikely that they originate in the giant fibers.

The second pathway coupling the motoneurons perhaps involves dendrodendritic connections between them. Dual electrotonic pathways between cells involving both dendrodendritic connections and presynaptic fibers have been demonstrated in a number of other systems (2, 4, 13).
DISCUSSION

The demonstration of rectification at the electrotonic synapse between the giant fibers and motoneurons depends primarily on the measurements of electrotonic spread and input resistances. These data are supported by the agreement between the observed amplitudes of the PSP’s due to orthodromic transmission of impulses and the amplitudes calculated from the input and transfer resistance data. The linear augmentation of the PSP in the motoneuron by hyperpolarization (Fig. 7) agrees quantitatively with the rectification demonstrated by current pulses. The effect of hyperpolarization might at first suggest that there was a chemically mediated component in the PSP. However, the time course and short latency of the PSP exclude any significant contribution from this mode of transmission. An interesting conclusion is that even linear changes in PSP amplitude produced by current pulses cannot be taken as proof of chemically mediated transmission.

It is probable that the structural basis for coupling of giant fibers and motoneurons is the fusion of their junctional membranes to form “tight junctions” as has been shown in numerous other instances (cf. references 4 and 13). It will be of considerable interest to see whether electron microscopic examination reveals any asymmetry that will correlate with the rectification.

Steady-State Measurements and Connectivity

A more complex equivalent circuit than that in Fig. 6 A is shown in Fig. 8, and represents the motoneurons and two giant fibers on one side of the body. Each of the 40 motoneurons is shown as coupled to both giant fibers by means of a voltage-dependent resistance, $R_s$, and a second fixed resistance in series, $R'_s$. $R'_s$ represents the longitudinal resistance of the axon from the recording site to the terminals on the motoneurons. The motoneurons are also connected to each other by resistances, $R''_s$, representing the pathway for electrotonic coupling in addition to that provided by the giant fibers.

Obviously, the use of $R'_s$ and $R''_s$ is a considerable simplification from the reality of a branched core conductor system with distributed resistance and capacity. Nonetheless, it is made clear that the effect of the longitudinal axonal resistance is to reduce the measured degree of rectification to less than the actual value at the junctions. (Similarly any voltage drop across a common extracellular resistance would tend to reduce the measured degree of rectification.) Since the giant fiber spike would be expected to propagate actively into the terminals, the agreement between calculated and observed PSP’s in the motoneurons suggests that the coupling measurements were little affected by electrotonic decrement along the giant fiber. In the coupling
experiments, the electrodes were separated by about 0.3 mm or less. Measurements using two electrodes showed that the giant fiber space constant was about 3 mm in the region of its synapses with the Mauthner fibers (1), although the space constant would presumably become shorter as the giant fiber tapered and branched in approaching the motoneurons.

It is possible that the cell resistance of the giant fiber, $R_{gf}$, largely represents the resistance of nonjunctional membrane of the terminals and fine axonal branches. In this case a more realistic equivalent circuit would be for $R_{c}'$ to connect the recording site to the terminals as in Fig. 8, but for $R_{gf}$ to go from the terminals to ground. In the circuit of Fig. 8 or in the alternative just suggested, $R_{c}'$ as well as $R_{e}, R_{mn}$, and $R_{gf}$ could be calculated from the resistance measurements if $R_{c}'$ were neglected. Except for $R_{mn}$ the exact values would depend on the equivalent circuit assumed. However, the data do not make the calculations practical, because the value of $R_{c}'$ depends on the difference between two much larger resistances that are inaccurately measured.

Since each giant fiber is connected to some 40 motoneurons, the resistance represented by all the coupling pathways in parallel represents an appreciable part of the input resistance of the fiber. Fig. 6 D indicates that for small hyperpolarizations, the shunt resistance through each coupling resistance and
motoneuron would be about 30 megohms (taking $R'_c$ as zero). For 40 motoneurons the total shunt path would have a resistance of 0.75 megohm. Since the input resistance of the giant fiber is about 0.5 megohm, the actual cell resistance, $R_{sf}$ in Fig. 8, would be about 1.5 megohms. This value would be decreased if $R'_c$ were not negligible. The difference between input and cell resistances had no effect on the measurements of $R_e$. For hyperpolarizations in the giant fiber, $V_{sf}$ was measured in separate experiments using two electrodes (Fig. 3 B) and $R_{sf}$ did not enter into the calculation of $R_e$ (equation 2). For hyperpolarizations in the motoneuron, spread occurred from only one motoneuron into the giant fiber, and the input resistance of the giant fiber was a good approximation of its “effective” cell resistance, that is, the resistance that the motoneuron “saw” on the far side of the coupling resistance (equation 4).

For large hyperpolarizations, the input resistance of the giant fiber ($V_{sf}/I_{sf}$) increased (Fig. 3 B), and this change is ascribable to increases in the coupling resistances. From Figs. 8 and 6 D the input resistance should increase from about 0.5 megohm to 0.75 megohm as hyperpolarization is increased to about 80 mV. The observed increases are in good agreement with this prediction. As the measurement of $R_e$ during hyperpolarization in the giant fiber did not depend on $R_{sf}$, the change in input resistance provides further confirmation of rectification in $R_e$. It will be recalled that, if $R_e$ were constant, $R_{sf}$ and the input resistance of the giant fiber would have had to decrease during hyperpolarization to explain the observed decrease in transfer resistance.

The equivalent circuit of Fig. 8 indicates that the two giant fibers are coupled by way of the motoneurons. From Fig. 6 B a steady-state hyperpolarization of 50 mV in one giant fiber would produce about 0.8 mV in each motoneuron. Since all 40 motoneurons would act in parallel for spread to the other giant fiber, there would be about 0.5 mV in that fiber, if one takes $R_e$ as 30 megohms for transmission to the second giant fiber and $R_{sf}$ as 1.5 megohms. Experimental verification of this calculation has yet to be obtained.

In the equivalent circuit of Fig. 8 the linearly arranged motoneurons are shown as only connected to their nearest neighbors; undoubtedly they are connected to more cells since the antidromic potentials in them are fairly smoothly graded. If one excludes coupling by way of the giant fibers, the parallel sum of the resistances of all the coupling paths into one motoneuron would be not less than about 13 megohms, since the maximum graded potential is about 6 mV, motoneuron spike amplitude is about 90 mV, the input resistance of a motoneuron is about 0.9 megohm, and the motoneuron time constant appears to be short (cf. equation 2). The resistance of the path con-
necting two particular motoneurons would, of course, be considerably higher. These results indicate that coupling between motoneurons would have little effect on the observed changes of PSP amplitude during hyperpolarization of a single cell (Fig. 7). There would be very little hyperpolarization of adjacent motoneurons, less than 5% of that in the polarized cell, and any small change in the PSP in them would be greatly attenuated in spreading back into the polarized cell.

Although it is difficult to be confident of resting potential measurements made deep in neural tissues, the interior of the giant fibers in the hatchetfish usually appeared to be about 20 mv negative to that of the motoneurons. Injury may well have contributed to this difference, which would have tended to move the junctions towards their higher resistance condition. The question of possible differences in normal resting potentials between pre- and postsynaptic cells is also relevant to nonrectifying electrotonic synapses. If such differences could be demonstrated, it would be difficult experimentally to determine whether there were continuous currents running between cells or whether there was a compensating potential across the synapses. If the resting potentials were due to potassium concentration differences, and the junctional membranes were selectively permeable to potassium, a compensating potential would develop across the junctions and there would be no resting current flow.

In connection with the preceding paragraph, one may raise the possibility that if the junctional membranes generate potentials, changes in these potentials could contribute to or even be entirely responsible for the observed differences in transfer resistance. By taking the transjunctional currents as equal in the two cases, it is easy to show that a junctional membrane with a fixed resistance, but a variable, current-dependent battery, can in principle be substituted for a membrane with a variable resistance but no internal battery. However, the junctional membrane would be required to generate relatively large internal potentials, if its resistance were fixed. For example, a 90 mv relative depolarization of the giant fiber caused a calculated halving of the junctional resistance (Fig. 6 D). If the junctional resistance had remained constant, the same transjunctional current would have required a potential across the junctional resistance of 180 mv. Only 90 mv of this potential was applied, requiring that the membrane itself generate the remaining (in this case equal) potential of 90 mv. It is unlikely that such large potentials could be generated by the junctional membranes, particularly in view of the probable absence of large differences in ionic concentrations between the cytoplasms on either side of the junction. However, differentiating electrically between the two mechanisms would require experimentally difficult high frequency measurements.
Time Courses

The time courses of the giant fiber spike and PSP in the motoneuron are very similar. Both equivalent circuits (Figs. 6 A and 8) indicate that the PSP should be distorted by the changes in junctional resistance during the spike, and that additional distortion would be introduced if these changes were delayed at either the onset or termination of the spike. Distortion could also be produced by the motoneuron or junctional membrane capacities, which are omitted from the equivalent circuits. The time constant of the motoneuron appears to be quite short, which would tend to minimize distortion and also explain why the PSP did not outlast the giant fiber spike. The degree of distortion is difficult to evaluate accurately because the precise time course of the giant fiber spike in the presynaptic terminal is unknown. Loading of the spike-generating membrane in the terminals by the junctional resistances would tend to reduce spike duration as well as amplitude, and occasionally PSP's in the motoneurons had a slightly earlier peak and more rapid falling phase than the simultaneously recorded spike in the somewhat distant part of the giant fiber (Fig. 5 E).

The short duration of the graded antidromic depolarizations in the giant fiber was noted above. The discrepancy between these potentials and hyperpolarizations electrotonically spread from the motoneurons is ascribable to shortening of the giant fiber time constant by delayed rectification in the nonjunctional membrane of the fiber processes. It should be noted that the presumed delayed rectification in the giant fiber processes is unrelated to and would not affect the measurement of rectification at the giant fiber, motoneuron synapse. The rectifying properties of the synapse were derived from transfer resistance measurements and from augmentation of the PSP in the motoneuron by hyperpolarization. Neither kind of measurement could have involved the delayed rectification.

The graded antidromic potentials in the motoneurons are considerably longer lasting than the PSP's due to giant fiber spikes (Fig. 2). This result would not be expected, since the time constant of the postsynaptic cell, i.e. the motoneuron, is the same in both cases, and the duration of recorded spikes in giant fibers and motoneurons is about the same. One explanation of the difference in time course is that the spread between motoneurons occurs by way of dendrodendritic connections at some distance from the cell body where the postsynaptic time constant is longer. This explanation suggests the reasonable corollary that the giant fiber synapses are axosomatic on the motoneurons.

Antidromic Transmission across the Synapse

The magnitude of antidromic transmission of impulses from motoneurons to giant fibers was much less than orthodromic transmission (Fig. 5 E and F)
and was also less than predicted by the steady-state measurements of electrotonic coupling. For antidromic transmission from a single motoneuron, one may take the effective cell resistance of the giant fiber as equal to the input resistance, that is 0.5 megohm. If the amplitude of the motoneuron spike were 90 mV, the coupling resistance, $R_c$, would be 60 megohms from Fig. 6 D, and the antidromic response in the giant fiber would be about 0.7 mV from equation 3. The potentials in the giant fiber following stimulation of a single motoneuron were always considerably smaller than this value, and their amplitudes were not accurately determined (Fig. 5 F). This discrepancy between antidromic transmission of impulses and of steady-state potentials can be resolved if the slow time course of the electrotonically spread potentials is taken into consideration. As a first approximation, the giant fiber can be considered as isopotential with an input time constant of 5 msec; i.e., the approximate time constant of potentials spread from motoneuron to giant fiber (Fig. 5 B). If the spike in the motoneuron were a rectangular pulse 1 msec in duration, the antidromically transmitted impulse would be attenuated by a factor of five over the values calculated from steady-state measurements. This degree of attenuation reduces the predicted size of the antidromic response down to the limit of sensitivity of the measurements made to date.

The potentials recorded in the giant fibers when many motoneurons were activated by antidromic stimulation reached about 5-8 mV before antidromic invasion of the giant fibers occurred. Monitoring the size of the antidromic volleys indicated that these responses were generated by 20-40 motoneurons. In this case the effective cell resistance of the giant fiber "seen" by the active motoneurons would have been greater than when a single motoneuron was active, because there was less shunting by coupling resistances to inactive motoneurons (see Fig. 8). When 20 motoneurons were active, the coupling resistance would be one-twentieth that for a single motoneuron, and from equation 3 there would be about 18 mV antidromic depolarization. Similarly when 40 motoneurons were active, there would be about 45 mV antidromic depolarization. These values are considerably larger than the maximum observed graded potentials of about 8 mV. As in the case of a single active motoneuron, the discrepancy between antidromic transmission of impulses from many motoneurons and steady-state coupling can be accounted for by the capacity of the giant fiber. Furthermore, the larger depolarizations in the giant fiber caused by many active motoneurons could have caused delayed rectification, as noted in the preceding section, and this factor would have led to a further reduction in antidromic transmission.

The assumption of isopotentiality of the giant fiber probably does not hold during transmission of impulses from the motoneurons, and there is likely to be decrement of brief subthreshold potentials along the giant fiber from terminals to recording site. The apparent firing level at which the giant fiber spike arose from the graded antidromic potentials was about one-half the
firing level for direct or synaptic stimulation (Fig. 4D and Fig. 3H in reference 1). This result indicates that there is a common longitudinal resistance from recording site to the site of initiation of the antidromic impulse and suggests that the lower apparent firing level is due to decrement of the brief, graded responses. In agreement there is some evidence for decrement of the Mauthner fiber PSP in passively spreading along the giant fiber from recording site to terminals. In respect to antidromic transmission of impulses most of the effect of decrement along the fiber would have been taken into consideration in the above calculations by using the time constant of electrotonically spread potentials for the time constant of the giant fiber.

It is perhaps useful to recapitulate why orthodromic transmission of impulses is greater than antidromic. First, there is rectification in the junctional membrane. The coupling resistance decreases significantly when the giant fiber is firing and increases when the motoneuron is firing, there being a fourfold change over the potential range covered by a spike on either side of the junction. Second, the capacity of the giant fiber is greater than that of the motoneuron. Thus, a given synaptic current produces a smaller potential in the giant fiber than in the motoneuron. Third, the effective cell resistance of the giant fiber is lower than that of a motoneuron if one considers antidromic transmission from a few motoneurons. Again, a given synaptic current produces a smaller potential in the giant fiber. If antidromic transmission occurs from many motoneurons, the effective cell resistance of the giant fiber is greater than that of a motoneuron, which would favor antidromic transmission. However in this case, delayed rectification in or near the terminals may reduce the resistance of the giant fiber. Finally, the giant fiber spike at the terminals could be somewhat larger than the motoneuron spike, although there are data that suggest the spikes are approximately equal.

The relatively greater attenuation in antidromic transmission of impulses would probably be less pronounced if it could be measured between giant fiber terminals and motoneurons because of decrement in the giant fiber. Impulses initiated in either giant fiber or motoneuron propagate actively only so far as the synapses, which are presumably on or close to the motoneuron somata, and relatively far from the recording site in the giant fiber. Thus the antidromic response in the giant fiber is decremented by passive spread over a longer distance than the orthodromic PSP in the motoneuron. Because of membrane capacity this factor would be exaggerated for the brief potentials due to impulses as compared with steady-state measurements.

Reflex Function

The finding of electrotonic coupling between neurons controlling the pectoral fins of the hatchetfish was predicted from the functional advantage of such coupling in mediating rapid, synchronous movements of the fins. As already
noted, similar coupling has been found in many other instances in which neurons have the same requirements for synchrony (2–6, 13). Where there is evidence in these other cases, it indicates that junctional resistance is constant, which is not unexpected for a synchronizing synapse that can act in either direction. Of particular relevance are the electromotor neurons of the electric catfish. These cells are coupled by way of presynaptic fibers, but the coupling resistance is constant (6). In contrast the resistance of the giant fiber, motoneuron synapse in the hatchetfish varies with transjunctional potential; i.e., the synapse rectifies so as to favor transmission of excitation from giant fibers to motoneurons. The significance of electrical transmission at this synapse may be that it shortens the latency of the motoneuron response, while the rectification may be functional in reducing antidromic transmission. The pathway coupling the motoneurons in addition to that provided by the giant fibers probably synchronizes motoneuron firing and would not be expected to rectify. However, the low degree of coupling between pairs of cells would make this prediction difficult to verify.

Rectifying electrotonic junctions have also been found between retinula and eccentric cells of the *Limulus* ommatidium (15), and at the crayfish giant motor synapses (11). In the *Limulus* ommatidium the rectification operates so that the junctional resistance increases from its resting value when the eccentric cell becomes relatively depolarized. The opposite potential change has little effect on junctional resistance. In the crayfish the junctions are of high resistance at rest, but decrease in resistance when the giant axons are depolarized, which favors impulse transmission to the motor fibers. The degree of rectification is about 10-fold, which is greater than in the hatchetfish. As in the hatchetfish, there is little synaptic delay and the PSP amplitude is markedly affected by polarizing currents, although there are no published data as to linearity. The resistance of the junctional membrane in the crayfish appears to be near its maximum value in the absence of polarizing current. In contrast, the junctional resistance in the hatchetfish is at an intermediate value in the absence of polarization. The motor axon in the crayfish is small and was reported to be easily injured. Perhaps depolarization of the motor axon during electrode penetration shifted the voltage-current characteristic, and thereby was responsible for the high value of the resistance in the apparently resting condition.

From the data in this and the preceding paper, the following picture of the escape reflex can be proposed. An approaching predator excites the Mauthner cells by way of eighth nerve or lateral line inputs; one Mauthner cell becomes active, and all four giant fibers are then excited, which in turn excite the pectoral fin adductor motoneurons and musculature on both sides. The firing of the Mauthner cell also excites the contralateral axial musculature and inhibits the contralateral Mauthner cell (cf. reference 10). This
pattern of activation is supported by high speed films showing that when hatchetfish jump (in response to tapping the aquarium or moving a dummy predator through the water), the pectoral fin musculature on both sides and the axial musculature on one side contract together. The complete down-stroke of both fins and the full tail movement can be completed in less than 20 msec.

At the crayfish motor giant synapse, rectification appears to prevent antidromic propagation from the motor fiber to lateral or medial giant fibers when the motor fiber is excited by other inputs. In the hatchetfish, the motoneurons can be excited by inputs other than the giant fiber (Fig. 1 F), and the fish can also make small movements with its pectoral fins that appear to involve only a few motoneurons. Perhaps rectification prevents antidromic excitation of the giant fibers during these small movements which would otherwise tend to become much larger. In the escape reflex, the Mauthner fibers would activate the giant fibers which would in turn activate most or all the motoneurons. The coupling between motoneurons is not so close as to prevent a few motoneurons from being independently active. This coupling could, however, provide a strong synchronizing influence once many of the neurons became active during the escape reflex. Furthermore, some motoneurons appear to be less closely coupled to the others; that is, they show relatively little antidromic depolarization even when many other motoneurons are excited (Fig. 2 B). Perhaps these neurons are specifically involved in the small movements.

The rarity of rectification in known electrotonically coupled systems may be a result of the fact that most of these systems are synchronously active under virtually all conditions, and rectification would tend to oppose synchronization. The pectoral fin system of the hatchetfish may differ, because it has the dual function of mediating a fast synchronized escape reflex and normal swimming movements. The present finding of a rectifying electrotonic synapse extends the known repertoire of the vertebrate nervous system. Rectification provides an additional mechanism for reducing antidromic transmission at electrotonic synapses, and helps to overcome one functional objection to a role for electrical transmission in integrative action.

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