parasite affected the activity of the host also. No specific enzyme was found in the free living forms of *M. hapla,* but negative results for amylase, pepsin and cellulase were obtained[8]. Acid phosphatase was observed in 10-day-old larvae of *M. javanica* and fresh larvae of *Tylenchulus semipenetrans;* and the activity increased in the older larvae, thus showing the role of enzymes in ageing of the parasites[5]. Erlanger and Gershon[9] observed acid phosphatase activity in soil nematodes, and the activity varied at different stages of nematodes. Sex ratio of plant nematodes has been found to vary under different physiological and ecological conditions. Sex ratio of *Ditylenchus dipsaci* changed when cultured on callous tissues of different hosts[10]. Tyler[11] noted that percentage of males of *Meloidogyne* in tomato increased during malnutrition and other stresses on the host plant, more fertilized eggs are produced. In these observations, the males showed greater activity of both the phosphomonoesterases than the females. There were differences of 28.76% in the alkaline phosphatase and 60.36% in acid phosphatase activity of the 2 sexes of *M. lucknowica.* Thus sexual variations are related to the physiology of sex in the plant nematodes.

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Innervation by W-type retinal ganglion cells of superior colliculus neurons projecting to pulvinar nuclei in cats

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Summary. Neurons of the cat superior colliculus (SC) sending their axons to the pulvinar nuclei were identified electrophysiologically as the ones responding antidromically to electrical stimulation of the pulvinar nuclei. They were located in the superficial layers of SC and found to be innervated by axons of W-type ganglion cells of the retina.

Anatomical studies have shown that there is an ascending projection from the superior colliculus (SC) to the thalamic pulvinar nuclei (Pul), which in turn projects largely to the visual association cortex[3–5]. The experiments reported here were made in cats for the purpose of knowing what types of retinal ganglion cells are responsible for visual impulses which are relayed by SC to Pul.

Material and methods. All the experiments were conducted in cats anesthetized with nitrous oxide (70% O_2:30% N_2O), paralyzed with continuous i.v. infusion of Flaxedil (20 mg/h) and artificially respirated.

Bipolar electrodes with exposed tips about 1.5 mm apart were introduced stereotaxically into the optic chiasm (OC), the optic tract (OT), 3 portions of the medial pulvinar nucleus[6] (Pul_m) and 2 portions of the lateral pulvinar nucleus[6] (Pul_l). Electrical stimuli were single pulses of 0.01–0.5 msec in duration with variable intensities. Extracellular spike discharges were recorded from SC with glass-coated tungsten microelectrodes.

During experiments the animal was fixed in a stereotaxic apparatus facing a tangent screen (99° × 99°) that was placed 1 m in front of his eyes. The pupils were dilated by topical application of phenylephrine chloride (Midrin-P, Santen Chemical). The cornea was protected with a contact lens having such a power as to give a clear projection of retinal landmarks onto the screen.

The visual stimuli were spots and slits of light (60 lux) projected onto the dimly lit screen (14 lux) from the mirror of a moving coil galvanometer which was driven by a function generator. Hand-held black rectangles and light spots from an ophthalmoscope (Pantoscope, Keeler) also provided stationary and moving stimuli.

Results and discussion. A total of 152 SC units responded to both OC and OT stimulation. The latencies to OT stimulation were distributed from 0.7 to 12.9 msec, with a pattern similar to that reported by Hoffmann[7] (figure 2, E). From these response latencies and the measured value of the distance from OC to OT (average 13.1 mm) conduction velocities of the OT fibres were calculated. They ranged from 0.9 to 67.4 m/sec and found to cover all the retinocollicular pathways so far reported[7,8].

Among 136 SC units activated by stimulation of OC and OT, 132 were also activated by stimulation of Pul_m and Pul_l. Of these, 120 showed transsynaptic responses which were characterized by a sign of EPSP and variable latencies of spike discharges. In other 12 units, responses to stimulation of Pul_m were found antidromic, although those to stimulation of Pul_l were transsynaptic. The antidromic response is exemplified in figure 1, A and B; the spike had a short and fixed latency with a clear step midway to the positive peak (an arrow in figure 1, A). When paired stimuli were applied with short intervals, the response to the 2nd shock was reduced in size, sometimes the A spike only. These SC cells followed repetitive stimulation of Pul_m up to 100 Hz. In one unit with spontaneous discharges, the collision test[9] successfully proved that its Pul_m-induced response was of antidromic origin; in other units no such test could be made because of paucity of spontaneous discharges.

The SC cells driven antidromically by Pul_m stimulation were found in the layer from which the OC-induced field response was recorded as a positive wave of intermediate amplitude. Referring to previous works[10–12] which studied the configuration of the SC field response to electrical

stimulation of the optic pathway as a function of the recording depth within SC, it was judged that the SC cells sending their axons to Pul_m lie in the stratum griseum superficiale.

Figure 1, C, shows the response latency histogram of SC units to Pul_m stimulation. The antidromic response latencies ranged from 0.45 to 2.4 msec with the average at 1.23 ± 0.61 msec (dark columns). In the antidromically driven unit, the latency to OT stimulation ranged from 5.0 to 11.8 msec (mean 7.43 ± 2.34 msec) (figure 2, E) and that to OC stimulation from 6.5 to 13.1 msec (mean 9.1 ± 2.56 msec). The axonal conduction velocities calculated ranged from 4.2 to 11.7 m/sec with an average of 7.9 ± 2.3 m/sec. This value corresponds to the conduction velocity of the slowest group of OT fibres. It is thus assumed that the SC cells driven antidromically from Pul_m are connected directly to retinal W-cell axons.

Among the 12 cells responding antidromically to Pul_m stimulation, 6 were subject to various tests for visual properties. All of them were responsive only to stimulus velocities less than 50°/sec, which is reported as a characteristic of the SC cells innervated by W-cell axons[7] (figure 1, C). 4 cells were driven from both eyes equally, while 2 cells from the ipsilateral eye only. Direction selectivity was tested in 4 out of 6 units; 3 were direction selective (figure 1, D) and the remaining one was not. 2 units examined with a stationary spot flashing on the receptive field centre showed phasic ON- and ON-OFF responses (figure 1, B). These properties are consistent with what is known as the major characteristics of SC cells activated by retinal W cells[7].

Using the horseradish peroxidase method, Kawamura and Kobayashi[13] demonstrated that larger cells in the stratum griseum superficiale contributed to the tecto-thalamic projection, which is directed almost exclusively to Pul_m, no projection being given to Pul_l and the inferior pulvinar. Confirming these anatomical findings, antidromic invasion of SC cells were obtained only from Pul_m.

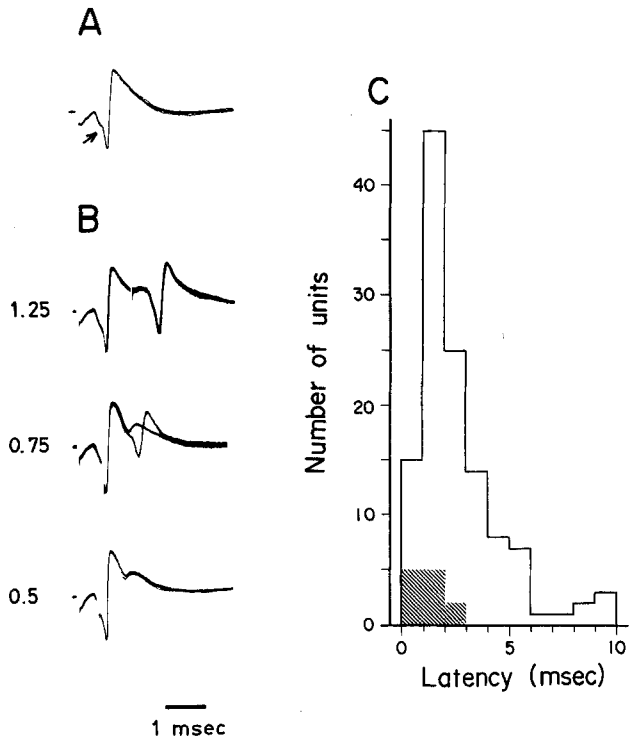

Fig. 1. *A* and *B* Antidromic responses of a SC cell to Pul_m stimulation. In each record, 5 sweeps were superposed. In *A*, the *A*–*B* step was indicated by an arrow. In *B*, paired shocks were applied with various intervals. Numerals to the right indicate shock intervals in msec. With a shock interval of 0.75 msec, the 2nd response occurred once in 5 sweeps and was the *A* spike only. With the shock interval reduced to 0.5 msec, the *A* spike failed to occur to the 2nd stimulus. *C* Latency histograms of SC cells to Pul_m stimulation. Histograms of orthodromic (open columns) and antidromic (dark columns) response latencies are presented together.

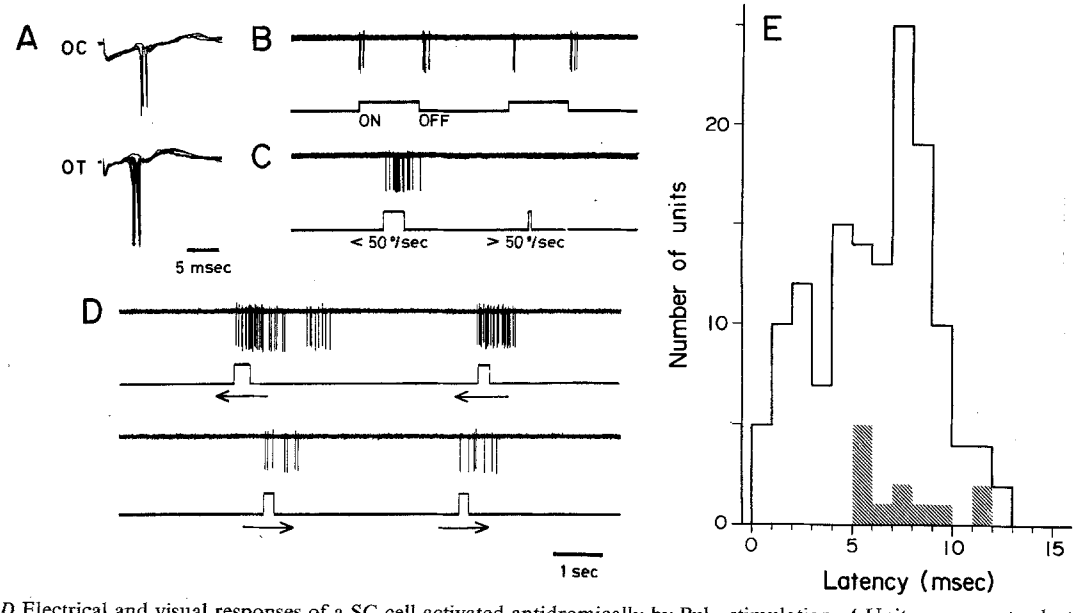

Fig. 2. *A*–*D* Electrical and visual responses of a SC cell activated antidromically by Pul_m stimulation. *A* Unit responses to electrical shocks applied to OC and OT. 5 responses were superposed. OC- and OT-latencies were 6.5 and 5.0 msec, respectively. Conduction velocities for the unit is calculated as 9.0 m/sec. *B* ON-OFF phasic responses to a 2° stationary spot flashed upon the receptive field. *C* Responses to a black rectangle ($1° \times 0.5°$) moving with speeds above and below 50°/sec. *D* Responses to a black rectangle ($1° \times 0.5°$) moving to right (→) and left (←). *E* Latency histogram of SC cells to OT stimulation. SC cells activated antidromically by Pul_m stimulation are distinguished by dark columns.

The present study has provided evidence that the tecto-pulvinar projection is originated from SC cells innervated by retinal W-type cells. The previous findings[14,15] that neurons in cat Pul_m are sensitive only to complex visual stimuli (figured stimuli and/or moving stimuli) may be accounted for, at least partly, by the fact that these pulvinar neurons are stimulated indirectly by retinal W-type ganglion cells which are known to have complex visual properties[16,17].

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Histamine release in dogs by Emulphor EL620[1]

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Summary. A vehicle containing ethanol and Emulphor EL620 lowers blood pressure and increases heart rate in morphine-chloralose anesthetized dogs. These effects are associated with histamine release caused by Emulphor EL620.

Emulphor EL620 is a polyoxyethylated vegetable oil which has been recommended as a component in an i.v. formulation for Δ^9-tetrahydrocannabinol[2,3], and this formulation has been used in studies of the pharmacological effects of cannabinoids[4–8]. Since Cremophor-EL, which is similar to Emulphor EL620, causes release of histamine in dogs[9], we investigated the formulation[3] containing Emulphor EL620 for this effect.

Methods. Mongrel dogs of either sex, 6–12 kg b.wt, were anesthetized with sodium pentobarbital, 35 mg/kg i.v. (18 dogs) or with morphine, 1 mg/kg i.m., followed by chloralose, 100 mg/kg i.v. (30 dogs). A cannula was placed into a fermoral artery for measurement of blood pressure with a transducer (Statham, model P23G) and the electrical output of this operated a biotachograph (Narco Biosystems, model B1200) for measurement of heart rate; both variables were recorded continuously with a physiograph (Narco-Biosystems, model 4CFM). Where appropriate, blood pressure, heart rate and latent period are expressed as the average ±SD. 6 pentobarbital and 10 morphine-chloralose anesthetized dogs were injected twice, 20 min apart, with ethanol, 0.03 ml/kg i.v. Each dose was twice the amount of ethanol administered in the formulation containing Emulphor EL620. The latter was prepared in a formulation[3] consisting of 5% Emulphor EL620, 5% ethanol and 90% sodium chloride solution (0.9%) for injection into a femoral vein. Injections were given twice, 20 min apart, and the dose of ethanol and Emulphor EL620 in each was 0.015 ml/kg. In 4 additional morphine-chloralose anesthetized dogs, 5 ml of blood was drawn from a femoral vein at 6 and 1 min before, and 1, 7 and 20 min after injection of the formulation (3 dogs) or compound 48/80, 1 mg/kg i.v. (1 dog). In 1 other dog, the formulation was given twice with a 10-min interval between treatments; blood was drawn at 6 and 1 min before, and 1 min after the 1st dose, and at 2 and 10 min after the 2nd dose. All blood samples were centrifuged for 5 min at 374×g; the plasma was separated and frozen until analyzed for histamine.

The assay for plasma histamine was adapted from the procedure of Beaven et al.[10]. The histamine in a 50-µl sample was labelled by methylation with tritiated S-adenosyl methionine using histamine methyl transferase isolated from guinea-pig brain[11]. The radiolabeled histamine was measured with a Beckman LS-250 scintillation spectrometer. The lower level of sensitivity of the assay was 4 ng histamine base/ml of plasma.

Results. There were no effects on blood pressure or heart rate in any of the dogs injected 2 times with ethanol, at a dose twice that administered in the formulation containing Emulphor EL620. We thus conclude that the effects described below for the formulation are due to Emulphor EL620.

In each of 12 pentobarbital anesthetized dogs, Emulphor EL620, 0.015 ml/kg i.v., caused sustained hypotension. The average blood pressure was 129±4.5 mm Hg (range, 110–160 mm Hg) before treatment, and was decreased an average of 60±3.5% (range, 38–85%). Heart rates averaged 143±51 beats/min, and, in 9 of the dogs, there was an average increase of 16±4.8% (range, 2–45%), followed by a sustained decrease of 21±2.3% (range, 14–35%); in the remaining 3 dogs, there was only a sustained decrease of 29±5.3% (range, 10–37%). The latent period, i.e., the time from injection of Emulphor EL620 to onset of cardiovascular changes, averaged 1.8±0.14 min (range, 1.5–2.5 min). After a 20-min interval, all 12 dogs were again given Emulphor EL620, 0.015 ml/kg i.v., and it had no further effect.

In 20 morphine-chloralose anesthetized dogs, blood pressure and heart rate averaged 113±3.2 mm Hg (range, 95–130 mm Hg) and 97±2.4 beats/min (range, 85–120 beats/min), respectively. In 15 of the dogs, Emulphor EL620, 0.015 ml/kg i.v., caused sustained decreases in blood pressure averaging 32±2.9% (range, 13–43%) and sustained increases in heart rate averaging 85±6.3% (range, 35–139%).

In 2 of the remaining 5 dogs, blood pressure and heart rate were not affected and, in another 2, blood pressure was unchanged, but there was either a transient (+41%) or a sustained (+70%) increase in heart rate. In the 5th dog blood pressure was increased transiently by 17% followed by a sustained decrease of 13%, and there was a sustained increase in heart rate of 95%. For the 18 of 20 dogs in which