Brain Research, 620 (1993) 310–316

BRES 25778

The wind-elicited escape response of cockroaches (*Periplaneta americana*) is influenced by lesions rostral to the escape circuit

Andrew P. Keegan and Christopher M. Comer

Neuroscience Group, Department of Biological Sciences, University of Illinois at Chicago, Chicago, IL 60680 (USA)

(Accepted 18 May 1993)

Key words: Escape behavior; Insect; Interneuron, giant; Locomotion

When the escape response of the cockroach (*Periplaneta americana*) is triggered by wind, it is mediated by the cercal-to-giant interneuron pathway and leg motor circuitry, within the abdominal and thoracic portions of the ventral nerve cord. We have found that a lesion rostral to the thorax (transection of a cervical connective) produces specific changes in wind-evoked escape. Lesioned animals reliably displayed short-latency responses to wind. However, the orientation of the initial turning component of escape was altered and the duration of subsequent running was reduced. Preliminary physiological study suggests that changes in the orientation of escape reflect changes in the integration of wind-sensory signals by thoracic circuitry. These findings imply that rostral centers influence sensorimotor integration underlying wind-evoked escape.

In the central nervous system, many sensorimotor functions are primarily controlled by pathways at one 'level', but are influenced by circuitry at other levels. Such influence may be subtle or profound and the form it takes probably depends upon behavioral context. In vertebrates, some examples include: modulation of mammalian somatic reflexes by catecholaminergic systems[2], alterations of spinally controlled reaching movements in cats by descending visual pathways[1], or hormonally sensitive modulation of spinal and brainstem reflex loops for sexual behavior in rodents[21]. In several behaviors in invertebrates, especially insects, modulation of circuitry in segmental ganglia by pathways descending from the brain has been described. Examples include defensive reflexes in crickets and sexual reflexes in mantids[12,27]. For most of the above examples, we possess some understanding of either the behavioral contexts in which sensorimotor functions are modified, or the cellular mechanisms involved, but not an understanding of both. We describe here an example from an invertebrate system in which segmental sensorimotor circuits are subject to influence by descending pathways and for which it may be possible to describe the behavioral context in which they are influenced and the cellular mechanisms of that influence.

A well-known insect behavior with essential circuitry at segmental (abdominal and thoracic) levels is the wind-evoked escape response of orthopteroid insects such as cockroaches and crickets. Wind receptors on the abdominal cerci activate giant interneurons (GIs) in the abdominal portion of the ventral nerve cord (VNC) and the GIs activate thoracic interneurons and leg motor neurons to produce evasive behavior[23,24,32] (see Fig. 1). The evasive running response is preceded by a turning movement which orients the insect away from the predator's attack[5]. While more rostral neural pathways are involved in escape responses evoked through other modalities such as antennal mechanoreception[4,10,11], rostral influences on the pathway for wind-evoked escape have received little attention.

Neuronal circuitry rostral to the thorax is generally assumed to be non-essential for wind-evoked escape behavior. For example, it has been noted that decapitated cockroaches respond behaviorally to wind directed at the cerci[14] and in physiological studies, it has been found that impulses in the GIs continue to activate leg motor neurons following decapitation[23]. How-

Correspondence: C.M. Comer, Department of Biological Sciences, University of Illinois at Chicago, Box 4348, Chicago, IL 60680, USA.

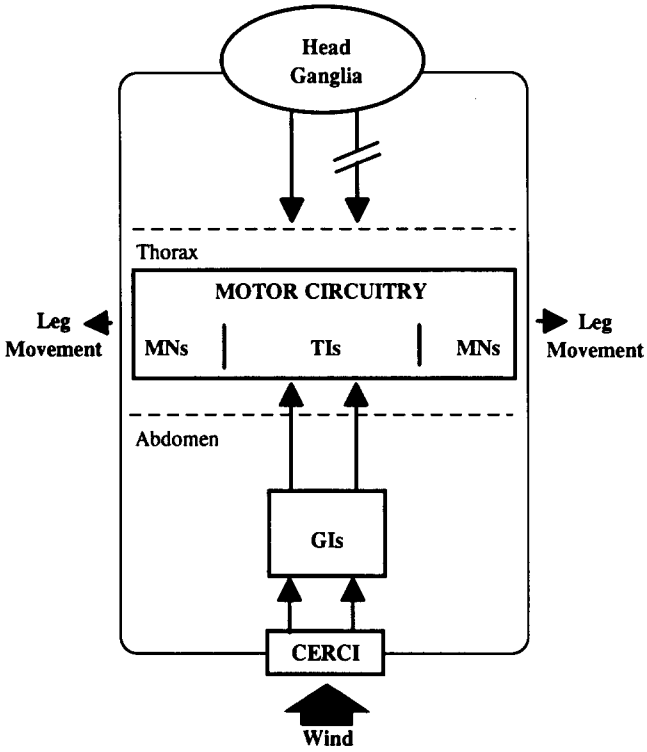

Fig. 1. Schematic illustration of circuitry for wind-elicited escape in relation to lesions made in this study. Neural circuitry is enclosed within the figure, with wind as input and movements of left and right legs as outputs. GIs, giant interneurons; TIs, thoracic interneurons; MNs, motor neurons. The interrupted line indicates a transection of the nerve cord (right connective) rostral to the thoracic level.

ever, despite responsiveness to wind, the behavior of decapitated animals is grossly abnormal: they do not engage in spontaneous locomotion and their evasive responses consist of short jumps without sustained running[14] (also Keegan and Comer, unpublished observations). We now have analyzed the effects of less drastic rostral lesions on wind-evoked escape behavior. Surgical transection of one cervical connective did not eliminate spontaneous locomotion, nor did it block turning and running in response to wind stimuli. Nonetheless, we observed specific changes in the execution of both the turn and run components of wind-evoked escape after lesion. Preliminary electrophysiological analysis suggests that cervical lesions do not significantly alter sensory information in the GIs, but rather the sensorimotor transformation within the GI-to-motor pathway. Some of the results have appeared in abstract form[15].

Adult male cockroaches, *Periplaneta americana*, were housed in plastic cages ($20 \times 28 \times 15$ cm) with screen tops and fed rat chow and water ad libitum. Animals were used only if their sensory and motor appendages such as legs, cerci and antennae were fully intact and if they were highly responsive to wind in prelesion testing.

For behavioral analysis, an animal was placed in an arena (45 cm diameter) and allowed to acclimate for 15 min. Animals typically engaged in spontaneous locomotion during this period: circling about the arena in one direction or the other. Because we intended to make unilateral lesions, this spontaneous motor activity was recorded so that any imbalances would be detected. To do this we continuously monitored the direction of locomotion and counted the total number of turns made (full circling movements clockwise or counterclockwise).

To assess wind-elicited escape, methods similar to those of previous studies[7] were used. Briefly, wind puffs were generated by a fan and delivered through a tube with a shutter at its end. When the shutter was opened, a wind puff with a peak velocity of 1.2 m/s and a rise-time of 95 ms was produced. This wind is known to stimulate cercal wind receptors, but is not sufficient to stimulate other mechanoreceptors that may trigger escape such as those associated with the antennae[30,31]. When an animal was stationary and its cerci were positioned at a standard point in front of the wind tube, the shutter was opened. Responses were viewed with an overhead high-speed video camera (Xybion SVC-02) and transcribed to tape with a Sony VO-5800 deck at a rate of 120 pictures per s. They were later analyzed from 'frame-by-frame' analysis.

At least 2 min elapsed between trials. No more than 30 consecutive trials were performed with an animal during 1 day. Behavioral studies were completed within 7 days of lesion. For each trial, the latency, wind stimulus angle, angle of the initial turn and length of the subsequent run were measured. Latency was estimated by counting the number of frames from shutter opening (indicated by a light emitting diode) to the animal's initial movement (each frame equivalent to 8.3 ms).

To make a cervical lesion, an animal was anesthetized with carbon dioxide and restrained ventral side up. Iridectomy scissors were inserted through a lateral puncture in the neck and the blade cut through cuticle and one cervical connective. Controls were handled similarly, except damage was done to cuticle and surrounding tissue only. Following lesion, a mixture of phenylthiourea and penicillin/streptomycin was applied to the lesion site[28] and dental wax was used to seal the cuticle. Animals were active within 30 min.

When postlesion testing was complete, the site of the lesion was verified first by gross dissection and then from histological examination of the nerve cord. The

TABLE I

Wind-evoked escape behavior of experimental (lesioned) and control (sham lesioned) animals both before and after lesion

L and R refer to animals where the cervical connective was cut on the left or right side, respectively. *N*, number of animals; *n*, number of trials, *R*, response frequency (percentage of trials on which responses occurred), *Lat*, average behavioral latency (expressed as number of video frames between wind onset and response) with time equivalent in ms given in parentheses to the right.

Group	*Prelesion*				*Postlesion*		
	N	*n*	*R (%)*	*Lat (ms)*	*n*	*R (%)*	*Lat (ms)*
Lesion/L	4	120	91	6.5 (54)	282	89	6.7 (56)
Lesion/R	4	120	93	6.3 (52)	286	93	7.2 (60)
Controls	3	90	96	6.1 (51)	198	91	6.4 (53)

brain and thoracic cord were extracted and fixed in Carnoy's solution and 12 μm cross sections were stained with eosin.

For electrophysiological experiments, an animal was anesthetized with carbon dioxide and legs and wings were removed. It was pinned to a wax platform dorsal side up and positioned so the cerci extended over the edge for clear reception of wind. For recording GI activity, the VNC was exposed at the abdominal level and unipolar silver-wire electrodes were placed on each connective between the 4th and 5th abdominal ganglia. For recording leg motor activity, similar electrodes were placed around nerve 6 of the mesothoracic ganglion. (Root 6 is almost purely motor[22] and is known to contain axons to posterior coxal levator muscles that flex the leg[20]. Flexion of the mesothoracic legs is known to be important in determining the direction of the initial escape turn made in response to frontal wind puffs[19].) Multi-unit signals from the electrodes were amplified by conventional methods and digitized at 10 kHz for storage and later analysis. When impulses were counted, only those that were $\geq 15\%$ of the maximum spike height were included.

Wind puffs for physiology experiments were generated using a wind machine that produced puffs with similar dynamics to those used in behavioral testing (measured with the same hot-wire anemometer). Following 20–30 prelesion wind trials, one of the cervical connectives was transected. After 30 min, 20–30 postlesion trials were recorded. In control animals the connective was manipulated without inflicting any obvious damage and neural activity was subsequently recorded.

Behavioral results are based on eight animals with unilateral cervical lesions and three controls. All experimental animals were found to have one connective

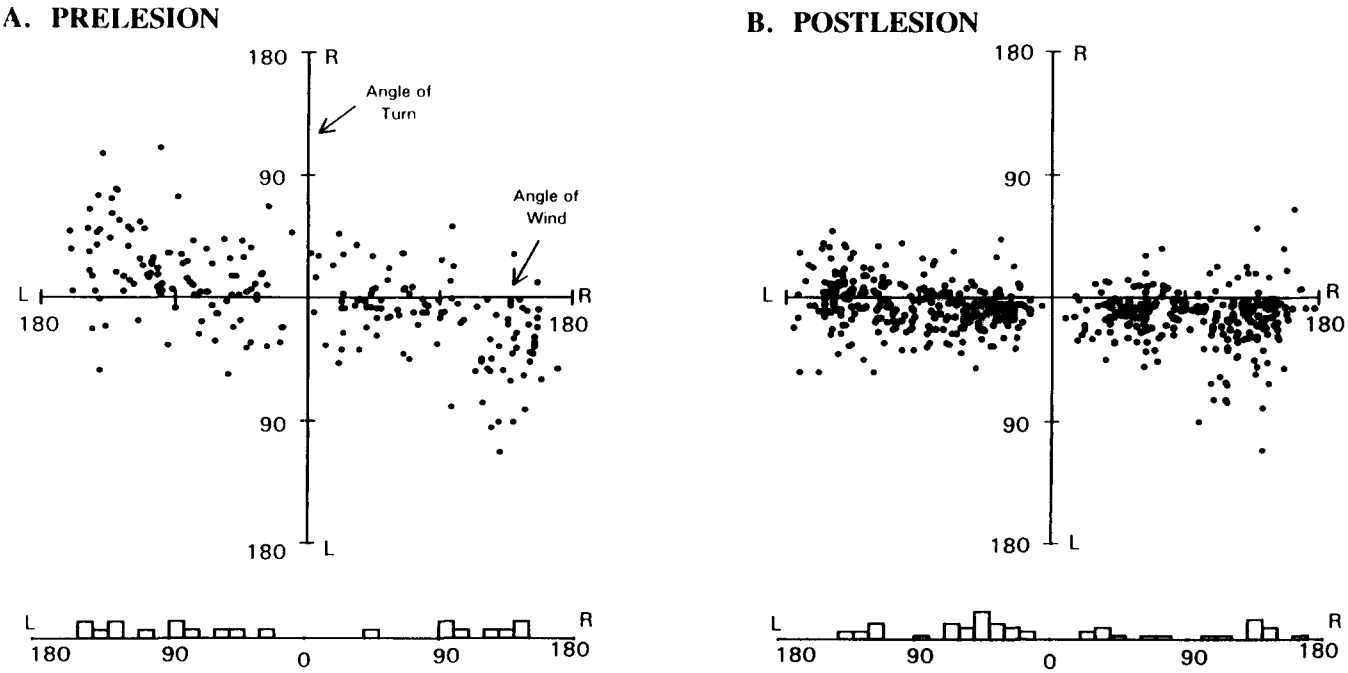

Fig. 2. Influence of unilateral (right) cervical lesion on the initial turning component of wind-evoked escape. Each plot gives pooled responses of eight animals (four animals with right side lesions, and four with left side lesions whose data were normalized to reflect the lesioned side as being on the right). Angle of turn is plotted vs. angle of wind stimulus as shown at top. On the horizontal axis, L and R refer to winds from left and right side of animal (wind from front, 180°; wind from rear, 0°). Vertical axis gives angular amplitude of initial pivot to the left (L) or right (R). Histograms at bottom show the distribution of trials on which no response to wind was observed.

severed upon postmortem dissection. In addition, histological sections confirmed unilateral neural damage in lesioned animals, but not in shams.

All lesioned animals engaged in spontaneous locomotion following surgery. While they could walk and turn either left or right, we noted a bias to circle in a direction away from the lesioned side. This was confirmed by counts of turns made during acclimation periods. Net turns (number of turns to the right − turns to the left) were always close to zero prior to lesion (mean = −4 for all experimental animals). Net turns after lesion increased significantly in the direction away from the lesioned side (right lesion group mean = −35, a left bias; left lesion group mean = +34, a right bias). Controls were unbiased in their spontaneous locomotion both before and after surgery.

A summary of data retrieved from the video analysis of wind-evoked escape is in Table I. The percentage of escape responses triggered by wind did not change significantly as a result of lesion (2×2 χ^2-test). Average latency differed by less than one video frame and this was not statistically significant (Mann–Whitney *U*-test).

Although animals were highly responsive after lesion, their wind-evoked turning was altered. All animals showed similar lesion effects and, as with spontaneous locomotion, changes in behavior were clearly related to the side of lesion, i.e., patterns of turning seen in responses to wind from each side of an animal following a lesion to the left cervical connective were a mirror image of patterns seen on each side following lesion of the right cervical connective. Thus, data have been normalized to represent all animals as if lesioned on the right side of the nervous system. Pooled turning data (angle of evasive turns plotted as a function of wind angle), both before and after lesion, are shown in Fig. 2.

The prelesion data (Fig. 2A) resemble other published data from normal cockroaches[5,7] and show that most frequently animals turned away from the wind source (points in upper left or bottom right quadrants of the graph). However, after lesion (Fig. 2B), responses to winds from the left side (opposite the lesion) tended to be directed to the left (ipsiversively) rather than the right (contraversively). The percentage of ipsiversive turns made in response to left winds was 23% before lesion and 62% after lesion ($P < 0.001$, 2×2 χ^2-test). This means that lesioned animals typically turned toward, rather than away from, wind sources on the side opposite lesion. Responses to wind from the right (side of lesion), on the other hand, showed a slight decrease in ipsiversive turns from 23% prelesion to 16% postlesion (difference not significant). It is also evident in Fig. 2 that animals tended to make turns of smaller angular size following lesion.

Three things are important to note about changes in turning. First, the bias in wind-evoked turning was in the same direction as that in spontaneous locomotion: lesions on one side caused heightened turning to the contralateral side in spontaneous behavior and during evasive turning. Second, the bias was not an absolute motor bias. Lesioned animals were still capable of turning in either direction (spontaneously and when stimulated by wind). Third, this bias does not appear to generalize to all sensory modalities: when tested with a different sensory input (tactile stimulation of the pronotum or legs) animals with this lesion either displayed a turning bias in the opposite direction (a tendency to turn toward the side ipsilateral to the lesion), or no bias at all[15,16].

Change was not restricted to the turn component of wind-triggered escape. In most prelesion responses, running persisted for at least 20 video frames (166 ms) from stimulus triggering (after that, most animals were out of the camera's view) and animals usually translated themselves by at least one body length in that time period. This translational component of escape was significantly reduced following lesion: in response to winds from either side, animals often stopped or paused soon after turning. This can be seen in the pooled data of Fig. 3. Fewer postlesion responses persisted to the 20th frame and fewer resulted in transla-

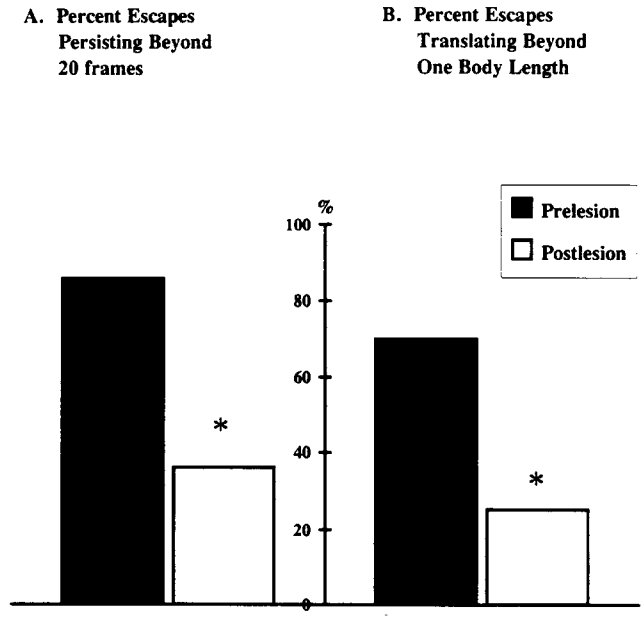

Fig. 3. Influence of unilateral cervical lesion on the run component of wind-evoked escape. A: percentage of escapes persisting for more than 20 video frames (166 ms); prelesion, 86%, postlesion, 36%. B: percentage of responses which translated animal more than one body length from initial position; prelesion, 70%; postlesion, 25%. Asterisk, significant difference at $P < 0.001$, 2×2 χ^2-test.

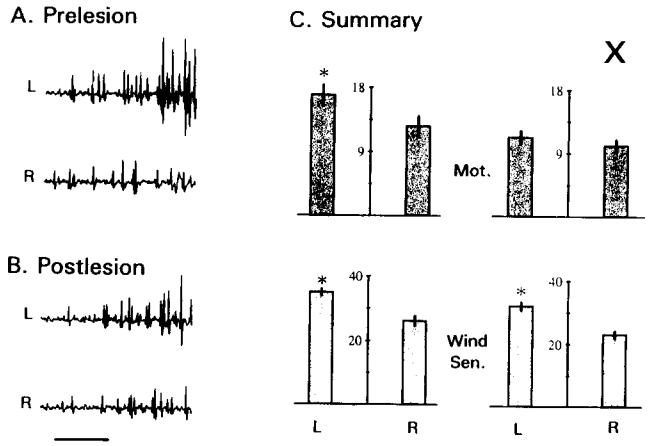

Fig. 4. Influence of unilateral (right) cervical lesion on wind-evoked sensory and motor impulse activity. A: example of left/right difference in wind-evoked motor activity. Traces labelled L and R were recorded from left and right mesothoracic roots as described in text. Wind was from front left (135°). B: mesothoracic motor activity recorded at same gain, but 30 min after section of the right cervical connective. Calibration bars apply to both (40 ms, 300 μV). C: laterality of activity in ascending abdominal wind interneurons (Wind Sen.) and thoracic motor neurons (Mot.) summarized for all animals ($n = 10$) both before and after cervical lesion. L and R, left or right abdominal connective or thoracic root. Activity was evoked in response to front left winds. Height of each bar is average number of evoked large impulses ± S.E.M. (vertical line). See text for a definition of large impulse. X, side of lesion. Asterisk, statistically significant difference at $P < 0.05$, paired Wilcoxon sign test.

tion of an animal by more than one body length ($P < 0.001$ 2×2 χ^2-test).

Wind-evoked neural activity ascending within the VNC contains directional information. For example, the GIs ipsilateral to a wind source are more active than those contralateral to a wind source[8,29,32]. Thus one possible explanation for a shift in wind-evoked turning would be that unilateral cervical lesions alter the directional information ascending to thoracic levels, principally via the GIs. An alternative explanation would be that wind-evoked sensory signals might have normal directional information, but its translation into motor signals could be altered. We therefore compared the wind-evoked impulse activity of large amplitude ascending units on the left and right side of the abdominal nerve cord, as well as wind-evoked thoracic motor activity both before and after making unilateral cervical lesions. Fig. 4 shows a sample of electrophysiological data and a summary from all 10 lesioned animals.

Prior to lesion, wind puffs from one side produced measurable laterality differences in both ascending wind-sensory activity and in thoracic motor activity. Fig. 4A shows wind-evoked motor activity in response to a wind puff from the front left. After cutting the right cervical connective, wind-evoked activity in the ascending pathway containing GI signals was still lateralized, but motor activity was no longer clearly lateralized in response to wind from the left (Fig. 4B). This finding was consistent in all animals studied physiologically (Fig. 4C). The change documented in this figure was specific to winds from the left (side opposite the lesion); wind puffs from the right side continued to produce significant left/right differences in both sensory and motor activity (Wilcoxon paired test).

Most available information about rostral centers and escape behavior has come from physiological observations of animals with complete disconnection of the head ganglia. Huber has noted in crickets[13] that the kicking response to cercal stimulation is enhanced following removal of the brain, but is suppressed during electrical stimulation of certain brain regions. In cockroaches, Ritzmann and Camhi[23] found that electrically evoked impulses in GIs can activate leg motor neurons after decapitation, but they noted an enhancement of motor neuron activation by the GIs under these conditions. Both of these observations suggest a net inhibitory effect of the head ganglia on escape circuitry. We saw no clear indication that descending inhibition was removed by our unilateral lesions: animals were not significantly more likely to respond to wind after lesion, nor did they respond more quickly (Table I).

Nonetheless, not all the effects of rostral centers on escape are likely to be inhibitory. For example, although decapitated animals respond to wind, the fact that their responses are not sustained has been interpreted to mean that a descending loop provides excitatory feedback for the running component of the escape response[14]. Recent experiments have also described units in the cockroach corpora pedunculata that discharge in relation to locomotion and appear to be associated anatomically with brain regions that give rise to descending pathways[17]. Interruption of descending (perhaps tonic) drive could be related to our observation that forward translation in the run phase of escape was reduced following cervical lesion (or to asymmetries in spontaneous locomotion, see below). While the changes in escape behavior we observed do not allow conclusions about descending inhibitory vs. excitatory control, they do indicate that *the form of the escape turn and run is not determined solely by ascending wind sensory signals arriving at the thoracic ganglia, but by signals descending to the thoracic ganglia as well.*

Lesions of the VNC that have been reported in past work to shift the directionality of wind-evoked escape were unilateral lesions made at the abdominal level, i.e., cutting one abdominal connective[7], or deleting one or more GI axons from the abdominal cord with pronase[6,8,9]. Such effects might be expected from abdominal lesions, because they directly disrupt the cercal-

GI-motor pathway. However, the lesions made here were not directly within that pathway.

It is important to note that cervical lesions not only altered wind evoked turning, but also produced effects not seen following unilateral abdominal lesions: a bias in spontaneous locomotion. Many previous studies have reported similar locomotor biases as a result of lesions rostral to the thoracic ganglia[3,12,27]. The relationship of any tonic motor bias to alterations in wind-evoked escape turns is not yet known. Indeed, the observed bias in spontaneous locomotion need not be interpreted solely as a motor bias. For example, animals usually locomote with one antenna contacting a wall and the bias in locomotion could be related to 'favoring' of one antenna as a result of the unilateral lesion – this possibility will require further investigation.

Nonetheless, the observed changes in direction of escape turning did not reflect loss of motor competence to turn in one direction. In spontaneous movement and in wind-evoked turning, animals retained the ability to turn in both directions (e.g. Fig. 2). Furthermore, when evasive turns are evoked through other modalities, such as touch, animals with unilateral rostral lesions may show no bias in turning, or even a bias opposite to that seen here[15,16]. Therefore the changes in wind-evoked escape turning following rostral lesions do not appear to represent global effects on turning motor circuits, but rather effects related to specific aspects of sensorimotor processing. Another difference between the effects of unilateral cervical lesions and those made at abdominal levels is that animals with an abdominal connective cut make ipsiversive turns in response to winds from the side of the lesion[7], whereas sectioning a cervical connective caused the animals we studied here to turn ipsiversively in response to winds from the side opposite the lesion. Furthermore, the shift to ipsiversive turning produced by unilateral abdominal cord section was virtually complete[7], but the shift to ipsiversive turning produced by unilateral cervical lesions was considerably less complete (34% contraversive turning remained, see Fig. 2). These differences suggest that lesions at the cervical level interrupt a descending pathway(s) that is non-essential to escape and which is not as strictly lateralized with respect to thoracic motor circuitry as the ascending wind-sensory (GI) system.

The physiology of wind-evoked impulse activity at mesothoracic root 6 showed a correlation with lesion-induced changes in wind-evoked turning behavior. On average, an animal's motor root activity contralateral to a cervical lesion was no longer predominant following presentation of a wind puff from the side contralateral to the lesion. This is the same side on which animals responded more often by turning into, rather than away from a wind stimulus. Presumably then, this modulation of the circuit for wind-triggered escape by rostral centers occurs at the level of thoracic motor circuitry. For example, touching the antennae can activate large caliber interneurons that descend to thoracic levels and appear to be capable of initiating escape turns independent of the GI system[4,8,11,31]. Recent work has indicated that identified thoracic interneurons which receive wind-sensory input via the GIs also receive convergent inputs from other sensory modalities, including touch[26].

It seems reasonable to expect that descending sensory signals should influence the GI-to-motor system. For example, the direction of a turn may be modified by antennal contact with a barrier either prior to (Kono and Comer, unpublished observations), or during a wind-evoked escape response[26]. In the presence of a barrier, a turn toward a wind source may be the appropriate strategy for successful escape. Further study of interactions between antennal touch information and wind sensory information may provide an understanding of some of the behavioral contexts for modification of wind-evoked escape.

While the cellular details of descending modulation of escape are not yet known, it should be possible to record from specific thoracic motor cells and determine the action of both tonic and phasic descending signals on the GI-to-motor transformation. Escape circuits have often been regarded as among the 'simplest' of neurobehavioral systems. However, the data presented here, as well as other recent examples in which escape responses are modified according to behavioral context[18,25], suggest that they will continue to serve as tractable models for some of the most interesting phenomena of organismal behavior.

This work was supported by a Grant from the National Science Foundation (No. BNS-8909051) to C.M.C. We thank Drs. J. Dowd, E. Gose, A.D. Murphy and W. O'Neill for comments on an earlier version of this manuscript.

1 Alstermark, B., Eide, E., Gorska, T., Lundberg, A., Pettersson, L.G., Visually guided switching of forelimb target reaching in cats, *Acta Physiol. Scand.*, 120 (1984) 151–153.
2 Barnes, C.D., Fung, S.J., Pompeiano, O., Descending catecholaminergic modulation of spinal cord reflexes in cat and rat, *Ann. NY Acad. Sci.*, 563 (1989) 45–48.
3 Bethe, A., Nervous system of arthropods, *J. Comp. Neurol.*, 8 (1898) 232–238 (translation).
4 Burdohan, J.A., Comer, C.M., An antennal-derived mechanosensory pathway in the cockroach: descending interneurons as a substrate for evasive behavior, *Brain Res.*, 535 (1990) 347–352.
5 Camhi, J.M., Tom, W., The escape behavior of the cockroach *Periplaneta americana* I. Turning response to wind puffs, *J. Comp. Physiol. A.*, 128 (1978) 193–201.
6 Comer, C.M., Analyzing cockroach escape behavior with lesions of individual giant interneurons, *Brain Res.*, 335 (1985) 342–346.
7 Comer, C.M., Dowd, J.P., Escape turning behavior of the cock-

roach. Changes in directionality induced by unilateral lesions of the abdominal nervous system, *J. Comp. Physiol. A*, 160 (1987) 571–583.

8 Comer, C.M., Dowd, J.P., Multisensory processing for movement: Antennal and cercal mediation of escape turning in the cockroach. In R. Beer, R. Ritzmann and T. McKenna (Eds.), *Biological Neural Networks in Invertebrate Neuroethology and Robotics*, Academic Press, NY, 1993, pp. 89–112.

9 Comer, C.M., Stubblefield, G.T., Effects of lesioning individual giant interneurons on the direction of the escape turning response in the adult cockroach, *Periplaneta americana*, *Soc. Neurosci. Abstr.*, 14 (1988) 310.

10 Comer, C.M., Dowd, J.P., Stubblefield, G.T., Escape responses following elimination of the giant interneuron pathway in the cockroach, *Periplaneta americana*, *Brain Res.*, 445 (1988) 370–375.

11 Comer, C.M., Mara, E., Murphy, K.A., Getman, M., Mungy, M.C., Multisensory control of escape in the cockroach *Periplaneta americana*. II. Patterns of touch-evoked behavior, *J. Comp. Physiol. A*, in press.

12 Huber, F., Neural Integration (Central Nervous System). In M. Rockstein (Ed.), *The Physiology of Insecta, Vol. II*, Academic Press, New York, 1965, pp. 333–406.

13 Huber, F., Brain controlled behaviour in orthopterans. In J.E Treherne & J.W.L. Beament (Eds), *The Physiology of the Insect Central Nervous System*, 1965, pp. 233–246.

14 Hughes, G.M., Neuronal Pathways in the insect central nervous system. In J.E. Treherne and J.W.L. Beament (Eds.), *The Physiology of the Insect Central Nervous System*, New York, Academic Press, 1965, pp. 79–112.

15 Keegan, A.P., Mara, E., Comer, C.M., Expanding the boundaries of the cockroach 'escape circuit', *Soc. Neurosci. Abstr.*, 17 (1991) 1245.

16 Mara, E., Behavioral and neural characterization of tactually-elicited escape in *Periplaneta americana*, (M.S. thesis, University of Illinois at Chicago), 1991.

17 Mizunami, M., Weibrecht, J.M., Strausfeld, N.J., A new role for the insect mushroom bodies: place memory and motor control. In R. Beer et al. (Eds.), *Biological Neural Networks in Invertebrate Neuroethology and Robotics*, Academic Press, New York, 1993 pp. 199–225.

18 Nolen, T.G., Hoy, R.R., Initiation of behavior by single neurons: the role of behavioral context, *Science*, 226 (1984) 992–994.

19 Nye, S.W., Ritzmann, R.E., Motion analysis of leg joints association with escape turns of the cockroach, *Periplaneta americana*, *J. Comp. Physiol.*, 171 (1992) 183–194.

20 Pearson, K.G., Iles, J.F., Discharge patterns of coxal levator and depressor motorneurones of the cockroach, *Periplaneta americana*, *J. Exp. Biol.*, 52 (1970) 139–165.

21 Pfaff, D.W., Features of a hormone-driven defined neural circuit for a mammalian behavior, *Ann. N.Y. Acad. Sci.*, 563 (1989) 131–147.

22 Pipa, R.L., Cook, E.F., Studies on the hexapod nervous system. I. The peripheral distribution of the thoracic nerves of the adult cockroach, *Periplaneta americana*, *Ann. Entomol. Soc. Am.* 52 (1959) 695–710.

23 Ritzmann, R.E., Camhi, J.M., Excitation of leg motor neurons by giant interneurons in the cockroach *Periplaneta americana*, *J. Comp. Physiol. A.*, 125 (1978) 305–316.

24 Ritzmann, R.E., Pollack, A.J., Identification of thoracic interneurons that mediate giant interneuron-to-motor pathways in the cockroach, *J. Comp. Physiol. A*, 159 (1986) 639–654

25 Ritzmann, R.E., Tobias, M.L., Fourtner, C.R., Flight activity initiated via giant interneurons of the cockroach: evidence for bifunctional trigger interneurons, *Science*, 210 (1980) 443–445.

26 Ritzmann, R.E., Pollack, A.J., Hudson, S.E., Hyvonen, A., Convergence of multimodal sensory signals at thoracic interneurons of the escape system of the cockroach, *Periplaneta americana*, *Brain Res.*, 563 (1991) 175–183.

27 Roeder, K.D.: The control of tonus and locomotor activity in the praying mantis (*Mantis religiosa* L.), *J. Exp. Zool.*, 76 (1937) 353–374

28 Schneiderman, H.A.: Insect Surgery. In F. Wilt and N. Wessells, N. (Eds.), *Methods in Developmental Biology*, T.Y. Crowell, New York, pp. 753–766.

29 Smith, S.R., Wheeler, B.C., Dowd, J.P., Comer, C.M., Investigation of a multi-cellular neural code for directed movement, *Proceedings of the 13th Annual IEEE Conference on Engr. Biological Medicine*, 1991, pp. 457–459.

30 Stierle, I.E., Getman, M.E., Comer, C.M., Comparison of wind-elicited evasive behavior mediated by giant interneuron (GI) and non-GI pathways in the cockroach, *Periplaneta americana*, *Soc. Neurosci. Abstr.*, 14 (1988) 689.

31 Stierle, I.E., Getman, M.E., Comer, C.M., Multisensory control of escape in the cockroach *Periplaneta americana*. I. Initial evidence from patterns of wind-evoked behavior, *J. Comp. Physiol. A*, in press.

32 Westin, J., Langberg, J.J., Camhi, J.M., Responses of giant interneurons of the cockroach *Periplaneta americana* to wind puffs of different directions and velocities, *J. Comp. Physiol. A*, 121 (1977) 307–324.