

to 15 kg) (25) and females are often hornless in the two smallest species (< 8 kg: *C. maxwelli* and *C. monticola*) (26). Similarly *Gazella* contains two large species (> 40 kg) where females' horns are well developed, but in the smaller *G. thomsoni* (18.4 kg), females' horns are often vestigial (27). In the monospecific *Oreotragus* (12.9 kg), females have horns in only 1 of 11 subspecies (26).

Being straighter and thinner, the horns of females are relatively more specialized as stabbing weapons than are males' horns and as such may function more exclusively as defensive weapons against predators. The correlation between body weight and the presence of horns in females may be a consequence of the relation between body weight and antipredator behavior in antelopes: smaller species rely on crypsis or flight while large species often show direct defense against predators (6). Defense is more effective in larger species because the larger species are bigger than most predators and, in particular, females of large species are very much larger than predators of their young (11, 13). Thus, horns are most likely to be of value to females in species of larger body weight. The utility of horns as antipredator weapons has received relatively little attention, probably because of the emphasis on males in previous studies and because observations of effective defense are uncommon (1). While the antipredator functions of horns may only be secondary in males, effective defense has been observed by both sexes in many of the better studied species (9, 11, 12, 13). Horns are likely to confer a selective advantage even if they prevent predation only a few times over an average lifespan.

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16. Adult specimens of both sexes of nearly all 72 species of the 25 African genera were available either in the British Museum of Natural History, Field Museum of Natural History, National Museum of Kenya, or the Oxford University Museum. Deformed horns were excluded.
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19. Males of genera where females have horns do not differ from males of genera with hornless females either for basal area (comparison of slope:  $F = 0.483$ , d.f. = 1, 21, not significant; elevation:  $F = 0.046$ , d.f. = 1, 22, not significant) or reach (slope:  $F = 2.159$ , not significant; elevation:  $F = 0.288$ , not significant). If females are compared only to the males of their respective genera, the difference in elevation of basal area remains the only difference between males and females ( $F = 9.868$ , d.f. = 1, 23,  $P < 0.01$ ).
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22. Body weights of females are from (18). Group size and habitat (forest, intermediate, or open) are from (6). Sexual dimorphism is male weight divided by female weight. Each variable was considered separately and then in a multivariate analysis with the use of the logistic regression program available from SAS [F. Harrell, in *SAS Supplemental Library User's Guide*, P. S. Reinhardt, Ed. (SAS Institute, Cary, N.C., 1980), p. 83]. Only the log of body weight and the log of group size were significantly correlated with the presence of horns when entered separately ( $\chi^2 = 15.19$ ,  $P < 0.0001$ , and  $\chi^2 = 9.85$ ,  $P < 0.002$ ). Large females and those in large groups are more likely to have horns. However, weight and group size are correlated ( $r = .79$ ,  $n = 25$ ) (also see 6) and when all variables are entered in a backward elimination model, body weight is the only variable significant to  $P < 0.05$ .
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19 February 1982; revised 31 January 1983

## Corollary Discharge Provides Accurate Eye Position Information to the Oculomotor System

**Abstract.** *The saccadic system accurately compensates for perturbations of eye position produced by microstimulation of the superior colliculus. This requires that information about the stimulation-induced change in eye position be provided by an extraretinal source—either proprioceptive endings in extraocular muscles or a centrally generated corollary discharge. It is shown that compensation remains intact after elimination of extraocular muscle proprioception, demonstrating that corollary discharge provides accurate eye position information.*

Precise information about the position of the eyes in the orbit is required for localization of visual targets (1) and is an essential component of current models of the oculomotor system (2). The question of whether eye position signals originate from a central copy of the oculomotor command or arise peripherally from extraocular muscle proprioceptors is a fundamental issue in oculomotor physiology.

A century ago Helmholtz concluded that knowledge of eye position was derived from a "measure of the effort of will required to move the eyes" (3). Such

an internal signal representing an intended change in eye position was referred to as corollary discharge by Sperry (4). Recently, physiological studies have shown that a number of brain areas contain neurons which have tonic firing rates correlated with eye position (5). These neuronal populations are functionally close to the final oculomotor output and project to other centers involved in the control of eye movements. Such physiological and anatomical properties suggest that these neurons generate a corollary discharge which could be used to represent eye position. Unlike most

other structures under motor control, the eyes are not subject to changing external loads, so a given oculomotor output always produces the same movement. Because of this tight coupling of motor output with the resulting movement, a corollary of the motor command would indeed accurately reflect eye position.

On the other hand, mammalian extraocular muscles are endowed with well-developed proprioceptors (6). Noting the presence of these receptors, Sherrington (7) proposed that extraocular muscle proprioception was the source of eye position information. Recent physiological studies have demonstrated that afferent fibers from extraocular muscles carry signals corresponding to muscle length and tension and are distributed to almost every known visuomotor center (8).

In this study we tested the hypothesis that corollary discharge alone provides accurate eye position information. After elimination of extraocular muscle proprioception (9), we used a saccadic tracking task (10) which required rapid and accurate feedback about eye position. A monkey looked at a lighted fixation target in an otherwise dark room. As this target was extinguished a saccadic target was presented for 50 msec at a selected location in the visual field. On randomly selected trials, after the sac-

cadic target was extinguished, but before the onset of the saccade to it, eye position was changed in a direction away from the target by microstimulation of the superior colliculus. After a brief pause at the new eye position, the animal made a compensatory saccade to the original saccadic target location. The compensatory saccade was different from the saccade that would have been generated based solely upon a retinal error signal (distance and direction of the target retinal image from the fovea). For compensation to occur, the saccadic system must be informed of the stimulation-induced change in eye position by an extraretinal source. If extraocular muscle proprioception is an essential source of eye position information, without it the oculomotor system would not be capable of generating an accurate compensatory saccade. On the other hand, if corollary discharge is used as an accurate eye position signal, the animals would continue to compensate in the absence of extraocular muscle proprioception.

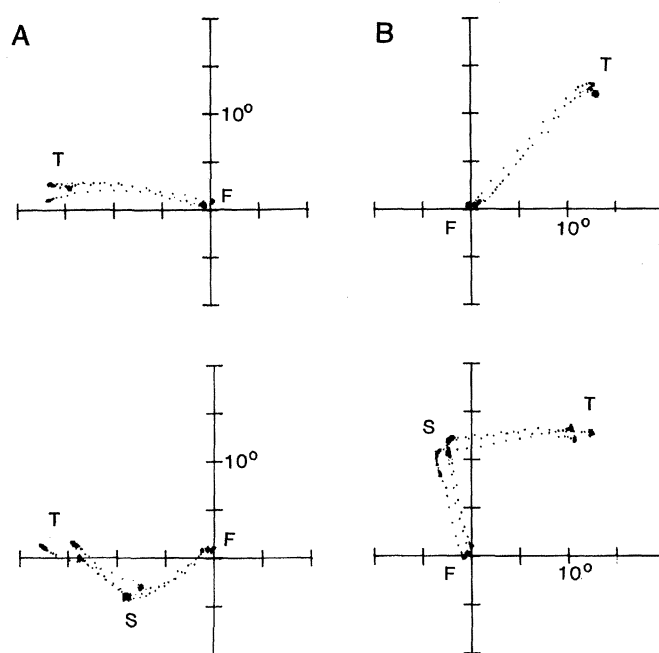
Two rhesus monkeys were used in the experiment. Each was implanted with scleral search coils (11) and trained to make saccades to visual targets (12). Skull cylinders were implanted over appropriately placed small craniectomies for microelectrode stimulation of the su-

perior colliculus. Display of visual targets, monitoring of eye position, delivery of reward, and stimulation of the colliculus were computer-controlled (13). Prior to surgery, each animal was tested in the stimulation-compensation task by using systematically varied target locations. Then, with a subtemporal approach, the ophthalmic nerves were transected bilaterally at their junction with the trigeminal ganglion, eliminating extraocular muscle proprioception. Complete transection was ascertained postoperatively by loss of the corneal blink reflex, then confirmed postmortem by the absence of labeling of trigeminal ganglion sensory neurons after injection of horseradish peroxidase into the extraocular muscles. Intraoperative damage to the oculomotor nerves was ruled out by normal action of the extraocular muscles postoperatively (14). After surgery each monkey was tested in the stimulation-compensation task.

Preoperatively, compensation was tested for three collicular stimulation sites using two to five target locations per site. Each compensatory saccade exhibited a normal magnitude-velocity relation and followed a 20- to 60-msec fixation at the end of the stimulation-induced saccade. Compensation was accurate, showing an average error of 4.5 degrees, ranging from 1.6 to 6.8 degrees, varying with target location and stimulation vector. This performance is comparable to that described in an earlier, more extensive study of stimulation-compensation behavior (13). After elimination of extraocular muscle proprioception, compensation remained intact (Fig. 1). Four collicular sites were stimulated using three to six target locations per site. As was the case preoperatively, each compensatory saccade had a normal waveform and followed a brief fixation after the stimulation-produced saccade. Accuracy of compensation was not significantly different, showing an average error of 4.0 degrees, ranging from 2.1 to 7.3 degrees.

Postoperative compensation is possible only if information about the stimulation-induced eye movement is still available. Three possible sources of this eye position signal are the retina, extraocular muscle proprioception, and a centrally generated corollary discharge. In this study the dark experimental environment and the absence of the saccadic target during and after the stimulation-induced saccade prevented retinal feedback about change in eye position. Transection of the ophthalmic nerves eliminated extraocular muscle proprioception. Thus, eye position information

Fig. 1. (A) and (B) represent postoperative trials at two different collicular stimulation sites, performed in the absence of extraocular muscle proprioception and plotted in a plane Cartesian coordinate system. Trials were performed in total darkness, with initial fixation at lighted target *F*. Coincident with the offset of *F*, saccadic target *T* was presented for 50 msec. Saccades *FT*, shown on the upper axes, are three saccades to target location *T* without collicular stimulation. Shown on the lower axes are three stimulation-compensation trials using the same target locations. In these trials, after the offset of target *T* but before saccade *FT* could be initiated, the eyes were driven away from the target to location *S* by collicular stimulation. After a brief pause at *S*, compensatory saccades *ST* were made to the location of the (now absent) target *T*. This compensation could have occurred only if the saccadic system received information about the stimulation-induced change in eye position from *F* to *S*. Since retinal and proprioceptive feedback about these eye movements were not available, a centrally generated corollary discharge must have provided the eye position information. If the saccadic system had not been informed of the stimulation-produced movement from *F* to *S*, saccades with the same vector as *FT* but starting from eye position *S* would have been expected.



must have been provided by a centrally generated corollary discharge. This experiment, while demonstrating that corollary discharge provides accurate eye position information, does not address the possibility that extraocular muscle proprioception may provide information for some oculomotor functions (15).

Although previous investigations have suggested that motor systems distribute corollaries of their output (16), none has unequivocally demonstrated a functional role for these internal signals. This study, however, clearly demonstrates that the oculomotor system generates a corollary discharge which accurately represents the intended motor act and is used in generating further movements.

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21 December 1982; revised 5 May 1983

## Subthreshold Excitatory Activity and Motoneuron Discharge During REM Periods of Active Sleep

**Abstract.** A striking paradox of the rapid eye movement periods of active sleep, which are typically characterized by the exacerbation of somatomotor atonia, is the occurrence of muscle twitches and jerks. The purpose of this study was to examine the specific motoneuron membrane potential processes responsible for these myoclonic patterns of activity. In lumbar motoneurons, examined intracellularly in the cat prepared for long-term study, these processes consisted of recurrent depolarizing membrane potential shifts and spontaneous action potentials that were either full-sized or of partial amplitude. In addition, the invasion of antidromically induced spikes into the soma was often blocked. Hyperpolarizing potentials were evident in the intervals between spontaneous spikes. Hyperpolarization was also observed immediately before depolarization and spike activity, in contrast to the gradual depolarization of the motoneuron membrane potential that always occurred during wakefulness. Thus, during rapid eye movement periods, in conjunction with muscle twitches and jerks, a strong excitatory input is superimposed on a background of inhibitory input. The unique patterns of membrane potential change that arise thus seem to result from the simultaneous coactivation of excitatory and inhibitory processes.

Certain patterns of motor activity seem, from a behavioral perspective, to be based on abnormal motor mechanisms. Specifically, the myoclonic twitches and jerks that predominate during the periods of rapid eye movements (REM's) of active sleep do not resemble normally controlled motor processes, nor do they fulfill any known purpose; they are, however, a constant and prominent feature of this state. We were therefore interested in determining the responsible motoneuron membrane potential processes that give rise to these "normally" occurring patterns of apparently "abnormal" motor activity.

To explore the underlying motoneuron membrane potential changes we recorded intracellularly, during sleep and wakefulness, from antidromically identified lumbar motoneurons in the unanesthetized, undrugged, normally respiring cat. The details of the procedures have been reported elsewhere (1).

Experiments were performed on six adult cats prepared for long-term record-

ing. Glass micropipettes were used to record intracellularly from alpha motoneurons of the lumbar spinal cord. The micropipettes, which were filled with 2M potassium citrate, had tip resistances of 5 to 15 megohms. All recordings from the 47 motoneurons studied met established standards for monitoring intracellular activity (that is, antidromic identification; membrane potential  $\geq 55$  mV; spike height  $\geq 55$  mV; and data from cells recorded for at least 10 minutes and across a minimum of two behavioral states) (1).

We have previously described phasic episodes of postsynaptic inhibition, revealed by the presence of a depression of motoneuron excitability and a decrease in motoneuron input resistance, which occur during REM periods (2). These postsynaptic inhibitory processes are characterized by sequences of hyperpolarizing shifts in the membrane potential which are the result of the summation of discrete inhibitory postsynaptic potentials (3). However, during certain REM