Course correction circuitry translates feature detection into behavioural action in locusts

H. Reichert, C. H. F. Rowell & C. Griss

Department of Zoology, University of Basel, Rheinsprung 9, CH-4051 Basel, Switzerland

Like other flying animals, locusts must maintain course stability despite turbulence and motor errors. Accordingly, they must detect course deviations and then correct them. The compound eyes, the ocelli and the cephalic wind hairs all detect different sensory consequences of flight instability[1–3], and descending interneurones bring this information to the thorax[4–9]. One such interneurone of this population (the tritocerebral commissure giant neurone) is known to elicit correctional steering[10]. Here we characterize three additional pairs of descending deviation detector neurones and show how their information is translated into altered drive to the flight motoneurones. The central pattern generator for flight[11–13], modulated by proprioceptive feedback[14], gates the signal of the detector neurones in thoracic premotor interneurones[9], ensuring that the flight motoneurones are affected only during flight. Further, the gating process transforms the phase-independent information of the deviation detectors into a phase-dependent signal modulated at wing-beat frequency, and transfers it to those flight motoneurones active at the time and appropriate to the corrective action required.

We recorded intracellularly, with dye-filled microelectrodes, from the three pairs of descending interneurones and their identified postsynaptic cells in the thoracic ganglia of locusts (*Locusta migratoria* (L. 1758)) during flight motor activity[12], to correlate their morphological and functional characteristics. Course deviations were simulated by an artificial horizon, provided with a central wind jet (Fig. 1*b*). In some experiments the ocelli were stimulated independently via light pipes that excluded light from other sources[9]. These procedures established the role of the three interneurones as highly specific absolute deviation detectors, which integrate sensory information from the compound eyes, the ocelli and the wind hairs. Table 1 summarizes the excitatory input characteristics of the three cells.

We have named these interneurones DNI, DNM and DNC, so called because they receive input from the ipsilateral, medial and contralateral ocelli, respectively (Fig. 1*a*). All have been in part described previously, but because of confusion in the literature (Table 2), we find it necessary to rename them. Input from the compound eye confers on these cells sensitivity to large field movement and to direction of movement. Figure 1*c* shows an example of the specific nature of compound eye input. A simulated roll deviation presented to the compound eyes results in excitation of the left DNC only if the roll is clockwise and away from the horizontal position. For a roll of this specific type of given amplitude, the response of the cell increases with increasing divergence of the artificial horizon from the horizontal. The cell does not respond to a clockwise roll towards the horizontal, nor does it respond to an anticlockwise roll towards or away from the horizontal. This cell also responds with similar specificity to deviations in the pitch and yaw axes (Table 1). The optimal combination of compound eye stimulation for this cell is what the animal would experience during a diving, banked turn to the side contralateral to the axon.

Excitatory input from the wind hairs or ocelli complements the compound eye input in an aerodynamically compatible and directionally specific manner. For example, a cell which is excited by a visually simulated yaw to the left is also excited by wind directed towards the animal's head from the right which would in nature accompany this yaw. If compound eye input causes the cell to be excited by a simulated clockwise roll, it

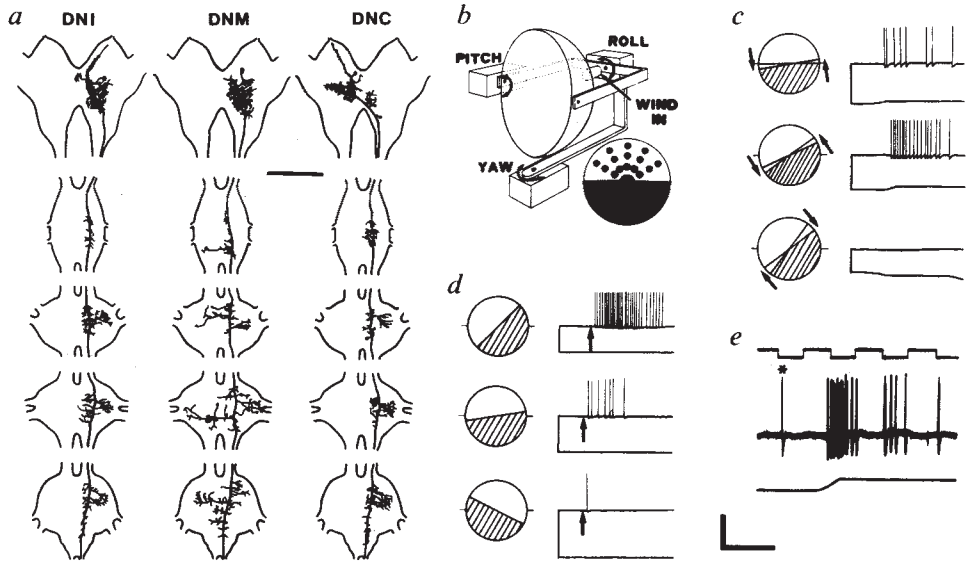

Fig. 1 The descending deviation detector neurones DNI, DNM and DNC. *a*, Morphology and nomenclature. The units are so named because they receive input from the ipsilateral, medial and contralateral ocelli, respectively, where ipsi- and contralateral are defined as the side on which the axon of the interneurone runs. This is clearly reflected in the location of the input arborizations in the brain. In the remaining ganglia, DNM is distinguished by its bilateral projection, while DNI and DNC project only unilaterally and are very similar in structure. All three units are paired. We distinguish individual units as left and right with reference to the connectives in which the axon descends to the thorax. Drawings are based on 20–30 successful intracellular fills (either Lucifer yellow[35] or hexamine cobalt chloride with subsequent intensification[36]) of each unit. Scale bar, 800 μm. *b*, Stimulus apparatus producing simulated course deviations. The animal's head was placed at the centre of a remotely steerable translucent hemisphere, on which was painted the opaque pattern shown, representing an artificial horizon and a structured visual field; the whole was illuminated diffusely with daylight from above and behind. The hemisphere, which was also equipped with a central air tube for simulation of wind on the head in flight, could be rotated around all three orthogonal axes and thus simulate pitch, roll and yaw deviations. *c*, Response of the left DNC to simulated clockwise and anticlockwise rolls away from (upper two records) and towards (lower record) the normal flying position. The sketches show the initial and final positions of the horizon, and the arrows the direction of its rotation. Only clockwise rolls away from the normal position evoke graded responses. Upper traces: intracellular recording, scale bars 50 mV, 200 ms; lower traces: roll monitor, vertical scale bar 75°. *d*, Response of same unit as in *c* to three identical frontal wind stimuli (onset at arrows) during maintained simulated visual rolls to various positions, as shown in the sketches. Calibrations as in *c*. Non-preferred orientations of the visual world inhibit the response to wind. *e*, Response of a DNI (centre trace, intracellular recording, scale bars 5 mv, 200 ms) to a visually simulated roll (lower trace, vertical scale bar 75°) and to simultaneous ON/OFF stimulation of the ipsilateral ocellus by means of a separate light pipe (upper trace, light at 565 nm modulated by 1 $\log_{10}$ unit, upward going = ON, duty cycle 200 ms). Ocellar OFF stimuli evoke only single spikes when presented alone (asterisk). These summate with the response induced by visually signalled roll. Ocellar ON stimuli are functionally incompatible with the visual information, as they imply a roll in the opposite direction; they strongly inhibit the visual response, which otherwise would consist of a long phasic-tonic burst, as in the middle record of *c*.

Table 1 Excitatory input characteristics of deviation detector neurones

	DNI	DNM	DNC
Ocellus producing reliable* excitation with OFF stimulation	Ipsilateral	Medial	Contralateral
Most effective visually simulated deviation (that is, via compound eyes)	Roll ipsilateral Pitch down Yaw ipsilateral	Pitch down	Roll contralateral (Pitch down)† Yaw contralateral
Most effective wind direction corresponds to	Yaw ipsilateral	Pitch down	Yaw contralateral

Data were derived from 60–100 experiments on each cell.
* DNI received additionally weak input from the medial ocellus, and DNM from both ipsi- and contralateral ocelli. In a fraction of the population, these weak inputs can evoke spikes.
† Weak response.

will also be excited by selective darkening of the right ocellus, which would in nature be darkened by such a roll[15].

The response properties of a deviation-detecting neurone are not, however, shaped simply by summation of excitation from three sensory sources. Mutual inhibition between the different sensory inputs is also important, and prevents a response if there is a conflict in information content. For example, Fig. 1*d* shows that the position of the visual world can alter the response of the cell to a constant wind stimulus. If the horizon is rolled to a 'visually preferred' position (Fig. 1*d*, top records), indicative of a specific deviation, and held there, the response to a frontal wind stimulus is strong (here 34 action potentials). If the horizon is held in a near-horizontal position (Fig. 1*d*, middle records), the same wind stimulus produces only a modest excitation (7 action potentials). Finally, if the horizon is rolled to a 'visually antipreferred' position, one which does not represent a deviation of interest to this cell, its response to the identical wind stimulus is virtually eliminated (1 action potential). Similarly, Fig. 1*e* shows that the ocellar input can inhibit the response to compound eye input. Darkening of the left ocellus produces an action potential in the left DNI, which responds visually to an anticlockwise roll. However, selective illumination of the same ocellus via a light pipe strongly inhibits the response of the neurone to an otherwise effective visual roll stimulus. Inhibition of the wind input by ocellar input[8] and inhibition of the compound eye input by wind input can be demonstrated in similar ways. These inhibitory relations reflect the relative probability of error for a conflict occurring in natural conditions (for example, turbulence could affect the wind hairs without producing a course deviation; the eyes would experience no change in stimulus, and the two inputs to the interneurone would convey conflicting information were it not for the inhibition). Taken together, these interactions ensure that the firing of any of these neurones is a reliable indication of a specific and well-defined deviation from course. Each neurone responds optimally to that specific combination of sensory stimuli which would impinge on the compound eyes, ocelli and wind hairs during a specific deviation. The units are formally multimodal, but we feel that this is not a useful description; the feature detected by these neurones, their 'emergent modality', is course deviation.

The cells central to the transformation of the firing of these deviation detectors into appropriate steering manoeuvres are a population of thoracic interneurones[9] that have three important characteristics[16] (see Fig. 2*e*): (1) they are postsynaptic to specific descending deviation detectors, (2) they are powerfully presynaptic to specific flight motoneurones and (3) they receive

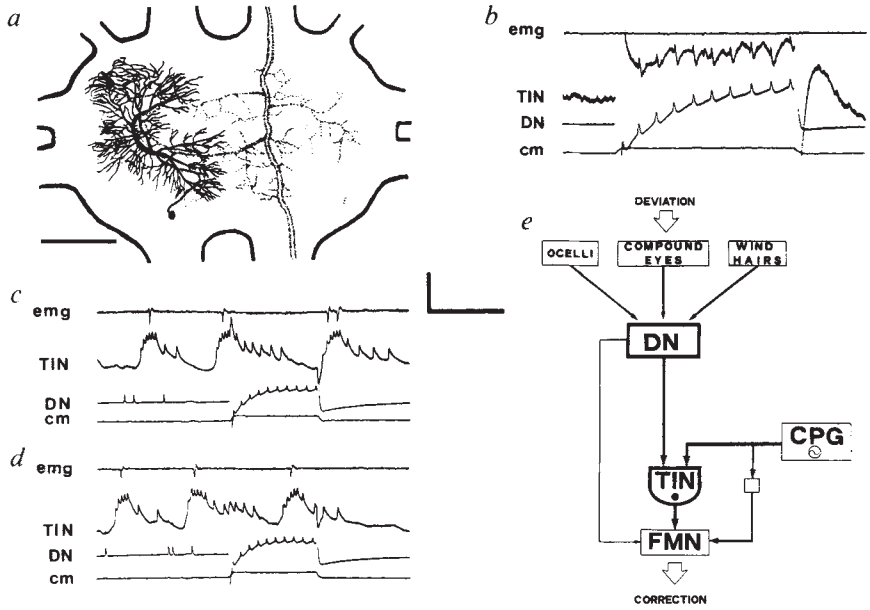

Fig. 2 Flow of information from the deviation detector neurones (DN) to the flight motoneurones (FMN) is gated in the thoracic interneurones (TIN) by summation with a phasic signal derived from the central pattern generator for flight (CPG). ***a***, Presynaptic DNM (dotted) and representative postsynaptic TIN (solid) in mesothoracic ganglion (scale bar, 300 μm). ***b***, Simultaneous intracellular recordings in a non-flying locust (scale bars 2 mV and 50 mV, respectively, 50 ms) from the TIN and DN shown in *a*, and an extracellular recording from the dorsal longitudinal depressor muscle ('emg') which serves as a monitor of flight activity. A depolarizing current of 10 nA ('cm') injected into DN evokes 10 action potentials, which in turn evoke small 1 : 1 excitatory postsynaptic potentials (e.p.s.ps) in the TIN. (The small high-frequency deflections on the TIN trace are artefacts caused by capacitative coupling between the two electrodes. The input bridges of both recording amplifiers are balanced, but the high current necessary to evoke a brisk burst of action potentials from an axon penetration results in a sizeable stimulus artefact.) ***c***, ***d***, Same experiment as *b*, but during flight motor output, indicated on the emg trace by periodic depressor muscle potentials. Scale bars 20 mV (TIN), 100 mV (DN) and 100 ms. The TIN receives strong synaptic drive from the CPG, depolarizing in the depressor phase. Note that in *c* and *d* the current pulses come at different points in the wing-beat cycle. During the depressor phase, e.p.s.ps evoked by spikes in the DN increase the number of action potentials in the depolarized TIN by ~50%, which in turn influences the postsynaptic FMN (not shown). Visual stimuli can cause a spike rate in the DN 50% greater than that induced here by current injection, so that this figure underplays the potential effect. During the elevator phase, the same DN activity does not evoke spikes in the TIN, and thus has no effect on the FMN postsynaptic to that cell. ***e***, Summary diagram indicating the main relationships in the circuitry discussed here. Thickness of arrows is roughly proportional to the relative strength of the interaction. The TIN population is symbolized as an AND-gate, reflecting its role. CPG drive reaches the FMNs not only via the TINs, but also by other interneurones which do not receive DN input.

Table 2 Synonomy of the deviation detector neurones

Interneurone			Ref.
DNI	DNC	DNM	This work
O_3	O_2	—	28–30
ip 1	cl 1	—	31
O_2	O_1	—	32
i.o.	c.o.	—	33
VIP 1	VCP 1	—	6
O_3	—	O_3	8
O_4	—	—	34
Fast ipsilateral, Fast ipsilateral (medial)	Fast contralateral	Fast medial	9

Deviation detector neurones have been mentioned under different names by previous authors; all used *Schistocerca* rather than *Locusta*, but we can detect no differences. Their descriptions were based on partial dye fills (for example, thoracic ganglia but not the brain, or vice versa) and either incomplete physiological data or none at all. The only complete structure previously given[8] is actually derived from two separate units, one filled in the brain and one in the thoracic ganglia. Normally, the oldest nomenclature would be adopted (in this case the $O_{1...n}$ system[28]). This is no longer practical, as is evident from the table. Abbreviations: $O_{2,3,...}$, ocellar interneurone 2, 3, . . .; ip 1, ipsilateral 3rd-order ocellar neurone; cl 1, contralateral 3rd-order ocellar neurone; i.o., ipsilateral ocellar neurone; c.o., contralateral ocellar neurone; VIP, ventral nerve cord unit, ipsilateral to its soma, protocerebral soma; VCP, ventral nerve cord unit, contralateral to its soma, protocerebral soma.

rhythmically alternating excitatory and inhibitory drive from elements of the flight central pattern generator[13], at phase angles characteristic for each interneurone. One such thoracic interneurone is shown in Fig. 2*a*. In a non-flying animal, the firing of the presynaptic DNM evokes only subthreshold synaptic potentials in this thoracic interneurone (Fig. 2*b*). Postsynaptic flight motoneurones are consequently not affected and no behaviour results. However, in a flying animal the flight central pattern generator is operative and delivers rhythmic drive to the thoracic interneurones. If the deviation detector is now fired (Fig. 2*c*, *d*), the synaptic input it evokes in the thoracic interneurone summates with central pattern generator drive, causing the interneurone to fire an increased number of action potentials. This augmentation occurs only when the drive is depolarizing (Fig. 2*c*, *d*). Because of the rhythmic nature of the depolarizing and hyperpolarizing potentials evoked by the flight central pattern generator in the thoracic interneurone, the tonic deviation signal is transformed into phasic activity of the premotor interneurones. Sensory information is gated at the level of the thoracic interneurone. The appropriate postsynaptic flight motoneurones will consequently receive additional drive, phase-coupled with the wing-beat cycle, for the duration of the descending deviation signal. This additional phase-coupled motor drive can either alter the phase of firing of one of a pair of homologous bilateral motoneurones, or alter the number of action potentials in the discharge of affected motoneurones, or even recruit an otherwise silent motoneurone. These comprise the mechanisms which have been shown to operate during steering behaviour by intact locusts[17–20]. All the flight motoneurones and the premotor interneurones are spiking cells with all-or-none threshold characteristics, clearly a prerequisite for this mechanism. We describe elsewhere[16] circuitry in which the CPG-evoked depolarizations in the premotor interneurones summate with inhibitory postsynaptic potentials derived from the deviation detector neurones, rather than with their excitatory postsynaptic potentials, but which has similar effects on the firing pattern of the motoneurones. That summation of inputs to spiking premotor interneurones could lead to gating of the signal to motoneurones has been postulated previously[21], but not demonstrated.

The deviation detector neurones also make direct connections with the flight motoneurones[8,9]. We have shown elsewhere[16] that these connections produce much smaller postsynaptic potentials than those mediated by premotor interneurones. This is understandable in view of the need to avoid flight muscle contraction when the animal is not flying; the direct input to the flight motoneurone must be small enough to avoid firing it, even though the motoneurone receives an almost continuous barrage of sensory excitatory postsynaptic potentials from other sources. In flight, however, these direct postsynaptic potentials are also subject to a similar gating process by the flight central pattern generator, which drives the motoneurones directly[13] as well as the premotor interneurones.

Thus, at the thoracic level, a two-stage gating process ensures that the intrinsically phase-independent information from the deviation detectors: (1) elicits a response in the flight motoneurones only when the flight central pattern generator is operative, that is, in the behavioural context of flight, (2) is phase coupled, that is, is transformed into a phasic signal modulated at wing-beat frequency and (3) is automatically applied to the appropriate group of flight motoneurones which are active at the time of arrival of the signal, obviating the need for any further decision-making in the brief interval between stimulus and response.

The complete steering behaviour of the animal involves not only the modulation of wing beat treated here, but also movements of head, abdomen and hind legs[11,22–24]. It remains unclear whether the circuitry described here participates in these elements. However, we have evidence that the information brought to the thoracic ganglia by the other deviation-sensitive interneurones is processed in the same way as described above. Furthermore, the circuit principles documented here seem to be generally applicable and significant. In vertebrates, for example, locomotor control involves descending pathways from projection areas in the brain to segmental motor centres in the spinal cord and thence to spinal motoneurones[25]. Moreover, a gating process has been postulated and ascribed to the level of the segmental interneurones[26,27]. This scheme is comparable in detail to the circuitry documented here for the locust (see Fig. 2*e*). Indeed, we expect that similar principles of neuronal organization will be found in all animals that need to integrate a rhythmic motor output with non-phase-locked exteroceptive information, a need that occurs in most forms of locomotion.

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