had 2 small apertures, one used to accommodate the polyvinyl plastic tubing by which the gas mixtures were delivered, the other used to pass the ECG leads from the animal to the polygraph and to allow for continuous flushing and exchange of gases. Gas was allowed to flow at 4 l/min, permitting complete equilibration of the gas in the box within 1 min of switching to a new gas mixture. Oxygen content of the gas mixture, delivered from cylinders of O_2 and N_2 by an adjustable multiple flow meter, was continuously monitored by a Beckman model E2 Oxygen Analyzer, allowing control of the O_2 concentration to within 0.5%.

Initially, compressed air was allowed to flow for at least 15 min to permit attainment of a steady, basal HR. At the desired time, delivery of the experimental gas was started and maintained at 21% (20.6 $\pm$ 0.5%) O_2 for a 15 min control period, then changed to 10.0 $\pm$ 0.5% O_2 for 5 min, and finally returned to 21% O_2 for a 10 min recovery period. Throughout, HR was sampled at $^1/_2$-min-intervals. To test the effects of blocking drugs, either 0.5 ml 0.9% NaCl alone, or 0.2 mg atropine sulfate or 5.0 mg propranolol hydrochloride (Inderal, Ayerst) dissolved in 0.5 ml 0.9% NaCl, was injected i.p. Animals were then placed in the boxes and, after 15 min to allow stabilization of HR, the 15 min control period with 21% O_2 was begun. (In preliminary tests, 0.2 mg atropine blocked for at least 1 h the bradycardia produced in a pentobarbital-anesthetized 400 g rat by stimulation of the right vagus with squarewave impulses at 15 Hz, 2 msec duration, 8 V. 5.0 mg propranolol blocked the tachycardic effect of 0.125 µg isoproterenol [Isuprel, Winthrop] given i.v.) The order of administration of drugs was randomized among the animals, and an interval of at least 2 days was allowed between drug trials on each animal.

24 trials of hypoxia were conducted on the untreated rats, i.e. 5 trials on each of 4 animals, 4 trials on one. In addition, 5 trials on each of the 5 animals were conductet with 0.9% NaCl injection, and 4 trials per animal were made with each of the blocking drugs. Analysis of variance of the data obtained during control periods indicated that the variances among animals were not significant, i.e. $p > 0.25$. Hence, HRs measured in the 20–25 trials for each sample point, i.e. at each $^1/_2$-min-interval, were pooled and reported as a mean value for that point plus or minus the standard error of the mean.

Results (figure). In the untreated rats, HR averaged 330 beats per min (bpm) during the latter part of the control period (21% O_2). HR began to rise within $^1/_2$ min from the onset of administration of 10% O_2, and attained a maximum, steady level around 415 bpm at about 3 min. On resumption of 21% O_2, HR fell to its control level within 3 min. Injection of saline increased resting HR by 10–15 bpm, but did not affect the pattern of response to hypoxia. From a level of 340 bpm, HR rose steadily to a peak of 420 bpm after 3 min of exposure to 10% O_2. When 10% O_2 was replaced by 21% O_2, HR returned to its resting level in about 3 min. Atropine raised the resting HR to 420–440 bpm. HR increased steadily in response to 10% O_2, peaking after 3 min at 470–480 bpm. When 21% O_2 was resumed HR decreased to its previous resting level in about 2 min. It then continued to decrease slightly, stabilizing at 390–410 bpm. Propranolol lowered resting HR to approximately 300 bpm and greatly modified the response to 10% O_2. HR rose transiently by 15 bpm for the first 1–2 min of exposure, then decreased gradually to its resting level where it remained. On resumption of 21% O_2 HR fell to about 270 bpm in the first minute; it then gradually increased and stabilized at the previous resting level.

Discussion. The fact that atropine markedly raised the resting heart rate (HR) implies a fairly high degree of vagal tone in the normal resting animal. The reduction in the absolute magnitude of tachycardia in response to hypoxia (maximum mean increase of 60 bpm with atropine vs. 80 bpm with saline) probably indicates that, starting from a greatly elevated level, HR was approaching an upper limit when it increased to 480 bpm in response to hypoxia. The failure of atropine to alter qualitatively the hypoxic tachycardia strongly indicates that inhibition of vagal tone plays little role in the response. The decrease in resting HR following injection of propranolol suggests some beta-adrenergic control of the heart in the normal resting rat. Since propranolol essentially blocked the hypoxic tachycardia, this response is apparently due almost entirely to beta-adrenergic stimulation (cardiac sympathetic innervation and circulating catecholamines). The transiency of the slight tachycardia at the beginning of the period of hypoxia suggests that it differs from the normal tachycardic response. As this slight rise in HR disappeared during continued exposure to 10% O_2, the possibility of incomplete block of the adrenergic receptors by the propranolol is unlikely. No ready explanation for the post-hypoxic bradycardia in the propranolol-treated animals is at hand, but mediation of the bradycardia by a baroreceptor reflex may be ruled out since preliminary unpublished observations indicate that arterial pressure does not rise in response to hypoxia in untreated or propranolol-blocked rats.

Sustained and transient cortical neurones in area 18 of the cat

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Summary. The majority of the simple cells in area 18 of the cat's visual cortex gave pure transient responses to flashing slits of light. A few simple cells gave sustained responses to stationary slits of light. All of the complex cells encountered gave transient responses.

Neurophysiological investigations of the receptive field properties of the relay cells in the lateral geniculate nucleus (LGN) of the cat have revealed 2 major groups of cells, X or 'sustained' and Y or 'transient' cells[2–5]. Ikeda and Wright[6] have classified cells in area 17 of the visual cortex as 'sustained' or 'transient' and reported that the 'sustained/transient' classification is independent of the 'simple/complex' classification.

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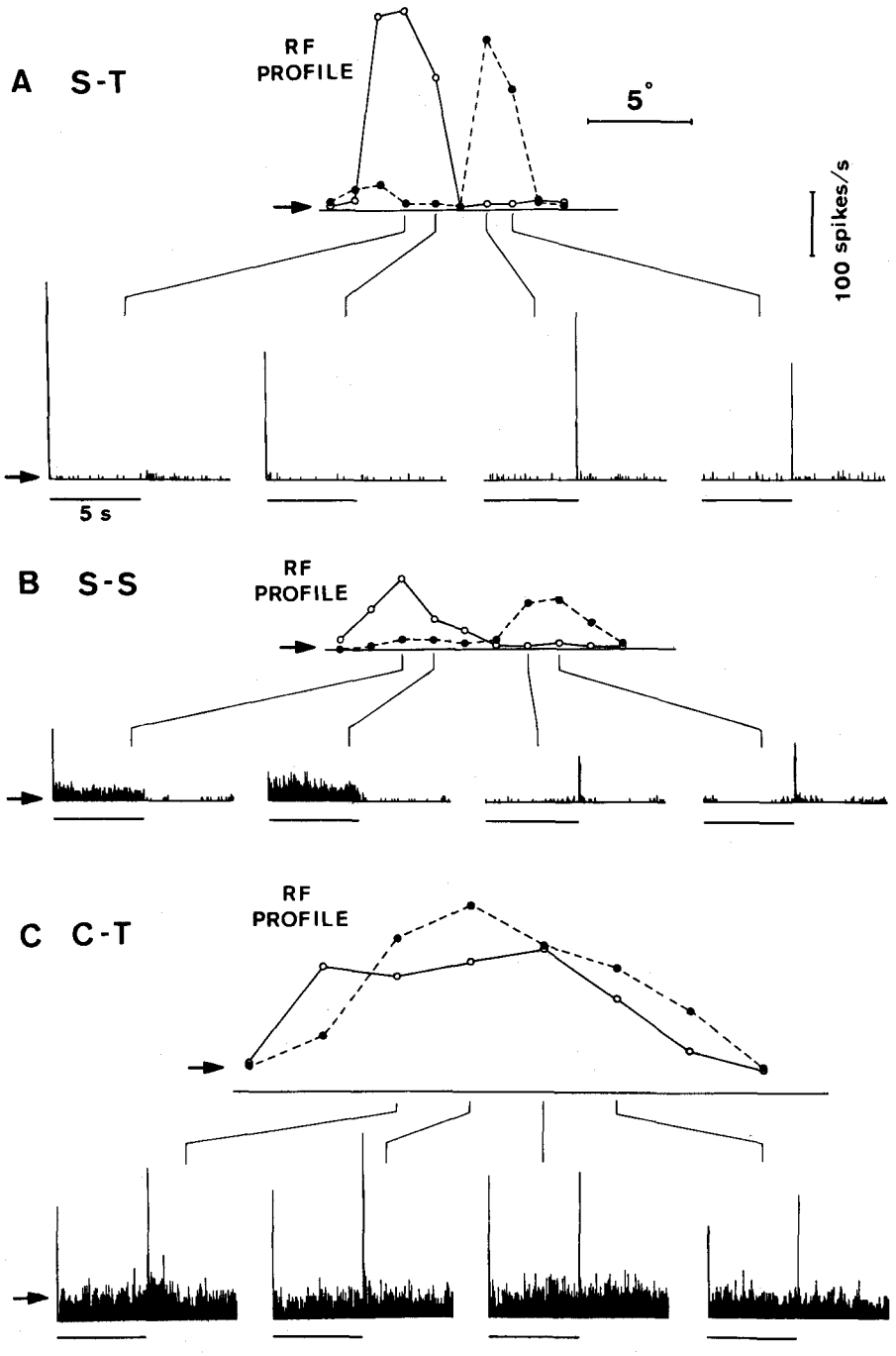
2 B. G. Cleland, M. W. Dubin and W. R. Levieck, J. Physiol., Lond. *217*, 473 (1971).
3 K.-P. Hoffman and J. Stone, Brain Res. *32*, 460 (1971).
4 K.-P. Hoffman, J. Stone and S. M. Sherman, J. Neurophysiol. *35*, 518 (1972).
5 J. Stone and B. Dreher, J. Neurophysiol. *36*, 551 (1973).
6 H. Ikeda and M. J. Wright, Exp. Brain Res. *22*, 363 (1975).

It is well established that the peristriate area 18 receives a direct input from the LGN[7–14]. At least the majority of the simple and complex cells in area 18 were monosynaptically excited by electrical stimulation in the LGN[3,15,16]. Moreover, the axons reaching area 18 from the LGN belong exclusively to Y-cells[5,16]. Therefore, it is expected that the majority of the simple and complex cells in area 18 would be classified as 'transient' cells. The aim of the present paper is to check the above prediction.

Material and methods. 12 adult cats weighing 2.5–3.5 kg were used. Cannulations of the trachea and the right cephalic vein and craniotomy were performed under fluothane (2.5–3.5%) anesthesia. Ears and woundlips were locally anesthetized with 1% xylocaine. To prevent



Classification of cortical cells in area 18 into 3 types. Average response histograms were obtained from 'simple-transient' (S-T), 'simple-sustained' (S-S), and 'complex-transient' (C-T) cortical cells. Each histogram was obtained using a flashing stationary slit of light, optimally oriented and placed in the position indicated in the receptive field (RF) profile, and was collected over 20 presentations of the slit of light. Horizontal bar below each histogram: presentation of a slit of light for 5 sec. Arrows: spontaneous firing rate. Slit dimensions: 15° × 0.6° in A, and 15° × 1.5° in B and C. Receptive field location from the vertical meridian and from the horizontal meridian: 14° lateral and 20° below in A, 20° lateral and 20° below in B and 10° lateral and 25° below in C. Receptive field profiles were obtained from the responses to a slit of light. Ordinate: peak evoked response (spikes/s). Abscissa: distance across the receptive field at right angles to the prefered orientation. Open circles: on response. Filled circles: off-response.

eye movement, the animal was paralyzed with a continuous perfusion of gallamine triethiodide (6 mg/kg per h) and d-tubocurarine (1 mg/kg per h). The animal was artificially ventilated with 70% N_2O and 30% O_2 with additional fluothane 0.5%. The body temperature was maintained at 37–38 °C by a thermostatically controlled heating blanket.

The animal was fixed in a Bishop type stereotaxic apparatus facing a 114 cm tangent screen with a luminance of approximately 10 cd/m^2. The pupils were dilated by applying tropicamid and phenylepherine chloride (Midorin, Santen Chemical). A pair of contant lenses were selected from a family of lenses prepared in $^1/_8$ diopter steps, so that the retina was focussed on the screen using direct projection method[17]. From the position of the blind spots projected by the method, the area centralis of each eye was marked on the screen using the data of Vakkur et al.[18]. A slit of light of adjustable dimensions and orientation was projected on the screen. It could be moved back and forth by a pair of mirrors attached to moving coil galvanometers. A mechanical shutter, operated electrically, was employed for flashing the slit of light. The intensity of the slit of light was about 60 cd/m^2.

Single units were recorded extracellularly by a glass-coated tungsten microelectrode inserted through the dura matter. The trephine hole in the cranium was sealed with soft bone wax to minimize brain movement. Once a unit was isolated, its optimal orientation was determined using slits of light moved in a direction perpendicular to the slit's orientation. Thereafter, a flashing slit of light, oriented parallel to the receptive field orientation, was used for the stimulus. Average response histograms (bin width = 10 ms) were obtained over 20 presentations of this stimulus. The spontaneous firing of a unit was also collected with background illumination alone. The average response of the unit was compared with the spontaneous firing, in order to classify the unit as 'sustained' or 'transient'.

Receptive fields were also classified as simple or complex cells according to the criteria set by Hubel and Wiesel[19, 20]. We considered therefore as simple cells only those orientation specific units whose receptive fields could be subdivided into spatially distinct on- and off-areas, and whose responses were maximal with stimuli filling an entire on- or off-area. We considered as complex cells those orientation specific cells which either did not show a separation of the receptive field into excitatory areas, or which did not show summation of the stimulus effect over such separate areas.

Results and discussion. All units were recorded from an area within Horsley Clarke coordinates anterior 3–5 and lateral 4–5 mm, in order to avoid the border between area 17 and 18. And also to avoid recording from the upper bank of the supra-splenial sulcus, electrodes were advanced up to 2 mm from the cortical surface. Most units of the sample had their receptive fields located 7–20° lateral from the vertical meridian and 8–25° below the horizontal meridian. We have always checked up on the matter that receptive fields of units recorded from an area within lateral 3 and anterior 3–5 mm were located nearer to the vertical meridian than those of the sampled units. The cells of the sample were actually registrated in area 18.

We observed 3 characteristic types of responses to flashing slits of light within receptive fields. These are shown in average response histograms of the figure, with arrows to indicate the mean spontaneous firing rate of the unit. The first type of the unit is illustrated by the histograms in the figure A. It gave a transient response to a flashing slit of light. After the transient response, the firing rate immediately fell to the spontaneous level. The receptive field was subdivided into spatially distinct on- and off-areas.

The second type of the unit is illustrated by the histograms in the figure B. It had a receptive field profile similar to the first type. It gave a sustained response to a slit of light within on-area. The response remained more than 10 spikes/sec above the spontaneous rate after 5 sec. Within off-area, this unit gave a transient response to off-stimulus of the slit of light.

The third type of the unit is illustrated by the histograms in the figure, C. It gave a transient response to both on- and off-stimuli of a slit of light within its receptive field. The first and second types were classified as simple cells, and the third as complex cells. Of 137 units encountered, 42 were simple cells, 73 were complex cells, and the remaining 22 cells were not classified in the simple/complex scheme either. Of 42 simple cells, 34 gave pure transient responses to flashing slits of light, and only 8 gave sustained responses to stationary slits of light within on-area. All of the complex cells encountered gave transient responses. Therefore, of simple and complex cells in area 18, 93% were of transient type.

In contrast to area 18, in area 17, 61% out of simple and complex cells were transient type and sustained responses were observed both in the simple and complex cells[21]. Therefore, 'transient' cells were much more frequent in area 18 than in area 17. Another difference was that sustained responses were not observed in the complex cells encountered in area 18.

The great majority of area 18 cells gave pure transient responses. This is consistent with the fact that Y-cells constitute the only input from the LGN to area 18. However, our data have shown that a few simple cells in area 18 give sustained responses, which X-cells are likely to give.

7 L. J. Garey and T. P. S. Powell, Proc. R. Soc. *B 169*, 107 (1967).
8 L. J. Garey and T. P. S. Powell, Proc. R. Soc. *B 179*, 41 (1971).
9 M. Glieckstein, R. A. King, J. A. Muller and M. Berkley, J. comp. Neurol. *130*, 55 (1967).
10 S. K. Niimi and J. M. Sprague, J. comp. Neurol. *138*, 219 (1970).
11 A. C. Rosenquist, S. B. Edwards and L. A. Palmar, Brain Res. *80*, 71 (1974).
12 S. Rossignoll and M. Colonnier, Vision Res., suppl. *3*, 329 (1971).
13 K. Toyama, K. Matsunami, T. Ohno and S. Takashiki, Exp. Brain Res. *21*, 45 (1974).
14 M. S. Wilson and B. G. Cragg, J. Anat., Lond., *101*, 679 (1967).
15 K. Toyama, K. Maekawa and T. Takeda, Brain Res. *61*, 395 (1973).
16 F. Tretter, M. Cynader and W. Singer, J. Neurophysiol. *38*, 1099 (1975).
17 S. Yamane, S. Kaji and N. Sugie, J. physiol. Soc. Japan *33*, (1971).
18 G. J. Vakkur, P. O. Bishop and W. Kozak, Vision Res. *3*, 289 (1963).
19 D. H. Hubel and T. N. Wiesel, J. Physiol., Lond. *160*, 106 (1962).
20 D. H. Hubel and T. N. Wiesel, J. Neurophysiol. *28*, 229 (1965).
21 H. Ikeda and M. J. Wright, Exp. Brain Res. *22*, 385 (1975).