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CALCIUM CONDUCTANCE AND FIRING PROPERTIES OF SPINAL MOTONEURONES IN THE TURTLE

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SUMMARY

1. The contribution of Ca^{2+} conductance to the firing properties of motoneurones was investigated in transverse slices of the turtle spinal cord.

2. In the presence of tetrodotoxin (TTX), tetraethylamonium (TEA) in low extracellular concentration (less than 5 mM) promoted Ca^{2+} spikes. In higher concentrations of TEA, a suprathreshold depolarizing current pulse was followed by an after-discharge of Ca^{2+} spikes riding on a Ca^{2+} plateau potential.

3. The Ca^{2+}-dependent plateau was also promoted by Cs^+, 4-aminopyridine (4-AP) and apamin. However, Ca^{2+} spikes during plateaux were an order of magnitude faster when promoted by Cs^+ or 4-AP rather than TEA, and apamin did not promote Ca^{2+} spikes at all.

4. Ca^{2+} plateaux but not Ca^{2+} spikes were blocked by nifedipine.

5. In normal medium all effects of the transient Ca^{2+} influx during action potentials were attributable to its influence on the slow after-hyperpolarization. The nifedipine-sensitive, sustained Ca^{2+} influx was expressed exclusively as plateau potentials and only under conditions of reduced K^+ current.

6. It is concluded that the transient and the sustained Ca^{2+} fluxes in spinal motoneurones are curtailed by different K^+ conductances. The two Ca^{2+} responses are suggested as being mediated by two different types of Ca^{2+} channels.

INTRODUCTION

The response properties of spinal motoneurones are potentially more complex than observed under standard experimental conditions. During various forms of spinal seizures motoneurones can generate plateau potentials and bursts intrinsically (Schwindt & Crill, 1980*a*; Schwindt, Spain & Crill, 1984); similar properties are also induced by serotonin *in vivo* (Hounsgaard, Hultborn, Jespersen & Kiehn, 1984) and *in vitro* (Hounsgaard & Kiehn, 1985). Ca^{2+} conductance is known to contribute to the response properties of motoneurones by mediating a transient Ca^{2+} influx during action potentials (Barrett & Barrett, 1976; Harada & Takahashi, 1983; Walton & Fulton, 1986; Hounsgaard, Kiehn & Mintz, 1988). In addition, however, recent findings suggest that a sustained Ca^{2+} influx is responsible for the plateau potential

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in motoneurones (Schwindt & Crill, 1980*b*; Hounsgaard & Kiehn, 1985). It has also been shown that the plateau potentials can be promoted by procedures that shift the equilibrium potential for K^+ in the depolarizing direction (Schwindt & Crill, 1980*a*) or reduce K^+ conductance (Hounsgaard & Kiehn, 1985).

Using the same preparation and based on the results presented in the preceding paper (Hounsgaard *et al.* 1988) Ca^{2+}-mediated response properties in turtle spinal motoneurones have been investigated. The results suggest that the Ca^{2+} influx during action potentials and during plateau potentials is mediated by two different Ca^{2+} conductances and bounded by different K^+ conductances.

METHODS

See the preceding paper (Hounsgaard *et al.* 1988).

RESULTS

Two regenerative responses mediated by Ca^{2+}

The preceding paper showed that Ca^{2+} conductances contributed to the response properties of turtle motoneurones. In normal medium only a small fraction of the inward current during action potentials was mediated by Ca^{2+} conductances since all regenerative responses disappeared after application of TTX (10^{-6} M). In this section we investigate Ca^{2+} conductances in motoneurones in which Na^+ responses have been eliminated by TTX and Ca^{2+} responses enhanced by reducing various K^+ conductances.

Ca^{2+} spikes and Ca^{2+} plateaux. The basic properties of the two Ca^{2+}-mediated responses found in motoneurones, are illustrated by the effects of extracellular TEA shown in Figs 1 and 2. In the experiment in Fig. 1*A–C* the duration of the action potential and the amplitude of the slow after-hyperpolarization (AHP) in normal medium (Fig. 1*A*) was increased in medium containing 5 mM-TEA (Fig. 1*B*). The regenerative response that remained after addition of TTX (Fig. 1*C*) had a lower amplitude, a slower rate of rise, a longer duration and a higher threshold.

These results are in agreement with the finding that TEA reduces the K^+ conductance responsible for the repolarizing phase of the action potential and for the fast AHP in motoneurones (Barrett & Barrett, 1976; Schwindt & Crill, 1980*b*; Walton & Fulton, 1986; Hounsgaard *et al.* 1988). As originally shown in motoneurones in the frog (Barrett & Barrett, 1976), this effect of TEA leads to an enhancement of the Ca^{2+} component of the action potential and thereby the Ca^{2+}-dependent slow AHP.

In many nerve cells, however, the full effect of TEA on K^+ conductance is only attained at concentrations approaching 30 mM. The sweeps in Fig. 1*D* and *E* illustrate the development of the response properties after increasing the concentration of TEA from 5 to 30 mM in medium containing TTX. It is seen that brief depolarizing current pulses are followed by a prolonged after-discharge of spikes riding on a plateau potential. The plateau response responsible for the after-discharge always required substantially higher concentrations of TEA than were needed to generate Ca^{2+} spikes. In addition the plateau never occurred with

depolarizing current pulses subthreshold for Ca^{2+} spikes and Ca^{2+} spikes were always riding on the plateau. The duration of the action potentials and the voltage plateau was sensitive to the holding current. The first response in fig. 1*D* was generated without holding current by a brief current pulse. The duration of both the plateau and the action potentials was increased when the pulse was repeated after applying a holding current of +0·4 nA (second train in Fig. 1*D*).

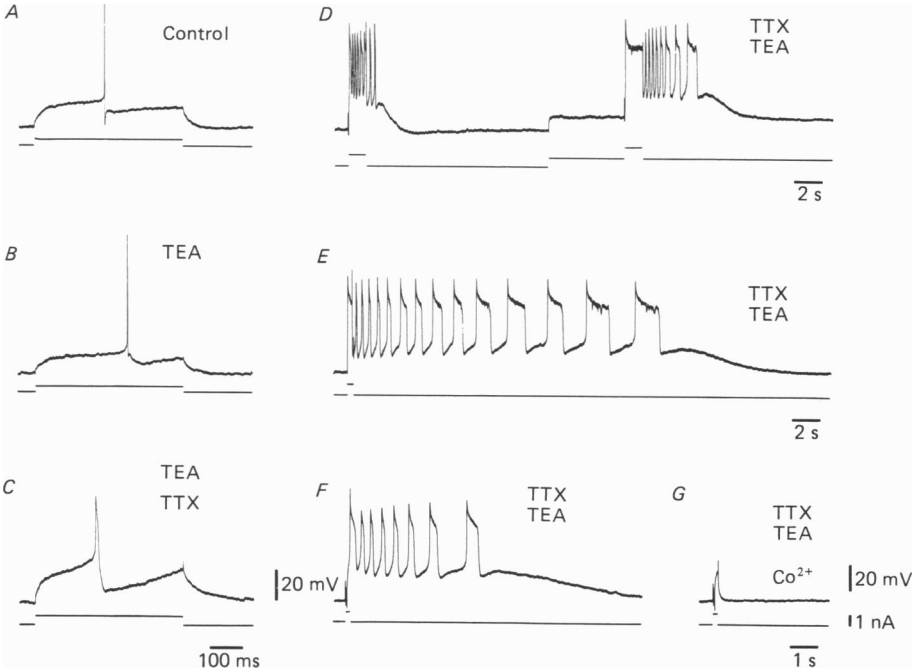

Fig. 1. Ca^{2+} spikes and Ca^{2+} plateaux in the presence of TEA. *A–C*, TEA in low concentration. *A*, control response in normal medium. *B*, prolonged action potential, increased slow AHP and reduced fast AHP 6 min after addition of TEA (5 mM). *C*, remaining regenerative response after addition of TTX (1 μM). *D–G*, TEA in high concentration. All experiments in the presence of TTX (1 μM). *D*, depolarizing bias current increases the duration of Ca^{2+} spikes and plateau potential following depolarizing current pulse, 10 min in TEA (30 mM). *E*, increased duration of action potentials and plateaux after 45 min in TEA (30 mM). *F–G*, Ca^{2+} dependence of spikes and plateaux with TTX and TEA. *F*, control (TEA, 30 mM; TTX, 1 μM). *G*, 20 min after addition of Co^{2+} (3 mM). *A–C*, *D* and *E*, and *F–G* from three different cells.

The duration of spikes and plateaux also increased with the concentration of TEA. The sweep in Fig. 1*D* was recorded after 10 min in medium containing 30 mM-TEA while the sweep in Fig. 1*E* was obtained in steady state after 45 min.

The Ca^{2+} dependence of the spike and the plateau was substantiated by the blocking effect of Co^{2+} and Mn^{2+}. The plateau and the spikes generated in the presence of 25 mM-TEA in Fig. 1*F* were completely eliminated after 30 min in medium in which Ca^{2+} was replaced with Co^{2+} (Fig. 1*G*).

The properties of the Ca^{2+} plateau are illustrated in more detail in Fig. 2. A plateau response, as shown in Fig. 2*A*, could be terminated by a brief hyperpolarizing pulse

(Fig. 2*B*). The elimination of the plateau required a hyperpolarizing current pulse above a certain minimal amplitude and duration. The elimination process is illustrated by the difference between the voltage trajectory produced by a hyperpolarizing current pulse applied during the plateau and again after the plateau was eliminated (Fig. 2*C*).

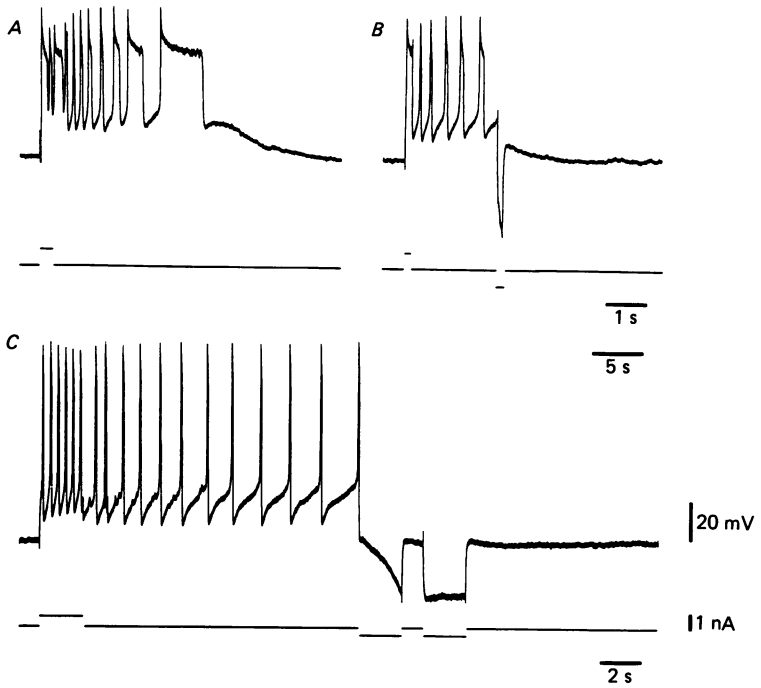

Fig. 2. Bistability in high TEA (30 mM) and TTX (1 μM). *A*, plateau response initiated by depolarizing pulse. *B*, plateau potential eliminated by hyperpolarizing current pulse. *C*, voltage trajectory during hyperpolarizing current pulse from the plateau and from rest. *A* and *B* are from the same all, *C* from different cell.

Ca^{2+} responses promoted by 4-aminopyridine (4-AP), Cs^+ and apamin. The preceding paper (Hounsgaard *et al.* 1988) demonstrated that two K^+ conductances and a Q-like conductance in spinal motoneurones were differentially sensitive to TEA, Cs^+, 4-AP and apamin. In the present experiments we found that all these agents promoted the generation of Ca^{2+}-mediated plateau potentials. While the properties of the plateau were found to be independent of the blocker used, the agents clearly differed in their ability to promote Ca^{2+} spikes. This is illustrated by the experiments in Fig. 3, all performed in the presence of TTX. In the presence of Cs^+ (10 mM), as with TEA, the plateau was always associated with spikes (Fig. 3*A*). With 4-AP (5 mM in Fig. 3*B*) the plateau tended to have a lower threshold than the Ca^{2+} spikes and could in fact be evoked in isolation. Finally, although 0·1 μM-apamin readily promoted the generation of plateaux (Fig. 3*C*) Ca^{2+} spikes could not be generated with any combination of holding current and current pulses.

The difference between Ca^{2+} spikes promoted by Cs^+, 4-AP and TEA are illustrated

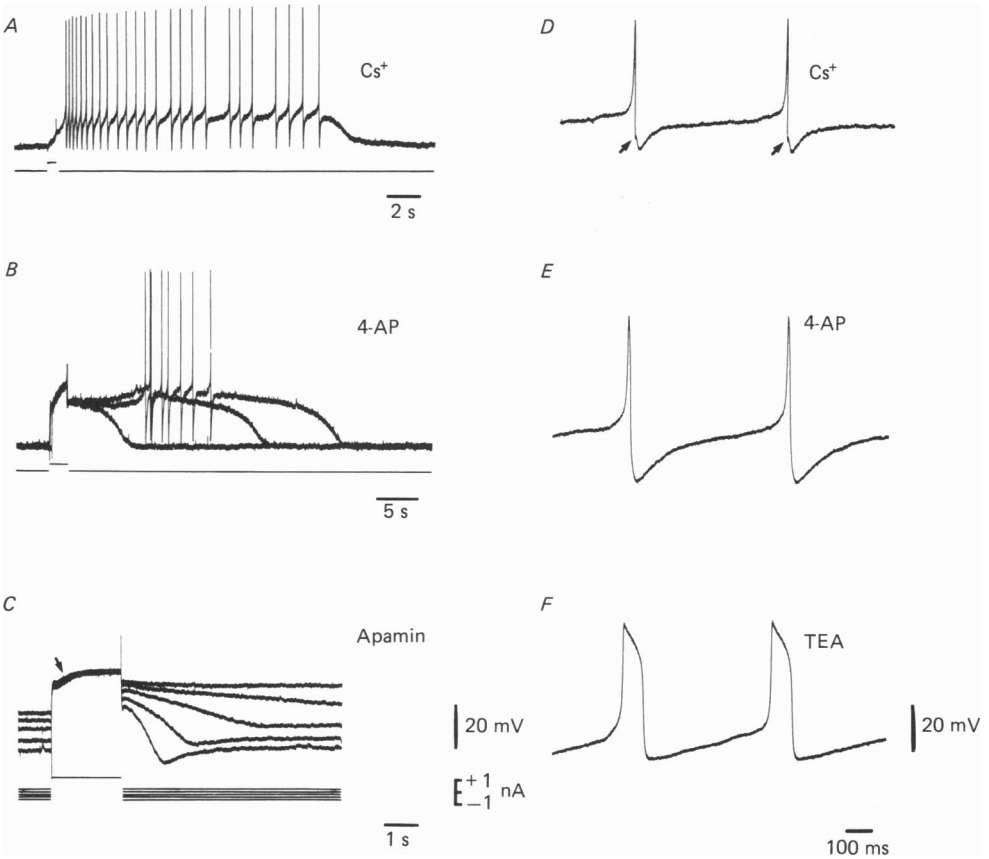

Fig. 3. Ca^{2+} plateaux and Ca^{2+} spikes with Cs^+, 4-AP, apamin and TEA. All experiments in the presence of TTX (1 μM). *A–C*, plateau potentials induced by depolarizing current pulses in the presence of Cs^+, 10 mM (*A*), 4-AP, 1 mM (B) and apamin, 0·1 μM (*C*). In *B* numbers indicate order of three successive sweeps generated with a 60 s interval. *C*, a constant depolarizing current level during activating pulses from different levels of bias current results in uniform plateau onset during pulses (arrow) but different voltage and duration after pulses. *D–F*, Ca^{2+} spikes in the presence of Cs^+, 10 mM (*D*), 4-AP, 1 mM (E), and TEA, 30 mM (F). Note that a fast AHP is distinguishable in *D* (arrows). *A* and *D*, *B* and *E*, and *C* from separate cells. *F* from the same cell as Fig. 2*C*.

in Fig. 3*D–F*. The sweeps were taken during plateaux in the cells also illustrated in Figs 3*A* and *B* and 2*C*. Ca^{2+} spikes promoted by Cs^+ (Fig. 3*D*) were characterized by the preserved fast AHP (arrows) and by their relatively brief duration and low amplitude. Both 4-AP and TEA block the fast AHP and enhance the slow AHP (Hounsgaard *et al.* 1988). In agreement Fig. 3*E* and *F* show Ca^{2+} spikes without fast AHPs and with enhanced slow AHPs. The main difference between Ca^{2+} spikes promoted by 4-AP and TEA is that the duration of Ca^{2+} spikes in the presence of TEA is an order of magnitude longer than seen with 4-AP.

Figure 3 also serves to illustrate some general properties of the plateau response that were found not to depend on the K^+ conductance blocker used. Although the membrane potential quickly returned to rest following the termination of the

plateau, there was clearly a long-term effect of plateaux on the membrane properties. The three superimposed sweeps in Fig. 3*B* were generated in succession with a current pulse of 1 nA applied at 60 s intervals. With a repetition interval of 3 min or more such fluctuations were eliminated. These findings parallel the frequency-dependent decay of Ca^{2+} spikes in cultured spinal cord neurones (MacDonald & Schneiderman, 1986) and may best be explained by a slowly eliminated inactivation of Ca^{2+} channels during the plateau.

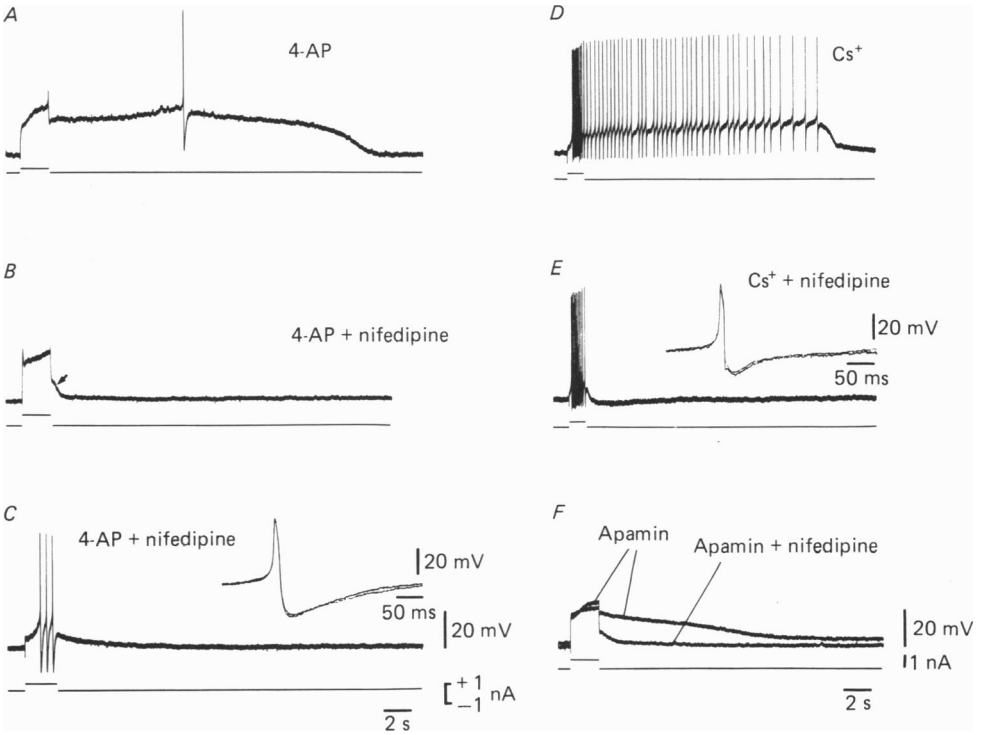

Fig. 4. Nifedipine blocks Ca^{2+} plateaux. All experiments in the presence of TTX (1 μM). *A–C*, Ca^{2+} spikes and Ca^{2+} plateaux with 4-AP (1 mM). *A*, control response without holding current. *B*, plateau eliminated by nifedipine (1 μM). *B*, without holding current. *C*, with depolarizing holding current of +0·7 nA. *D* and *E*, Ca^{2+} spikes and plateau with Cs^+ (10 mM). *D*, control response. *E*, plateau eliminated by nifedipine (1 μM). Insets in *C* and *E* show superimposed Ca^{2+} spikes before and after addition of nifedipine. *F*, plateau in the presence of apamin (10^{-7} M) eliminated by nifedipine (1 μM).

As a parallel to the slow elimination of the plateau during hyperpolarizing current pulses (Figs 2*B* and *C* and 5*D*), the onset of the plateau was slow and depended on the amplitude of the depolarizing current. In addition, as illustrated in Fig. 3*C* for apamin (arrow), the onset of the plateau was independent of the holding current as long as the net current during the depolarizing current pulse was kept constant. As already shown for TEA in Fig. 1*D* the duration of the plateau clearly varied with the holding current (Fig. 3*C*).

Nifedipine blocks Ca^{2+} plateaux. It is conceivable that both Ca^{2+} spikes and Ca^{2+} plateaux are generated by a single type of Ca^{2+} channel. On the other hand, the differential potency of K^+ channel antagonists to facilitate the two response modes raised the possibility that more than one type of Ca^{2+} channel was involved. This hypothesis was supported by the finding that plateaux but not Ca^{2+} spikes were reduced by nifedipine – an organic Ca^{2+} channel blocker which preferentially affects slow-inactivating L channels (McCleskey, Fox, Feldman & Tsien, 1986). Figure 4*A–C* shows the effect of nifedipine on Ca^{2+} responses in the presence of 4-AP. The plateau response in Fig. 4*A* was reduced to an inconspicious after-depolarization after 30 min in nifedipine (10^{-6} M; arrow in Fig. 4*B*) and was no longer facilitated by a depolarizing holding current (Fig. 4*C*). A similarly selective elimination of the Ca^{2+} plateau was seen in the presence of Cs^+ (Fig. 4*D* and *E*), apamin (Fig. 4*F*) and TEA (not shown). The degree of selectivity in the effect of nifedipine is illustrated by the superimposed sweeps of Ca^{2+} spikes before and after application of nifedipine in the insets in Fig. 4*C* and *E* for 4-AP and Cs^+. The onset of the plateau during current pulses and the plateau following pulses disappeared in parallel in medium containing nifedipine. This is illustrated by the superimposed sweeps in Fig. 4*F* from an experiment employing 0·1 μM-apamin as a K^+ conductance blocker.

Ca^{2+} conductance and firing properties

In the preceding section Ca^{2+} spikes and Ca^{2+} plateaux were shown to be latent properties of spinal motoneurones. The two regenerative responses were differentially promoted by different blockers of K^+ conductances and the plateau was selectively eliminated by nifedipine. Based on these results this section attempts a qualitative treatment of how the conductance changes underlying Ca^{2+} spikes and Ca^{2+} plateaux contribute to the firing properties of motoneurones.

Among the determinants of intrinsic firing properties in motoneurones most attention has probably been given to the Ca^{2+}-dependent slow AHP (Kernell, 1965; Calvin & Schwindt, 1972; Baldissera & Gustafsson, 1974). It was noted above that the amplitude of the slow AHP was increased by extracellular TEA in low concentration (less than 5 mM). Under these conditions, which prolong the action potential and promote Ca^{2+} spikes, the degree of adaptation of firing frequency during a depolarizing current pulse was also increased. This is illustrated by the response to a depolarizing current pulse in normal medium and after addition of TEA. In the presence of 1 mM-TEA (Fig. 5*B*) the firing frequency adapts from a higher initial level to reach the same frequency by the end of pulse as in normal medium (Fig. 5*A*). These observations agree with the proposed contribution of the Ca^{2+}-dependent slow AHP to the firing properties of motoneurones (Barrett & Barrett, 1976). It is quite possible however, that the conductance underlying the slow AHP has an equally important role controlling the Ca^{2+} plateau. In agreement with findings in motoneurones in the cat (Zhang & Krnjevic, 1987) the preceding paper (Hounsgaard *et al.* 1988) showed that apamin blocked the slow AHP without otherwise affecting the action potential. When added to normal medium apamin not only reduced the early adaptation but also promoted a subsequent acceleration of the firing frequency to a higher steady-state level (Fig. 5*C*). With a depolarizing holding current firing continued after the current pulse and could be terminated by a brief

hyperpolarizing current pulse (Fig. 5*D*). Note the course of the voltage trajectory during the hyperpolarizing pulse.

A major part of the response to apamin was nifedipine sensitive and therefore attributable to release of the Ca^{2+} plateau rather than the reduced slow AHP *per se*. Figure 6 shows the response to a depolarizing current pulse in normal medium (Fig. 6*A*), in the presence of apamin (Fig. 6*B*) and in the presence of both apamin and nifedipine (Fig. 6*C*). Both the increased firing rate and the phase of accelerated firing promoted by apamin (Fig. 6*A*) is seen to be partly counteracted by nifedipine (Fig. 6*C*).

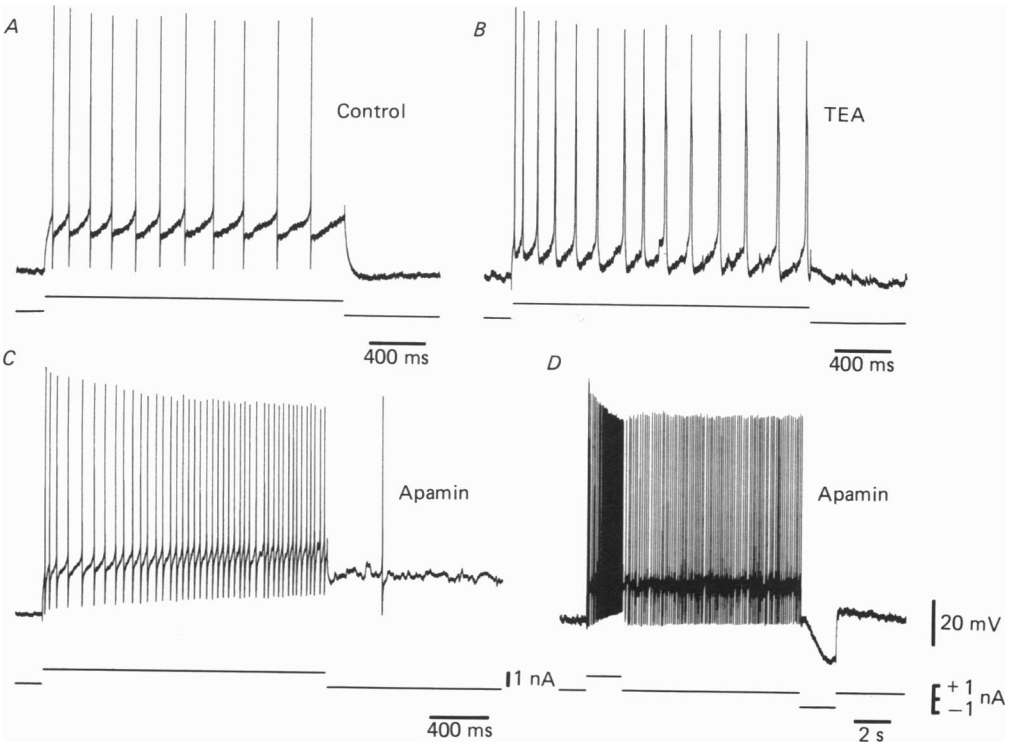

Fig. 5. Change in firing pattern with TEA (*A* and *B*) and apamin (*C* and *D*). *A*, the configuration of action potential and firing pattern in normal medium. *B*, with TEA (1 mM) the action potential is prolonged, the fast AHP reduced and the slow AHP and adaptation increased. Note increased level of synaptic noise. *C* and *D*, response of motoneurone after addition of apamin (0·1 μM) to normal medium. *C*, initial adaptation of firing frequency followed by acceleration during depolarizing pulse from rest. *D*, with a depolarizing bias current a brief depolarizing pulse induced a sustained plateau, finally eliminated by hyperpolarizing pulse.

Activation of the voltage plateau did not only result from an increased input resistance resulting from block of K^+ channels. Figure 7 shows an experiment performed after K^+ in normal medium was increased from 5 to 10 mM. This caused a depolarization of about 5 mV and a significantly lower input resistance. In addition the amplitude of the fast and the slow AHP was reduced (Hounsgaard *et al.* 1988). Under these conditions adaptation proceeded normally during a depolarizing current

pulse of low intensity (Fig. 7*A*). At higher current intensity however, the early adaptation was followed by a phase of accelerating firing frequency (Fig. 7*B*). The underlying plateau, revealed after addition of TTX, was blocked by nifedipine (Fig. 7*C*).

Having demonstrated that a Ca^{2+}-dependent voltage plateau can be released in motoneurones by procedures that reduced outward K^+ current, it was of interest to

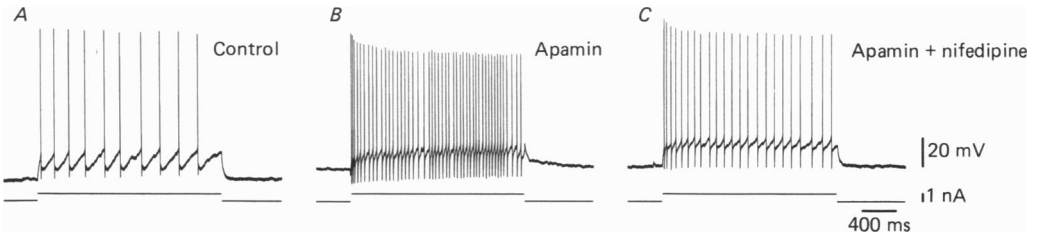

Fig. 6. Contribution of slow AHP and Ca^{2+} plateau to adaptation. *A*, control response in normal medium. *B*, after addition of apamin (0·1 μM) early adaptation is followed by accelerated firing. *C*, acceleration is eliminated and adaptation restored after addition of nifedipine (1 μM).

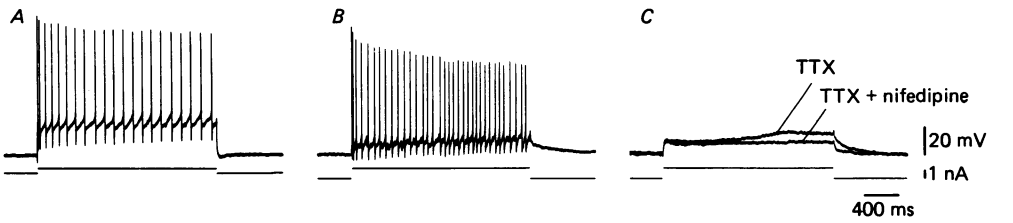

Fig. 7. Firing pattern in medium containing high K^+ (10^{-2} M). *A*, adaptation of firing with low stimulus intensity. *B*, initial adaptation followed by acceleration with high current intensity. *C*, plateau during depolarizing current pulse after addition of TTX (1 μM) eliminated by nifedipine (1 μM).

investigate the role of the underlying Ca^{2+} conductance in normal medium. The action potential and the fast and the slow AHP were found to be unaffected by the addition of nifedipine to normal medium. The firing pattern of motoneurones also appeared unaffected by nifedipine. Figure 8*A* and *B* show *f–I* plots for the first to the fourth interspike interval from the onset of depolarizing current pulses and in steady state for a range of currents. No significant difference was detected between the plots in normal medium (Fig. 8*A*) and in the presence of nifedipine (Fig. 8*B*). The influence of the nifedipine-sensitive Ca^{2+} conductance on the *f–I* curve was finally assessed indirectly by comparing the plots for the first few interspike intervals before and after addition of apamin to normal medium. As shown in Fig. 8*C* apamin increased the slope of the curves but left the break between primary and secondary range unchanged. It thus seems unlikely that the apamin-sensitive, Ca^{2+}-dependent K^+ conductance and the nifedipine-sensitive Ca^{2+} conductance are the sole determinants of the sigmoid *f–I* relation that characterizes motoneurones.

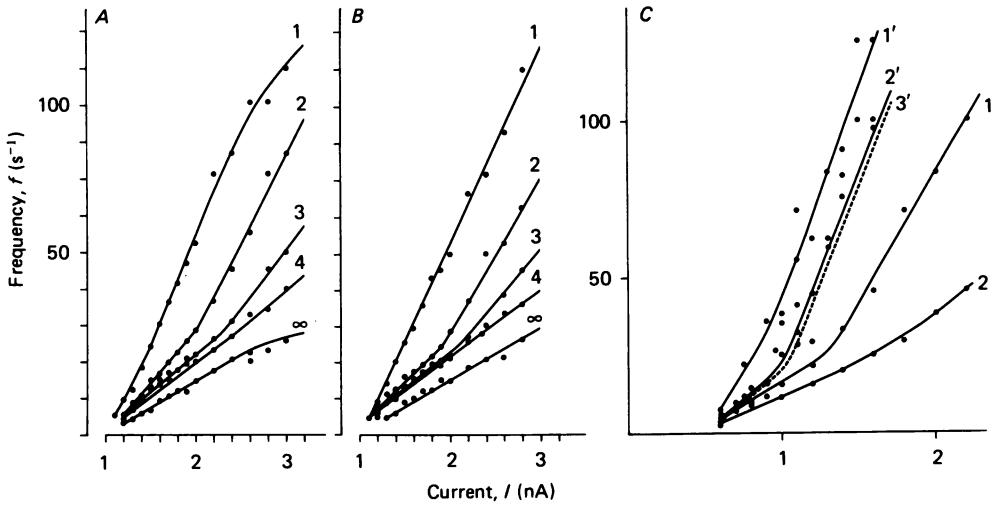

Fig. 8. Effect of nifedipine and apamin on the *f–I* relation. *A* and *B*, effect of nifedipine. *C*, effect of apamin. *A*, *f–I* relation in normal medium for the first four interspike intervals and in steady state after 10 s (1, 2, 3, 4 and ∞). *B*, *f–I* relation for the same cell after 30 min in nifedipine (10^{-6} M). *C*, *f–I* relation for the first two interspike intervals in normal medium (1 and 2) and for the first three interspike intervals after 15 min in apamin, 10^{-7} M (1′–3′).

DISCUSSION

The data presented show that Ca^{2+} conductance contributes to the response properties of turtle motoneurones in two ways. The transient influx of Ca^{2+} during action potentials is mainly responsible for the slow AHP while the sustained Ca^{2+} influx can generate a depolarizing voltage plateau. The two Ca^{2+}-dependent response modes are normally curtailed by different K^+ conductances and can be promoted relatively independently by changing the balance between the K^+ conductances. These findings suggest that the firing properties of spinal motoneurones are more complex than normally assumed and accessible to regulation.

K^+ conductances and regenerative Ca^{2+} responses

In normal medium Ca^{2+} currents in turtle motoneurones are masked and restricted by K^+ conductances. The transient Ca^{2+} influx is mainly curtailed by the delayed rectifier K^+ conductance. As was first found in the frog (Barrett & Barrett, 1976), the Ca^{2+} influx only contributes significantly to the action potential when the delayed rectifier is reduced (Fig. 1*A* and *B*). This is also an absolute requirement for the generation of pure Ca^{2+} spikes. The agents that promote Ca^{2+} spikes in the presence of TTX also increase the duration of the action potential and reduce the fast AHP when added to normal medium in the same concentrations.

The sustained Ca^{2+} influx, responsible for the plateau potential is mainly curtailed by K^+ conductances other than the delayed rectifier. While TEA in low concentration (less than 5 mM) reduced the delayed rectifier and promoted Ca^{2+} spikes, a much higher concentration of TEA was needed for the generation of Ca^{2+} plateaux.

Plateaux were promoted more readily by less selective K^+ blocking agents like Cs^+ and 4-AP. Moreover, apamin, which blocks the Ca^{2+}-dependent K^+ conductance responsible for the slow AHP but has no effect on the delayed rectifier (Hounsgaard *et al.* 1988), promoted Ca^{2+} plateaux but not Ca^{2+} spikes. It is unlikely that the apamin-sensitive K^+ conductance alone controls the sustained Ca^{2+} influx. In other cell types this particular K^+ conductance is insensitive to TEA, Cs^+ and 4-AP, which all promote plateau potentials in turtle motoneurones. Although these agents reduce the delayed rectifier, their potency in this respect correlates poorly with their ability to promote Ca^{2+} plateaux.

It seems quite possible then that the plateau potential under normal conditions is curtailed by the sum of several K^+ currents and that removal of any one of these will promote the plateau. It is also worth noting that the unspecific reduction of K^+ currents imposed by moving the equilibrium potential in the depolarizing direction (Fig. 7) promotes plateau potentials but not Ca^{2+} spikes. This experiment also shows that plateau potentials can be generated when the input resistance decreases rather than increases.

Ca^{2+} conductances in motoneurones

Ca^{2+} spikes and Ca^{2+} plateaux have been observed in spinal motoneurones from many vertebrates (Barrett & Barrett, 1976; Schwindt & Crill, 1980*a*,*b*; Walton & Fulton, 1986). Based on the present findings it seems likely that Ca^{2+} conductance in motoneurones is mediated by more than one type of Ca^{2+} channel. Assuming that K^+ conductance blockers do not interfere with Ca^{2+} channel inactivation, the Ca^{2+} conductances underlying Ca^{2+} spikes and Ca^{2+} plateaux are clearly of the slow-inactivating types. Both Ca^{2+} spikes and Ca^{2+} plateaux can last longer than 1 s in turtle motoneurones. In addition the duration of both responses increases when the depolarizing holding current is increased (Figs 2*B* and 3*C*). These observations seem to exclude the possibility that the Ca^{2+} spike and Ca^{2+} plateau involve channels with rapid inactivation like T channels (Nowycky, Fox & Tsien, 1985).

The finding that the Ca^{2+} plateau potential in motoneurones is eliminated by nifedipine supports the possibility that this response is mediated by the slowly inactivating L channels (Nowycky *et al.* 1985). It is unlikely, however, that this channel is also responsible for the generation of the Ca^{2+} spike and the transient Ca^{2+} influx during action potentials, in particular since the blocking effect of nifedipine is known to increase with depolarization (Bean, 1984; Reuter, Porzig, Kokubun & Prod'hom, 1985). It seems likely therefore that both L- and N-like Ca^{2+}channels co-exist in spinal motoneurones and contribute distinctly differently to the response properties of these nerve cells.

Ca^{2+} conductances and firing properties

The experiments suggest that Ca^{2+} conductance contributes to the firing properties of spinal motoneurones in two ways. In agreement with the findings in motoneurones of other species (Barrett & Barrett, 1976; Walton & Fulton, 1986) the transient Ca^{2+} influx during action potentials influence adaptation and the *f–I* relation by controlling the slow AHP. In contrast, the only effects of the nifedipine-sensitive Ca^{2+} conductance are those directly related to the plateau potential generated by the

Ca^{2+} influx. While the effects of the transient Ca^{2+} influx are readily detectable in normal medium, Ca^{2+}-mediated plateaux are generated only when the K^+ current across the membrane has been reduced. It is of interest to note that although the transient and sustained Ca^{2+} fluxes may be mediated by separate Ca^{2+} channels and curtailed by separate K^+ conductances, they are not independent. The sustained Ca^{2+} influx can be promoted by reducing the apamin-sensitive K^+ conductance, which is controlled by the transient Ca^{2+} influx.

This and the preceding paper have described the firing properties of spinal motoneurones in the turtle. It seems likely that the underlying set of ion conductances is shared with spinal motoneurones from other vertebrates. Of particular interest is the finding that plateau potentials in both the cat and the turtle are latent properties probably mediated by a common Ca^{2+} conductance and curtailed by a common set of K^+ conductances. The experiments reported here show that spinal motoneurones possess latent response properties of considerable complexity. It has already been noted that some of these properties are uncovered by certain neurotransmitters (Hounsgaard & Kiehn, 1985). It is quite possible that the intrinsic firing properties of motoneurones are regulated over a wide range as part of normal motor behaviour.

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