Low-Voltage Activated T-Type Calcium Currents Are Differently Expressed in Superficial and Deep Layers of Guinea Pig Piriform Cortex

JACOPO MAGISTRETTI AND MARCO DE CURTIS
Department of Experimental Neurophysiology, Istituto Nazionale Neurologic Carlo Besta, 20133 Milano, Italy

Magistretti, Jacopo and Marco de Curtis. Low-voltage activated T-type calcium currents are differently expressed in superficial and deep layers of guinea pig piriform cortex. J. Neurophysiol. 79: 808–816, 1998. A variety of voltage-dependent calcium conductances are known to control neuronal excitability by boosting peripheral synaptic potentials and by shaping neuronal firing patterns. The existence and functional significance of a differential expression of low- and high-voltage activated (LVA and HVA, respectively) calcium currents in subpopulations of neurons, acutely isolated from different layers of the guinea pig piriform cortex, were investigated with the whole cell variant of the patch-clamp technique. Calcium currents were recorded from pyramidal and multipolar neurons dissociated from layers II, III, and IV. Average membrane capacitance was larger in layer IV cells [13.1 ± 6.2 (SD) pF] than in neurons from layers II and III [8.6 ± 2.8 and 7.9 ± 3.1 pF, respectively]. Neurons from all layers showed HVA calcium currents with an activation voltage range positive to −40 mV. Neurons dissociated from layers III and IV showed an LVA calcium current with the biophysical properties of a T-type conductance. Such a current displayed the following characteristics: 1) showed maximal amplitude of 11–16 pA/pF at −30 mV, 2) inactivated rapidly with a time constant of ~22 ms at −30 mV, and 3) was completely steady-state inactivated at −60 mV. Only a subpopulation of layer II neurons (group 2 cells; circa 18%) displayed an LVA calcium current similar to that observed in deep layers. The general properties of layer II-group 2 cells were otherwise identical to those of group 1 neurons. The present study demonstrates that LVA calcium currents are differentially expressed in neurons acutely dissociated from distinct layers of the guinea pig piriform cortex.

INTRODUCTION

Ion channels operated by cell-membrane voltage are known to control electroresponsiveness of neurons by regulating the efficacy of postsynaptic potentials and by setting the neuronal output modalities. In particular, voltage-dependent calcium conductances influence excitability properties of central neurons by shaping their firing and by promoting intracellular propagation of excitation between dendrites and soma and vice versa (Llinás 1988). Moreover, calcium influx through voltage-gated channels controls several biochemical processes and regulates the expression of genes involved in survival, development, and plasticity changes of neurons (Ghosh and Greenberg 1995). Two main classes of calcium currents with different biophysical and pharmacological properties have been described in neurons of the mammalian nervous system (Tsien et al. 1988), i.e. high-voltage activated (HVA) and low-voltage activated (LVA) currents. Both types of currents and the underlying ion channels have been demonstrated in pyramidal neurons of the hippocampus (Fisher et al. 1990; Kay and Wong 1987; Magee and Johnston 1995; Mogul and Fox 1991) and in neurons from deep layers of the neocortex (de la Peña and Geijo-Barriento 1996; Friedmann and Gutnick 1987; Sayer et al. 1990; Sutor and Ziegglansberger 1987). The specific functional roles of such conductances depend on a variety of factors including their specific biophysical properties, their location in the different neuronal membrane compartments, and their possible spatial association with other conductances. It is currently believed that HVA currents are mostly involved in synaptic release (McCluskey 1994; Takahashi and Momiyama 1993) and in the control of other cell-function regulatory mechanisms by promoting calcium fluxes and prominent intracellular calcium-concentration increases, whereas LVA currents are implicated in dendritic amplification of distal synaptic inputs (Magee and Johnston, 1995; Huguenard 1996) as well as in the generation of regenerative spikes underlying somatic burst firing (Huguenard 1996; Jahnsen and Llinás 1984; Llinás and Yarom 1981).

Although LVA calcium currents have been studied in several cell systems over many years, their properties and expression in mammalian neocortex and paleocortex neurons are incompletely characterized. This may be due to the following: 1) the small amplitude of LVA currents in comparison to HVA calcium currents; 2) their relatively rapid kinetics, which cause space-clamp problems in branched cells such as cortical neurons; and 3) the lack of satisfactorily specific pharmacological blockers.

The differential expression of HVA and LVA calcium conductances in distinct populations of neurons within the same cortical region, is believed to underlie specific intrinsic firing patterns and characteristic synaptic integrative properties that influence the dynamic network organization of the cortex, thereby affecting its information-processing function (Connors and Gutnick 1990). The present study investigates the possibility of a nonhomogeneous expression of calcium currents in different subpopulations of cortical neurons. The piriform cortex was chosen as model for this study because it is composed of three unusually distinct cellular layers that can be selectively isolated for preparing acutely dissociated neurons, and because the presence of a low-threshold, calcium-dependent potential in deep but not in superficial neurons was reported in an in vitro study performed on slices (Tseng and Haberly 1989). The analysis of calcium currents...
with the whole cell variant of the patch-clamp technique in neurons acutely dissociated from layers II, III, and IV of the guinea pig anterior piriform cortex demonstrated that LVA calcium currents are selectively expressed in deep layers and in a small subpopulation of layer II neurons. Part of the results have been communicated in abstract form (Magistretti and de Curtis 1996).

**Methods**

**Cell preparation**

Female Hartley guinea pigs (7–38 days old) were anesthetized with an intraperitoneal injection of ketamine (200–250 mg/kg) and decapitated. The brain was quickly extracted under hypothermic conditions and submerged in an ice-cold solution (dissection buffer) composed of (in mmol/l): 115 NaCl, 3 KCl, 3 MgCl₂, 0.2 CaCl₂, 20 piperazine-N,N'-bis(2-ethanesulphonic acid) -1.5 Na (PIPES-Na), and 25 glucose (pH 7.4 with NaOH, bubbled with pure O₂). The two hemispheres were separated and cut with a McIlwain tissue chopper into 500-μm-thick slices. The section plane was normal to the longitudinal axis of the lateral olfactory tract. Slices that included the anterior piriform cortex were transferred into a 90-mm-diameter Petri dish coated with Sylastic (Dow Corning) and filled with ice-cold dissection buffer. The examination of the sliced fresh tissue at low magnification allowed easy recognition of the lamination of the piriform cortex (Fig. 1A). In each experiment one of the layers (either II, III, or IV) was dissected from each slice under microscopic control. The tissue fragments obtained were then transferred into a 20-ml stirring flask filled with the dissection buffer added with 1 mg/ml pronase (protease type XIV, Sigma, St. Louis, MO) and continuously bubbled with O₂. The flask was submerged in a thermostated bath at 34°C and gently stirred for 15 min. The enzymatic reaction was stopped by removing the solution and by rinsing the tissue three times with a solution (dissociation buffer) containing (in mmol/l): 113.5 NaCl, 3 KCl, 3 MgCl₂, 20 PIPES-Na, 3 ethylene glycol-bis (β-aminoethyl ether) -N,N',N'-tetraacetic acid (EGTA), and 25 glucose and also 2 mg/ml bovine serum albumin (Sigma fraction V) (pH 7.4 with NaOH). The tissue fragments were then placed in a holding chamber kept at room temperature and filled with a continuously oxygenated perfusion buffer, composed of (in mmol/l): 130 NaCl, 3 KCl, 2 MgCl₂, 2 CaCl₂, 10 N-2-hydroxyethylpiperezine-N'-2-ethanesulphonic acid (HEPES), and 25 glucose (pH 7.4 with NaOH). When needed, tissue fragments were removed from the chamber, resuspended in 2 ml dissociation buffer, and triturated with a few passages through Pasteur pipettes of progressively decreasing tip diameter. After sedimentation of the undissociated tissue, the supernatant was transferred into the recording chamber, on a concanavaline A (Sigma type V)-coated, 15-mm-diam round coverslip. The dissociated cells were allowed to settle down for 15 min before starting the recordings.

The recording chamber was mounted on the stage of an Axiovert 100 microscope (Zeiss, Oberkochen, Germany), and the cells were observed at ×400 magnification. Recordings were performed exclusively from neurons with a recognizable pyramidal or multipolar shape. The morphology of the cells was pyramid-like in the case of layer II (Fig. 1, B and C) and multipolar-like in the case of layer IV (Fig. 1, C and D). Cells from layer III had either pyramid-like or multipolar shape (not shown).

**Patch-clamp recordings**

The recording chamber was initially perfused with oxygenated perfusion buffer and then, after the wash-out of cell debris, with an oxygenated extracellular solution suitable for isolation of calcium currents, containing (in mmol/l) 88 choline-Cl, 40 tetraethylammonium (TEA)-Cl, 3 KCl, 2 MgCl₂, 5 CaCl₂, 3 CsCl, 10 HEPES, 5 4-aminopyridine, and 25 glucose (pH 7.4 with HCl). Perfusion rate was about 0.5 ml/min. Patch pipettes were fabricated from thick-wall borosilicate glass capillaries (GC 150-7.5, Clark Electromedical Instruments, Reading, UK) by means of a Sutter
P-87 puller (Sutter Instruments, Novato, CA). The pipette solution contained (in mmol/l) 78 Cs methanesulphonate (CsMeSO₃, obtained by neutralizing CsOH with equimolar methanesulfonic acid), 40 TEA-Cl, 10 HEPES, 10 EGTA, 20 phosphocreatine di-Tris salt (PC), 2 adenosine 5’-triphosphate (ATP)-Mg, 0.2 guanosine 5’-triphosphate (GTP)-Na, and 1 adenosine 3’,5’-cyclic monophosphate (cAMP) as well as 20 U/ml creatinephosphokinase (CPK) (pH adjusted to 7.2 with TEA-OH). In some experiments on layer IV and layer III neurons, PC was substituted by equimolar CsMeSO₃ and ATP, cAMP, GTP, and CPK were omitted to accelerate the rundown of HVA calcium currents, that under these conditions was complete in <10 min. No differences in LVA calcium-current voltage dependence and kinetics were noticed with the two intracellular solutions. The patch pipettes had a resistance of 4–6 MΩ when filled with the above solutions. Tight seals (>1 GΩ) and the whole cell configuration were obtained according to the standard technique (Hamill et al. 1981). Voltage-clamp recordings were performed at room temperature (22°C) by means of an Axopatch 1-D amplifier (Axon Instruments, Foster City, CA.). Cell capacitance was estimated by reading out the cell capacitance value after cancelling the whole cell capacitive transients evoked by a 10-mV hyperpolarizing voltage step with the amplifier compensation section. Series resistance (usually ~12–18 MΩ and always <25 MΩ) was routinely compensated by 50–70%. Voltage protocols were commanded and current signals were acquired with a 486 PC connected to an Axon DigiData 1200 interface, using the Clampex program of the pClamp 6.0.2 software (Axon Instruments). In all recordings the general holding potential was −70 mV. Current signals were filtered at 5 kHz, digitized at 20–50 kHz and leak subtracted via an on-line P/4 protocol.

Data analysis

Current traces were analyzed by means of the Clampfit program. Times to peak were measured from the onset of command voltage steps. Calcium permeability ($P_{Ca}$) was calculated from peak current amplitudes ($I_{Ca}$) by applying the constant-field equation in the form

$$P_{Ca} = I_{Ca} \times (RT/4F)^{1/2} \times [(1 - \exp(-2FV_{m}/RT))/\{[Ca^{2+}]_{o} - [Ca^{2+}]_{i} \exp(-2FV_{m}/RT)\}]$$

in which the nominal intra- and extracellular calcium concentration values (0 and 5 mM, respectively) were introduced. Data were fitted to exponential functions using Clampfit or to Boltzmann functions ($1/1 + \exp((V - V_{1/2})/k))$ using Origin 3.06 (MicroCal Software, Northampton, MA). Average values were expressed as mean ± SD, and statistical significance was evaluated by means of the two-tail Student’s $t$ test for unpaired data.

Results

Two morphologically distinct, but electrophysiologically homogeneous, types of principal neurons with small pyramidal and semilunar somata have been described in layer II (Haberly 1983). We probably recorded from both types of cells, because the size and shape of their somata could not be distinguished after the dissociation procedure. Pyramidal and multipolar neurons are found in layer III, whereas in layer IV (also termed endopiriform nucleus) large multipolar neurons represent the only morphological type of principal cell (Haberly 1983). No recordings were performed in the present study from bipolar cells or other small neurons interpretable as interneurons. Voltage-activated calcium currents were recorded in 39 neurons from anterior piriform cortex layer II, 12 neurons from layer III, and 29 neurons from layer IV. Different postnatal (P) age groups were similarly represented in the neurons from different layers: for P7–P16, P17–P26, P27–P36, and P37–P38 age groups the number of layer II and layers III–IV cells was 20 and 19, 11 and 13, 5 and 7, and 3 and 2, respectively.

Whole cell calcium currents show layer-specific expression of an LVA transient component

Typical whole cell voltage-dependent currents recorded from layers II, III, and IV neurons with our intra- and extracellular solutions are shown in Fig. 2. The superfusion with 200 μM CdCl₂ abolished these currents (not shown), which therefore were identified as calcium currents. In most layer II neurons (Fig. 2, top left panels) calcium currents could
be evoked with test depolarizing voltage steps to −40 mV or above, whereas in all layer III and IV (Fig. 2, bottom left and right panels, respectively) and in a small percentage of layer II cells (Fig. 2, top left panel) the threshold for calcium-current activation was much lower, around −60 mV. In all cases the current-voltage (I-V) relationships (Fig. 2) showed a peak between 0 and +10 mV. Layer III and IV neurons showed a prominent shoulder in the peak-amplitude I-V (●) between −60 and −20 mV, no longer visible when the I-V relationship was constructed by measuring the current amplitude at the end of a 300-ms voltage pulse (□). The shoulder at negative potentials in the I-V curve was not observed in most layer II neurons. Only in a minority of layer II cells (top right panel in Fig. 2) the I-V relationship closely resembled that of layer III and layer IV cells. No differences in current expression were observed among different postnatal age groups (see previous section).

Figure 3A shows typical whole cell calcium-current traces from three other representative layer II and IV neurons. In the majority of layer II cells (left traces) a detectable current appeared at −40 mV, whereas in a small number of layer II cells (middle traces) and in all layer IV neurons (right traces) a calcium current was already present at −60 mV and became fast-decaying at more positive potentials. Layer

**FIG. 3.** Time courses of whole cell calcium currents in layer II and IV neurons. A: total voltage-activated calcium-current traces recorded at different test potentials (T.P.) in 2 neurons from layer II (left and middle) and one from layer IV (right). ● and □, time points at which the peak amplitude (Iₚ) and the amplitude at 120 ms past the peak (I₁₂₀) were respectively measured at the test potential of −40 mV. B: scatter plots of the time-to-peak (tp) as a function of the ratio I₁₂₀/Iₚ (R₁₂₀/Iₚ) of the currents recorded at −40 mV in layer II, III, and IV neurons. In 3 additional cells from layer II no detectable calcium currents were present at the T.P. of −40 mV. ●, ○, ­, and △, experimental points obtained from the sample traces shown in A.
TABLE 1. Average values of cell capacitance and whole cell calcium-current shape parameters

<table>
<thead>
<tr>
<th>Layer</th>
<th>n</th>
<th>Capacitance, pF</th>
<th>$I_p - I_{120}$, pA/pF</th>
<th>$I_{120}$, pA/pF</th>
<th>$R_{120}$, pA/pF</th>
</tr>
</thead>
<tbody>
<tr>
<td>Layer II</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Group 1</td>
<td>33</td>
<td>8.7 ± 2.9</td>
<td>-1.24 ± 0.93</td>
<td>-1.91 ± 1.3</td>
<td>0.61 ± 0.14†</td>
</tr>
<tr>
<td>Group 2</td>
<td>6</td>
<td>8.0 ± 3.0</td>
<td>-0.75 ± 3.4*</td>
<td>-0.67 ± 1.1</td>
<td>0.16 ± 0.015*</td>
</tr>
<tr>
<td>Layer III</td>
<td>12</td>
<td>7.9 ± 3.1</td>
<td>-9.81 ± 5.24*</td>
<td>2.12 ± 2.2</td>
<td>0.15 ± 0.11*</td>
</tr>
<tr>
<td>Layer IV</td>
<td>29</td>
<td>13.1 ± 6.2*</td>
<td>-7.95 ± 3.4*</td>
<td>-1.55 ± 1.3</td>
<td>0.155 ± 0.11*</td>
</tr>
</tbody>
</table>

Values are means ± SD; n = number of observations. Test potential of -40 mV (see Fig. 2 for voltage protocols.) Current amplitudes normalized to cell capacitance. * Statistically significant difference with respect to layer II-group 1 neurons; P < 0.0005. † n = 30. $I_p$, peak current amplitude; $I_{120}$, current amplitude at 120 ms past the peak. $R_{120}$, ratio of $I_{120}$:$I_p$.

III neurons showed currents comparable to those of layer IV cells. As a first screening we analyzed the whole cell calcium currents recorded at -40 mV; for each cell we measured the current peak amplitude ($I_p$), the time-to-peak (tt$p$), and the current amplitude 120 ms past the ttp ($I_{120}$). The ratio between $I_{120}$ and $I_p$ ($R_{120}$) was calculated and taken as an inverse index of current decay. The ttp was then plotted for each cell as a function of $R_{120}$ (Fig. 3B). In all neurons from layers III ($n = 12$) and IV ($n = 29$) both ttp and $R_{120}$ fell below 20 ms and 0.3, respectively, with the ttp fluctuating around an average value of about 12 ms. On the contrary, fixing the same two limit values in the graphic

![Figure 4](http://jn.physiology.org/)

**FIG. 4.** Layer-specific isolation of T-type currents via subtraction procedure. The currents evoked by voltage steps preceded by a 500-ms conditioning prepulse (C.P.) at either -100 mV (A) or -60 mV (B) in representative neurons from layer II-group 1, layer II-group 2, layer III, and layer IV are shown. A–B: traces resulting from the subtraction of the currents preceded by the -60 mV C.P. from those preceded by the -100 mV C.P. The currents obtained at 3 voltage test pulses (-50, -30, and -10 mV, as illustrated at the bottom) are superimposed. Right column: average, normalized I-V relationships of the currents used for and yielded by the subtraction procedure in neurons from the different piriform-cortex layers. In each graphic the mean peak amplitudes of the currents obtained with a C.P. at either -100 mV (●) or -60 mV (○), and of the currents returned by the subtraction (▲) are illustrated. For each cell the current values have been normalized to the absolute value of the maximal observed one (I). The number of observations is 13 for layer II-group 1, 3 for layer II-group 2, 4 for layer III, and 7 for layer IV. SDs (always <20% of the mean) have been omitted for clarity.
relative to layer II neurons (---) allowed identification of two cell groups: a large majority of neurons (n = 33) in which both ttp and R_{120/p} were higher, and a minority of neurons (n = 6) in which both ttp and R_{120/p} were lower than the two limit values. This result means that most layer II neurons had calcium currents more slowly activating and less completely decaying than observed in layers III–IV neurons, whereas in a few layer II cells calcium current activation and inactivation rates were comparable to those of deep-layer neurons. We preliminarily distinguished between layer II neurons having ttp and R_{120/p} higher or lower than 20 ms and 0.3, respectively, and referred to the first as group 1 neurons and to the second as group 2 neurons. An example of calcium currents from a layer II-group 2 neuron is shown in the middle column of Fig. 3A.

The existence of a prominent, transient current component in layer IV and III, but not in most layer II neurons, was confirmed by estimating the amplitude of such component as the difference I_0 - I_{120} at the test potential -40 mV. As summarized in Table 1, this quantity (normalized to the cell capacitance) was much higher in layer IV, layer III, and layer II-group 2 neurons than in layer II-group 1 neurons, whereas I_{120} was not significantly different in the four groups.

Table 1 also shows that the average cell capacitance was significantly higher in layer IV than in layers II and III neurons, consistent with results reported by Banks et al. (1996) for in situ piriform-cortex neurons. On the contrary, the cell capacitance was not significantly different in group 1 and group 2 neurons from layer II. These results strongly suggest that neurons from piriform-cortex deep layers express an LVA T-type calcium current, which is not observed in a large subpopulation of layer II neurons.

Layer-specific isolation of an LVA transient calcium current

To confirm the above conclusion we performed experiments in which the test pulses were preceded by a 500-ms prepulse either at -100 or at -60 mV. The traces obtained with the prepulse at -60 mV were subtracted from those recorded after the prepulse at -100 mV. The results of this procedure in four representative cells are shown in Fig. 4. The subtraction returned very little if any current in layer II-group 1 neurons, whereas an evident fast-decaying
current in layer IV, layer III, and layer II-group 2 neurons was demonstrated (traces a–b in Fig. 4). The average I-V relationships of the currents used for the subtraction (● and ○) and of those obtained after the subtraction (▲) are illustrated in the right column of Fig. 4. In layer IV, layer III, and layer II-group 2 neurons the I-V relationship of the currents yielded by the subtraction had a threshold at about −60 mV, just like the I-V of total currents, and peaked around −30 mV. In layer II-group 1 neurons the subtraction returned tiny currents whose I-V had both threshold and peak at much more positive potentials. The peak amplitudes of the isolated LVA currents at −30 mV were 11.8 ± 6.6 (SD) pA/pF (layer II-group 2 neurons), 16.1 ± 7.0 pA/pF (layer III neurons), and 11.2 ± 5.3 pA/pF (layer IV neurons). Because it is known that LVA T-type calcium currents are steady-state inactivated at relatively negative potentials, these data confirm that layers IV and III neurons express an LVA T-type current that is missing in most layer II neurons.

Voltage dependence and kinetic properties of LVA T-type currents in the different layers

We further characterized the kinetic properties of this current in layer IV, layer III, and layer II-group 2 neurons. The current was isolated either by the subtraction procedure or after waiting for the complete run-down of high-voltage activated currents (see METHODS). The voltage dependence of steady-state inactivation was studied by means of a standard voltage protocol (Fig. 5A). The voltage dependence of activation was analyzed either by means of a tail-current protocol (Fig. 5B) or by deriving permeability values from the peak-current amplitudes (see METHODS). Figure 5 shows current traces recorded in two representative layer IV neurons (A and B) and the average graphics for layer II-group 2, layer III, and layer IV cells (left to right in C, respectively). In all of the three groups of neurons the steady-state inactivation was complete at −60 mV, whereas the activation began near −60 mV and was nearly maximal at −25 mV.

Figure 6 shows the voltage dependence of the current decay rate. The decay part of the currents could be fitted by a single exponential, with a time constant that became progressively faster at more positive potentials (Fig. 6A). Panel B shows the average voltage dependence of the decay time constant (τdec) for layer II-group 2, layer III, and layer IV neuron currents (□, ○, and ●, respectively). In all three neuronal groups τdec displayed a marked voltage dependence, falling from ~90 ms at −60 mV to ~22 ms at −30 mV or above. All these features are typical of a T-type current. On the contrary, in layer II-group 1 neurons, when the above described subtraction procedure returned measurable currents, these currents showed much slower decay time constants (~120 ms at −30 mV: Fig. 6B,▲).

DISCUSSION

The present study demonstrates that LVA T-type calcium currents are segregated in subpopulations of principal neurons in the guinea pig piriform cortex. The experimental preparation utilized, namely the acutely isolated neuron, allowed satisfactory voltage-clamp conditions of the fast LVA current, because most of the dendritic arborization, with the exclusion of the large proximal dendrites, was eliminated by the dissociation procedure. We found an LVA calcium conductance in all freshly dissociated pyramidal and multipolar neurons of the deep layers III and IV. In the large majority of layer II neurons (group 1) no measurable LVA current was present; only a small subpopulation (group 2) represented by 18% of layer II cells displayed an LVA current similar to that observed in deep-layer cells. The biophysical properties of the LVA calcium current in piriform-cortex cells are similar to the T-type calcium current previously described in sensory neurons (Carbone and Lux 1984), in thalamic neurons (Coulter et al. 1989; Hernandez-Cruz and Pape 1989), in pyramidal cells and interneurons of the cornu Ammonis in the hippocampus (Fraser and McVicar 1991;
LVA calcium currents in piriform cortex neurons were studied, revealing specific functions. Unlike superficial neurons, deep pyramidal neurons exhibit higher membrane capacitance and larger somatic membrane surface, allowing a selective expression of an LVA calcium current in their soma and proximal dendrites. These currents are important in the generation of low-threshold calcium spikes, which contribute to intrinsic bursting activity. Deep neurons are more excitable due to the presence of LVA currents, making them more susceptible to synaptic inputs. The specific physiological role of these currents in facilitating synaptic potentials was further strengthened by the demonstration that DAPs mediated by activation of a T-type calcium current regulate plastic cellular changes that promote long-term potentiation. Intrinsically bursting activity and prominent DAPs have been demonstrated in deep pyramidal cells of the neocortex, indicating the importance of LVA calcium currents in deep layers.
REFERENCES


