morphologically well suited for possible ionic interactions between molecular layer elements. They are densely packed, fine (0.1 to 0.3 μm) nonmyelinated axons passing through Purkinje dendrites, separated by a narrow extracellular space with few intervening glial elements (*16*). These characteristics might allow the PF's to be particularly sensitive to changes in $[K^+]_o$.

Changes in $[K^+]_o$ have been hypothesized to be functionally significant in several neuronal systems (*17*). Our results support the proposal that the excitability of the presynaptic afferent PF's is influenced by increases in $[K^+]_o$ resulting primarily from activity in postsynaptic elements. Accumulation of extracellular potassium could lead to membrane depolarization, which, if large enough, could inactivate the sodium channel and slow and possibly block conduction (*18*). Whether such changes occur during normal cerebellar activity is open to question. Granule cells discharge small bursts of action potentials at frequencies above 500 Hz (*19*). We demonstrated changes in PF frequency-following for short bursts at lower firing frequencies (ten impulses at 50 Hz). If changes in $[K^+]_o$ occur during normal activity in the cerebellum, then a mechanism might operate that could inhibit the afferent PF's in the microsurround of a Purkinje cell, by changing the extracellular ionic environment.

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11. That the tips of K^+ sensing microelectrodes are large in comparison with the extracellular space results in predictable delays in detecting evoked increases in $[K^+]_o$ [Orkand, *Fed. Proc. Fed. Am. Soc. Exp. Biol.* **39**, 1515 (1980)]. With brief (<1 second) trains of stimuli, the evoked $[K^+]_o$ increases sum, but do not reach a steady state, and the final stimulus is followed by a further, slightly delayed, increase in $[K^+]_o$. Similar considerations explain the tendency of the K^+ microelectrode to underestimate the $[K^+]_o$, which actually accumulates. For these reasons, when evoked changes in $[K^+]_o$ were related to changes in the final field potential of a train (Fig. 1H), the maximum value of $[K^+]_o$ was utilized even though it occurred slightly after the final field potential. Quantitatively similar results were obtained by testing the field while changing the steady state $[K^+]_o$ by altering the superfusion solution (Fig. 2, A and B).
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Instrumental Control of Cardioacceleration Induced by Central Electrical Stimulation

Abstract. *Each of four monkeys* (Macaca mulatta) *was operantly conditioned to slow and to speed heart rate through a shock-avoidance procedure. During these sessions, electrical brain stimulation that produced tachycardia and pressor responses was delivered on alternate, 64-second segments to one of several brain regions. All animals were able to attenuate the increases in heart rate produced by brain stimulation during the slowing sessions when posterior hypothalamic and striatal regions were stimulated but not when anterior hypothalamic or subthalamic areas were stimulated. During speeding or control sessions during which heart rate was monitored, brain stimulation continued to increase heart rate.*

A number of experiments have examined the effect of electrical stimulation of the brain (ESB) on cardiovascular changes (*1*). With few exceptions [for example, (*2*)] nonhuman primates have been anesthetized and unable to interact with the environment (*3*). Therefore, there was no chance to observe the possible relationships among ESB, cardiovascular changes, and behavior. Our research was designed to investigate these relationships and to delineate the neural structures involved in cardiovascular control. We trained four monkeys (*Macaca mulatta*) to raise and to lower heart rate to avoid an electric shock to the tail (*4*); we then determined whether they could modulate cardiovascular function while receiving ESB, which produced tachycardia and pressor responses.

Sessions consisted of a 256-second baseline phase plus a 1024-second phase of either conditioning or heart rate monitoring (control) (*4*). The blood pressure signal was detected by a pressure transducer (P23 DB Statham) attached to a catheter permanently inserted in the abdominal aorta. The signal was controlled by computer (Raytheon 704), which also controlled experiments and recorded heart rate and systolic and diastolic blood pressure. [We report derived mean pressure—the sum of systolic pressure + 2 (diastolic pressure) divided by 3.] Animals were signaled to slow heart rate by a red cue light or to speed it by a green

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cue light. Correct performance was signaled by a yellow light, and incorrect performance was punished by a 10-mA, 0.45-second shock to the tail delivered on an 8-second fixed-interval schedule. Monitoring was unsignaled and unreinforced. The monkeys were housed in Plexiglas restraining chairs in standard primate booths for the duration of the experiments.

After training was completed, an acrylic platform containing holes drilled to specified anterior-posterior and lateral coordinates (*5*) corresponding to one of four brain regions (*6*) was stereotaxically mounted on the monkey's skull and cemented in place under aseptic conditions. One week later the animal was intramuscularly injected with 0.1 mg of phencyclidine per kilogram of body weight; the skull was drilled through the holes in the platform. Bipolar electrodes were lowered into the brain 1 mm at a time until an active site was found (*7*).

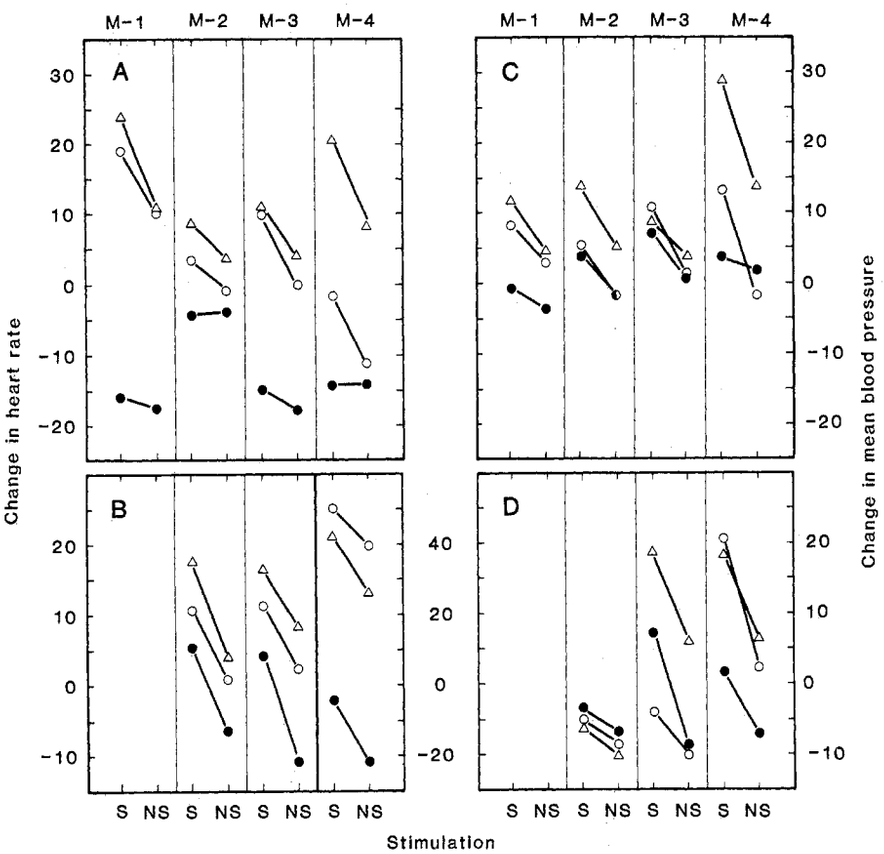

Fig. 1. Changes in heart rate (A and B) and mean blood pressure (C and D) during speeded (triangles), control (open circles), and slowed (closed circles) sessions after (A and C) posterior hypothalamic (*8*) and (B and D) anterior hypothalamic (*8*) stimulation (*S*) or nonstimulation (*NS*).

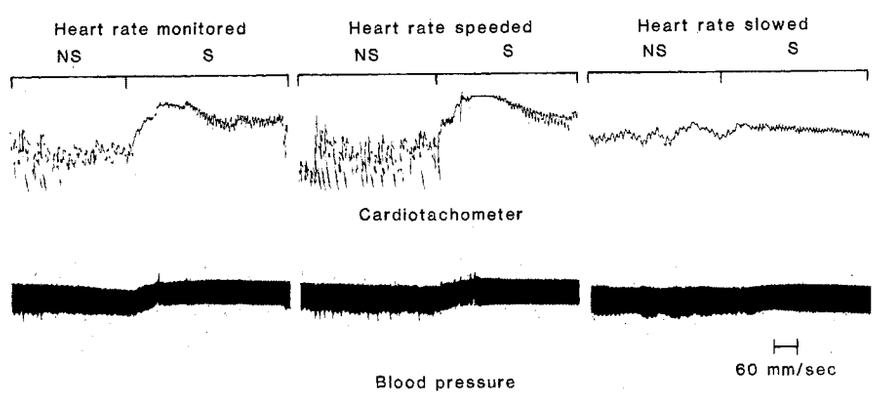

Fig. 2. Change in heart rate and change in mean blood pressure during posterior hypothalamic stimulation (*S*) and nonstimulation (*NS*) segments. The ESB was in effect for all portions of Fig. 2 labeled S.

Cardiovascular changes were examined in the fully awake animal during experimental and control sessions in which ESB was delivered to one of the sites throughout alternate 64-second segments. Sessions were carried out daily in sets of three—one control, one slow, and one speed session per set on a particular brain area. Two brain areas were examined each day on alternate days. Sessions within a set were counterbalanced to minimize any chance of any decrease in sensitivity to ESB as a result of repeated presentations.

At the completion of the sessions, lesions were produced at the electrode sites with a radio frequency lesion generator (50 mA for 60 seconds). The animal was killed and perfused with 20 percent Formalin in saline. The brains were embedded in paraffin, sectioned to 5 μm, and stained in hematoxylin and counterstained in eosin (*8*).

Data were analyzed by subtracting each stimulation (S) and nonstimulation (NS) score on each segment from the baseline for the session to produce difference scores for heart rate and blood pressure. Two (S and NS) by three (control, speed, and slow) analyses of variance were carried out for each site in each monkey and followed by Duncan's tests (*9*) where significant statistical interactions were found.

Each animal was able to attenuate or even abolish the effects of the ESB on HR in the striatum or posterior hypothalamus during slowing, but was not able to attenuate the ESB effects in the anterior hypothalamus or subthalamic nucleus (Fig. 1, A and B). Significant interactions between stimulation condition and session type were found for each animal [M-1: $F(2, 92) = 15.26$, $P < .005$; M-2: $F(2, 137) = 11.08$, $P < .01$; M-3: $F(2, 146) = 5.29$, $P < .01$; M-4: $F(2, 99) = 23.14$, $P < .001$]. Duncan's tests showed that ESB did not have differential effects between speed and control conditions; however, both of these conditions differed from the slow condition ($P < .05$) (Fig. 2). Figure 1 shows that there was no selective attenuation of ESB effects on heart rate during slow sessions when ESB was delivered to the anterior hypothalamus (Fig. 1, B and D). There were no significant condition-by-session interactions for animals 2 and 4 (M-1 died before the completion of the experiments in the anterior hypothalamus and subthalamic nucleus). In M-3 this interaction was significant [$F(2, 89) = 4.09$, $P < .025$]; the difference between stimulation and nonstimulation was greater under the slow condition than under fast and control conditions.

The changes in blood pressure after ESB of the posterior hypothalamus did not parallel those seen in heart rate (Fig. 1, C and D). Analyses of variance and subsequent Duncan's tests indicated some attenuation of blood pressure during slow sessions in animals M-1 and M-4. Stimulation of the anterior hypothalamus produced changes in blood pressure similar to those seen in heart rate (Fig. 1), and there was no attenuation of either during slow sessions. Analyses of variance and Duncan's tests indicated that the differences between stimulation and nonstimulation were greater in slow conditions than in (i) speed and control conditions for M-2, (ii) control for M-3, and (iii) speed for M-4. Thus no attenuation of blood pressure occurred during slow conditions.

The results of striatal stimulation were similar to those seen after posterior hypothalamic stimulation. This was true for blood pressure as well as heart rate. Thus, the change in heart rate was attenuated during slow but not during speeded or control sessions, and blood pressure did not change across conditions. The effect on heart rate of stimulating the subthalamic nucleus was similar to that of stimulating the anterior hypothalamus. Strong effects were seen on heart rate, and the animals were not able to attenuate these effects during slowing.

The data show that the ability of an animal to significantly attenuate its cardiovascular responses to stimulation of sites in the striatum or posterior hypothalamus was selective to the modality on which a negative reinforcement contingency was placed—heart rate but not blood pressure. The attenuation was not the result of threshold changes after repeated stimulation, since (i) counterbalancing controlled for order effects, and (ii) analysis of variance of changes in heart rate during the first stimulus segment in the control sessions with the last stimulus segment for each brain area showed neither segment differences nor segment differences as function of the brain area in any animal.

Smith *et al.* (*10*) have shown that lesions in areas in the baboon corresponding to our anterior hypothalamic sites create deficits in an animal's ability to generate a conditioned emotional response (a change in heart rate or blood pressure in response to a stimulus paired with an electric shock). It is possible in our experiments that affective responses elicited by stimulation in these areas overrode the reinforcement strength of shock avoidance. The striatal or posterior hypothalamic stimulations were overridden because they are associated with cardiovascular effects only. Cardiovascular efferents to the posterior hypothalamus may involve only outputs from the anterior hypothalamus (*10, 11*). The subthalamic nucleus is a complex area that mediates motor as well as cardiovascular functions (*12*) and that may receive selective input from the anterior hypothalamus and striatum. One conclusion is clear. Central nervous and autonomic interactions in the unanesthetized animal vary and can be modified by training. Differences in response to ESB suggest that a variety of mechanisms operate within the brain to mediate plasticity such as that seen here. The heart rate responses, even in the face of ESB, can differ as a function of environmental contingencies and the demands made on the animal.

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8. Histologies were carried out at the experimental pathology laboratory, Herndon, Virginia, according to their standard protocol. Striatal penetrations included the caudate nucleus, the lenticular nucleus, and the lateral portion of the area tegmentum. The anterior hypothalamus included the dorsomedial anterior hypothalamus and the anterior lateral hypothalamus. The posterior hypothalamus included the mammillary bodies and the posterior dorsomedial hypothalamus. The subthalamic nucleus included the subthalamic nucleus and the parafascicular nucleus.
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Pigeons with a Deficient Sun Compass Use the Magnetic Compass

Abstract. *Homing pigeons that had never seen the sun before noon could not use the sun compass in the morning; nevertheless they were homeward oriented. When such birds carried magnets, however, they were disoriented, suggesting they were using a magnetic compass. These findings indicate that the magnetic compass is available to pigeons whether or not the sun compass has been established and that the magnetic compass is apparently the first source of compass information.*

Experiments with birds whose internal clock was phase-shifted demonstrate that the sun compass is used by homing pigeons whenever the sun is visible (*1*). This compass system is learned rather than innate (*2*), and we therefore studied that learning process. Tests indicated that knowledge only of the descending part of the sun's arc was not sufficient to establish the sun compass for the entire day. Young pigeons that had observed the sun only in the afternoon and that were tested in their subjective morning did not react to the shifting of their internal clock but departed homeward oriented (*3*).

These findings led to a question about how such birds orient if they are not able to use the sun compass. One possibility is that they rely on the earth's magnetic field for compass information (*4, 5*). If this were so, then attaching magnets to them might cause disorientation. A group of experimental pigeons was prevented from seeing the sun in the morning; they grew up in a light-tight room in a natural photoperiod and were allowed to enter their aviary or fly around their loft only in the afternoon after the culmination of the sun. Control pigeons grew up in an identical room, but these birds had access to their aviary all day and were released for exercise flights at various times of day. Both groups had a series of training flights in which flocks were released, up to 30 km in the cardi-

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