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The Anterior Basal Lobe and Control of Prey-Capture in the Cuttlefish (*Sepia officinalis*)

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CHICHERY, M. P. AND R. CHICHERY. *The anterior basal lobe and control of prey-capture in the cuttlefish* (Sepia officinalis). PHYSIOL BEHAV 40(3) 329–336, 1987.—The predatory behaviour of the cuttlefish comprises several stages: prey-detection, orientation, translation and prey-seizing. In this neuroethological study, lesions to the anterior basal lobe were made by an electrolytic method and the animals were allowed to attack their prey in an unrestricted way so that motor defects, functional recovery and the emergence of new adaptative behavioural strategies could be studied. Lesions to the central region of the anterior basal lobe suppress the orientating behaviour, thus only prey situated in the frontal visual field can be seized. Less extensive lesions in this region are associated with similar defects. Without head orientation, the cuttlefish still rotates with its fins. This rotation, however, is usually underestimated, tentacular ejection thus missing the prey. Dorsal lesions cause an underestimation of tentacular strike often associated with defects in maintaining ocular convergence. These results demonstrate the heterogenous function of the anterior basal lobe and its complex role in the control of predatory behaviour.

Cephalopod *Sepia* Lesion Motor control Motor programs Predatory behaviour Anterior basal lobe Visual guidance

THE cuttlefish (Mollusca, Cephalopoda) is a highly active predator, whose locomotion is often characterized by abrupt acceleration or deceleration brought about by the synergistic action of the fins and the funnel. Movements may be in any direction: forwards, backwards or to the side.

Prey is detected by visual input [18], the latter playing an important role in the various stages of predatory behaviour [13,18].

The cephalopod eye is similar in structure to that of vertebrates: a classic example of convergent evolution. It is highly mobile, on account of a complex extraocular musculature, a differentiated area in the posterior part of the retina functioning as a fovea. The statocysts have been extensively studied in recent years [5–7]. Their organisation reflects the vertebrate labyrinth, being adapted to detect linear and angular accelerations [5,6].

The central nervous system of cephalopods is by far the most highly evolved of the molluscs, convergent evolution with the vertebrates often being emphasized [22]. The neuroanatomy of this group is now very well known [20, 26–30]. Numerous studies of the vertical lobe system utilizing techniques of lesion in conjunction with discrimination learning experiments have permitted the elucidation of the perceptual performance and memory capacity of these animals [3, 4, 24]. Certain visuo-motor structures have also been extensively studied recently [8, 16, 20, 21, 25]. These structures, which are characterized by sensorial multiconvergence, are the peduncle and basal lobes. The motor and behavioural effects of electrical stimulation of these structures, in chronic experiments, have already been described [9,12]. There are few studies concerning motor and behavioural defects induced by ablation of motor centres [3,17].

The use of an electrolytic method, never used before with cephalopods, has permitted us to study the motor and behavioural defects provoked by lesions in this lobe.

METHOD

Sepia officinalis of between 200 and 1000 g wet weight were caught by local fishermen in the region of Luc-sur-Mer between May and October. The central nervous system comprises a central mass, containing supra-, peri- and sub-oesophageal lobes, flanked by two massive optic lobes. The supra-oesophageal mass is illustrated in Fig. 1. Numerous studies [16, 20, 21, 29] have shown that the basal and peduncle lobes share the same fundamental structure. The basal anterior lobe may be divided into anterior and posterior portions [29]. Each of these may be further divided into a basal region, the cortex of which contains some large neurons, and a dorsal region or spine containing a network of fine parallel fibres [29]. This lobe receives direct static input and optic input via the optic lobes. The afferent visual fibres are numerous, large and clearly visible in horizontal sections (Fig. 2). It is probable that this lobe also receives various sensory inputs from the arms, mantle and fins [8,21]. This

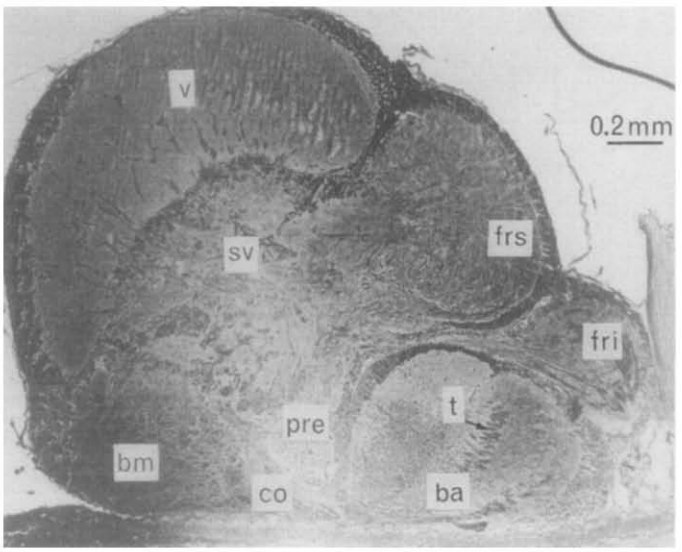

FIG. 1. Sagittal section of the supra-oesophageal mass. Cajal silver stain. ba: anterior basal lobe; bm: median basal lobe; co: ventral optic commissure; fri: inferior frontal lobe; frs: superior frontal lobe; pre: precommissural lobe; sv: subvertical lobe; t: ipsilateral dorsal optic to anterior basal tract; v: vertical lobe.

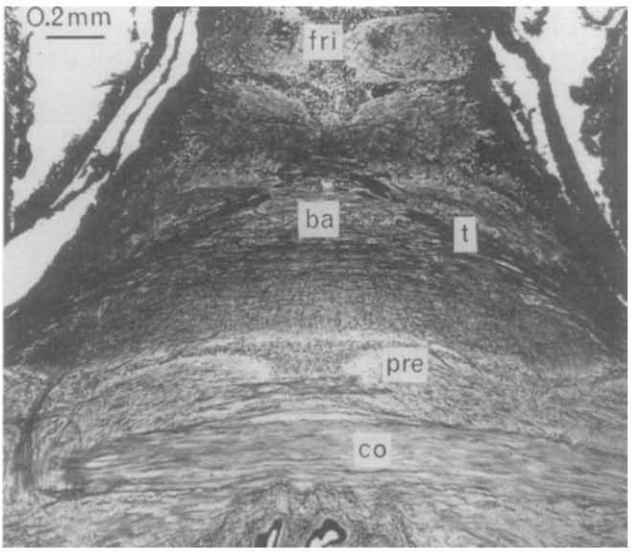

FIG. 2. Horizontal section of the supra-oesophageal mass. Cajal silver stain. ba: anterior basal lobe; co: ventral optic commissure; fri: inferior frontal lobe; pre: precommissural lobe; t: ipsilateral dorsal optic to anterior basal tract.

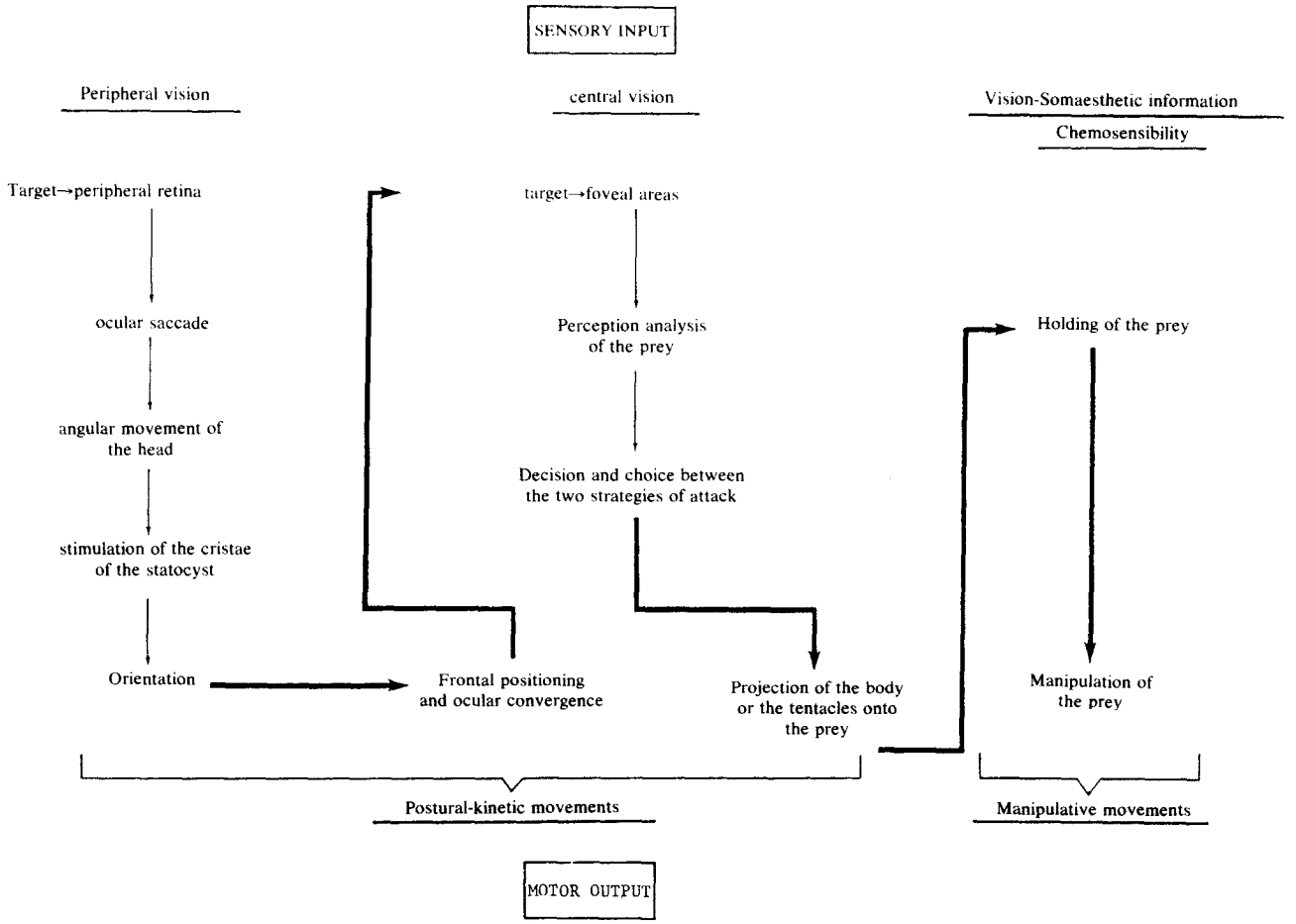

FIG. 3. Functional diagram of the different stages of the predatory behaviour.

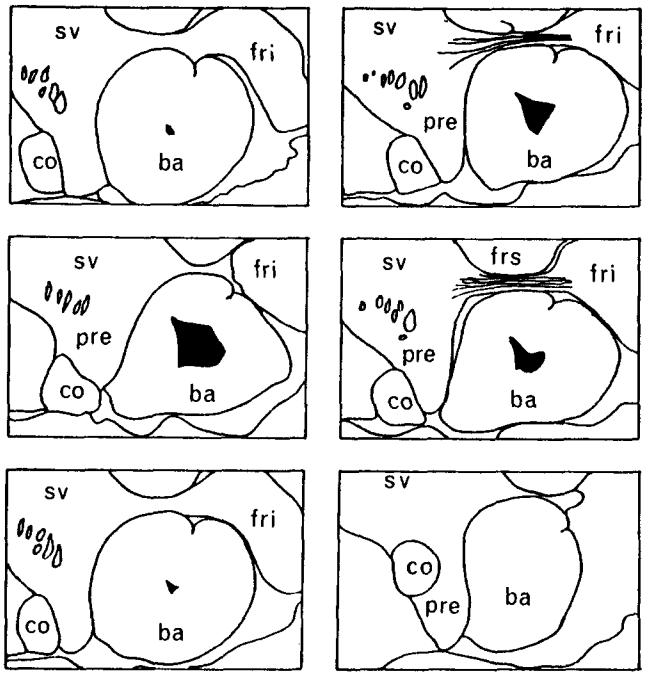

FIG. 4. Parasagittal and sagittal sections (serial sections; 200 μm between sections) through the supra-oesophageal mass. The maximal extent of this central lesions (in black) in the horizontal plane is about 800 μm. ba: anterior basal lobe; co: ventral optic commissure; fri: inferior frontal lobe; frs: superior frontal lobe; pre: precommissural lobe; sv: subvertical lobe.

sensory multiconvergence is associated with multidivergent outputs to various motor centres: the lateral pedal lobe (containing the motoneurons innervating the extraocular musculature), the posterior pedal lobe (containing the motoneurons innervating the funnel) and the palliovisceral lobe (controlling the movements of the mantle and the fins).

The predatory behaviour of the cuttlefish has been the subject of a few detailed studies [10, 13, 14, 18, 19], and can be split into three phases: attention, positioning, attack. The attention phase corresponds to the detection of the prey; it starts with an ocular saccadic movement followed by orientation of the head towards the prey and finally a rapid pivoting of the body, bringing the prey into the frontal visual field. The positioning phase is characterized by the taking up of a specific attack distance. Ocular convergence is maintained throughout this phase. Attack *per se* is achieved by one of two strategies: either by ejection of the two tentacles or by jumping on the prey. The different stages and characteristics of this predatory behaviour are outlined in Fig. 3.

The animals were rapidly anaesthetized by immersion in sea-water containing 2% ethanol. The dorsal part of the cranium was exposed. The positions of the sagittal plane and the anterior and posterior parts of the supra-oesophageal mass were localized using external morphological characteristics of the cranium. An electrolytic electrode (shaft diameter: 0.25 mm, contact diameter: 0.1 mm) was introduced into the cranium. The electrode was held in place by a small plate of inert plastic stuck to the cranium with tissue adhesive while the length of the electrode protruding was adjusted according to the size of the animal and to the depth of penetration desired [11]. Previously all lesion experiments have been involved the ablation of large parts of the supra-oesophageal mass. For this reason it was decided to destroy large parts of the anterior basal lobe without destroying it in totality by administering a current of 1 mA for 60 sec (Fig. 4). The electrode was then removed and the wound sealed with tissue cement. The animals were placed in experimental glass aquaria (1.50×0.80 m) and their motor defects were analysed from video recordings. To study behavioural defects, the prey (crabs and prawns) and predator were given the freedom of the aquarium so that defects from any stage of the attack could be studied as well as any recovery of function or the adoption of new strategies. Capture efficiency was evaluated using three criteria:

(1) total capture time (from detection to seizure of prey);
(2) percentage, first-time, capture success: controls: 85–90% [14,18];
(3) influence of the position of the prey before the attack (anterior visual field or posterior visual field).

Following experimentation (20–30 days), animals were sacrificed, the brain removed and fixed in 10% neutral formalin in sea-water and impregnated with silver [26].

The localisation and the extent of the lesions were studied in histological sections (20 μ). All lesions studied affected the sagittal, or slightly parasagittal, region of the anterior basal lobe (Fig. 4). No lesion, described here, directly affected the peri-oesophageal structures (e.g. lateral anterior basal lobe).

RESULTS

In this paper, only the principal results from 37 lesions affecting the anterior basal lobe are described. The principal motor effects of lesions to the anterior basal lobe and the disturbances of predatory behaviour are summarised in Table 1. Comparison of lesion sites with the corresponding motor activity and behaviour enables us to recognise five groups of defects (Table 2). A detailed analysis of predatory behaviour is summarised in Table 3, the varying number of attacks showed by the different lesioned animals depends on the size of the cuttlefish and the nature of the prey itself (crabs and shrimps). These results include only experimental animals having effected at least ten complete or incomplete attacks.

Group 1 results from extensive lesions affecting the central part of the anterior basal lobe. The orientating behaviour is profoundly altered and the ocular saccadic movement, the first criterion of prey detection and consequently head reorientation, no longer occurs. It must be pointed out that ocular convergence movements and the vestibulo-ocular reflex remained unaffected. The rapid rotation of the animal, normally assured by the synergistic action of the fins and funnel [1], is severely hindered, rendering body reorientation impossible or very difficult (Table 1). Thus, body rotation is usually insufficient and fails to bring the cuttlefish face to face with its prey. Tentacular ejection still occurs but is often unsuccessful (Table 3) due to directional miscalibration. It is noteworthy that this defect of calibration occurs in animals showing a total loss of rotation (e.g., lesion 17.85, Table 3), even when the prey is placed in the anterior visual field only very slightly to one side of the body axis (rotation in this case is not a prerequisite for prey capture in control animals as the angle of projection of the tentacles itself, in relation to the body axis, permits the necessary reorientation). In animals with this class of lesion, the failed tentacular ejection is sometimes accompanied by a jumping, the prawn being seized with the arms ("tentacular ejection + jumping" in

TABLE 1

SUMMARY OF THE MAIN MOTOR DEFECTS

Lesion	Orientation				Ocular Convergence	Capture		% First Time Capture Success	No. of Complete Attacks	Manipulation (crab)
	N	A	UN	OV		Anterior Movement	Seizure			
3.82		+			permanent	—	—	—	0	—
4.82	+				normal	normal	few	—	2	normal
5.82		+			normal	—	—	—	0	—
8.82		+			normal	—	—	—	0	—
9.82	+				normal	normal	normal	100%	10	normal
10.82	+				normal	normal	few	—	6	normal
11.82		+			permanent	—	—	—	0	—
12.82	+				normal	rolling	few	—	5	difficult
18.82			+		often no maintenance	normal	fr. fail.	70%	98	normal
19.82	+				often no maintenance	normal	fr. fail.	36%	33	normal
20.82	+				normal	normal	infr. fail.	83%	96	normal
22.82			+		often no maintenance	normal	fr. fail.	50%	21	normal
N.83	+				normal	normal	infr. fail.	78%	105	normal
P.83			+		normal	normal	fr. fail.	44%	8	not observed
1.83				+	often no maintenance	rolling	fr. fail.	50%	10	normal
3.83			+		often no maintenance	normal	fr. fail.	50%	11	normal
5.83	+				normal	normal	no eject. tent.	—	0	—
6.83	+				no maintenance	normal	—	—	0	—
9.83	+				normal	normal	normal	92%	39	normal
10.83	+				often no maintenance	rolling	normal	86%	41	normal
2.84	+				normal	rolling	infr. fail.	84%	15	normal
3.84	+				normal	normal	normal	90%	14	not observed
6.84	+				no maintenance	—	—	—	0	—
9.84				+	normal	normal	infr. fail.	83%	141	normal
10.84	+				normal	normal	normal	86%	39	normal
11.84			+		normal	normal	fr. fail.	56%	111	normal
1.85			+		normal	normal	normal	100%	8	normal
7.85	+				normal	normal	normal	86%	15	normal
8.85	+				normal	normal	normal	86%	16	normal
9.85	+				normal	normal	normal	100%	19	normal
11.85	+				no maintenance	—	—	—	0	—
12.85	+				normal	normal	normal	88%	18	normal
13.85	+				normal	normal	normal	92%	38	normal
16.85	+				often no maintenance	normal	fr. fail.	32%	23	normal
17.85		+			normal	normal	fr. fail.	50%	15	normal
18.85	+				often no maintenance	normal	fr. fail.	64%	44	difficult
19.85	+				often no maintenance	normal	few	—	4	normal

N: normal; A: absent; UN: under-estimated; OV: over-estimated; fr. fail.: frequent failure; infr. fail.: infrequent failure.

TABLE 2

MOTOR DEFECTS AND LOCALISATION OF THE LESIONS

Lesions	Most Frequent Defects	Site
Group 1		
3.82	Rotary motion	Middle of the ant.
5.82	impossible	basal lobe or large
8.82	or very	lesions in ant. ant. basal
11.82	difficult.	lobe or post. ant.
18.82		basal lobe.
P.83		
3.83		
11.84		
1.85		
17.85		
Group 2		
19.82	Defects in maintenance	Large lesions of
22.82	of ocular convergence	the dorsal parts
1.83	and under-estimation	of the ant.
6.83	of tentacular	basal lobe.
10.83	ejection.	
6.84		
11.85		
16.85		
18.85		
19.85		
Group 3		
12.82	Rolling during	Large lesions of the
1.83	the acceleration	ant. ant. basal
10.83	of the anterior	lobe with small
2.84	displacement.	lesions of the inf. cortex of the inf. frontal lobe.
Group 4		
3.83	Tentacular ejection	Lesions in the post.
5.83	impossible or difficult.	ant. basal lobe with large lesions in precom. lobe.
Group 5		
4.82	Very few defects.	Small lesions in the
9.82		frontal, dorsal or
10.82		ventral parts of the
20.82		ant. basal lobe.
N.83		
9.83		
3.84		
9.84		
10.84		
7.85		
8.85		
9.85		
12.85		
13.85		

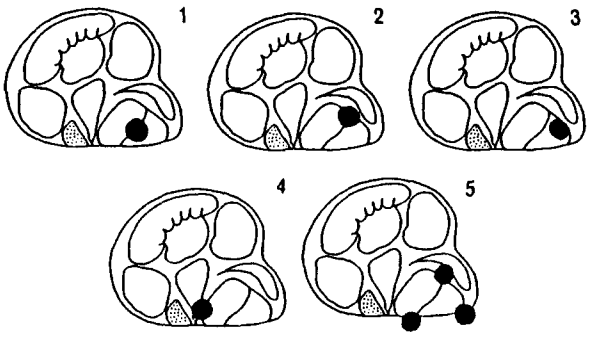

FIG. 5. Schematic representation of the localisation of the five groups of lesions (sagittal plane).

Table 3). This behaviour is very rarely observed in control animals and could correspond to the adoption of a new prey capture strategy. This adaptative strategy compensates for the failure of tentacular ejection when the prey was detected in the posterior visual field ($\chi^2=14.51$, $p<0.001$; no significant difference in control animals, $\chi^2=0.013$, $p>90$).

Group 2 comprises animals with extensive lesions to the dorsal parts (spines) of the anterior anterior basal and/or anterior posterior basal lobes. In these animals the orientation phase takes place normally but defects occur in the maintenance of ocular convergence during positioning. This defect often leads to the cuttlefish abandoning the attack. Such cessation of the attack is always followed by one or two vigorous forward expulsions of water between the arms. Prey attack, however, is still possible, though is often unsuccessful (Table 3), especially on the first attempt on account of an underestimation of prey distance (of up to 2 cm). The total time required for prey capture is noticeably increased and becomes far more variable (Table 3). In this group no significant difference appears in the accuracy of the attack in correlation with the position of the prey in the detecting phase, $\chi^2=0.02$, $p\geq 90$. As with group 1, a failed tentacular attack is sometimes accompanied by the adoption of jumping (Table 3).

Group 3 included animals having extensive lesions of the anterior portion of the anterior anterior basal lobe associated with some destruction of the ventral cortex of the inferior frontal lobe. In these animals spontaneous movements appear totally normal, though marked rolling movements are displayed during the acceleration implicated in the attack phase *per se*. This problem is all the more evident during the jumping attack on to crabs.

Group 4 animals have lesions of the posterior part of the posterior anterior basal lobe in addition to extensive destruction of the precommissural lobe. They all show difficulties in tentacular ejection.

Group 5 comprises animals with a variety of lesions that affect only a limited part of the anterior basal lobe arising from misjudgment of the positioning and depth of the electrode (dorsally, anteriorly and ventrally). No serious motor defects are evident in this group (Table 3). As with control animals, prey position has no significant influence on attack success, $\chi^2=0.24$, $p>50$.

The schematic localisation of all of these lesions is shown in Fig. 5.

None of the lesions of the anterior basal lobe, cited in the above five groups, had any effect on skin patterning, confirming results obtained by electrical stimulation [2,9].

TABLE 3

ANALYSIS OF PREDATORY BEHAVIOUR

	Prey Position Prior to Attack						
	Anterior Visual Field		Posterior Visual Field				
	Successful Attacks	Failed Attacks	Successful Attack	Failed Attacks	Mean Total Capture Time (in sec)	No. of Non-Maintenance of Ocular Convergence and Cessation of Attack	No. of "Tentacular Ejection + Jumping"
Lesions Group 1							
18.82	25	0	52	21	7.7 ± 3.3	3	2
P.83	2	0	2	4	11 ± 5.5	2	2
3.83	4	3	1	3	8.9 ± 5.5	2	2
11.84	23	9	30	49	5 ± 1.8	1	5
17.85	6	5	0	4	43.6 ± 26	0	7
Total	60	17	85	81		8	18
Lesions Group 2							
19.82	9	11	4	9	22 ± 11	11	0
22.82	3	3	8	7	9.6 ± 2.8	4	1
1.83	3	3	2	2	17.8 ± 7.4	4	1
6.83	0	0	0	0	—	30	0
10.83	14	4	21	2	3.6 ± 0.9	8	2
6.84	0	0	0	0	—	15	0
16.85	6	9	3	5	5.3 ± 1.4	4	0
18.85	15	6	13	10	7.5 ± 3.7	10	3
19.85	1	2	1	0	—	17	0
Total	51	38	52	35		103	7
Lesions Group 5							
9.82	4	0	6	0	3.8 ± 1.4	0	0
20.82	30	6	53	7	5.4 ± 2.9	0	1
N.83	27	7	60	11	5.6 ± 2.6	0	4
9.83	11	1	24	1	3.2 ± 1.4	0	1
3.84	4	1	9	0	8.2 ± 4.9	1	0
9.84	28	6	93	14	3 ± 1.5	0	0
10.84	16	0	20	3	2.8 ± 1	0	0
7.85	7	0	6	2	3.4 ± 1.2	0	0
8.85	3	2	11	0	3.6 ± 2.4	0	1
9.85	11	0	8	0	4.2 ± 1.3	0	0
12.85	5	0	11	2	3.1 ± 1	0	0
13.85	13	1	23	1	3.6 ± 1.6	0	0
Total	159	24	324	41		1	7
Controls							
1	6	1	11	1	2.2 ± 0.8	0	0
2	3	0	9	1	3.8 ± 1.6	0	0
3	4	0	7	0	2.8 ± 1	0	0
4	3	0	13	0	3.3 ± 1.4	0	0
5	9	0	15	1	4.3 ± 1	0	0
6	9	1	12	2	3.2 ± 1	0	0
7	6	2	15	1	2.5 ± 0.8	0	0
Total	40	4	82	6		0	0

DISCUSSION

Previous experiments involving acute and chronic electrical stimulation of the anterior basal lobe have shown that it plays an important role in the control of locomotion [2,9]. These new results confirm this role and underline the importance of this structure in the programming of the complex motor activities associated with predatory behaviour. Taking into account the histological localisation of lesions, the defects of orientating behaviour are possibly due to the partial or total destruction of the tracts originating in the optic lobe (ipsilateral dorsal optic to anterior basal tract). These fibres project into the central part of the anterior basal lobe, crossing over in the median anterior region [29]. These fibres could, therefore, trigger ocular saccadic movements and consequently the orientating behaviour as a whole. The structure of the anterior basal lobe is similar to that of the peduncle lobe. In the latter the projection of the optic fibres respects the retinotopic organisation of the optic lobe [20,21]. A recent reexamination of pathways to the peduncle lobe [8] shows that the visual input to the basal zone maintains the retinotopic organisation of the optic lobe whereas the visual input to the spine is more diffuse. A similar study concerning the anterior basal lobe would be very useful and could perhaps lead to the improvement of functional hypotheses.

Indeed, natural behaviour suggests the following successive operations leading to the estimation of the angular direction of the prey:

(1) Ocular saccadic movement, the amplitude of which is a function of the angular position of the target with respect to the foveal area.

(2) Head turning movement correlated with the new position of the eyes.

(3) Funnel reorientation: this new orientation is possibly calibrated in feed-forward control by direct static projections into the posterior pedal lobe [28]. These fibres could, as part of their function, carry sensory information concerning angular acceleration of head movement.

Defects in the initial phase of ocular saccadic movement could thus explain the lack, or the underestimation of the rotation necessary for successful prey capture. Defects in directional calibration of tentacular ejection, observed during attacks on prey in the anterior visual field, slightly to one side of the body axis, also indicate the importance of ocular saccadic movement in the correct programming of this final phase of the predatory behaviour. In this respect, it is interesting to note that, for mammals, several authors [15,23] have stressed the possible role of ocular saccadic movements in the programming of goal-directed movements of the anterior limbs.

Defects in the maintenance of ocular convergence and the rolling during the acceleration involved with jumping are both associated with a lesion of the dorsal part of the anterior basal lobe. In this region there is a network of fine parallel fibres with an apparent structural analogy with the cerebellar cortex of vertebrates [16]. These regions could provide a fine control of motor programs perhaps organised elsewhere (optic lobes, suboesophageal structures ?). The important correlation between the degree of ocular convergence and prey distance, as well as the remarkable visual depth perception of the cuttlefish [19], must be underlined. Defects of ocular convergence may therefore account for the underestimation of tentacular ejection.

These hypotheses must now be confirmed and expanded by experiments involving less extensive lesions in conjunction with studies of the temporal evolution of behavioural defects (recovery or deterioration) and the emergence of new adaptative strategies (such as jumping on prawns).

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