parable to any corollary discharges which may subserve normal visual perception. During the period of such involuntary eye movements, we are aware that the perceived world is never stabilized, suggesting that a corollary mechanism would no longer be operating effectively. In summary, it would appear that in normal conditions of active eye movements, the LGN of the cat does not participate in oculomotor-visual integration. The saccadic suppression may simply reflect the decreased excitability of LGN cells that would be caused by impulses conveyed by axons of the retinal ganglion cells.

HIROHARU NODA
W. ROSS ADEY
Department of Anatomy and
Brain Research Institute,
University of California,
Los Angeles 90024

References and Notes

1. R. W. Ditchburn, *Optica Acta* **1**, 171 (1955); F. C. Volkmann, *J. Opt. Soc. Am.* **52**, 571 (1962); P. L. Latour, *Vision Res.* **2**, 261 (1962); B. L. Zuber and L. Stark, *Exp. Neurol.* **16**, 65 (1966); G. W. Beeler, *Vision Res.* **7**, 769 (1967); A. Starr, R. Angel, H. Yeetes, *ibid.* **9**, 195 (1969); F. C. Volkmann, A. M. L. Schick, L. A. Riggs, *J. Opt. Soc. Am.* **58**, 562 (1968); L Mitrani, N. Yakinoff, St. Mateef, *Vision Res.* **10**, 411 (1970).
2. E. G. Gross, H. G. Vaughan, Jr., E. Valenstein, *Electroencephalogr. Clin. Neurophysiol.* **22**, 204 (1967); J. A. Michael and L. Stark, *Exp. Neurol.* **17**, 233 (1967); R. Chase and R. Kalil, *Vision Res.* **12**, 215 (1972).
3. H. Collewijn, *Vision Res.* **9**, 803 (1969); J. A. Michael and L. Stark, *Electroencephalogr. Clin. Neurophysiol.* **21**, 478 (1966).
4. E. Bizzi, *J. Neurophysiol.* **29**, 861 (1966); H. Kawamura and P. L. Marchiafava, *Arch. Ital. Biol.* **106**, 141 (1968); L. J. Malcolm, I. S. C. Bruce, W. Burke, *Exp. Brain Res.* **10**, 283 (1970); T. Ogawa, *Brain Res.* **38**, 211 (1972).
5. R. W. Sperry, *J. Comp. Physiol. Psychol.* **43**, 482 (1950); E. von Holst and H. Mittelstaedt, *Naturwissenschaften* **37**, 464 (1950); H. L. Teuber, in *Handbook of Physiology*, section 1, *Neurophysiology*, J. Field, H. W. Magoun, V. E. Hall, Eds. (Williams & Wilkins, Baltimore, 1960), vol. 3, pp. 1595–1668.
6. D. M. MacKay, *Nature* **225**, 90 (1970).
7. H. Noda, R. B. Freeman, Jr., B. Gies, O. D. Creutzfeldt, *Exp. Brain Res.* **12**, 389 (1971).
8. Similar to those used for the monkey by P. H. Schiller and M. Stryker, *J. Neurophysiol.* **35**, 915 (1972).
9. H. Noda and W. R. Adey, in preparation.
10. B. G. Cleland, M. W. Dubin, W. R. Levick, *J. Physiol. (Lond.)* **217**, 473 (1971); K.-P. Hoffmann, J. Stone, S. M. Sherman, *J. Neurophysiol.* **35**, 518 (1972).
11. W. Singer and O. D. Creutzfeldt, *Exp. Brain Res.* **10**, 311 (1970); W. Singer, E. Pöppel, O. D. Creutzfeldt, *ibid.* **14**, 210 (1972); K.-P. Hoffman, J. Stone, S. M. Sherman, *J. Neurophysiol.* **35**, 518 (1972).
12. E. Bizzi, *J. Neurophysiol.* **29**, 861 (1963); H. Kawamura and P. L. Marchiafava, *Arch. Ital. Biol.* **106**, 141 (1968); M. Jeannerod and P. T. S. Putkonen, *Exp. Brain Res.* **13**, 533 (1971); R. Corazza and C. Lombroso, *Electroencephalogr. Clin. Neurophysiol.* **19**, 323 (1970).
13. E. Bizzi and D. C. Brooks, *Arch. Ital. Biol.* **101**, 666 (1963).
14. E. Bizzi, *J. Neurophysiol.* **29**, 1087 (1966); H. Sakakura, *Jap. J. Physiol.* **18**, 23 (1968).
15. U. Büttner and A. F. Fuchs, *J. Neurophysiol.* **34**, 127 (1973).
16. Supported in part by U.S. Air Force contract AFOSRF 44620-70-C-0017, NIH grant 1 RO1 EY01051-01A1, and NASA grant 05-007-195.

9 October 1973

Torpor in an Andean Hummingbird: Its Ecological Significance

Abstract. *Field studies on an Andean hummingbird showed that nocturnal torpor occurs more frequently and lasts longer in the winter. Energy depletion does not seem to cause this yearly torpor cycle, and a photoperiodically controlled rhythm that enables the birds to automatically conserve energy in early evening for possible metabolic expenditures required later in the winter night is suggested.*

Daily torpor, in which the body temperature is lowered to near ambient, is known to occur in several groups of small endotherms, including some birds, bats, and rodents. Many workers have conducted laboratory studies on the physiology of torpor (*1, 2*), and some have calculated its savings in energy (*3*). However, because of the difficulty of studying organisms in their roosts or burrows, few field data are available that have been systematically gathered on torpid animals on a yearly basis. Nocturnal torpor may be employed occasionally in the field by energy-stressed incubating female hummingbirds, indicating that torpor may be used by normal, healthy individuals in certain emergency situations (*4*). However, I know of no study on birds that reveals the importance of torpor under natural conditions to males and nonbreeding females and to the population as a whole during the nonbreeding season. The need for this kind of information has been noted (*2, 4, 5*). This report shows that torpor occurs naturally in the field in both sexes of the Andean hillstar hummingbird, *Oreotrochilus estella estella,* that often its occurrence is not related to any detectable emergency conditions, and that its frequency and duration are seasonal.

In 1968 and 1970, I conducted field studies on several populations of *O. estella* in the southern Peruvian Andes between 3800 and 4300 m elevations. This species roosts in caves at night, clinging to surface irregularities in the walls and ceilings with their claws (*6*). Individuals are thus easily located and studied in their roosts with little disturbance. I located individuals by flashlight at night, determined their metabolic condition (nontorpid, entering torpor, torpid, and arousing from torpor) by observing breathing rates, erection of plumage, and general alertness, and took ambient and body temperatures (T_a and T_b, respectively) with a Schultheis quick-recording thermometer whenever feasible. When the difference between ambient and body temperatures was likely to be large, the bulb of the thermometer was first warmed in the hand before insertion in the bird. Unless a bird was just entering torpor, its T_b was usually only 0° to 2°C higher than T_a except when T_a was lower than 7°C (Fig. 1). I also noted the time of night, the sex (by plumage), and the identification number of the individual. I checked on each individual one to three times a night and recorded "torpor" or "nontorpor" for each night. During both winter and summer, evening observations were made between ½ hour after darkness and 23:00, and early morning observations were made between 02:00 and 05:00 (first light occurred at 05:15 to 05:30). Observations were made during the rainy summer months of September to March and the dry winter months of June to August. No individual was observed in both seasons. Within a season each individual was observed one to eight different nights, except one summer individual that was observed 15 nights and one winter individual that was observed 12 nights. Sample sizes and results are given in Fig. 2A. The summer individual observed 15 nights was torpid twice and nontorpid 13 nights; the winter individual observed 12 nights was torpid all 12 nights.

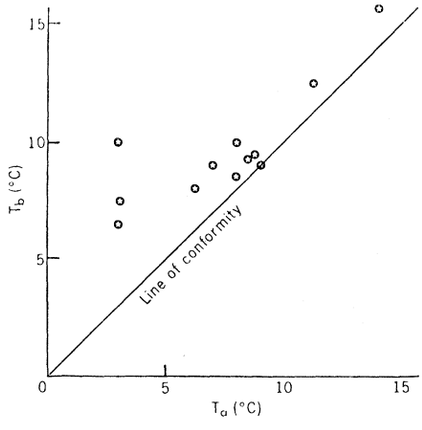

Fig. 1. Body temperatures of torpid *O. estella* at various ambient temperatures; T_a, ambient temperature; T_b, body temperature.

In summer and winter there were no differences between the sexes in the

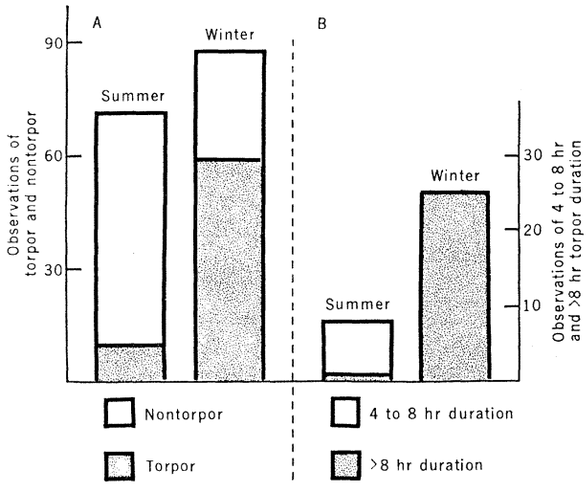

Fig. 2. Incidence (A) and duration (B) of torpor in two seasons in *O. estella*. Summer observations were made on 34 individuals: 13 males, 19 females, and 2 undetermined; of 92 observations, 72 were on nonnesting birds, and these are graphed at left. In the winter, 88 observations were made on 41 individuals: 17 males, 19 females, and 5 undetermined. Winter and summer populations differ significantly both in number of torpor observations and in duration of torpor bouts (chi-square with Yates's correction factor, $P < .001$).

incidence of torpor, with data on nesting females being omitted. Nesting females (eight individuals, 20 observations) were not seen torpid. Although Calder (*4*) discovered by continuous recording of nest temperatures that nesting females may enter nocturnal torpor, this was only under conditions of apparent food deprivation, and my observations suggest that nesting females resist entering torpor more than do the other members of the population (*7*).

The durations of torpor bouts in the field were estimated for both summer and winter. I observed 14 "natural" arousals from torpor in birds left undisturbed in their roosts: 12 began between 03:00 and 04:30. These data show that the time of the onset of arousal is relatively constant, and support suggestions (*4, 5*) that arousal is timed for a first-light search for food. By assuming that arousal began at 03:45, I estimated the durations of the 8 summer and 25 winter bouts of torpor for which I knew the time of entry. The estimates were accurate to ± 1 hour. Average durations of summer and winter bouts were about 7 and 10 hours, respectively. In Fig. 2B the durations of torpor bouts are assigned to two interval categories, 4 to 8 hours and more than 8 hours, and the number of bouts in each category was compared in summer and winter birds. Winter torpor bouts lasted significantly longer than did summer bouts. This is because the time of entry into torpor was variable and often late in the evening in summer birds, whereas in winter birds entry began soon after the birds entered their roosts. Also, winter nights were 15 to 90 minutes longer than summer nights: this usually accounted for much less than half the increase in winter torpor durations.

Some hummingbirds studied in the laboratory metabolically regulate their body temperatures while remaining in torpor, when T_a falls below some critical value (*8, 9*). I observed regulation in torpid *O. estella* under natural conditions—this is the first such field observation. In the field I determined the occurrence of metabolic regulation in two ways. First, body temperatures measured in the field were never lower than 6.5°C, except for one individual with T_b at 5.0°C, even though the temperatures in the roost were sometimes as low as 3.0°C (Fig. 1). Second, at ambient temperatures around 7°C torpid birds alternated half-minute periods of slow breathing with 2- to 10-minute periods of undetectable breathing; but below 7°C these periods of apnea ceased—this onset of a steady breathing indicates increased metabolic output and heat production. Time courses of laboratory records of torpid metabolism and body temperature of birds transported to California showed that T_b regulation occurred at ambient temperatures below 6.6°C, thereby supporting the field information (*10*). Other species studied in the laboratory begin their T_b regulation at higher temperatures, which correlate with the usual minimum temperature to which they are exposed in the field (*9*). In this study ambient temperatures lower than 6.5°C were recorded 11 times in hummingbird roosts. Thus, although T_b regulation in torpid *O. estella* begins at a lower ambient temperature than in any hummingbird species previously studied, thereby correlating well with its harsh ecological situation, it is puzzling that the limit of nonregulated torpor is not even lower.

These extensive field observations have established that torpor occurs normally in this species in both seasons and that its incidence and duration are greater in the winter. Why is torpor used so much more extensively by the birds in the winter than in the summer? What are the proximate cues for and the ultimate causes of torpor? The function of torpor seems to be the conservation of energy (*1*). Thus, if nocturnal ambient temperatures are lower in the winter, or if food is scarcer or less nutritious, or if daily energetic output is greater, one would then expect torpor to occur more extensively in the winter. Ambient temperatures in the roosts were recorded at each bout of torpor and had nearly identical ranges of 3.5° to 13°C in the summer, and 3° to 14°C in the winter. Although winter nocturnal temperatures at these elevations and this latitude are usually considerably below 0°C, the winter of this study was mild because of unusual cloud cover. Thus, temperature seems to be eliminated as a proximate cue for the increased usage of torpor that I saw in the winter, although it may act as an ultimate cause, that is, as an evolutionary selection pressure.

Low food availability also can be eliminated as a proximate cue for torpor. One of the winter populations of *O. estella* studied occurred in an area with introduced blooming *Eucalyptus* sp. trees. The blossoms were fed upon heavily, and four hummingbirds set up permanent feeding territories in the trees—a behavior not seen in wild populations, whose only food sources were *Chuquiragua spinosa* (Compositae) and insects (*10*). While the *Eucalyptus* blossoms produced copious nectar, *Chuquiragua* was poor in nectar and apparently provided only pollen for the hummingbirds. Yet the incidence and duration of torpor were the same in the *Eucalyptus* hummingbird population as in the *Chuquiragua* hummingbird populations, and much greater than in the summer populations that fed from several native flower blossoms that produce nectar (*10*).

Last, I compiled a time budget on two summer territorial individuals and two winter territorial individuals (in *Eucalyptus*). The energy budget calculated from the time budget and from metabolic data gathered in the laboratory showed that the summer birds consumed 30 to 40 kcal/day while the

winter birds consumed less than 20 kcal/day (*10*). Thus, activity level and energy output also cannot be the proximate cause for increased torpor in the winter.

The duration of a bout of torpor may be determined by the energy state of the bird in an interaction between "biological clock" and "biological fuel gauge" (*2, 4*). This is probably true for animals in certain emergency situations. However, the fact that in my study the *Eucalyptus* population of birds used torpor much more extensively than did summer birds regardless of unusually mild ambient temperatures and abundant food suggests that energy depletion is not used as a cue for torpor in the winter. Instead, an innate circannian pattern of torpor is suggested for this species. In the study areas there is about a 1.5-hour difference in daylight between the two seasons, which is adequate to serve as a photoperiodic cue for a circannian rhythm (*11*). The selection pressure for the evolution of a circannian torpor rhythm has been suggested above: the low ambient temperatures that usually occur in the winter. A circannian cycle of torpor is of special value to an organism living in a region where nocturnal temperatures are seasonally low but where the daytime climate gives no indication of the low nocturnal temperatures to come. Such a climate exists in the Peruvian Andes, where winter days are sunny and mild. In this study, when an individual *O. estella* entered its roost on a winter evening, the temperature was usually no lower than that in a typical summer roost. Yet in most winters the roost temperature probably falls to near freezing by midnight. If the bird enters torpor immediately upon roosting, it will have saved energy that can be spent later that night if ambient temperatures fall low enough to force T_b regulation. Thus, the peculiar climate of the tropical Andes has apparently selected for both a relatively low temperature limit to nonregulated torpor and an innate annual cycle of torpor in *O. estella*. The genetic responses of this species to this environmental selection pressure have enabled it to live year-round on the energetically demanding slopes and plains of some of the highest mountains in the world despite the fact that it is the smallest member of the avifauna of that region.

F. LYNN CARPENTER*
Museum of Vertebrate Zoology, University of California, Berkeley

References and Notes

1. J. W. Hudson, in *Comparative Physiology of Thermoregulation*, G. C. Whittow, Ed. (Academic Press, New York, 1973), vol. 3, pp. 97–165.
2. W. R. Dawson and J. W. Hudson, in *ibid.*, vol. 1, pp. 287–297.
3. V. A. Tucker, *J. Cell. Comp. Physiol.* **65**, 405 (1965); O. P. Pearson, *Condor* **56**, 317 (1954).
4. W. A. Calder and J. Booser, *Science* **180**, 751 (1973).
5. R. C. Lasiewski, *Physiol. Zool.* **36**, 122 (1963).
6. O. P. Pearson, *Condor* **55**, 17 (19[illegible] N. R. French and R. W. Hodges, *ibid.* **61**, 223 (1959).
T. R. Howell and W. R. Dawson, *ibid.* **56**, 93 (195[illegible] W. A. Calder, *ibid.* **73**, 314 (1971).
8. F. R. Hainsworth and L. L. Wolf, *Science* **168**, 368 (1970).
9. L. L. Wolf and F. R. Hainsworth, *Comp. Biochem. Physiol. A Comp. Physiol.* **41**, 167 (1972).
10. F. L. Carpenter, thesis, University of California, Berkeley (1972).
11. A. J. Marshall, in *Biology and Comparative Physiology of Birds*, A. J. Marshall, Ed. (Academic Press, New York, 1961), vol. 2, pp. 317–321.
12. I thank O. P. Pearson for guidance in this project. The Museum of Vertebrate Zoology and a Chapman grant from the American Museum of Natural History provided funding, equipment, and space.
* Present address: Department of Population and Environmental Biology, University of California, Irvine 92664.

24 September 1973; revised 3 November 1973 ■

Regeneration Electrode Units: Implants for Recording from Single Peripheral Nerve Fibers in Freely Moving Animals

Abstract. *Implantable electrode assemblies that become penetrated by regenerating axons were used to record signals from single sensory and motor nerve fibers associated with leg movement in unrestrained amphibians* (Xenopus laevis). *Such neuroimplants may provide a means for establishing the roles of various muscle afferents and efferents in posture and locomotion, and have potential clinical applications.*

Little direct information is available on the roles in locomotion of the individual sensory and motor nerve fibers supplying mammalian muscles. This is partly due to the difficulty of recording simultaneously from several single peripheral axons during free movement —a procedure requiring implantable arrays of electrodes that stay in place despite movement, while picking up signals from identified sensory and motor neurons. Regeneration electrode units were designed to satisfy these requirements.

We thought that it might be possible to separate small bundles of axons, for either recording or stimulation, by encouraging severed nerve fibers to regenerate through an implant perforated by cylindrical channels (*1*) having built-in electrodes (*2*). Ideally, such channels would be narrow enough to allow selective recording from single units while not greatly impeding regeneration, and would be sufficiently long to permit action potential currents to develop a recordable voltage between a central electrode and the tissue fluid; the peak-to-peak amplitude of the triphasic spike expected from one of the fibers in a channel of length L and diameter is D is $k(L/D)^2$, for small L and D (*3*).

Channeled implants were fabricated by etching silver strands 25 μm in diameter out of epoxy wafers with ferric nitrate, by boring holes through photoengraved electrode patterns, or by drilling through wires embedded in epoxy slabs with 100-μm bits (Sphinx microdrills; Swiss Instruments, Toronto).

The latter technique was simplest and gave satisfactory results. One end of a ribbon formed from ten Teflon-coated silver wires with core diameters of 77 μm (Medwire Corp., Mount Vernon, New York) was dipped in Epon 812 (Ladd Research Industries, Burlington, Vermont). The bulb of hardened epoxy was milled down to two flat faces, parallel to the ribbon of wires and 0.7 mm apart. A channel perpendicular to the flat faces was drilled through each wire (Fig. 1a), then cleaned ultrasonically. Thus, each channel was left with its own central wire electrode. The impedances of these electrodes were determined, and their noise level was predicted (*4*). Units with several leads having impedances less than 20 kilohms or more than 300 kilohms were discarded.

Figure 1a shows how regeneration electrode units were implanted in the thighs of *Xenopus laevis*, anesthetized by immersion in tricaine methanesulfonate (0.2 g/liter) (Fraser, Vancouver, British Columbia). A period of 12 to 25 weeks was allowed for regeneration.

The first evidence that bundles of axons had penetrated the implants was provided by light microscopy. This was corroborated by electron micrographs of channel cross sections (Fig. 1b), which confirmed that axons grow into channels as narrow as 25 μm. Of 17