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DIFFERENT FIRING PATTERNS GENERATED IN DENDRITES AND SOMATA OF CA1 PYRAMIDAL NEURONES IN GUINEA-PIG HIPPOCAMPUS

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SUMMARY

1. Intracellular recordings, taken from CA1 pyramidal cells in guinea-pig hippocampal slices, were used to examine the origins of repetitive and burst firing in these cells. Single action potentials were elicited by depolarizing current injection at somatic recording sites. In contrast, current injection during intradendritic recordings initiated burst firing in the dendrites. Burst firing could be elicited in the soma by direct depolarization of distal apical dendrites (> 150 μm from the cell body layer) with large extracellular polarizing electrodes.

2. Intracellular recordings were taken simultaneously from the apical dendrites and pyramidal cell somata with the intention of impaling the same neurone with both electrodes. Paired dendrite–soma recordings confirmed that rhythmic single action potentials were generated at the cell soma, whereas bursts of action potentials were initiated in the distal apical dendrites (> 150 μm from the cell body layer). Fast spikes in the dendrite often triggered fast spikes in the soma, but not all fast spikes in the dendritic burst were 'relayed' to the soma.

3. In paired recordings, when a dendritic action potential failed to elicit a full somatic action potential, a 'd-spike' was commonly recorded in the soma. Somatic d-spikes were uniform all-or-none responses that could be shown, in some cases, to trigger the full somatic action potentials.

4. Attenuated spikes could be recorded in the dendrites, triggered by action potentials initiated at the cell soma. Dendritic responses to somatic stimulation sometimes varied in amplitude, but always showed a direct correspondence with somatic action potentials.

5. Dendritic recordings taken closer to the pyramidal cell bodies (< 150 μM from the cell body layer) showed a 'transitional' region where single action potentials rather than burst discharges could be evoked. After-potentials of these single spikes differed from those associated with somatic spikes in that proximal dendritic spikes had depolarizing after-potentials. The observed shift from after-hyperpolarization to depolarizing after-potentials in intradendritic recordings taken progressively further from the cell body corresponds to the change from repetitive to burst firing.

6. The results indicate that activity of the CA1 pyramidal cell soma, presumably

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a reflection of its output, can be either burst or repetitive firing. Somatic 'bursts,' unlike the burst discharges seen in the apical dendrites or the burst discharges reported in CA3 cells, are not initiated locally. Rather, they appear to be simply a rapid spike-for-spike response by the soma to the fast spikes that form part of the apical dendritic burst.

7. The firing pattern of CA1 pyramidal cells depends upon the location of excitatory input to the cell: suprathreshold excitation of the distal apical dendrites elicits a local burst response that causes a burst response in the soma, whereas suprathreshold excitation of the soma and proximal dendrites results in repetitive firing in the soma. Thus, very different modes of output activity of the CA1 pyramidal cell can be elicited depending on the location of its excitatory inputs.

INTRODUCTION

The burst firing of hippocampal pyramidal cells, initially described over 50 years ago by Renshaw, Forbes & Morrison (1940), is one of the principal identifying features of these cells. In extracellular recordings, the bursts, often called 'complex-spikes' (Ranck, 1973), distinguish these cells from hippocampal interneurones (Fox & Ranck, 1981). Studies in hippocampal pyramidal neurones have suggested a possible contribution of burst discharges in the generation of pathological synchrony seen in epileptic neural tissue (e.g. Prince, 1978).

Recent studies in central neurones suggest that the differences in firing patterns across cell types can be attributed to quantitative differences in ionic conductances. The output pattern of neurones is generally determined by a sequential activation of their ionic conductances. For example, hippocampal CA3 pyramidal neurones commonly respond to a depolarizing stimulus with a burst of two to seven decrementing 'fast' action potentials riding on a slow depolarizing potential that lasts about 20–50 ms. The slow depolarizing potential appears to result from the summation of depolarizing after-potentials (DAPs) that follow individual 'fast' action potentials (Wong & Prince, 1978, 1981). The burst is followed by a prolonged after-hyperpolarization (AHP). Ca^{2+} entry underlies a portion of the DAP (the remaining portion is passive decay of the membrane potential), and activates potassium conductances that underlie early and late portions of the AHP (Hotson & Prince, 1980; Gustafsson & Wigstrom, 1981*a*; Wong & Prince, 1981; Lancaster & Adams, 1986; Lancaster & Nicoll, 1987; Numann, Wadman & Wong, 1987; Storm, 1987; Alger & Williamson, 1988). The CA3 cell burst, therefore, is a self-limiting event sustained by a sequence of voltage- and ion-gated conductance changes.

Ionic conductances determining a particular firing pattern may be modified by a number of factors. Membrane polarization (e.g. Wong & Prince, 1981; Llinás & Jahnsen, 1982; Jahnsen & Llinás, 1984*a*, *b*) or transmitter application (e.g. Madison & Nicoll, 1982; Haas & Greene, 1984, 1986; Malenka & Nicoll, 1986; Colino & Halliwell, 1987; Storm, 1989) may be necessary to activate or inactivate specific conductances which can convert the firing of a cell from repetitive to bursting mode (Benardo & Prince, 1982*a*, *b*; McCormick & Prince, 1986*a*).

Direct dendritic recordings also revealed bursting mechanisms in the apical dendrites of both CA1 and CA3 pyramidal cells (Wong, Prince & Basbaum, 1979; Benardo, Masukawa & Prince, 1982). Using a pair of intracellular electrodes in area

CA3, one electrode in an apical dendrite and the other in a cell soma, Wong *et al.* (1979) showed that a burst response elicited at either location resulted in a burst at the other location. Thus, the dendritic burst could amplify synaptic events, ensuring discharge of the soma. Although 'fast' spike components of the dendritic burst did not often appear to be directly propagated into the soma, the slow depolarizing 'envelope' served as an effective generator potential for somatic burst responses.

CA1 pyramidal cells differ considerably from CA3 pyramids. Bursting appears much less commonly in CA1 pyramidal cells than in CA3 neurones *in vivo* (e.g. Ranck, 1973) and *in vitro* (Schwartzkroin, 1975, 1978; Wong & Prince, 1978). Whereas burst responses are seen in apical dendritic recordings (Wong *et al.* 1979; Benardo *et al.* 1982), CA1 somata are generally reported to respond to direct depolarization only with repetitive fast spikes. Thus, plots of firing frequency *versus* stimulus current, derived from intrasomatic recordings in CA1 pyramidal cells, are linear over a wide range of input currents (Schwartzkroin, 1978; Gustafsson & Wigstrom, 1981*b*). In contrast to CA3 cells, in which burst responses can be recorded from both apical dendritic and somatic sites, bursting in CA1 neurones may be an exclusive property of the apical dendritic membrane. Our objective was to examine the origins of repetitive and burst firing in CA1 pyramidal cells using intracellular recordings from cell somata and their apical dendrites.

With paired intracellular recordings (one apical dendritic, one somatic) we found that burst responses can be recorded from both locations, but that somatic burst responses occurred only when the apical dendrites were caused to burst. A burst pattern of somatic action potentials resulted from the relay of fast dendritic spikes (as part of a dendritic burst response) to the soma. Direct somatic stimulation did not produce a burst response. We conclude that the different output patterns of CA1 pyramidal cells can depend solely upon the locus of excitation.

METHODS

Guinea-pigs (14–21 days old) were anaesthetized with halothane and decapitated. Each brain was rapidly removed from the skull, bisected, and allowed to sit briefly in ice-cold artificial CSF. A block of tissue (about 1–2 mm thick) was obtained from the hippocampus, removed from one hemisphere by blunt dissection. Slices (400–500 μm) were cut from the block using a McIlwain tissue chopper (Mickle Lab. Eng. Co., Gomshall, Surrey) and transferred to a nylon mesh in the recording chamber. The lower surface of the slice contacted a perfusing solution (NaCl, 124 mM; KCl, 5 mM; $CaCl_2$, 2 mM; $MgCl_2$, 2 mM; $NaHCO_3$, 26 mM; and glucose, 10 mM; pH = 7·4 when exposed to 95 % O_2–5 % CO_2). The upper surfaces were exposed to a warmed, moistened atmosphere of 5 % CO_2 in O_2. The temperature of the chamber was maintained at 37 °C.

Recording electrodes were pulled from 1 mm diameter fibre-filled glass capillary tubes and filled with 4 M potassium acetate or an HRP (horseradish peroxidase) solution (4 % Sigma type VI HRP dissolved in Tris, pH 8·6 / 0·2 M KCl). Electrodes had maximal tip resistances of 100–150 MΩ at 130 Hz after bevelling. Signals were amplified by a high input impedance amplifier with facilities for current injection using an active bridge circuit and for capacitance compensation (WPI M707). Extracellular stimulation was applied between two flattened coils of silver wire (wire diameter = 100 μm; length of long axis of coil = 2 mm; e.g. Jefferys, 1981). One coil was placed along the apical dendrites of CA1 and the other coil was placed, parallel to the first, along the alveus. The apical dendritic electrode was made the cathode to depolarize these elements. Spontaneous and evoked activity was written with a chart recorder or photographed from an analog oscilloscope. To improve the clarity of illustrations, photographic images were re-traced.

Recording and stimulating electrodes were placed under direct visual guidance into the desired strata of area CA1. Typically, a somatic recording electrode and either a dendritic recording electrode or a dendritic extracellular stimulating electrode were used for a given experiment.

Paired recordings were made using the methods described by Wong *et al.* (1979). Briefly, this involved impaling a pyramidal cell soma, followed by electrode penetrations, perpendicular to the cut surface of the slice, intended to impale dendrites 100–200 μm away from the apical margin of stratum pyramidale. Since it is possible that electrode penetrations intended for apical dendritic regions of the slice actually impale interneurones or displaced pyramidal cell somata, HRP was injected into the cell to permit direct visualization of the recorded element. Ionophoresis of HRP was accomplished by applying positive current pulses (0·75–1·0 nA, 350 ms 'on', 350 ms 'off') for 20 min. Intracellular activity could be recorded throughout the filling process. After injection, slices were maintained in the recording chamber for at least one hour before being transferred to fixative (3 % glutaraldehyde–1 % paraformaldehyde). These slices were then exposed to 5 % $CoCl_2$ to 'intensify' HRP-injected cells and then developed with diaminobenzidine and H_2O_2. Dendritic recordings were confirmed in all ten cases examined.

RESULTS

Patterns of spontaneous activity recorded at the soma

Burst discharges can be recorded from the somata of CA1 pyramidal cells *in vitro*. Figure 1 illustrates spontaneous activity recorded from the soma of a CA1 pyramidal cell. Occasional burst discharges are seen together with single spikes. Small 'spikelets' or 'd-spikes' (marked by dots below the traces) can be seen together with full action potentials in every burst shown. The presence of spikelets during the bursts suggest that these events are generated at sites away from the soma. In contrast, single spikes are most probably initiated at the somatic recording site, since they arose from a fixed threshold, and they were not triggered by spikelets. One exception is shown in the Fig. 1 (middle of top trace) where d-spikes are present in relation to a single spike. It is clear from the pattern of d-spikes in this instance that the soma would have fired a 'burst' of action potentials had more of the d-spikes been able to trigger full somatic action potentials. A notable feature of the pattern of full spikes and d-spikes within bursts, is that one or more d-spikes *follow* full somatic action potentials; the first event in each burst was a full action potential. From this, it appears that somatic spikes cannot follow the high rate of d-spikes within the burst.

Apical dendritic origin of burst discharges

To test the hypothesis that somatic burst responses occurred as a result of burst activity initiated in the apical dendrites, we attempted to directly depolarize the dendritic elements. As others have seen (Schwartzkroin, 1978; Gustafsson & Wigstrom, 1981*b*), direct depolarization of the soma elicited trains of action potentials that were related to the magnitude of the stimulus (Fig. 2*Aa* and *b*).

It was possible to evoke burst responses in CA1 somata by depolarizing the apical dendrites using extracellular electrodes (Fig. 2*B*). Polarizing pulses were delivered through a pair of flattened silver wire coils, one in the distal apical dendrites and the other in stratum oriens (see Methods). The response of the soma to strong dendritic depolarization was burst pattern firing and not repetitive firing. This is evident in Fig. 2*Ba*, where a long duration polarizing pulse resulted in two discrete burst events (compare with Fig. 2*Ab*). Bursts consisted of multiple fast spikes with an associated slow depolarizing event. Hyperpolarization of the soma concurrent with the dendritic depolarization, initially caused an increase in latency of the somatic burst response (Fig. 2*Bb*). Latency changes were most probably due to the spread of

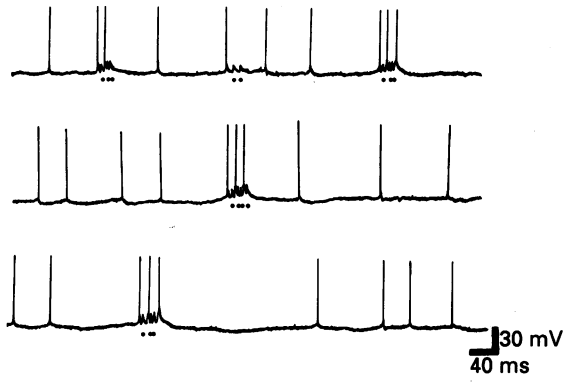

Fig. 1. Spontaneous activity recorded from the soma of a CA1 pyramidal neurone. Occasional burst discharges are seen together with single spikes. Note the presence of small 'spikelets' or d-spikes (marked by dots beneath the traces) together with full action potentials in every burst. The intervals between the end of a burst and the occurrence of a single spike are always much shorter than the burst to burst intervals.

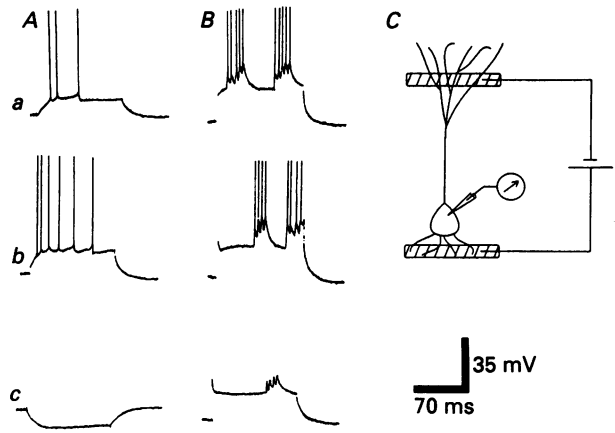

Fig. 2. Evoked repetitive and burst firing in CA1 pyramidal cell somata. *A*, responses of soma to two intensities of depolarizing current injection (*a* and *b*). Higher intensity stimuli cause repetitive firing, but not burst discharges. *Ac*, response to hyperpolarizing current injection. *B*, burst responses evoked in the soma (same cell) by extracellular stimulation of the apical dendrites. Compare double burst response to long duration dendritic stimulation (*Ba*) with repetitive firing induced by somatic stimulation (*Ab*). Concurrent hyperpolarization of the soma (*Bb* and *c*) increases the latency of the burst (current spread to dendrites) and causes somatic spikes to fail (local effect of hyperpolarizing current injection). Note the underlying d-spikes (*Bc*). *C* the drawing shows a recording electrode in the soma. Hatched bars represent the extracellular polarizing electrodes (apical dendritic electrode is the cathode).

hyperpolarizing current injected through the somatic electrode serving to diminish the depolarizing effect of the extracellular stimulus, delaying initiation of the dendritic burst. The shift in latency and all-or-none occurrence of the somatic burst suggest that the burst event was not sustained by synaptic potentials which could have been evoked by the extracellular stimulus. As the somatic hyperpolarization was increased, full somatic action potentials were successively dropped from the local

burst response, revealing the underlying d-spikes. Eventually, the somatic hyperpolarization was sufficient to prevent the appearance of any fast action potentials (Fig. 2*Bc*). All that remained were the d-spikes riding atop a 'depolarizing envelope'. The presence of the depolarizing envelope in the absence of fast spikes

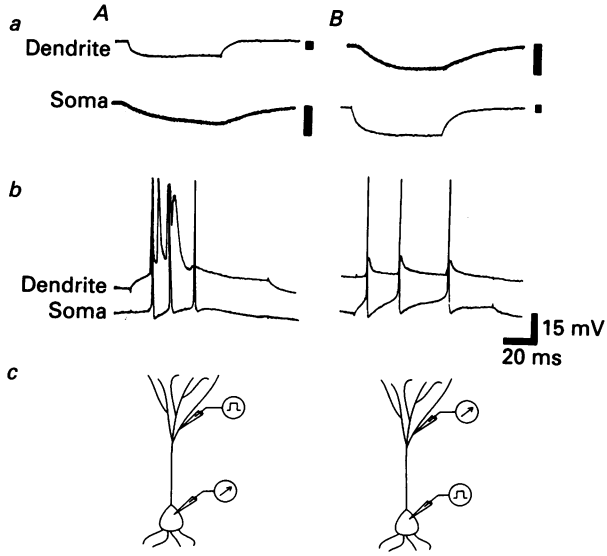

Fig. 3. Coupling of dendritic and somatic intracellular recordings. In each of the four paired recordings, the dendritic response is shown above the somatic response. *Aa*, hyperpolarizing current pulse injected through the dendritic recording electrode and the passive response recorded at the soma. *Ba*, hyperpolarization applied at soma is electrotonically conducted to the dendritic recording site. *Ab*, depolarization of the apical dendrite elicits a local burst response consisting of fast and slow components. The response of the soma is a burst of fast action potentials. *Bb*, depolarization of the soma produces repetitive firing. Attenuated spikes appear at the dendritic location. The drawings at the bottom (*Ac* and *Bc*) indicate which element was stimulated (apical dendrite in *A*; soma in *B*). Calibrations: vertical bars in top four traces (*A* and *B*, dendrite and soma) all 10 mV; time calibration same as bottom four traces: 20 ms.

suggests that it is associated with the dendritic burst and is passively conducted to the soma. The depolarizing envelope may contribute to action potential firing in the soma. Full spikes that dropped from the burst as a result of increased somatic hyperpolarization were the earliest spikes. In other words, it appeared that the depolarizing envelope permitted the soma to reach action potential threshold in response to later d-spikes. Across the different paired recordings, however, we noted considerable variability in the size of the depolarizing envelope (compare e.g. Figs 1 and 2 with Figs 3 and 4). This could be due to variations in the electrotonic separation of the recording electrodes.

Somatic responses to dendritic stimulation

To examine the relationship between dendritic and somatic firing more directly, paired intracellular recordings were taken from apical dendritic and somatic elements. As indicated in the Methods, attempts were made to impale dendrites about 170 μm away from the pyramidal cell layer. Dendritic recordings were

anatomically confirmed using HRP to visualize the recorded element (e.g. Wong *et al.* 1979). Figure 3 shows data from a pair of intracellular recording electrodes that were passively coupled. One electrode was placed in stratum pyramidale and the other electrode in distal stratum radiatum. Passive responses to hyperpolarizing

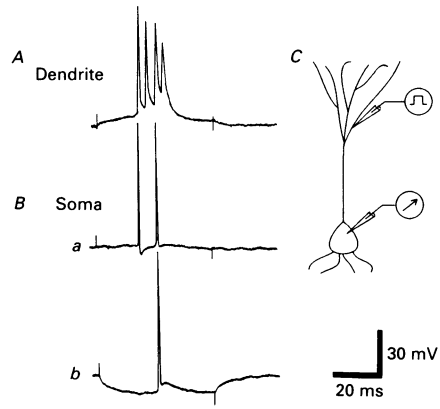

Fig. 4. Transmission of action potentials from dendrites to soma (electrode separation about 150 μm). *A*, apical dendritic recording of burst response to direct depolarization. *B*, somatic responses to dendritic bursts identical to that shown at top. *Ba*, the first and third spikes of the dendritic burst have been successfully relayed to the soma. No d-spikes are present to mark the times of occurrence of the second and fourth dendritic fast spikes. Note the decrease in the after-hyperpolarization following the second of the somatic spikes. Simultaneous somatic hyperpolarization is shown in *Bb*. Only the third dendritic fast spike is relayed to the soma. The hyperpolarization does not reveal d-spikes. Note the change in the after-potential to a depolarizing event. *C*, the drawing indicates the basic configuration for initiating action potentials in the apical dendrites.

pulses at either location demonstrate the coupling of the two electrodes (*a* in Fig. 3*A* and *B*). Direct excitation of the apical dendrite produced a local burst response (Fig. 3*Ab*). Fast spikes appeared in the paired soma recording, corresponding to fast spikes on the dendritic burst. Conversely, fast spikes initiated at the soma by current injection resulted in attenuated spikes at the dendritic recording site (Fig. 3*B*).

The correspondence of somatic fast spikes to dendritic fast spikes is illustrated in Fig. 4. Here, somatic responses to directly evoked dendritic bursts are shown at two levels of soma membrane potential. Note that somatic spikes were always triggered by dendritic spikes. A full somatic action potential with a pronounced post-spike after-hyperpolarization follows the first spike in the dendritic burst. The AHP appears to be long enough in duration to limit the high frequency following of dendritic spikes by the soma (Fig. 4*Ba*). Compare the 'spike–failure–spike–failure' pattern seen in Fig. 4 with the 'spike–d-spike' pattern seen in the bursts shown in Fig. 1. In support of the notion that the AHP acts to limit the frequency following capabilities of the soma is the fact that d-spikes or failed spikes were never the first event in a somatic burst unless the soma was hyperpolarized by current injection.

In the case illustrated in Fig. 4, an action potential generated in the dendrites did not elicit observable d-spikes in the soma. In the absence of observable d-spikes, the soma appeared to fire below threshold (Fig. 4*Bb*). Hyperpolarization of the soma (Fig. 4*Bb*) causes one of the spikes to fail, but does not reveal an underlying d-spike.

In another pair of coupled recordings, the dendritic recording was taken from an apical dendrite only 100 μm away from the soma. In this pair, d-spikes were recorded in the soma giving rise to somatic action potentials. Stimulation applied to the soma resulted in a full action potential at the dendritic location (Fig. 5*A*). In Fig. 5 (*B*,

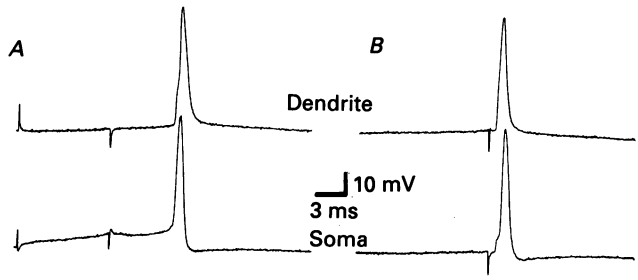

Fig. 5. Transmission of action potentials from dendrites to soma. In this pair of recordings, the dendritic electrode is located closer to the soma (electrode separation about 100 μm). *A*, a near-threshold depolarizing pulse delivered through the somatic electrode evokes a slow depolarization leading to a single action potential locally (bottom). This is seen at the dendritic site (top) as a spike arising from a spikelet (inflexion on rising phase). *B*, stimulus pulse delivered through dendritic electrode results in action potentials locally and at the somatic recording site (note inflexion on the rising phase). Only the break of this pulse is shown. The after-hyperpolarization becomes a depolarizing after-potential as recordings are taken further from the soma. This change corresponds to the change from single spiking to burst discharges seen at the two locations.

top), depolarization was applied to the dendritic element and an action potential was activated following the break of the pulse. It did not respond to this stimulus or to direct depolarization with a burst discharge, suggesting that the proximal dendritic region behaves more like the soma than the more distant apical dendritic membrane. A d-spike can be seen in the coupled soma underlying a full somatic action potential which arises from the d-spike (Fig. 5*B*, bottom).

Another conspicuous feature of the response recorded in the proximal dendritic region, illustrated in Fig. 5, is the shape of the after-potential. Although the proximal dendritic response resembles the somatic response in that both elements fire single spikes, we observed in ten cases out of ten trials that the dendritic action potential was followed by a pronounced depolarizing after-potential, not the after-hyperpolarization that followed spikes recorded in the soma. The gradual change in after-potential from hyperpolarizing at the soma to depolarizing in the dendrites arises from changes in the membrane properties along the soma–dendritic axis that could ultimately give rise to the burst firing pattern observed in the distal apical dendrites. It can be observed that during burst firing in the distal dendritic elements, successive fast spikes in the burst are elicited during the depolarizing after-potential of the preceding fast spike (e.g. Fig. 4).

Dendritic responses to somatic stimulation

When spikes were evoked directly in the cell soma, attenuated spikes (spikelets) could be seen in one-to-one correspondence in the dendritic recording (Fig. 3*Bb*; Fig. 6, where dendritic recordings were obtained 170 μm away from the cell body layer).

The spikelets seen in the dendrites in response to action potentials initiated at the soma were of several sizes. Figure 6*A* shows dendritic responses to single spikes evoked by depolarization of soma membrane. At low intensity of stimulation, a single action potential was activated in the soma. The somatic spike triggered a

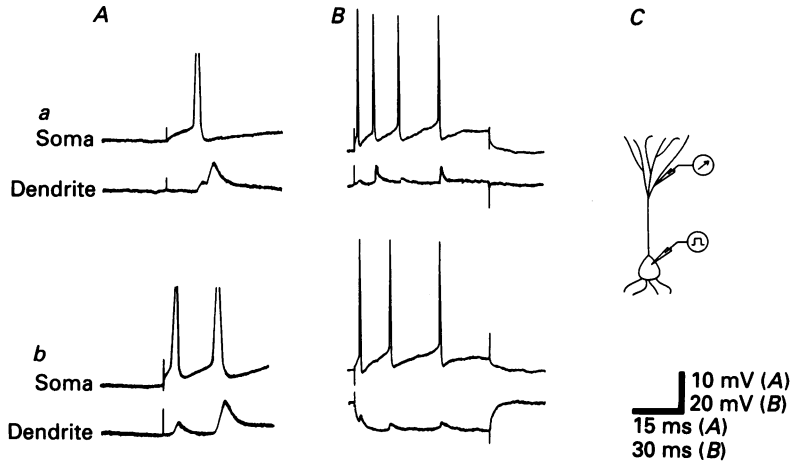

Fig. 6. Propagation of somatic action potentials to the dendrites. Action potentials initiated at the soma in each of two paired soma–dendrite recordings. *Aa* and *b*, pair of recordings show responses at two somatic stimulus intensities. Two attenuated spikes (spikelets) of different amplitudes are apparent, occurring together in relation to a single somatic spike (*Aa*) and occurring separately when stronger somatic stimulation produces two action potentials (*Ab*). *Ba* and *b*, in this pair of recordings the spikelets recorded in the dendrite were also of two discrete sizes. Concurrent dendritic hyperpolarization eliminated the large amplitude spikelets, suggesting that they originated at a site closer to the dendritic electrode than the smaller ones. *C*, the drawing indicates the basic configuration for initiating action potentials at the cell soma. Calibrations: 10 mV and 15 ms for *A*; 20 mV and 30 msec for *B*.

fractionated spikelet in the dendrite (Fig. 6*Aa*). On other occasions, somatic action potentials activated discrete dendritic spikelets with different amplitudes (Fig. 6*Ab*). Amplitude variations of dendritic spikelets triggered by somatic action potentials may result from the somatic spikes invading separate active dendritic zones (Spencer & Kandel, 1961; Schwartzkroin, 1977). The spikelets in a different recording pair also appeared to be two discrete sizes (Fig. 6*Ba*). The larger amplitude spikelets were dropped from the dendritic recording when hyperpolarizing current was applied through the dendritic electrode (Fig. 6*Bb*). This implied that the larger spikelets were due to activity at a region located closer to the dendritic electrode.

DISCUSSION

Burst pattern firing in cell soma reflects activity initiated in apical dendrites

While it is widely accepted that CA1 pyramidal cells are capable of burst discharges, the predominant response of cell somata to direct stimulation is repetitive firing (Schwartzkroin, 1978; Gustafsson & Wigstrom, 1981*b*). In contrast,

burst firing is the predominant response of apical dendritic regions to direct stimulation (Benardo *et al.* 1982). The association of 'd-spikes' with somatic bursts, suggested that the appearance of bursts in the soma may depend on dendritic activity (Spencer & Kandel, 1961; Schwartzkroin, 1977). With paired intracellular recordings, we showed that bursts could be recorded from *both* dendritic or somatic electrodes during direct stimulation of the apical dendrites. Somatic action potentials were found to correspond in a one-to-one fashion with fast action potentials initiated in the apical dendrites. In some instances, when a somatic action potential was not generated, dendritic spikes were not visible in the soma (e.g. Fig. 4). In other cases, however, dendritic spikes appeared as attenuated spikes or pre-potentials underlying full somatic action potentials (e.g. Figs 1 and 5).

The results suggest that a high safety factor for transmission of fast dendritic spikes to the soma accounts for the burst firing of CA1 pyramidal cell somata. With the soma capable of repetitive firing (Schwartzkroin, 1978; Gustafsson & Wigstrom, 1981*b*), it can follow bursts of action potentials initiated in the distal apical dendrites. Previous studies showed that burst firing can be elicited directly in the somata of CA3 pyramidal neurones. In contrast, the burst firing pattern of CA1 pyramidal cells depends upon the location of spike initiation and not upon local membrane conductances. Our results show that after-hyperpolarizations (AHPs) recorded in the soma can limit the ability of the soma to follow the high intraburst firing rates seen in the dendrites. Membrane depolarization or acetylcholine-induced suppression of the Ca^{2+}-dependent and other K^+ conductances underlying the after-hyperpolarization (e.g. Gustafsson & Wigstrom, 1981*a*; Benardo & Prince, 1982*b*; McCormick & Prince, 1986*b*; Lancaster & Nicoll, 1987; Madison, Lancaster & Nicoll, 1987; Storm, 1989; Williamson & Alger, 1990) should improve the ability of the soma to follow, with full action potentials, the high rate of firing associated with dendritic burst discharges. In addition, modification of somatic conductances, resulting in reduced AHPs, may be sufficient to permit the initiation of local burst discharges at the soma. To some extent decreases in the AHP may underlie the higher intraburst firing rate seen at the soma in intact animals (< 5 ms interspike intervals; Ranck, 1973; Fox & Ranck, 1981) as compared with *in vitro* data (about 10 ms interspike intervals; see Figs 1 and 4). Interestingly, in urethane-anaesthetized rats, complex spikes of CA1 pyramids can be selectively eliminated by iontophoretically applied atropine (Stewart, Luo & Fox, 1992). Atropine also eliminated spontaneous burst discharges of CA1 pyramidal somata *in vitro* (Benardo & Prince, 1982*c*).

Previous studies showed that Lucifer Yellow injected into one CA1 pyramidal cell can diffuse to other neighbouring cells (Andrew, Taylor, Snow & Dudek, 1982). This 'dye-coupling' effect suggests the presence of gap junctions between CA1 pyramidal cells (Taylor & Dudek, 1982; cf. Funch, Knowles & Schwartzkroin, 1981 and Knowles & Schwartzkroin, 1981). Given this finding, we cannot be certain whether our coupled recordings were obtained from the dendrites and soma of a single cell or whether these recordings were obtained from the dendrite of one cell electrotonically coupled to the soma of another cell. This uncertainty should not affect the major conclusion of this study: burst firing in the CA1 pyramidal cell is initiated in the distal apical dendrites, whereas cell somata mostly generate repetitive simple spikes. If dendrite–soma recordings were actually obtained from electrotonically coupled

cells, we would have underestimated the fidelity of communication between these cellular elements by assuming that the pair of recordings were obtained from a single cell.

Possible functional significance.

Neurones encode information for transmission to other neurones in the pattern of action potential traffic carried down their axons. Our data indicate that the CA1 pyramidal cell is able to indicate to its cellular targets which of two afferent systems is active. The response of the target neurones may vary depending upon their receiving a burst of action potentials or a stream of repetitive spikes.

The CA1 pyramidal cell is the first cortical neurone whose separate output modes can be shown to depend primarily on the site of activation of the cell. If particular firing patterns can be related to specific behaviour, new insights into the underlying active synaptic circuitry can be revealed. Although burst (complex spike) firing may occur more frequently during some behaviour (e.g. slow wave sleep), in general, both types of firing pattern can be seen during a given behaviour (Ranck, 1973). One instance where the different firing patterns may shed light on the 'normal' activity of the hippocampus is the spatial firing properties of these cells seen in chronically implanted rats. Pyramidal cells in CA1 (as well as other hippocampal regions) have been shown to fire at very high rates when a rat is in a particular place in its environment, and remain essentially silent when the rat is outside that place (O'Keefe & Dostrovsky, 1971; Muller, Kubie & Ranck, 1987). The high rates of firing within a stable 'place field' may consist exclusively of single fast spikes (J. B. Ranck, Jr, unpublished observations). Burst discharges (complex spikes) appear to be extremely rare at these times. Given the results of our experiments, it may be that activities elicited in CA1 pyramidal cells within their 'place field' are primarily activated by somatic or proximal dendritic inputs.

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