neurons was identified by characteristic field potentials evoked by orthodromic and antidromic stimulations. Successful penetrations were indicated by resting membrane potentials exceeding – 45 mV.

Results and discussion. Intracellular recordings were made from the Deiters' neurons as identified by characteristic field potentials. Figure A shows an example of a Deiters' neuron which could be driven antidromically from the oculomotor nucleus as well as orthodromically from the saccule. Latency for the EPSP was 0.8 msec and the orthodromic action potential could be triggered as early as 1.2 msec after the onset of stimulus. The antidromic spike was initiated after a delay of 0.3 msec. Such records provide further evidence that some of the Deiters' neurons are connected monosynaptically with the primary afferents from the saccule and project directly to the ipsilateral oculomotor nucleus. Figure B shows, however, that some Deiters' neurons could be connected both monosynaptically (1.0 msec) and disynaptically (1.8 msec) with the saccule. Figure C depicts that among 33 Deiters' neurons so recorded, 2 populations of cells could be distinguished. The first population (n = 14) responded monosynaptically to saccular stimulation with a mean latency of 1.14 ± 0.05 msec (SEM) but the second population (n = 19) responded with a mean latency of 2.14 ± 0.08 msec (SEM), and therefore probably driven disynaptically. In no case did any of the disynaptically driven cells respond to antidromic stimulation. The tri-neuronal nature of the sacculo-ocular reflex therefore remains unchallenged at least in so far as the vertical eye movements are concerned. Experiments are in progress to study the specific connections between the different vestibular nuclei and the extra-ocular motoneurons in the sacculo-ocular pathways.

Hypoxic tachycardia in the rat[1]

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Summary. Awake, freely breathing rats subjected to moderate hypoxia (10% O_2) manifest prompt tachycardia which is essentially unaffected by atropine and is blocked by propranolol, and is thus apparently mainly of sympathetic origin.

When exposed to moderate hypoxia, freely breathing dogs generally manifest tachycardia[2], while bradycardia is commonly observed in cats[3] and rabbits[4]. The present experiments were designed to characterize the change in heart rate (HR) in unanesthetized laboratory rats breathing 10% O_2, and to assess the roles of vagal and sympathetic influences in the response.

Methods. Under brief halothane (Fluothane, Ayerst) anesthesia, 5 male Sprague-Dawley rats (370–470 g) were each implanted with 3 Michel nickel-silver wound clips, widely spaced along the dorsal midline, for use as active electrocardiographic (ECG) and ground leads. At least 1 day intervened between the anesthesia and the first experimentation. At each recording session, the Michel clips were connected to a Grass 7P1A preamplifier whose output was used to trigger a Grass 7P4A cardiotachograph for recording of HR on a Grass 7D polygraph.

Experimental trials were conducted with the rats in clear plastic boxes of a size (16.5 × 8.5 × 8.5 cm) chosen to restrict movement without discomfort, as indicated by prompt cessation of any struggling. The top of each box

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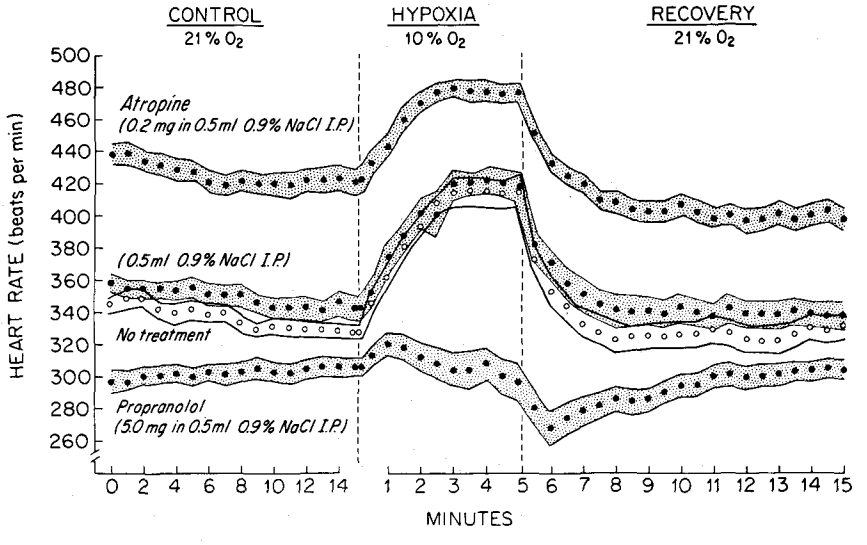

Heart rate responses to hypoxia produced by 10% O_2 in rats untreated (open circles) or injected i.p. (filled circles) with 0.5 ml of 0.9% NaCl alone, or with atropine (0.2 mg) or propanolol (5.0 mg). The circles represent the average values for all trials in all rats (numbers given in text) at each 1-min- (control) or $^1/_2$-min- (hypoxia and recovery) interval, while the accompanying lines indicate ± SE of the mean. Note that the time scale for the control period is compressed.

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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