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THE VENTRAL AND DORSAL LATERAL GENICULATE NUCLEUS OF THE RAT: INTRACELLULAR RECORDINGS *IN VITRO*

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SUMMARY

1. The membrane properties and the electrotonic structure of neurones in the ventral and dorsal lateral geniculate nucleus (l.g.n.) of the rat were studied using an *in vitro* slice preparation.

2. Following electrophysiological characterization, horseradish peroxidase (HRP) was injected intrasomatically and the morphological features of impaled cells were characteristic of principal neurones of the rat ventral and dorsal l.g.n.

3. Neurones in the ventral l.g.n. had a higher input resistance but similar membrane time constants (τ_0) and resting potentials than cells in the dorsal l.g.n.

4. Using a simple neuronal model, the electrotonic length (L) and the dendritic to somatic conductance ratio (ρ) were calculated and found to be similar for cells in both divisions of the l.g.n. The mean value of L (0·7) and ρ (1·5) suggest that both groups of neurones are electrotonically compact.

5. The width and after-hyperpolarization of directly evoked action potentials, but not their threshold or their amplitude, were different between cells of the ventral and dorsal l.g.n.

6. At potentials more negative than −55 mV, a slow rising and falling potential could be evoked in each neurone ($n = 310$) of the dorsal l.g.n. but only in three cells of the ventral l.g.n. ($n = 94$). The electrophysiological and pharmacological properties of this potential were identical with those of the low-threshold Ca^{2+}-dependent potential observed in other thalamic nuclei.

7. These results indicate that some of the passive and active membrane properties of ventral and dorsal l.g.n. neurones are different. The implications of these findings for the control of the integrative capability and the response of l.g.n. neurones to visual stimulation are discussed.

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INTRODUCTION

The ventral and dorsal lateral geniculate nucleus (l.g.n.). are of different embryological origin and differ substantially both anatomically and electrophysiologically (Jones, 1985). Thus in the rat the main efferents of the dorsal l.g.n. project to the visual cortex (Lashley, 1934; Nauta, 1954; Ribak & Peters, 1975), while the ventral l.g.n. innervates preferentially subcortical structures (Swanson, Cowan & Jones, 1974; Brauer & Schober, 1982). The morphological features of their constituent cells as shown in both Golgi (Grossman, Lieberman & Webster, 1973; Werner & Krüger, 1973; Kriebel, 1975; Mounty, Parnavelas & Lieberman, 1977) and horseradish peroxidase (HRP) preparations (Webster & Rowe, 1984; Brauer, Schober, Leibnitz, Werner, Lüth & Winkelmann, 1984) are also quite different, as it is their ability to process visual information (Burke & Sefton, 1966*a*,*b*; Hale & Sefton, 1978; Sumitomo, Sugitani, Fukuda & Iwama, 1979). However, because of methodological difficulties associated with intracellular recordings *in vivo*, comparatively little is known about the membrane properties of individual neurones in the ventral and dorsal l.g.n. and the way in which they may differ.

Taking advantage of the mechanical stability and the control of the ionic environment offered by an *in vitro* preparation we have now studied the membrane properties and the electrotonic structure of principal neurones in the ventral and dorsal l.g.n. of the rat. A preliminary report of some of these results has been published (Crunelli, Leresche & Pirchio, 1985).

METHODS

Slice preparation and recordings

L.g.n. slices were prepared from decapitated male Wistar rats (200 g) as described by Kelly, Godfraind & Maruyama (1979). Briefly, a block of tissue containing the l.g.n. was separated from the rest of the brain by two cuts made parallel to the plane of the optic tract. Using a Vibroslice (Campden Instrument), slices of the l.g.n. (250–400 μm thick) were then cut in the plane of the optic tract as it curves around the thalamus from the optic chiasma forward to the l.g.n. All these procedures were carried out at room temperature. The slices were then transferred to a recording chamber (modified from that described by Haas, Schaerer & Vosmansky, 1979) and perfused with a warmed (35 °C), continuously oxygenated (95 % O_2, 5 % CO_2) medium of composition (mM); NaCl, 134; KCl, 5; KH_2PO_4, 1·25; $MgSO_4$, 2; $CaCl_2$, 2; $NaHCO_3$, 16; 10 glucose. In all these procedures maximal care was taken to avoid excessive mechanical damage to the final few millimetres of the optic tract.

Intracellular glass micro-electrodes were filled with 1 M-potassium acetate or 4 % HRP in 0·5 M-Tris chloride. Potentials were recorded with a precision electrometer (model 707, WP Instruments) which was also used to inject current through the recording micro-electrode. Results were stored on a RACAL FM 4D tape recorder and later analysed with a PDP11/23 computer (Crunelli, Forda, Kelly & Wise, 1983).

Intrasomatic injection of HRP was performed by delivering pulses (300 ms, 2 Hz) of depolarizing current (1–4 nA) for 5–25 min. At the end of the injection the slice was allowed to survive for at least 1 h before being transferred into a fixative solution. It was subsequently placed in 0·1 M-phosphate buffer overnight and, on the following day, embedded in agar and cut at 60 μm. All sections were reacted with 3, 3′-diaminobenzidine and hydrogen peroxide and processed for light microscopic analysis. Recovered neurones were reconstructed using a camera lucida at $\times 400$ and later at $\times 1500$ for finer details.

Electrotonic analysis

Two methods (Brown, Perkel, Norris & Peacock, 1981*b*; Johnston, 1981) were used to calculate the electrotonic length (L) and the dendritic to somatic conductance ratio (ρ) (the neurone model is that of a lumped soma with a finite length, dendritic cylinder attached) (Rall, 1977). Both methods have already been described in detail elsewhere and shown to reproduce satisfactorily the passive electrical behaviour of different types of neurones (Brown *et al.*, 1981*b*; Brown, Fricke & Perkel, 1981*a*; Constanti & Galvan, 1983; Stafstrom, Schwindt & Crill, 1985). Following Rall's (1977) analysis, an electrotonic potential can be represented by the linear combination of exponential terms. Using only the first two terms of the summation, in the method of Brown *et al.* (1981*a*,*b*) ρ is calculated from the equation

$$\rho = \frac{\tau_0}{V_\infty}(a_0 + a_1) - 1, \tag{1}$$

where V_∞ is the size of the electrotonic potential at steady state, a_0 and a_1 are constants, and τ_0 is the membrane time constant.

An approximate measurement of L is then obtained from

$$L \simeq \pi \left[\frac{\rho/(\rho+1)}{(\tau_0/\tau_1)-1}\right]^{\frac{1}{2}}, \tag{2}$$

where τ_1 is the first equalizing time constant, and then more exactly by a trial and error substitution method from the equation

$$\rho + \alpha_1 \cot(\alpha_1 L) \tanh(L) = 0, \tag{3}$$

where

$$\alpha_1 = [(\tau_0/\tau_1) - 1]^{\frac{1}{2}}.$$

The second method is that of Johnston (1981). An initial estimate of L is obtained from $L = \pi/\alpha_1$ and then by a trial and error substitution method using the following equation:

$$\frac{a_1}{2a_0 - a_1} = \cot(\alpha_1 L)\left[\cot(\alpha_1 L) - \frac{1}{\alpha_1 L}\right]. \tag{4}$$

Knowing L, ρ can then be calculated from eqn. (3). From an analysis of eqn. (4) it is evident that this method can be used successfully when the denominator of the left-hand side is greater than zero. Practically we followed the suggestion of Brown *et al.* (1981*a*) of not using this method if the left-hand side of eqn. (4) was much greater than 5.

The values of τ_0, τ_1, a_0 and a_1 necessary to calculate L and ρ were obtained from plots of the derivative with respect to time ($\mathrm{d}V/\mathrm{d}t$) of hyperpolarizing electrotonic potentials lying in the linear portion of the steady-state voltage–current relationship (Rall, 1977). They were used for the electrotonic analysis only if the bridge could be accurately balanced at both the onset and offset of the current pulse. The linearity of the voltage–current relationship was also tested at several points prior to steady state to exclude the presence of transient active conductances. For each cell the initial portion (40 ms) of at least thirty electrotonic potentials was digitized at 10 kHz and averaged. The $\mathrm{d}V/\mathrm{d}t$ value of this averaged electrotonic potential was then calculated with the help of a computer program as

$$\left(\frac{\mathrm{d}V}{\mathrm{d}t}\right) \simeq \left(\frac{\Delta V}{\Delta t}\right) = \frac{V_2 - V_1}{t_2 - t_1},$$

for time intervals ($t_2 - t_1$, typically 1–5 ms) that were short enough so that the slope closely approximated the tangent to the electrotonic potential at a point midway between t_1 and t_2.

Semilogarithmic plots of $\mathrm{d}V/\mathrm{d}t$ *versus* time were then constructed (see Fig. 2) and τ_0 and τ_1 extracted from these plots with an exponential peeling technique. Briefly, in plots such as those in Fig. 2, the -1/slope of the line (least-squares criterion) fitting the latest data points represents the τ_0 of the cell. The difference between the earliest data points and the line is then plotted (▲ in Fig. 2) and the -1/slope of the line fitting these new set of points represents τ_1. The value a_0 and a_1 were estimated as the time-zero intercepts of these two lines.

RESULTS

The experiments described in this paper are based on 310 neurones of the dorsal and 94 neurones of the ventral l.g.n. from which stable, long-lasting intracellular recordings were made.

The ventral and dorsal l.g.n. could be distinguished easily from each other in our slices (Pl. 1 *A*) and in general the most rostral two to three slices containing the l.g.n. were used for each animal. The intrageniculate leaflet was difficult to see in the slice and in general we avoided recording in the 100–200 μm closest to the border limiting the dorsal from the ventral l.g.n. However, neurones impaled along electrode penetrations performed in what appeared to be the intrageniculate leaflet did not show any electrophysiological difference from the others and their results were then included in the total sample. While no difficulty was encountered in recording from neurones in the dorsal l.g.n., cells in the ventral were not easy to hold or even to impale without an almost immediate drop in input resistance (R_N). It is unlikely that this problem was simply the consequence of a small soma size. Although the cell bodies of ventral cells are indeed smaller than those in the dorsal l.g.n. (Brauer & Schober, 1973; Grossman *et al.* 1973; Mounty *et al.* 1977), the very same electrode that was unable to penetrate successfully ventral neurones could produce optimal impalement of the even smaller hippocampal granule cells *in vitro*.

Intrasomatic injection of HRP showed that impaled cells in both the dorsal and ventral l.g.n. possessed morphological features identical to those already described for projection neurones in the two portions of the nucleus. The neurone shown in Pl. 1 *B* was found close to the lateral border of the dorsal l.g.n. Both the size of the soma (540 μm^2), the number of primary dendrites and the shape of its appendages were similar to those of class A cells (Grossman *et al.* 1973) of the dorsal l.g.n. which are believed to be geniculo-cortical relay cells. The asymmetric dendritic field of this cell (note the lack of primary dendrites on the ventral side of the soma) is also characteristic of rat dorsal l.g.n. neurones that lie close to the lateral border of the l.g.n. (cf. Figs. 3 and 4 of Grossman *et al.* 1973). The neurone shown in Fig. 4 *C* was also impaled in the dorsal l.g.n. and possessed morphological features typical of class A cells. The area of the soma profile was 450 μm^2 and small spines were present on both proximal and distal dendrites. The camera lucida reconstruction shown in Fig. 6 *D* is from an HRP-injected cell in the ventral l.g.n. Although its over-all appearance was remarkably different from the two neurones described above, its morphological features were indeed very similar to those described in Golgi preparations for class A cells of the ventral l.g.n. (Mounty *et al.* 1977; Brauer *et al.* 1984). Its morphology was also comparable to that of ventral l.g.n. neurones retrogradely labelled following injection of HRP in the superior colliculus (Brauer & Schober, 1982).

Membrane properties and electrotonic analysis

The resting membrane potentials of neurones in the ventral and dorsal l.g.n. were similar (Table 1). The steady-state voltage–current (*V–I*) relationship of cells of both divisions of the l.g.n. was also similar (Fig. 1). It showed a linear region in the range of membrane potentials -55 to -75 mV and strong rectification in the depolarizing and the hyperpolarizing direction. From measurements taken in the linear region,

TABLE 1. Passive membrane properties of dorsal and ventral l.g.n. neurones

	Dorsal l.g.n.[a]	No. of cells	Ventral l.g.n.[a]	No. of cells
Resting potential (mV)	−60±1	44	−62±3	44
Input resistance (MΩ)	59±3	44	148±19*	44
Time constant (ms)	19·4±1·2	13	17·5±1·3	12
Electrotonic length (B)[b]	0·69±0·04	13	0·64±0·07	12
Electrotonic length (J)[b]	0·74±0·06	8	0·68±0·08	6
Dendritic to somatic conductance ratio (B)[b]	1·35±0·4	13	1·06±0·2	12
Dendritic to somatic conductance ratio (J)[b]	1·61±0·3	8	1·43±0·3	6

[a] Each value is the mean±S.E. of mean.

[b] Letters in parentheses refer to the method used to calculate the electrotonic length and the dendritic to somatic conductance ratio. B: Brown *et al.* (1981*a*); J: Johnston (1981).

* $P < 0·001$

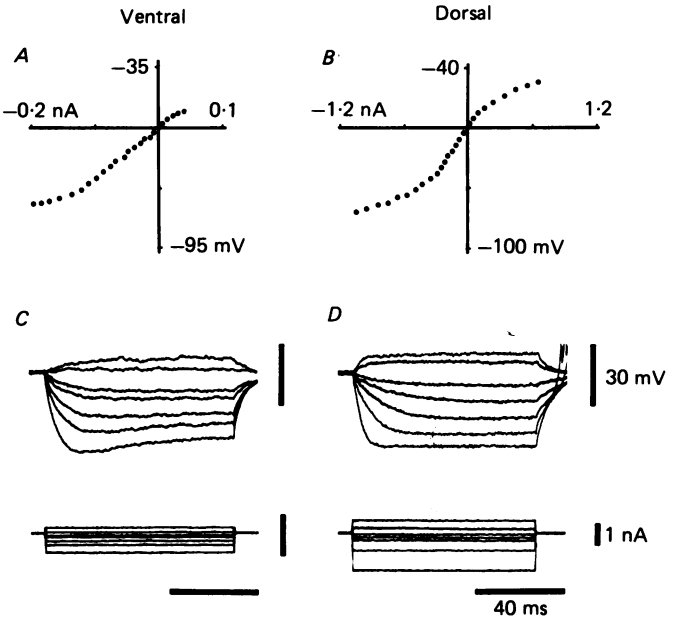

Fig. 1. Voltage–current (*V–I*) relationship of ventral and dorsal l.g.n. neurones. *B*, intracellular voltage records (upper traces) and corresponding current records (bottom traces) used to construct the *V–I* plots shown in *A*. In both neurones the membrane behaves linearly at potentials between −55 and −75 mV. Outside this range strong rectification occurs in both the depolarizing and hyperpolarizing region of the *V–I* relationship.

the input resistance of ventral neurones was observed to be higher than that of dorsal cells (Table 1). Because of the problems encountered in impaling ventral neurones, the possibility existed that the higher input resistance could simply be the consequence of a bias towards using micro-electrodes of smaller tip diameter. However, impalements carried out using the same electrode, in the same slice, in the two

divisions of the l.g.n. (six pairs of cells in two slices) showed ventral neurones to have a higher input resistance.

The membrane time constant (τ_0), the electrotonic length (L) and the dendritic to somatic conductance ratio (ρ) of neurones in the ventral and dorsal l.g.n. were similar (Table 1 and Fig. 2). The mean value of 0·7 and 1·5 for L and ρ respectively indicate

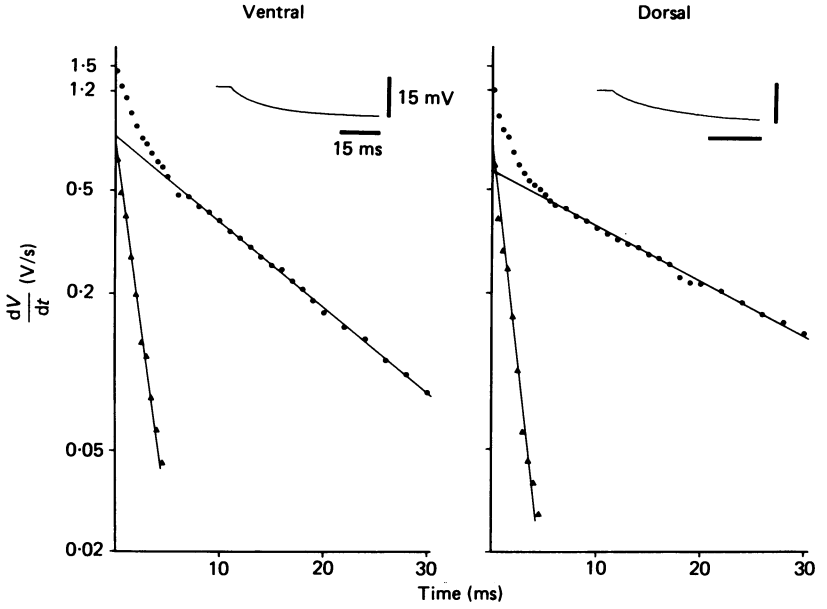

Fig. 2. Time constant of l.g.n. neurones. Semilogarithmic plots of dV/dt *versus* t obtained from the hyperpolarizing electrotonic potentials shown in the insets for a ventral and a dorsal neurone. The values dV/dt are indicated by the filled circles and the -1/slope of the line (least-squares criterion) fitting the clearly linear data points equals τ_0. The values of this line at earliest times are then subtracted from the corresponding data points and the results plotted as filled triangles. A second line is fitted to this new set of points and its -1/slope equals τ_1. The τ_0 of the dorsal cell is 17·9 ms and τ_1 is 1·2 ms. For the ventral cell τ_0 is 13·8 ms and τ_1 is 1·4 ms.

that neurones in both divisions of the l.g.n. are electrotonically compact. In more quantitative terms, the attenuation factor (H) was calculated from the equation $H = \cosh L$ (Jack, Noble & Tsien, 1975), and showed that the amplitude of a voltage signal applied at the soma would be attenuated by 19–24 % at the tip of the equivalent dendritic cyclinder.

Action potentials of dorsal and ventral cells were mediated by Na^+ channels, since they were completely abolished by addition of tetrodotoxin (TTX; 10^{-6} M) to the perfusion medium (Fig. 5*A*). The threshold of directly evoked action potentials was similar between neurones in the two divisions of the l.g.n. (Table 2) and also similar to the threshold for action potentials evoked by synaptic potentials (cf. Table 1 of Crunelli, Kelly, Leresche & Pirchio, 1987). Measurements taken at half-height showed no difference in the width of action potentials, but when measured at threshold for firing the width of action potentials of dorsal cells was longer than the one of ventral

cells (Table 2 and Fig. 3). This was partly a consequence of the different type of spike after-potentials observed sometimes in the two divisions of the l.g.n. (Fig. 3). In fact, although they could both reach the same membrane potential (−80 to −85 mV), the spike after-potential of the ventral neurones appeared to be faster than that of dorsal cells. In addition a notch was often present at the peak of the after-hyperpolarization of action potentials recorded in the ventral l.g.n.

TABLE 2. Properties of action potentials in dorsal and ventral l.g.n. neurones

	Dorsal l.g.n.[a]	Ventral l.g.n.[a]
Amplitude[b] (mV)	77 ± 1	74 ± 3
Width at threshold (ms)	1·0 ± 0·02	0·7 ± 0·02*
Width at 50% amplitude (ms)	0·5 ± 0·01	0·5 ± 0·01
Threshold for firing (mV)	14·8 ± 1·1	11·9 ± 1·8

[a] Each value is the mean ± S.E. of mean from thirty-five dorsal and twenty-three ventral cells.
[b] Measured from resting membrane potential.
* $P < 0·01$.

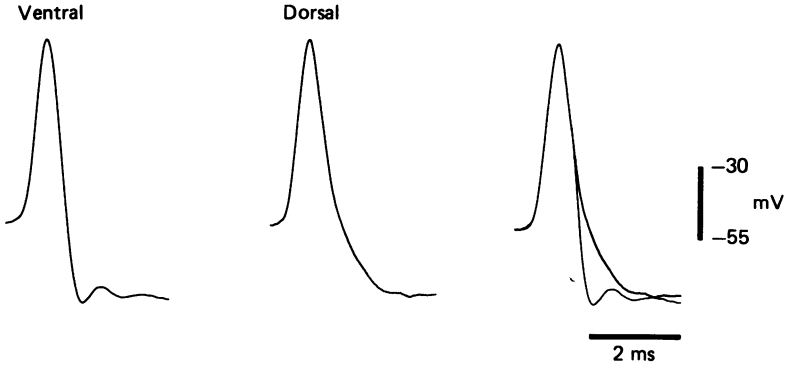

Fig. 3. Action potentials of l.g.n. neurones. Action potentials recorded from a ventral and a dorsal cell are shown in the left and middle trace respectively. The two potentials have been superimposed on the right-hand side. Note how the action potential of the dorsal cell is wider and its after-hyperpolarization reaches the same amplitude but it is slower in time than the one of the ventral cell.

Ca^{2+}-dependent potentials

The most striking difference between neurones impaled in the two divisions of the l.g.n. was the presence of a slow rising and falling potential which could be evoked at membrane potentials more negative than −55 mV in each cell of the dorsal (Fig. 4) but not of the ventral l.g.n. (Fig. 6). The electrophysiological and pharmacological properties of this potential were similar to those of the low-threshold Ca^{2+}-dependent potential recently described in thalamic nuclei of the guinea pig *in vitro* (Jahnsen & Llinás, 1984*a*) and of the cat *in vivo* (Deschênes, Paradis, Roy & Steriade, 1984). Thus, holding the cell at membrane potentials more negative than −55 mV, they could be evoked by pulses of depolarizing current (Fig. 4*A*) or following antidromic action potentials and excitatory post-synaptic potentials (e.p.s.p.s) (cf. Fig. 1 of

Crunelli *et al.* 1987). They could also appear as a rebound response following hyperpolarizing electrotonic potentials reaching membrane potentials more negative than −65 mV (Fig. 4*B*). Ca^{2+} potentials were both time- and voltage-dependent and after activation they showed refractoriness (to full amplitude) of 180 ± 12 ms (mean ± S.E. of mean). They were insensitive to TTX (10^{-6} M, $n = 8$) (Fig. 5*A*) but abolished in the presence of Co^{2+} (2 mM drop application, $n = 5$) (Fig. 5*A*) and Cd^{2+} (0·2 mM, $n = 3$) or when recording in a Ca^{2+}-free medium ($n = 3$) (Fig. 5*B*).

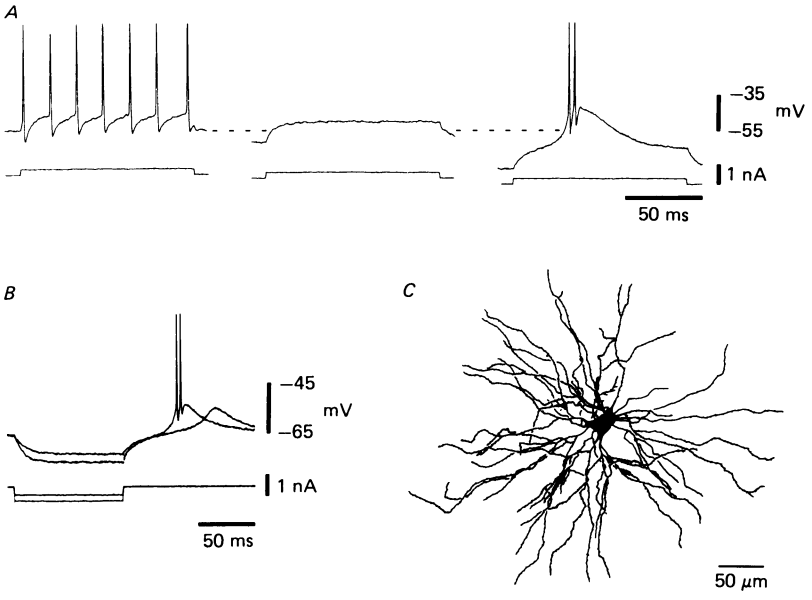

Fig. 4. The Ca^{2+}-dependent potential in neurones of the dorsal l.g.n. In *A*, at resting membrane potential (−55 mV, dashed line) a pulse of depolarizing current evokes repetitive firing (left trace). The cell is then hyperpolarized to −65 mV and the current pulse is not able to reach threshold for firing (middle trace). However when the cell is hyperpolarized to −80 mV the same pulse of current now evokes a Ca^{2+}-dependent potential which in turn reaches threshold for firing (right trace). These records were all obtained with an HRP-filled micro-electrode from the cell shown in Pl. 1*B*. In *B*, the Ca^{2+} potential is evoked as a rebound of an hyperpolarizing electrotonic potential while the cell is at resting potential (−65 mV). A bigger electrotonic potential (reaching −75 mV) evokes a Ca^{2+} potential of shorter latency and higher amplitude that now generates action potentials. The records in *B* were obtained with an HRP-filled micro-electrode from the dorsal l.g.n. neurone whose camera lucida reconstruction is shown in *C*.

In contrast, no such Ca^{2+}-dependent potential could be observed in all but three of the ninety-four neurones impaled in the ventral l.g.n. (Fig. 6). In two of these three neurones its amplitude was never bigger than 5 mV even when the cell was held at −95 mV. As clearly shown in Fig. 6*D*, ventral neurones lacking Ca^{2+} potentials appeared to have morphological features characteristic of projection neurones of this portion of the l.g.n. Since the low-threshold Ca^{2+} potential has been described as one of the conductances underlying the rhythmic oscillation of thalamic neurones (particularly the one at 5–6 Hz) (Jahnsen & Llinás, 1984*b*; Steriade & Deschênes,

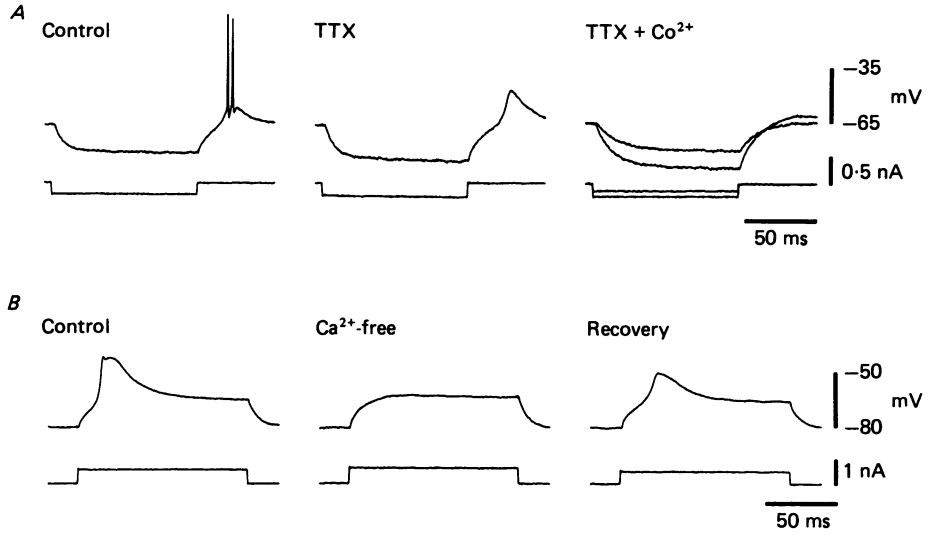

Fig. 5. Ionic dependence of the Ca^{2+} potential. In *A*, the Ca^{2+} potential is evoked as a rebound of a hyperpolarizing electrotonic potential. It is unaffected by tetrodotoxin (TTX, 10^{-6} M) but abolished by Co^{2+} (2 mM, drop application). The cell in *B* was superfused with a solution containing TTX (10^{-6} M) and the Ca^{2+} potential was evoked by a pulse of depolarizing current while holding the membrane potential at −80 mV. After 20 min perfusion with a Ca^{2+}-free medium, the Ca^{2+} potential is abolished and a partial recovery (with a current pulse of smaller amplitude) could be obtained before losing the cell, 30 min after switching back to the control solution.

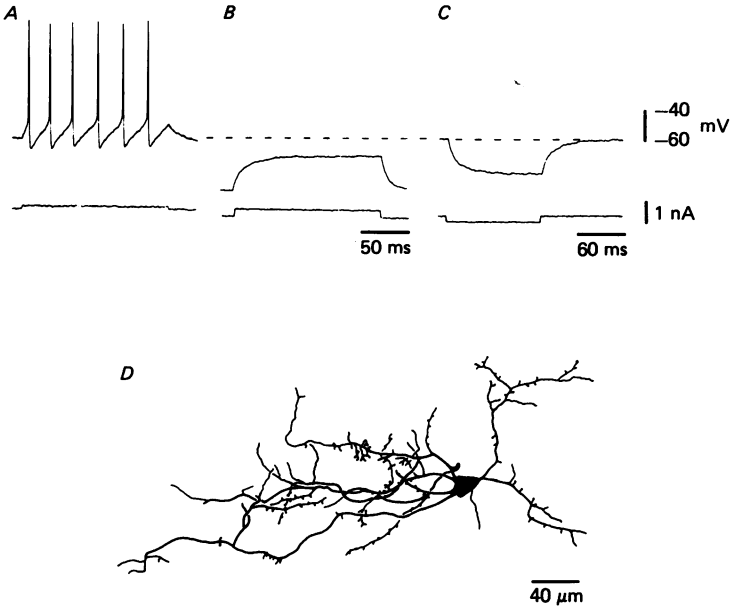

Fig. 6. Lack of Ca^{2+} potentials in a neurone of the ventral l.g.n. All records were obtained with HRP-filled micro-electrode from the cell shown in *D*. In *A*, repetitive firing is evoked at resting potential (dashed line) by a pulse of depolarizing current. In *B*, the cell is hyperpolarized to −90 mV but a pulse of even bigger amplitude than in *A* is not able to evoke a Ca^{2+} potential. *C* shows that no Ca^{2+} potential is evoked as a rebound of a hyperpolarizing electrotonic potential reaching −90 mV. *D* is the camera lucida reconstruction of the HRP-injected neurone in the ventral l.g.n. from which the records in *A*, *B* and *C* were obtained.

1984), it would appear unlikely that neurones in the ventral l.g.n. possess such rhythmic capability. In fact we have never been able to observe a 6 Hz firing pattern in ventral neurones, even although it was sometimes possible to trigger this activity in dorsal cells *in vitro* by electrical stimulation (single shock) of the optic tract (Fig. 7).

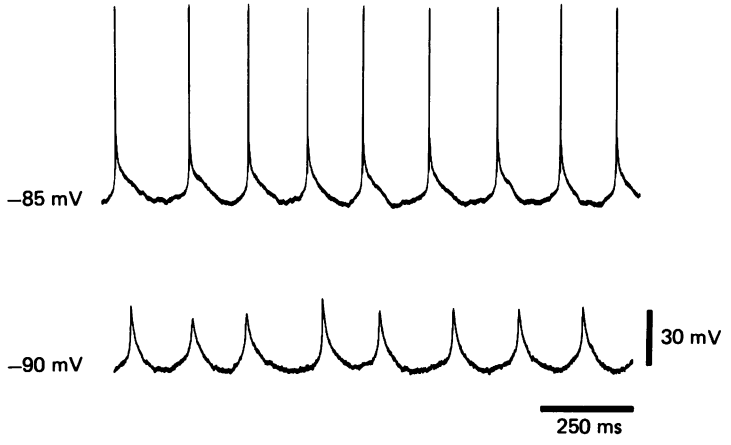

Fig. 7. Oscillation in a neurone of the dorsal l.g.n. The intracellular voltage records show rhythmic firing at a frequency of 5–6 Hz recorded from a dorsal l.g.n. neurone following electrical stimulation (single shock) of the optic tract, while holding the membrane potential at −85 mV (top trace) and at −90 mV (bottom trace) so that the Ca^{2+} potential could not reach threshold for firing.

DISCUSSION

The main conclusion of this investigation is that some of the passive and active membrane properties of principal neurones in the ventral and dorsal l.g.n. of the rat are different. Thus, although cells in both portions of the l.g.n. have similar resting potentials and τ_0 and are electrotonically compact, those in the dorsal l.g.n. have lower input resistances and action potentials that are wider, and often with a different after-hyperpolarization. In addition, they possess Ca^{2+}-dependent potentials which are de-inactivated by hyperpolarization and which are absent in ventral neurones.

Membrane properties of l.g.n. neurones

Two main groups of cells, class A and class B, have been identified in both the dorsal and ventral l.g.n. of the rat using Golgi impregnation techniques, with class A cells being the projection neurones and class B cells the presumed interneurones (Lieberman & Webster, 1972, 1974; Grossman *et al.* 1973; Mounty *et al.* 1977; Parnavelas, Mounty, Bradford & Lieberman, 1977). More recent studies have indeed confirmed this classification by showing class A cells of the dorsal and ventral l.g.n. to be the only cells labelled following HRP injections into the visual cortex and the superior colliculus respectively (Brauer & Schober, 1982; Webster & Rowe, 1984). Physiologically, two main groups of cells have also been identified, namely relay cells and interneurones (Burke & Sefton, 1966*a*,*b*; Sumitomo *et al.* 1979), although in some studies it has been suggested that presumed interneurones are clustered together not in the l.g.n. itself but mainly in the nucleus reticularis (Sumitomo, Nakamura &

Iwama, 1976). Our data show a remarkable similarity in the membrane properties of neurones recorded in the dorsal l.g.n. on the one hand and in the ventral l.g.n. on the other, and without any difference inside each group. Following intracellular injection of HRP, impaled neurones showed morphological features typical of those described for projection cells in the dorsal or ventral l.g.n. Thus, similar to the results obtained in other *in vitro* studies (Jahnsen & Llinás, 1984*a*), we too have been unable to record substantially from thalamic interneurones. Two possibilities can be taken into consideration in an attempt to explain such a finding. The first, and more likely, explanation is that because of their soma size and/or some particular feature of their membrane structure, class B cells were more easily damaged by impalement with our micro-electrodes. Secondly, it may be possible that the interneurones are more susceptible than the projection cells to the mechanical trauma caused by the slicing procedure and/or to the maintenance *in vitro*. This, however, seems unlikely since in other areas more extensively studied *in vitro* interneurones have been shown to be morphologically well preserved and to allow intracellular recordings (Nicoll & Madison, 1984). Moreover, in our l.g.n. slices the e.p.s.p.s evoked by stimulation of the optic tract were followed by an i.p.s.p. mediated by γ-aminobutyric acid (GABA) (Crunelli *et al.* 1987) which is the presumed transmitter of the majority if not all l.g.n. interneurones (Ohara, Lieberman, Hunt & Wu, 1983). This indicates that at least the GABA-containing interneurones are still functionally active. Notwithstanding, another possibility might be considered for the dorsal l.g.n., i.e. that some of the neurones we had recorded from without HRP electrodes might have been the short-axoned interneurones that had membrane properties identical with those of the HRP-classified principal cells. Physiologically, in the rat l.g.n. *in vivo*, interneurones have been distinguished from principal cells by the fact that they respond with a burst of action potentials to stimulation of the optic tract and optic radiation. Principal cells always respond with a single action potential that in the case of optic radiation stimulation is the antidromic spike. In both groups of cells this initial response is then followed by two or more bursts of action potentials at intervals of about 100 ms (Burke & Sefton, 1966*a*,*b*; Sumitomo *et al.* 1976). Thus it might be possible that a tonic input present *in vivo* could keep a set of l.g.n. neurones at a hyperpolarized level of membrane potential. As a consequence, these cells will respond to mono- and polysynaptic stimulation with a burst of action potentials generated by Ca^{2+} potentials (cf. Figs. 1 and 2 of Crunelli *et al.* 1987). It must be noted, however, that in the lateral thalamic nuclei of the cat *in vivo*, cells with physiological characteristics of interneurones have been shown to possess membrane properties different from relay cells (Deschênes *et al.* 1984).

The higher input resistance of ventral compared to dorsal l.g.n. neurones suggests that the efficacy of synaptic signals will probably be greater in the former cell group. The similarity in τ_0, on the other hand, indicates that temporal summation of synaptic potentials in the two portions of the l.g.n. is rather similar. Since τ_0 and L are similar in both cell groups and the specific membrane capacitance is thought to be a biological constant (1 μF/cm^2) (Cole, 1968; Jack *et al.* 1975), the higher input resistance of ventral cells cannot simply be the consequence of a different membrane resistivity but might probably also reflect the smaller surface area of ventral as opposed to dorsal l.g.n. neurones.

Jahnsen & Llinás (1984*a*) did not report a higher input resistance of cells in the ventral l.g.n. of the guinea-pig *in vitro*, where it appears they had recorded from at least ten cells (cf. Fig. 1 of Jahnsen & Llinás, 1984*a*). Nor did they observe a higher input resistance of neurones in the other ventral nuclei of the thalamus which are embryologically similar to the ventral l.g.n. (Jones, 1985). It may be that the difference in input resistance is simply due to the different species used (rats as opposed to guinea-pig) as indeed it has been shown for other cells in the central nervous system like the locus coeruleus (Williams, Henderson & North, 1985). Our mean value of τ_0 for dorsal and ventral cells is also higher than the value reported by Jahnsen & Llinás (1984*a*). Since these authors did not mention the method used to calculate τ_0, it is difficult to propose a reasonable explanation for this discrepancy. However, it should be noted that methods other than the one used in the present study have been suggested to lead to underestimations in the value of τ_0 (Rall, 1977).

In this study the methods used by Brown *et al.* (1981*b*) and Johnston (1981) to calculate L and ρ gave slightly different results, with the method of Brown *et al.* (1981*b*) consistently giving on average smaller values of L and ρ by 4% and 35% respectively. Similar discrepancies between the two methods have been reported for neurones of the hippocampus and the olfactory cortex and dorsal root ganglion cells in dissociated culture (Brown *et al.* 1981*a*,*b*; Constanti & Galvan, 1983). However, we felt that the method of Brown *et al.* (1981*b*) had to be used since the one of Johnston (1981) is not applicable to every cell for mathematical reasons. The mean values of L and ρ indicate that, despite their extensive arborization, both ventral and dorsal l.g.n. neurones are electrotonically compact, as are other vertebrate neurones studied *in vitro* (Brown *et al.* 1981*b*; Johnston, 1981; Constanti & Galvan, 1983; Stafstrom *et al.* 1985), including X and Y cells of the cat l.g.n. (Crunelli, Leresche & Parnavelas, 1986). This electrical compactness does not appear to be simply the consequence of the *in vitro* technique, since similar findings have been recently reported for cat l.g.n. neurones *in vivo* (Bloomfield, Hamos & Sherman, 1985). Optic nerve terminals are believed to synapse on different parts of the dendritic tree of rat l.g.n. cells, i.e. proximally for dorsal as opposed to distally for ventral neurones (Grossman *et al.* 1973; Stelzner, Baisden & Goodman, 1976). This observation has been taken as a basis to explain the different response of cells in the two portions of the l.g.n. to electrical stimulation of the optic nerve (Brauer & Schober, 1982). In the light of our results, it is clear that at least in the steady state, as far as location is concerned, the efficacy of optic-nerve-generated synaptic signals in the two cell groups is probably similar. However, as mentioned before, because of the high input resistance the synaptic signals will have a greater weight in the integration process at the soma of ventral compared to dorsal cells.

Lack of Ca^{2+} potentials in the ventral l.g.n.

The Ca^{2+}-dependent potentials present in neurones of the dorsal l.g.n. are indeed similar to those described in the l.g.n. and in other thalamic nuclei of the cat and guinea-pig both *in vitro* and *in vivo* (Jahnsen & Llinás, 1984*a*; Deschênes *et al.* 1984; Fourment, Hirsch & Marc, 1985). The role played by such potentials in the responsiveness of dorsal as opposed to ventral cells to electrical stimulation of the optic tract is discussed in detail in the following paper (Crunelli *et al.* 1987). The

absence of Ca^{2+} potentials in cells of the ventral l.g.n. is somewhat puzzling, and in contrast with the results of Jahnsen & Llinás (1984*a*) which have suggested that such potentials are a feature of thalamic relay cells but not of interneurones. It is unlikely that we have recorded preferentially from interneurones in this part of the l.g.n. Our experiments with HRP clearly show that impaled neurones had indeed those morphological features typical of projection cells of the ventral l.g.n. The fact that neurones in the ventral l.g.n. are developmentally different from those of the dorsal l.g.n. might explain their different membrane properties such as the lack of Ca^{2+} potentials. On the other hand, the ventral l.g.n. derives from the same group of cells that give origin to other ventral thalamic nuclei (Jones, 1985), which have indeed been shown to possess Ca^{2+} potentials, at least in cats and guinea-pigs (Deschênes *et al.* 1984; Jahnsen & Llinás, 1984*a*). The most likely explanation of our results is that ventral l.g.n. neurones in the rat develop differently from those of the rest of the thalamus and thus possess unique peculiarities in their membrane properties such as the lack of Ca^{2+} potentials and a higher input resistance.

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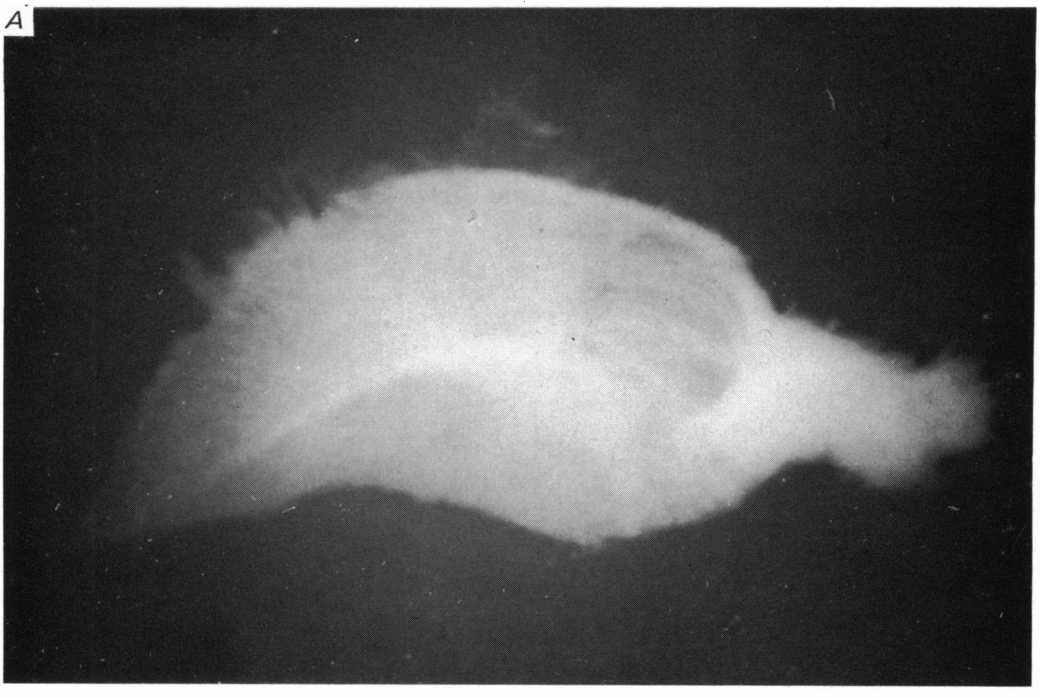

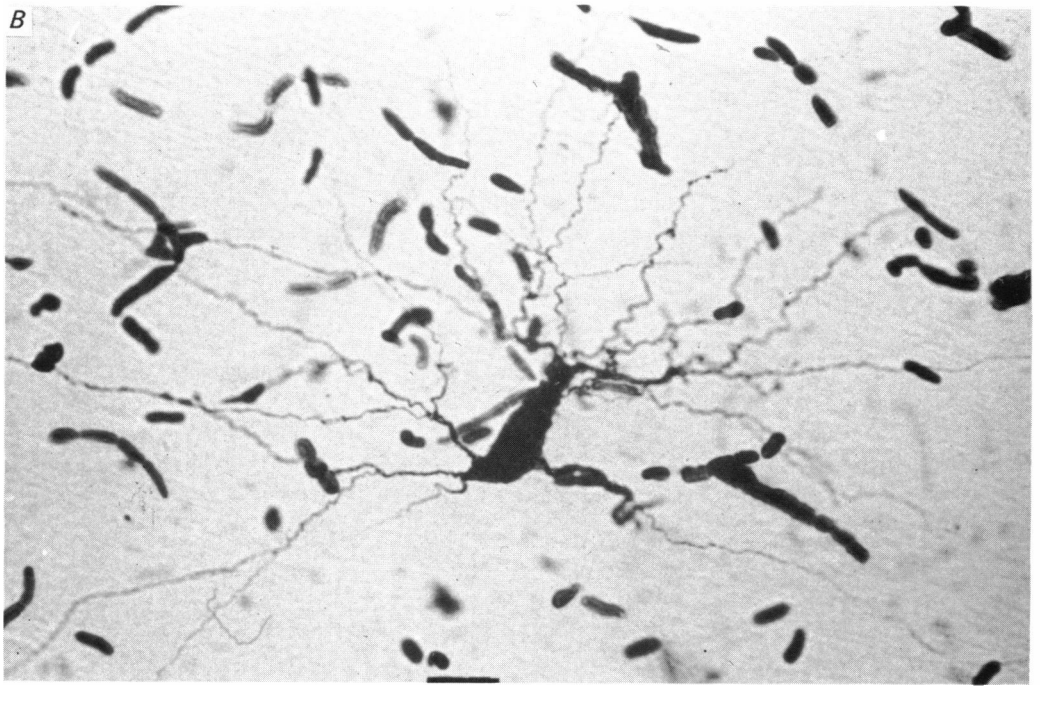

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EXPLANATION OF PLATE

A, typical slice (350 μm thick) of the rat l.g.n. (unfixed) containing the final portion of the optic tract (white band on the right-hand side of the slice), the ventral and the dorsal divisions of the nucleus (lateral is top, medial is bottom). *B*, HRP-injected cells of the dorsal l.g.n. from a 60 μm thick section. The asymmetric dendritic field is typical of cells lying close to the lateral border of the l.g.n. as this one was (lateral is bottom). Calibration bar equals 25 μm.