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Integration in Descending Motor Pathways Controlling the Forelimb in the Cat*

9. Differential Behavioural Defects After Spinal Cord Lesions Interrupting Defined Pathways from Higher Centres to Motoneurones

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Summary. The purpose of this study was to elucidate the relative role of the C3–C4 propriospinal neurones (PNs) and of neuronal networks within the forelimb segments for precise forelimb movements.

The effects of different spinal cord lesions were investigated on the ability of cats to retrieve food with the forelimb from the bottom of a narrow horizontal or vertical tube. The test movements, which are known to depend on the cortico- and/or rubrospinal tracts (Gorska and Sybirska 1980a, b), are subdivided into the *target-reaching movement* by which the paw is brought in contact with the food, and the *food-taking movement*, consisting of toe grasping and paw supination which are components of the movement by which the cat brings the food to the mouth.

The following lesions were made ipsilaterally to the tested limb:

1. A dorsal lesion in C5 interrupting the cortico- and rubrospinal input to the forelimb segments (four cats).
2. A dorsal lesion in C2 interrupting the cortico- and rubrospinal input to the C3–C4 PNs and the forelimb segments (four cats).
3. A ventral lesion in C5 interrupting the descending axons of the C3–C4 PNs and bulbospinal fibres to forelimb segments (three cats).
4. A ventral lesion in C2 interrupting bulbospinal fibres and ascending collaterals from the C3–C4 PNs (two cats).
5. A ventral lesion in C2 and a dorsal lesion in C5 sparing the cortico- and rubrospinal input to the C3–C4 PNs and their axons to the forelimb segments (one cat).
6. Hemisection in C2, except the dorsal column (two cats).

Severe short-term impairment of the food-taking movement was observed after lesion 1, 2, 5, or 6, but not after lesion 3 or 4. The target-reaching movement was severely impaired after lesion 2, 3, or 6 but not after lesion 1, 4, or 5. The defect after lesion 3 was not in the lifting of the limb but in the appearance of gross ataxia in aiming at the target with the paw.

It is postulated that the C3–C4 PNs can transmit to forelimb motoneurones the command for target-reaching but not for food-taking, which depends on direct activation of neuronal networks within the forelimb segments from the cortico- and/or rubrospinal tracts.

Long-term recovery of the limb lifting in the target-reaching movement occurred after all lesions. A partial recovery of the food-taking movement occurred after lesions 1 and 2 but not after lesions 5 and 6 which also included the ventral part of the lateral funicle; the recovery is assumed to depend on reticulospinal control of networks within the forelimb segments.

Key words: Forelimb movements – C2 or C5 spinal cord lesions – Cortico- and rubrospinal tracts – C3–C4 propriospinal neurones – Segmental interneurones

Previous reports in this series have dealt with the organization of connexions to the C3–C4 propriospinal neurones (PNs) which project directly to forelimb motoneurones. They are characterized by extensive monosynaptic excitatory convergence from different higher motor centres and from forelimb afferents

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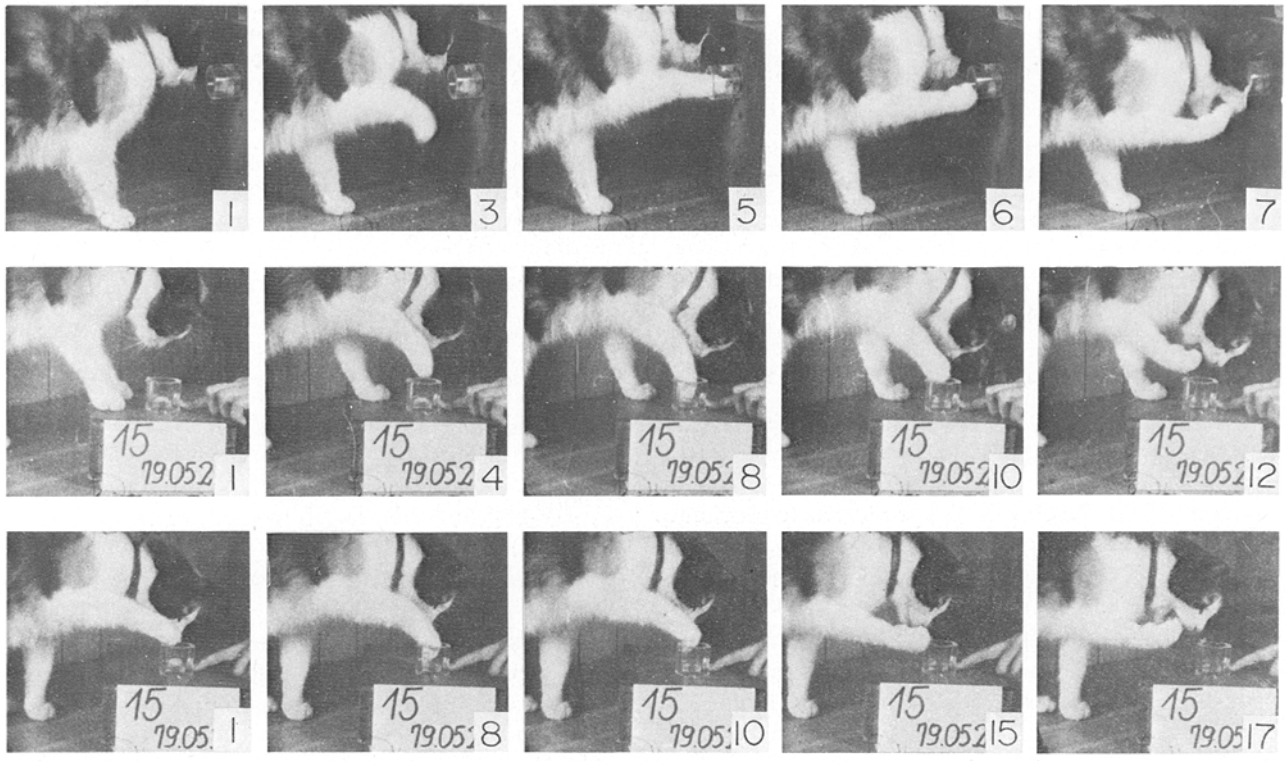

Fig. 1. Normal cat showing the movements performed in the HT and VT tests. *Upper row:* HT test. *Middle* and *lower rows:* VT test

(Illert et al. 1977, 1981); transmission is also controlled by different inhibitory mechanisms (Lundberg 1979). It has been postulated that the function of the C3–C4 PNs is to transmit excitation from higher centres to forelimb motoneurones and that the convergent effects from the forelimb, excitatory and inhibitory, have a regulatory role.

It is an attractive feature of the C3–C4 PNs that they can be isolated experimentally from other neuronal systems controlling forelimb motoneurones by spinal cord lesions. Such lesions were in fact originally used to define the C3–C4 PNs (Illert et al. 1977) but can also be used to investigate their role in motor behaviour. The present analysis is based on the fact that the axons of the C3–C4 PNs have a more ventral location in the spinal cord than the cortico- and rubrospinal tracts (Illert et al. 1977, 1978) which allows differential transection of either system of input to the forelimb segments.

It was necessary to investigate motor performance known to depend on the cortico- and/or rubrospinal tracts. Fortunately, suitable tests were available from the work by Gorska and Sybirska (1980a) who showed that forelimb movements required to retrieve a small morsel of food placed inside a narrow tube were severely impaired after pyramidotomy and to some extent also after rubral lesions. We have compared the performance in these tests before and after different spinal cord lesions. It will be shown that the C3–C4 PNs are important for the precise forelimb movements through space by which the cat reaches a target with the paw. On the other hand, the food-taking movement, implying not only grasping of the food with the toes but also supination of the paw which used to bring the food to the mouth does not depend on the C3–C4 PNs but on interneuronal systems within the forelimb segments directly activated by the cortico- and/or rubrospinal tracts. Preliminary reports have been published (Alstermark et al. 1979a, b).

Methods

Behavioural Tests

Throughout the investigation we used tests 3 and 4 by Gorska and Sybirska (1980a). In the former the cat retrieves a piece of fish from the bottom of a horizontal tube (30 mm diameter, 35 mm deep) which is placed at shoulder level 150 mm above the floor (Fig. 1) (henceforth HT test). In test 4 the same tube is placed vertically on a low platform 90 mm above the floor (henceforth VT test). To reach the tube in the HT test the cat lifts the paw from the ground and wrist extension occurs during the lifting phase. When the opening is reached the paw is inserted into the tube in a pronated position. The morsel of fish is then grasped by a

combined movement of plantar flexion and adduction of the toes. Supination of the paw occurs during the withdrawal from the tube by retraction of the whole limb. During further supination combined with wrist flexion the cat then brings the food to the mouth; coordinated head movements are also made (Fig. 1). In the VT test the cat directs the paw to the tube with the wrist in a more or less flexed position (Fig. 1). The paw is then brought downward to the bottom of the tube and the food is grasped and brought to the mouth as in the HT test. In both these tests the normal cat removes the food with the greatest ease in one swift movement independently of its original position in relation to the tube. In both tests the movements were made in a variety of postural situations also when lying down (Fig. 6). We also used, but less regularly, test 1 by Gorska and Sybirska (1980a), in which the cat is in a cage behind a grid of vertical bars 5 cm apart through which it reaches out for food placed 5 or 10 cm in front of the bars.

The experimental procedure followed closely that described by Gorska and Sybirska (1980a). Co-operative cats were chosen and the preoperative training lasted about one week. In each test 10–20 pieces of fish were placed successively in the tube and eaten after retrieval. The side of the best limb was chosen for surgery; when necessary usage of the other limb was discouraged with a sleeve on the paw. Testing was resumed 6 to 7 days after surgery and continued for many months; during the first month five times a week, during the second twice a week and later usually once a week. Moving pictures (32/s) were taken frequently. Postoperatively the cats were reinforced even after partial movements in order to prevent extinction.

Surgery

The surgery was made aseptically under sodium-pentobarbital (Nembutal, Abbott, 35 mg/kg) anaesthesia after 0.5 mg atropine sulfate premedication. During the surgery as well as for several hours afterwards the respiration and the electrocardiogram were recorded and the cats were at the same time connected to a respirator, which could be started immediately, if necessary. The cervical vertebral column was exposed dorsally through a longitudinal incision of the skin and longitudinal division and retraction of the cervical musculature. The spinal cord was exposed through partial removal of the dorsal arch of the relevant vertebra(e). The dura was opened and the dorsal spinal cord lesions were made with watchmakers forceps under visual guidance with the aid of a binocular dissecting microscope. The lesions involving the ventral part of the spinal cord only, were made with a minute hook which was inserted through an opening in the pia, which had been made with watchmakers forceps. The wounds were closed with separate muscle and skin sutures, and the cats were given procaine penicillin for 3 days postoperatively. Fluid and electrolyte therapy was given by subcutaneous injections until the cats started to drink spontaneously. Micturition and defecation were sustained, whenever necessary. The cats were returned to their home cages as soon as possible after the operations and generally within 3 days.

Electrophysiology

Investigations of behavioural defects after corticospinal lesions have revealed that a remarkable recovery can occur both in monkeys and in cats if only a small fraction of the fibres escape the lesions (cf. Lawrence and Kuypers 1968; Gorska and Sybirska 1980b). Histological controls of lesions in the spinal cord do not suffice to decide whether or not a lesion in the dorsal part of the lateral funicle has caused complete transection of the cortico- and rubrospinal tracts.

In the majority of the animals the effect of the lesions on conduction in the corticospinal and rubrospinal tracts was controlled electrophysiologically at the end of the observation periods. After laminectomy in Th 8–10 the dorsal column was removed over a few segments and the remaining spinal cord divided in two halves from which bipolar recording of descending volleys was made (Laporte et al. 1956). Tungsten electrodes were inserted in the pyramids to a position where corticospinal volleys were evoked at 10 μA (cf. Lance 1954; Illert et al. 1976a); 200 μA stimuli gave maximal volleys. Tungsten electrodes were also inserted bilaterally in the red nuclei (Hongo et al. 1969). The main comparison of the discharges in the two spinal halves was made with stimulation ventral to or in the red nuclei. By stimulation of the interpositorubral fibres in this region a monosynaptic rubrospinal volley is evoked in the contralateral spinal half (Baldissera et al. 1972b). In the same region axons from the opposite red nucleus are also stimulated after their crossing giving a direct rubrospinal volley in the ipsilateral spinal half. The ventral stimuli were preferred since they provide a convenient way of obtaining *maximal* rubrospinal volleys (Baldissera et al. 1972b). Stimulation in and around the red nucleus can also evoke disynaptical discharges in descending pathways located in the ventral spinal cord (Baldissera et al. 1972a). These discharges are easy to differentiate from rubrospinal volleys since they are also evoked, and at lower strength, from regions dorsal to the red nucleus. As an added precaution we also at the end of the experiments divided the spinal halves in dorsal and ventral quadrants and confirmed that the "extrarubral" discharges were conducted ventrally.

The size of the bipolarly recorded volley depends on the degree of extracellular shunting. To compare the discharges on the two sides we used the method described by Clamann and Henneman (1976) and measured the reduction of the amplitudes of the discharges when a 5 KΩ resistor was connected in parallel with the spinal half.

Although this investigation relates to the forelimb we chose to record the descending discharges in the thoracic spinal cord since it was much more difficult to obtain reliable records in the forelimb segments where the spinal halves are difficult to dissect. For control we recorded cortico- and rubrospinal volleys in C7 with a tungsten electrode and compared the amplitudes of the discharges with those evoked after collision with a maximal antidromic volley from Th8. From many different recording sites in the dorsal part of the lateral funicle we found identical amplitude relationship between these discharges, suggesting that there is no differential location of fibres projecting to fore- and hindlimb segments. As an additional precaution we also recorded in the forelimb segments in some of the lesioned cats. As control for the corticospinal tract we used the negative cord dorsum potential and for the rubrospinal tract the positive potential recorded by a tungsten electrode inserted into the dorsal part of the lateral funicle (cf. Fig. 7).

Histology

The electro-physiological acute experiments, which terminated the behavioural testing, were finished by passing current through the brain stem electrode network at relevant sites. The cats were then infused intra-arterially with physiological saline followed by 10% buffered formalin solution. The cervical vertebral columns and spinal cords were removed in one piece and placed in 10% buffered formalin solution for several weeks before the lesioned segments were extricated, embedded in paraffin and cut serially in 16 μm transverse sections. The sections were stained according to Klüver and Barrera (1953) and reconstructions of the lesions were made by projecting the extent of the degenerated tissue of the various relevant sections on to drawings of the spinal cord. The brain stems were removed and placed in 10% buffered formalin

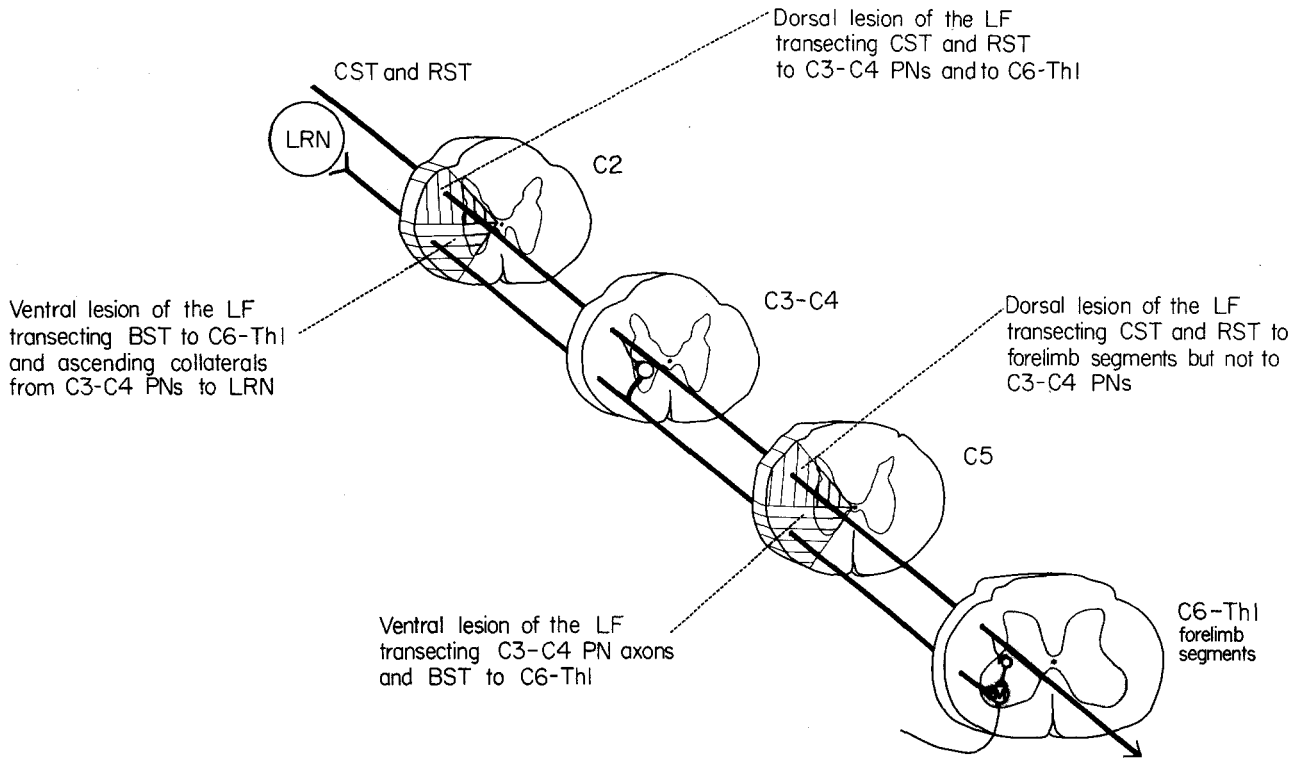

Fig. 2. Schematic drawing of the cervical spinal cord showing the different lesions and the trajectories of the relevant fibre tracts. Abbreviations: Corticospinal tract – CST; rubrospinal tract – RST; bulbospinal tracts – BST; propriospinal neurone – PN; lateral reticular nucleus – LRN

solution before being serially freeze-sectioned in 100 μm transverse sections. The positions of the electrode tracks were reconstructed and cell counts were made after Nissl staining, and representative sections were photographed.

Results

The dominating excitation of the C3–C4 PNs appears to be from the corticospinal and rubrospinal tracts and since both these tracts are located dorsally in the lateral funicle while the axons of the C3–C4 PNs descend in the ventral part of the lateral funicle (Illert et al. 1977, 1978), it is possible to make differential lesions as shown in Fig. 2. The descending axons of the C3–C4 PNs can be interrupted by a ventral C5 lesion which does not interfere with the cortico- and rubrospinal fibres to the forelimb segments (C6–Th1). A dorsal C5 lesion, on the other hand, does not interfere with the axons of the C3–C4 PNs but eliminates the direct cortico- and rubrospinal input to the forelimb segments. It is also possible to compare the effects of transections of the cortico- and rubrospinal tracts in the C2 and C5 segments, in the former case removing the input both to C3–C4 PNs and the forelimb segments, in the latter only to the forelimb segments.

In addition to the four lesions indicated in Fig. 2 we have in one cat combined a ventral C2 lesion with a dorsal C5 lesion and also in two cats investigated the effect of hemisection in C2. Histological controls of the lesions are shown in Fig. 3 and in Figs. 4, 7, 9 together with the electrophysiological controls.

The behavioural effect of each type of lesion will be described below in six subsections, each followed by a short summary. The results obtained in the HT test are summarized in Table 1 which lists the deficits in the test movements and the recovery. Distinction is made between the *target-reaching movement* by which the animal reaches the food with the paw, and the *food-taking movement*. The latter consists of toe grasping and supination of the paw which are part of the movement which enables the animal to bring the food to the mouth (cf. below). The purpose of Table 1 is to give an overview of the main results. The table does not include qualitative differences of the movements after the different lesions. For example, the execution of target-reaching after the dorsal C2 lesion was very different from that found after the C2 hemisection (cf. below). The results obtained with

Table 1

	Target-Reaching movement			Food-Taking Movement			
Lesion	normal/ defective	Deficiency lifting/ aiming	Recovery (aiming) near complete/ partial	normal/ defective	Recovery near-complete/ partial	Postsurgical observation period, months	Lesion Intended size/ too large or small
C5D: 1	+/			/+	+/	7	/S
C5D: 2	+/			/+	/+	7	+/
C5D: 3	+/			/+	/+	4.5	+/
C5D: 4	+/			/+	+/	10	+/
C2D: 1	/+	+/+	/+	/+	/+	6	/L
C2D: 2	/+	+/+	/+	/+	+/	2	+/
C2D: 3	/+	+/+	/+	/+	/+	8.5	/L
C2D: 4	/+	+/+	/+	/+	+/	6	+/
C5V: 1	/+	/+	0	+/		6.5	+/
C5V: 2	/+	/+	0	+/		6	+/
C5V: 3	/+	/+	0	+/		9	+/
C5V: 4	/+	/+				5.5	
C2V: 1	+/			+/		6	+/
C2V: 2	+/			+/		5.5	+/
C2V C5D	+/			/+	0	8.5	/L
C2H: 1	/+	+/	+/	/+	0	8	/S
C2H: 2	/+	+/	+/	/+	0	7	/S
C3H (after C2V C5D)	/+	+/	/+	–	–	2	/L

the VT test are equally important but more difficult to quantify since individual animals developed different compensatory motor strategies (cf. below).

A. Dorsal C5 Lesion

This lesion was made in four cats and the histological controls are shown in Fig. 3 (C5D 1, 2, 3, 4) and in Fig. 4H. Figure 4H shows that the dorsal C5 lesion gave considerable retrograde cell loss in the contralateral red nucleus. In the ventrolateral part of the red nucleus there was a profound loss of large and medium size cells, while many cells remained in the dorsomedial region which corresponds closely to the forelimb region of the red nucleus as delimited by Pompeiano and Brodal (1957); it is important for the interpretation of our results that this region was largely spared from retrograde cell loss after transection of the rubrospinal axons in C5 (cf. Discussion). The electrophysiological controls (Fig. 4) revealed complete transection of the corticospinal and rubrospinal tracts in cats 2–4. The corticospinal tract was completely transected also in cat 1 while 60% of the rubrospinal tract escaped the lesion in this cat.

In these electrophysiological controls simultaneous recording was made from both dissected spinal halves (except the dorsal columns) in Th8–9. Records in Fig. 4A–E are from cat 2; the effect of stimulation of the pyramid and the red nucleus on either side was compared, traces marked a are from the right and b from the left spinal half. The effect of pyramidal stimulation in Fig. 4D and E offers no problem since stimulation of the left pyramid evoked no discharge in the right spinal half showing complete transection of the corticospinal tract. The discharges evoked by rubral stimulation requires a more detailed description since it has been shown (Baldissera et al. 1972a, b) that stimulation in the red nucleus may give:

(i) By stimulation of interpositorubral fibres a monosynaptically evoked discharge in the contralateral rubrospinal tract as shown in Ab (arrow in upper pair) at 100 μA stimulation of the right red nucleus;

(ii) A directly evoked volley in the contralateral rubrospinal tract, illustrated in Ab (arrow in lower pair) at 200 μA (note latency shortening from corresponding arrow-marked trace at 100 μA);

(iii) A directly evoked volley in the ipsilateral rubrospinal axons stimulated after their crossing at the level of the red nucleus. This discharge is exemplified by the trace in Bb;

(iv) Discharges conducted in ventral spinal pathways which are monosynaptically evoked from extrarubral systems. The discharge in Aa at 200 μA belongs to this category (cf. legend).

Observe in records Ba the absence of discharge (i) and (ii) in the right spinal half on stimulation of the left red nucleus and in records Aa the absence of discharge (iii) in the right spinal half on stimulation of the right red nucleus. The lack of these three discharges shows that *the rubrospinal tract was completely inter-*

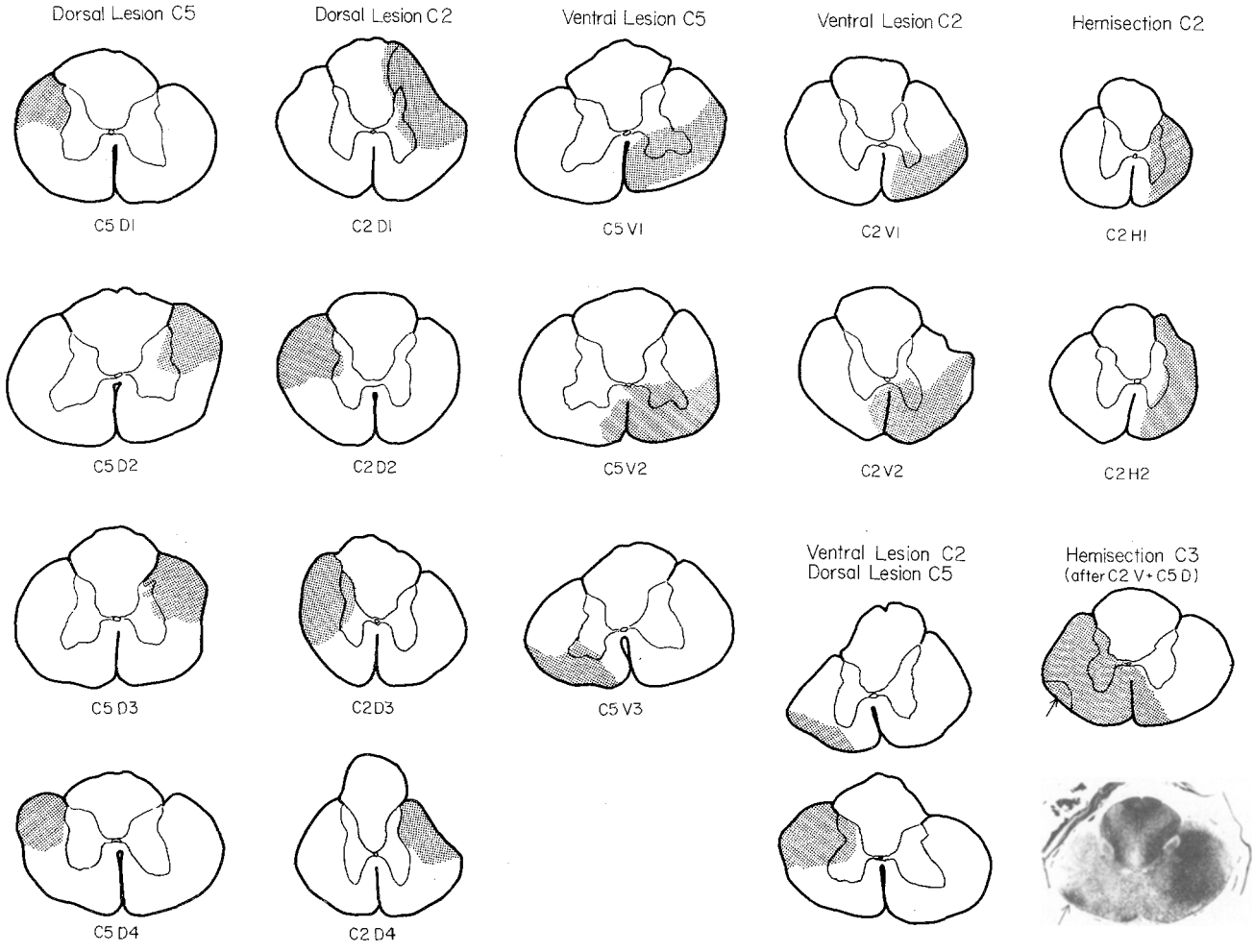

Fig. 3. Histological control of the maximal extension of lesions in all cats assessed from the lesions proper and the degeneration caudal to it. Lower right microphotograph is from rostral C4 less than 1 mm caudal to the C3 hemisection. Note the undegenerated fibre bundle (arrow) which is assumed to originate largely from cell bodies of C3–C4 PNs located caudal to the hemisection. The location of these axons is also indicated (arrow) in the drawing above the microphotograph. In unlesioned animals this region extends somewhat more dorsally (Illert et al. 1977, 1978); C3–C4 PNs with more dorsal axons presumably degenerated as result of the previous combined C5 dorsal lesion which extended somewhat ventrally, and the ventral C2 lesion which transected the ascending collaterals (Alstermark et al. 1981)

rupted by the lesion. Repetitive stimulation was used in Fig. 4C to show that the recording conditions in both spinal halves were equivalent (cf. legend).

All cats were able to lift and insert the paw into the horizontal as well as the vertical tube in the first trial (7 days); already in the beginning of the 2nd week insertion into the horizontal tube was made with a movement that was virtually indistinguishable from the normal one (Fig. 5). The VT test seemed to be somewhat more difficult and although all cats inserted the paw after 7 days it was a few days longer until they were able to reach the bottom.

Although the cats successfully reached the food with their paw in both tests they were unable to grasp and remove it in the normal way. They nevertheless succeeded in removing the food from the tube by other techniques. The food was raked out from the horizontal tube, dropped to the floor and was retrieved with the mouth (Figs. 5, 6B). In the VT test the cats pressed the food against the wall of the tube with the paw and pulled it up (Fig. 6A). In both tests the cats managed in removing the food either in the session when they reached it or in the following session.

Even if the cats were unable to grasp the food, hold it and take it to the mouth in the normal way, some movements appeared which seemed to represent fragments of the normal ones. In cats 2 and 3 very small paw supination without toe flexion was observed 2–3 weeks after surgery and slight toe flexion appeared about one week later. In cat 4 traces

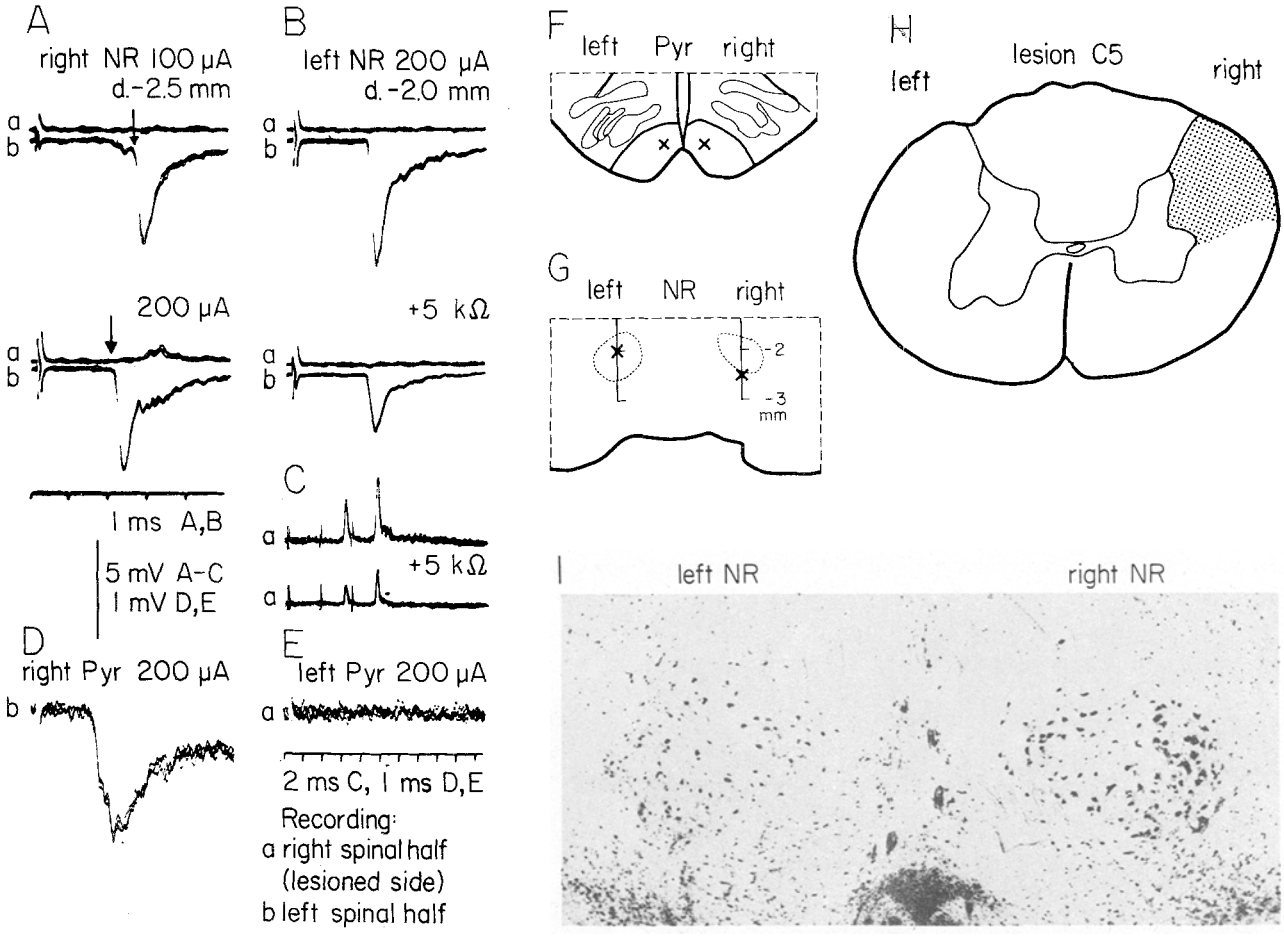

Fig. 4A–I. Electrophysiological and histological controls from cat C5D2. **A–E** Recording from the dissected spinal halves (except dorsal column), traces marked **a** and **b** are from right and left spinal halves, respectively; negative deflections are upwards in recordings from the right and downwards from the left spinal half. Side of stimulation in the red nucleus and pyramid as well as stimulus strengths are indicated above the records. The different discharges in the left spinal half are explained in small print section in text. After recording **A–C** the spinal halves were divided in dorsal and ventral quadrants; as expected (Baldissera et al. 1972a, b) the discharges recorded from the left spinal half in **A, B,** and **D** were conducted in the dorsal quadrant while the synaptically evoked discharge in the right spinal half (**Aa** at 200 µA and **Ca**) was exclusively in the ventral quadrant. The similarity in recording conditions in the two spinal halves was ascertained by comparing the short-circuiting effect by 5 KΩ on the discharge in **B** and **C;** note that a train of stimuli was applied in **C,** which by temporal facilitation gave a discharge adequate for this test

of both components of the movement were observed already during the 2nd week. In the former two cats there was only a slight and slow further improvement; toe grasping in particular remained deficient. At the end of the observation periods both cats sometimes brought the food to the mouth with the paw but they probably managed by hooking the food on the claws rather than with proper toe grasping. In cat 4 the recovery was also slow but after 6 months it could use proper toe grasping to take the food. In cat 1 with an incomplete lesion of the rubrospinal tract the recovery was faster and more complete. Slight toe grasping and paw supination were observed the 2nd week and the movements gradually improved so that food was taken to the mouth from the vertical tube already in the 5th week. After 4 months it could take the fish to the mouth from the horizontal tube in more than 50% of the trials; the toe grasping and paw supination required for this movement all appeared to be normal.

In summary, transection of the cortico- and rubrospinal tracts in C5 did *not impair* the ability to make the precise forelimb movements through space which are required for the insertion of the paw into the tube. By contrast the lesion gave inability of grasping with the toes and the associated supination of the paw required to take the food to the mouth, but there was some recovery with time; a virtually complete recovery was found in one animal with partial transection of the rubrospinal tract.

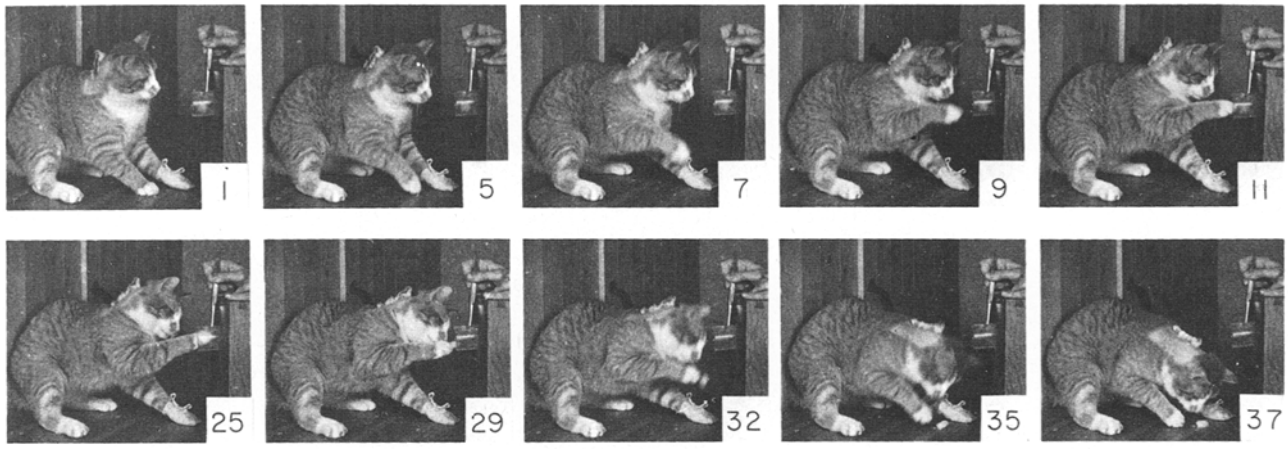

Fig. 5. Performance in the HT test of cat C5D2 8 days postoperatively. The photographs are taken from the moving film, speed 32 frames/s; frame number is indicated in each picture. Observe the precision in the reaching movement; the accurate timing of the wrist extension enabling the forepaw to be inserted in one swift movement. Note in the food-taking movement the virtually complete loss of toe grasping and paw supination resulting in an incapability of holding the morsel which drops (32) to the floor

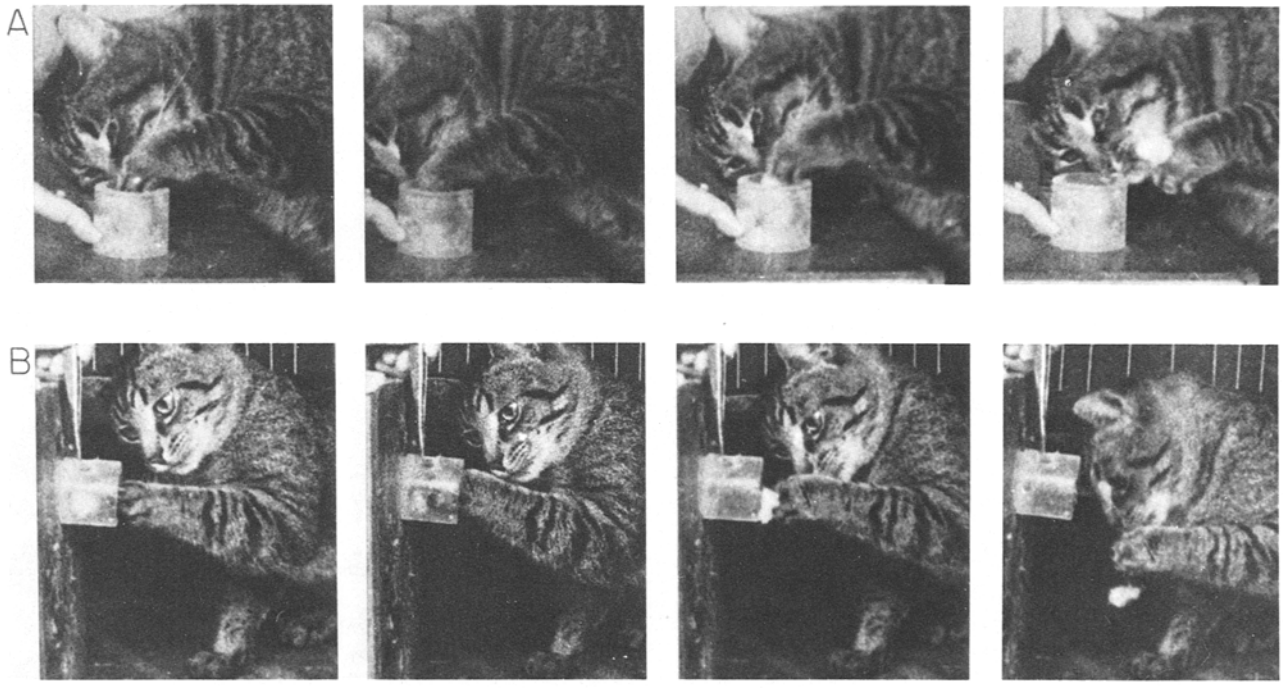

Fig. 6A, B. Performance in cat C5D3 2 months postoperatively in the VT (**A**) and HT (**B**) tests. Note absence of toe grasping and removal of food by raking and pressing movements. Observe in the right frames the morsel of fish in the air

B. Dorsal C2 Lesion

The histological controls of the four cats in this series are shown in Fig. 7G and Fig. 3 (cats C2D, 1, 2, 3, 4). Note that in cat 3 the lesion extended rather ventrally in the lateral funicle and that part of the dorsal column was damaged in cat 1. The lesion gave a virtually complete loss of large and medium sized cells, both in the fore- and hindlimb region of the right red nucleus (Fig. 7H). Electrophysiological controls as in Fig. 3 made in cats 1 and 2 revealed complete transections of the cortico- and rubrospinal tracts.

The records in Fig. 7 are from cat 2 in which we not only recorded from the dissected spinal halves in Th 8–9 but also in the forelimb segments where cord dorsum potentials as well as

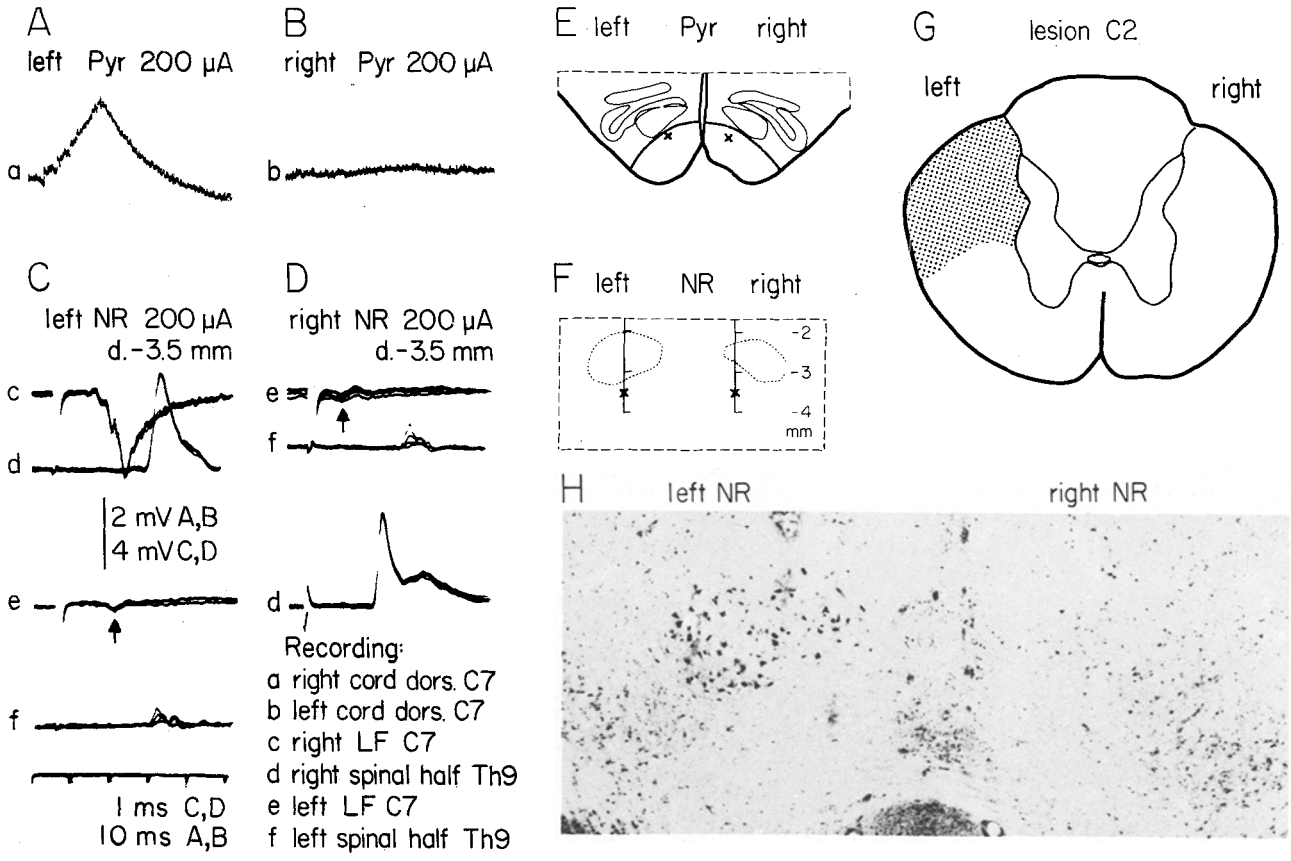

Fig. 7A–H. Electrophysiological and histological controls from cat C2D2 in which the effect of the lesion was controlled not only by recording from dissected spinal halves in Th8–9 as in Fig. 4 but also by recording in forelimb segments. Recording position in C7 or Th9 is indicated for each record with code (a–f) below record **D.** Upward deflection signals negative potential in all recordings. **A–B** Records of the cord dorsum potential in C7 on the right **Aa** (intact) and left **Bb** (lesioned) side when stimulating the contralateral pyramid. **C–D** Stimulation of the left and right NR as indicated. Recording was made on either side with a tungsten electrode inserted into the lateral funicle in C7 to a depth of 1.3 mm and from dissected halves in Th8–9. Arrow in **Ce** and **De** indicates the remote recordings (lesioned side) of the volley in the right (intact side) rubrospinal tract (see text). Controls as in Fig. 4 showed that the discharges in **Cf** and **Df** were mediated by ventrally located fibres. **E–F** Reconstruction of stimulus sites from histological sections, positions are indicated with crosses. **G** The maximal extension of the lesion in the white matter assessed from the degeneration caudal to the dorsal LF lesion in C2. Microphotograph **H** shows profound loss of large and medium sized cells in the entire right NR

recordings with a tungsten electrode inserted into the lateral funicle were used to test conduction in cortico- and rubrospinal fibres. For each trace the recording position is given (a–f, with key below record, Fig. 7D). The four different discharges (i)–(iv) defined above in the small print section in relation to Fig. 4 will be used to describe the effect of rubral stimuli. Stimulation ventral to the left red nucleus gave the expected synaptically evoked rubrospinal volley (i) in the right spinal half (Cd) but not the direct rubrospinal volley (iii) in the left spinal half (Cf); note that the ventral stimulus site (Fig. 7F) in the left red nucleus would be optimal for activating rubrospinal axons after their crossing. It is therefore concluded that the rubrospinal fibres projecting to the caudal spinal cord were completely transected by the C2 lesion. The discharges in Cf and Df are of type (iv), cf. legend. Records Cc shows the large rubrospinal volley recorded from the right lateral funicle in C7. There is no corresponding discharge in the left lateral funicle on stimulation of either the right or left red nucleus. The small potentials recorded from the *left* lateral funicle in Ce and De were recorded also outside the expected location of the rubrospinal tract and from the left dorsal column. These are remote records of the volley in the right rubrospinal tract as is also proven by the fact that the potential in Ce is time-linked to the synaptically evoked rubrospinal volley in Cd and the potential in De to the direct rubrospinal volley (discharge iii) in Dd; a reverse time relationship would have been expected for a discharge in rubrospinal fibres escaping the lesion in the left lateral funicle. It is therefore concluded that the lesion gave complete transection also of the rubrospinal fibres to the forelimb segments.

The movements required to lift and insert the paw into the tube were severely impaired both in the HT and VT tests but with considerable differences in recovery time between the individual cats. In the HT test cats 1, 3, and 4 performed infrequent phasic lifting movements during the 2nd week but only managed to lift the paw a few centimetres from the floor. The ability to lift the limb gradually recovered and the opening of the tube was reached during the 4th week. At the same time these movements

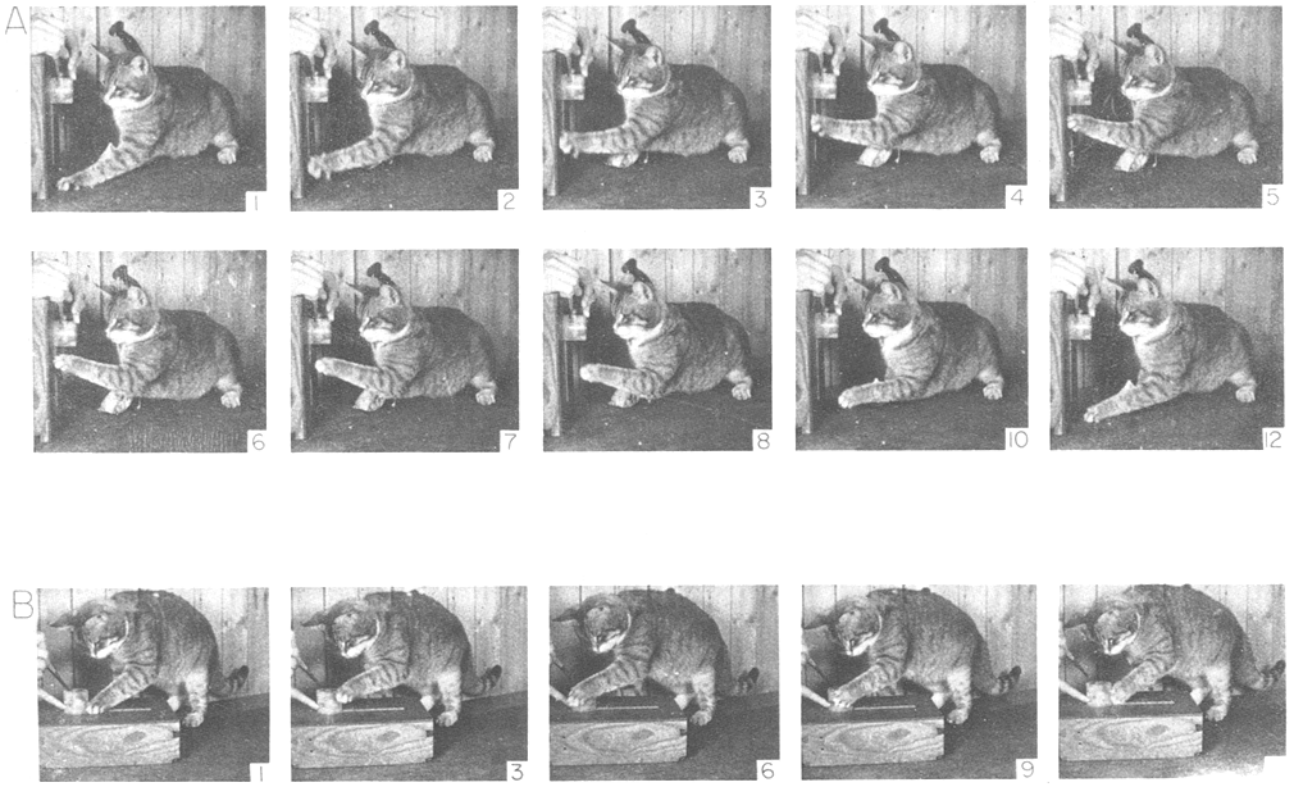

Fig. 8A, B. Performance in cat C2D2 18 days postoperatively. The photographs are consecutive (32 frames/s). **A** The movement consists of a rapid phasic lifting of the forelimb, note also wrist extension throughout the lifting phase and toe abduction. The paw did not reach tube level and the limb appears to drop passively to the floor, now without toe adduction. **B** shows an unsuccessful trial in the VT test. Observe the lack of precision when the cat tries to reach the tube opening and absence of wrist flexion

became more tonic allowing the cats to maintain the limb in an elevated position. Soon afterwards they sometimes managed to insert the paw into the tube and occasionally remove the fish by raking. Even if the lifting movement thus recovered it was characteristic that the aiming at the tube opening lacked the precision found in normal cats or in cats with a C5 dorsal lesion. They frequently placed the paw outside the tube or on the edge of the tube and could not make the proper correcting movements which are so easily and quickly made by normal cats. Instead of a purposeful correction the C2 lesioned cats often made many phasic scratching-like movements in the vertical direction while maintaining the paw in the wrong position relative to the tube opening. A gradual improvement in the ability to reach the food then occurred over several months and at the end of the observation periods all three cats had fair target-reaching movements although the aiming never reached the same precision as that in normal cats or cats with a dorsal C5 lesion.

A faster recovery of limb lifting was found in cat 2. Already in the beginning of the 2nd week it made occasional small lifting movements in the HT test and at the end of this week the lower edge of the tube was occasionally reached but the movement was phasic and jerky. The limb appeared to fall down from the maximally elevated position (Fig. 8A, successive frames 32/s). In the beginning of the 3rd week the paw was sometimes lifted to the opening of the tube and could be maintained for a short time but without insertion. At the end of this week the cat sometimes inserted the paw and it first succeeded in raking away the food during the 4th week. Also at this stage the aiming in most trials lacked precision and the scratching-like movements close to the tube opening were pronounced.

In the VT test cats 1 and 4 did not succeed in inserting the paw in the vertical tube during the first 5 weeks (Fig. 8B). At the end of this period they were able to rake away a piece of food only when it was placed at the level of the opening of the tube. In the 7th week the paw was inserted half distance to the bottom, which occasionally was reached in the 12th week and then they also managed to remove the food either by pressure against the tube as described in section A or by hooking on the claws. There was a similar slow recovery in cat 4 but the results were

more difficult to assess since the cat was unwilling to participate in this particular test. Cat 3, on the other hand, already the 12th day sometimes managed to insert the paw half distance to the bottom which was reached on the 20th day. At the same time removal was achieved by hooking on the claws. When cats 1, 3, and 4 ultimately succeeded in removing the fish in the VT test they used another strategy than the normal cats and the cats with a dorsal C5 lesion. The latter could stand at some distance from the tube and stretch the limb forward in a more or less horizontal direction with the wrist in a flexed position. When cats 1 and 3 used the horizontal limb movement they could only rake away the fish when it was placed in the upper part of the tube. Unlike the normal cats they seemed unable to combine the wrist flexion with a forward movement of the extended limb. They succeeded in inserting by standing closer to the tube and using a more perpendicular downward movement of the whole limb. Cat 2 reached the opening of the vertical tube in some trials at the end of the 2nd week but often failed (Fig. 8B). After three weeks this cat frequently reached the bottom and sometimes succeeded in removing fish by hooking it on the claws. Contrary to cats 1, 3, and 4 this cat used not only the "perpendicular technique" but also managed like a normal cat to stand at some distance from the tube, extend the limb towards the tube in a more horizontal direction and insert the paw with the wrist in a flexed position.

With regard to the ability to bring food to the mouth the cats 1, 3, and 4 resembled the animals with a dorsal C5 lesion. A few weeks after the partial recovery of the ability to reach the food with the paw some toe grasping and also paw supination appeared. There was some improvement over the next few months but toe grasping, in particular, remained weak and to the end of the observation periods (5.5 and 10 months) cats 1 and 3 never managed to hold the food securely with the toes. However, when they hooked the fish with the claws, supination and wrist flexion was sufficiently good to allow them to take the food to the mouth. The recovery in cat 4 was also slow but somewhat more complete since this cat after 6 months could take the food by grasping and hold it.

A faster and more complete recovery was observed in cat 2. Toe flexion and also some supination was observed after 1 month. These movements gradually improved and at the end of the 2nd month the cat managed to take the food to the mouth in both tests in a normal way although it occasionally lost it. These findings were unexpected in view of the previous results by Gorska and Sybirska (1980a, b) but our suspicion that the lesion was incomplete was falsified by the electrophysiological control (Fig. 7).

In summary, the dorsal C2 lesion gave severe impairment of the movements required for lifting the limb and inserting the paw into the tube. A gradual slow recovery occurred and the food was reached with the paw after 3–12 weeks, but the aiming at the tube opening lacked the normal precision. Toe grasping and paw supination required to take the food to the mouth were absent for many weeks. The subsequent recovery resembled that observed after the corresponding C5 lesion; a faster and more complete recovery was found in one of the cats.

C. Ventral C5 Lesion

This lesion was made in three cats and the histological controls are shown in Fig. 9K and Fig. 3 (cats C5V 1, 2, 3). All cats were controlled electrophysiologically. Transmission in the rubrospinal and corticospinal tracts appeared to be normal on the lesioned side. This is illustrated in Fig. 9 (cf. legend) showing antidromic field potentials in both red nuclei, descending discharges evoked from two sites within and ventral to the red nuclei (Fig. 9C–F) and the discharges evoked from the pyramids (Fig. 9G–H). Cat 3 was used in another experimental series and tried in the HT and VT tests 2 months after surgery. It is included in the series since its motor behaviour was identical with that of the two other cats to which the following description refers.

Cats 1 and 2 were tested 6 days after surgery and were able to insert the paw and remove the fish both in the HT and VT tests. However, even if the cats could lift and insert the paw into the tube there was from the onset a striking ataxic defect in the movement showing up as a very frequently occurring inability in precise aiming at the tube opening. The paw was usually placed outside the tube or at the edge of the tube, the correcting movement then very often went slightly too far in the opposite direction as is shown in Fig. 10A. The correcting movements were made close to the tube opening with extended wrist and the cats usually managed to insert after one or two attempts. If the cat did not succeed to insert after three to five attempts, it withdrew the limb and immediately tried again. The initial error in aiming could be in any direction above, below or sideways of the tube opening. The ataxia was pronounced in the HT test, but was observed also in the VT test. In both tests the ataxia persisted inside the tube which gave difficulty in grasping the food in the VT test. The cats solved this problem by placing the paw with the fifth toe outside the edge of the tube, thus stabilizing the position of the paw (Fig. 10B).

Both in the HT and the VT tests there was some improvement with time. After a few months the

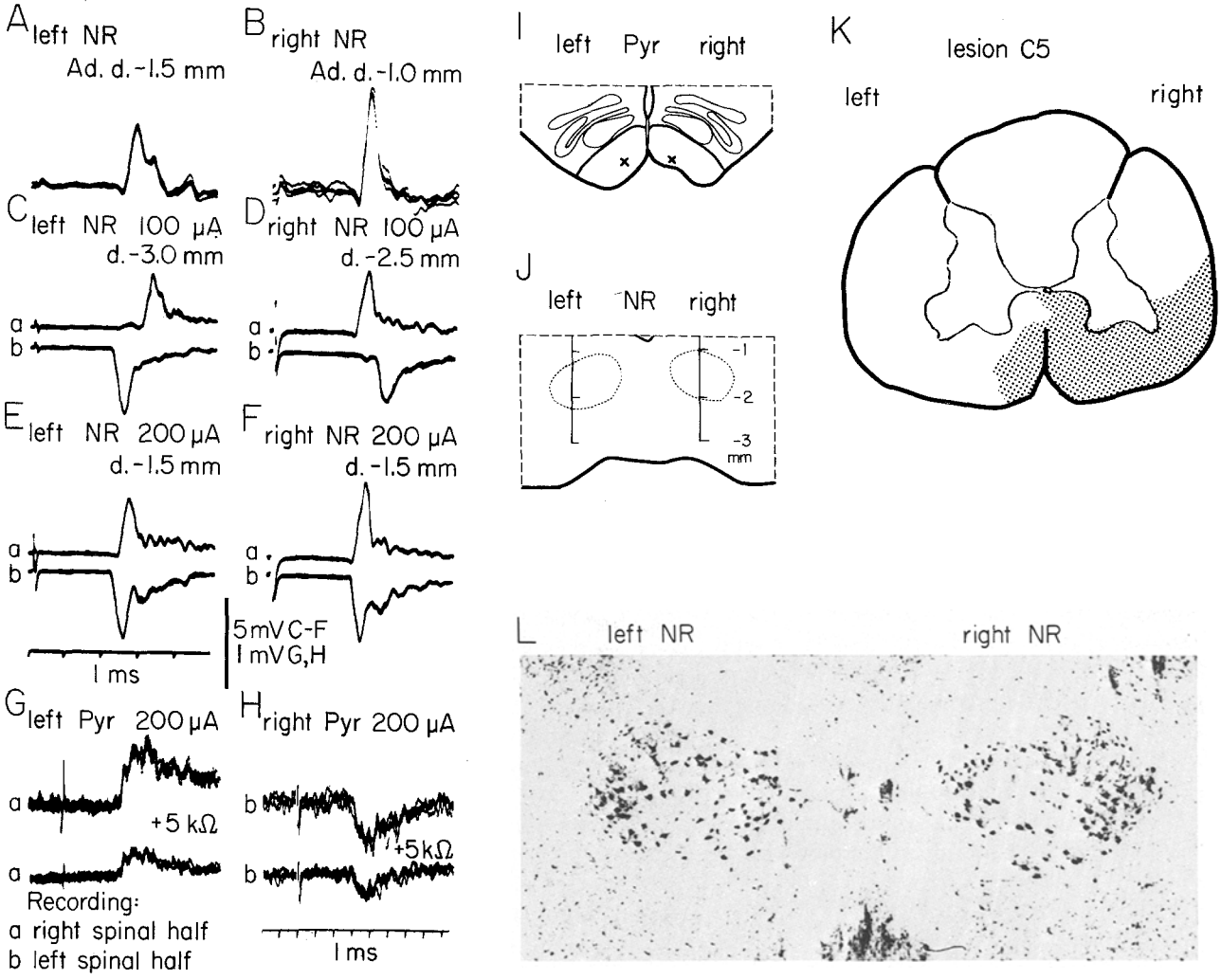

Fig. 9A–L. Electrophysiological and histological control from cat C5V2. **A, B** The antidromic field potential evoked in the left and right NR by stimulation of the contralateral spinal half in Th9. **C–H** Volleys recorded in the dissected spinal halves in Th9 on stimulation of the contralateral NR (**C–F**) and Pyr (**G, H**). Note the difference in depth of stimulating sites in **C, E** and **D, F**. Observe the equal effect of shunting with 5 kΩ in **G** and **H** (cf. Methods). **I, J** Histological reconstruction of the stimulating electrode tracks in NR (only depth scale) and Pyr (crosses). **K** The maximal extension of the lesion in the white matter assessed from degeneration caudal to the ventral LF lesion in C5, notice the partial involvement of the contralateral ventral funicle. **L** Microphotograph of NR demonstrating the similarity in cell density on the left and right side

ataxia was less pronounced if the cats were in a fixed position relative to the target, but if the target was moved sideways the ataxia reappeared. Tested in this way the ataxia remained at a virtually constant level through the entire 6 months observation periods.

The ataxia also appeared in test 1 particularly when the food was placed 10 cm in front of the bars. There was some improvement in the 2nd month but even after 5 months one of the cats averaged more than two movements with the paw in the target area for each attempt to reach the food. The other cat showed rather little ataxia in this test after a few months but it reappeared and was very pronounced if the fish was placed on a turntable and moved slowly in front of the animal. This task offered no problem for a normal cat or for cats with a dorsal C5 lesion.

Already in the first session when the cats reached the food with the paw, they performed the toe flexion and supination used in grasping the food and bringing it to the mouth (Fig. 10B). Both cats could grasp the fish and lift it to the mouth in the 2nd week but one of them often kept the paw on the floor while eating from it. Although this cat appeared to have good ability in grasping the food it continued throughout to eat with the paw at the floor level or lifted a few cm while the other cat usually ate the food from the paw at the tube level (HT test).

In summary, after a ventral C5 lesion the cats

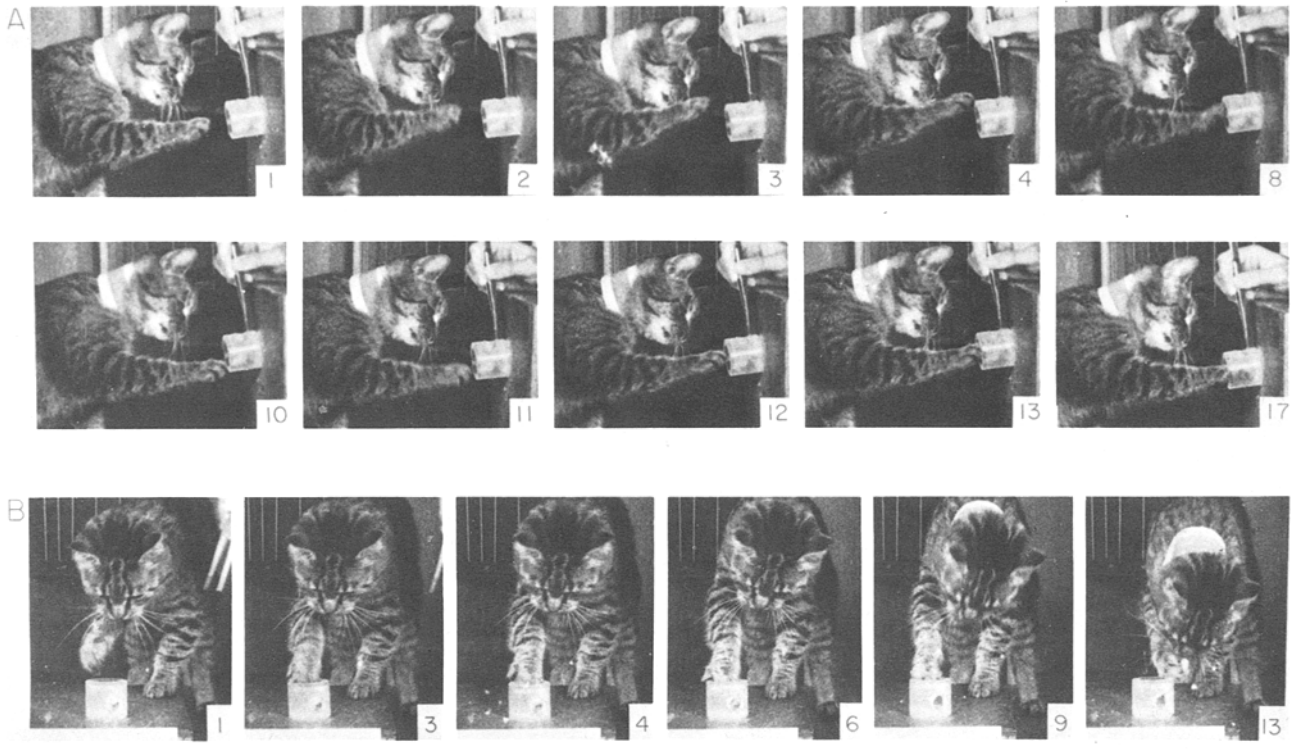

Fig. 10A, B. Motor performance in cat C5V1 one month postoperatively. The photographs are taken from the moving film 32 frames/s. Frame number is indicated in each picture. **A** The error in the trajectory of the reaching movements is seen already in frame 2, giving an overshoot. Corrective movement results in an undershoot (frame 10) before insertion (12–17). **B** To maintain the position during grasping the cats used their fifth toe to stabilize the paw (6). The retained ability of grasping is shown in frame 13. The morsel is grasped with toe flexion and brought to the mouth with the paw supinated

retained the ability to grasp food and bring it to the mouth showing virtually normal toe flexion and paw supination. They also retained the ability to lift the limb but a severe ataxic defect was evident when aiming at the target. The ataxia remained at an almost constant level for many months.

D. Ventral C2 Lesion

Histological controls from the two cats with ventral lesions at this level are shown in Fig. 3. Electrophysiological controls at the end of the observation periods showed that the lesions did not interfere with conduction in the corticospinal and rubrospinal tracts (not illustrated).

When the cats were first tested 6 and 7 days after surgery the movements in the HT and VT tests were performed in the same way as before operation including grasping the food and taking it to the mouth. They were examined for 5 and 6 months and throughout these periods there was no indication that the lesions had influenced the motor performances in the tests.

In summary, a ventral C2 lesion did not influence the movements required to reach, grasp and bring food to the mouth.

E. Ventral C2 and Dorsal C5 Lesion

The lesions were made in one operation and their extent is shown in Fig. 3. Because of severe postural disturbance the cat was tested during the second week with the body supported in a hammock. Already in the first trial after 8 days the cat could lift the limb and reached the tube level with the paw in the HT test but did not insert it. After 11 days the paw was inserted in one swift direct movement but the food was not removed. Removal from the horizontal tube by raking was first achieved after 13 days and regularly after 20 days. In the VT test the cat placed the paw on the tube opening after 11 days. The paw was inserted into the tube 2 days later and the food was removed after 20 days. Removal was made by pressure against the tube wall like cats with a dorsal C5 lesion. It did not use the "perpendicular technique" (cf. section B) to insert the paw in the

vertical tube but extended the limb forward and flexed the wrist. After the initial period the insertion movements were made with the same great ease and precision as before surgery.

Throughout the 9 months observation period the fish was removed from the horizontal tube with a flat paw without any sign of toe grasping and paw supination, and the food was always taken with the mouth from the floor. In the VT test there was possibly occasional slight toe flexion and paw supination from the 2nd month but it remained at the same low level to the end of the observation period. The cat never succeeded in grasping and lifting the food to the mouth.

When a hemisection in C3 (Fig. 3) was added to the previous C2 ventral and C5 dorsal lesions there was initially total loss of the movements. Very slight limb lifting in the HT test appeared only in the beginning of the 3rd week. The paw was lifted to the tube level without insertion after 4 weeks. The histological control in Fig. 3 shows an undegenerated fibre bundle in C4 which probably consists largely of axons of descending PNs originating caudal to the hemisection (cf. legend).

In summary, the cat with a combined ventral C2 and dorsal C5 lesion retained the ability to project the limb through space to the target but had lasting inability to grasp the food and bring it to the mouth.

F. C2 Hemisection, Except the Dorsal Column

The histological controls showed that the intended hemisection was incomplete in cats 1 and 2, with sparing of the medial part of the ventral funicle (Fig. 3). In cat 3 the hemisection in C3 was added to the previous ventral C2 and dorsal C5 lesions; in this case the lesion included the contralateral ventral funicle.

Despite severe postural disturbances cats 1 and 2 were able to lift the paw to the tube level in the HT test already in the 2nd week and they managed occasionally to insert the paw in the horizontal tube in the beginning of the 3rd week. It was a surprising finding that the recovery was faster than in any of the cats with a dorsal C2 lesion. However, the character of the lifting movement was clearly different from that observed in a normal cat or in the cats with a C5 or C2 dorsal lesion. In the early recovery phase after the dorsal C2 lesion, the cats lifted the paw towards the horizontal tube with a rapid phasic movement and when they failed to reach the tube level they did not maintain the limb in an elevated position but the paw appeared to fall back to the floor. By contrast the lifting movement of the hemisected cats was slow both in onset and execution and had a tonic character. It seemed to require a great effort of the animal to initiate the lifting of the limb but when they succeeded they could maintain the limb for a considerable time in the maximally elevated position even if they did not reach the tube. They did reach the tube level after some weeks and the precision of aiming at the opening was reasonably good, certainly better than after the C2 dorsal lesion and without indication of the "scratching movements" observed after that lesion (section B). The paw or the toes were frequently pressed against the lower inner edge of the tube which gave wrist and/or toe dorsiflexion and pronounced toe spreading (Fig. 11). Correspondingly, if the cats during lifting hit the outer lower part of the tube with the paw dorsum the result was a pronounced wrist and toe ventriflexion. These movements had the appearance of being passive, indicating rather flaccid distal muscles. It seems probable that the aiming at the tube opening to a greater extent than in normal animals depended on the proximal muscles. Figure 11 illustrates a phenomenon which was regularly observed in cat 1. When the paw came to rest on the inside of the lower tube wall, lifting movements, sometimes repetitive resembling stepping, occurred in the other limb. This movement of the left limb might be a crossed reflex evoked by pressure on the plantar surface. When the left limb was lifted then the dorsiflexion of the wrist and toes decreased in the right limb (Fig. 11, frames 22, 26) indicating active ventriflexion of these joints. This was probably a purposeful response, allowing raking when the food was reached. Our interpretation is that a poor ability to activate the toe and wrist flexors after the lesion was compensated by the response induced by lifting the other limb.

In cat 3 with a large lesion recovery was much slower. It only reached the lower level of the horizontal tube after one month and never managed to insert the paw during the 2 months observation period.

In the VT test cats 1 and 2 succeeded in lifting the limb, placing the paw on the upper part of the tube opening already during the 2nd week but with pronounced toe spreading. Cat 1 managed to insert the paw in the 3rd week and to remove the food in the 5th week; rather regular food removal was achieved in the 6th week. The recovery of cat 2 was somewhat slower but after some months it removed the food fairly regularly. Throughout the observation periods toe speading sometimes prevented insertion. Insertion into and withdrawal from the vertical tube was always made with the "perpendicular technique" (cf. section B). Cat 3 never managed to insert the paw.

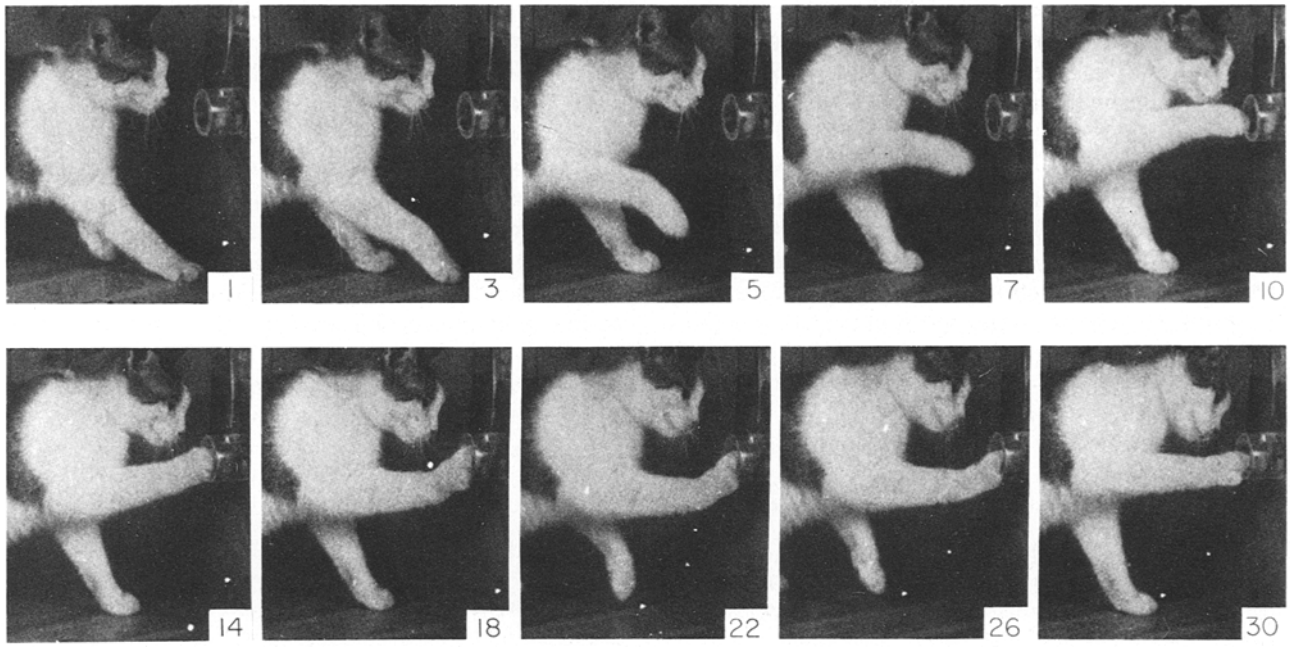

Fig. 11. Motor performance in cat C2H1 6 weeks postoperatively. The photographs are taken from the moving film, speed 32 frames/s. Frame number is indicated in each picture. See text

Neither in the HT nor in the VT test did we observe any sign of toe grasping or paw supination when the cats reached the food with the paw.

In summary, the C2 "hemisection" impaired the ability to retrieve the food in both tests. The recovery of the ability to lift the limb was faster than after the C2 dorsal lesion, and the aiming at the tube opening was also more precise. The lifting movement was slow and tonic in contrast to the phasic movements after the C2 dorsal lesion. There was no recovery of the toe grasping and supination which normal cats use to bring food to the mouth.

G. Other Effects of Spinal Lesions

In the initial period after the dorsal C2 and C5 lesions the cats occasionally walked on the dorsum of the foot as has been found after rubral or pyramidal lesions (Gorska and Sybirska, pers. commun.). Standing appeared to be normal except in cat C2D 1 in which the ipsilateral hindlimb tended to slide outwards.

Standing was normal after the ventral C2 or C5 lesions but during locomotion we sometimes observed abnormal co-ordination between hind- and forelimbs in one of the cats with a ventral C5 lesion.

Severe disturbance of posture and locomotion was observed after C2 hemisection and after the combined C2 ventral and C5 dorsal lesions, but considerable improvement occurred after 2–3 weeks (Denny-Brown 1966). It is unlikely that the postural disturbances had any major effect on test performance. For example, even when the hemisected cats were unable to stand they were able to retrieve food with the contralateral limb (cf. Methods) and the cat with the double lesion could use the ipsilateral limb when the body was supported in a hammock.

Discussion

The fact that the axons of the C3–C4 PNs are located ventral to the cortico- and rubrospinal tracts (Fig. 2) allowed us to eliminate the input from the cortico- and rubrospinal tracts to the forelimb segments by a dorsal C5 lesion which did not interrupt the C3–C4 PN projection to forelimb motoneurones. It is necessary to consider the possibility that the dorsal C5 lesion nevertheless might have had an effect on transmission via the C3–C4 PNs. Axonal transection causes retrograde changes and may lead to cell loss, which is very profound in case of the rubrospinal tract (Monakow 1909–10, Keller and Hare 1934). The cortico- and rubrospinal input to the C3–C4 PNs is from axons which also project further caudally to the forelimb segments (Illert et al. 1978; cf. also Shinoda et al. 1976, 1977). Accordingly, a dorsal C5

lesion transects one branch of the fibres projecting to the C3–C4 PNs. Our assumption has been that the very existence of the projection to the C3–C4 PNs gives protection from retrograde degeneration so that the cortico- and rubrospinal input to the C3–C4 PNs may continue to function even when descending branches are transected one to two segments caudally. Support for this assumption is given by the remarkable difference in retrograde cell loss following C2 and C5 transection of rubrospinal axons since the forelimb region of the red nucleus (Pompeiano and Brodal 1957; Nyberg-Hansen and Brodal 1964; Shinoda et al. 1977) was largely spared from retrograde cell loss after the latter lesion but not after the former.

There was almost certainly some retrograde cell loss also in the forelimb region of the red nucleus after the C5 dorsal lesions. This finding does not necessarily indicate that rubrospinal neurones supplying C3–C4 PNs were lost. While it has been shown that the entire rubrospinal input to the C3–C4 PNs is from axons which continue to the forelimb segments, it cannot conversely be assumed that all rubrospinal axons to the forelimb segments send collaterals to the C3–C4 PNs; the findings by Shinoda et al. (1977) rather suggest that this is not the case. In one of the cats with a C2 dorsal lesion we found a virtually complete retrograde loss of large and medium sized cells in the contralateral red nucleus. We are puzzled by this finding since rubral neurones project to the facial nucleus (Courville 1966) and to the lateral reticular nucleus (Corvaja et al. 1977; Kitai et al. 1974; Walberg 1958); furthermore some rubrospinal neurones send collaterals to the nucleus interpositus (Anderson 1971).

The early postsurgical effects of a dorsal C2 lesion that completely transects the corticospinal and rubrospinal tracts resemble those found by Gorska and Sybirska (1980a, b) after pyramidotomy and destruction of the red nucleus or after pyramidotomy alone. Although tracts other than the cortico- and rubrospinal tracts, both ascending and descending, are interrupted as well, we assume that the defects are due to the transection of these tracts or one of them. During the late postsurgical observation period our results differ rather dramatically from those of Gorska and Sybirska (1980b) since the recovery is faster and more complete after the spinal lesion. We will first discuss the early motor defects observed in the first few weeks after surgery and then at the end consider possible reasons for the faster recovery after the spinal lesions.

It is convenient to distinguish two main components in the motor performance: (a) *The target-reaching movement,* by which the cat reaches the food with the paw. (b) *The food-taking movement* consists of toe flexion and adduction combined with supination of the paw and we believe that they are components of a common motor pattern which is either triggered by the contact with the food or commanded into action at the estimated time of contact with the food (cf. below). The assumption that these movements are part of a common motor pattern is based on the observation that during recovery from the dorsal lesions, fractions of them regularly *occur together* for many months even if the movements are too weak to permit taking of the food and thus are useless to the animal. The *conjoint occurrence of fragments* of these movements after lesions is taken to indicate a partial recovery of the food-taking movement even if the animal never brings the food to the mouth. Since the results obtained after dorsal and ventral C5 lesion indicate a differential control of the target-reaching movement and the food-taking movement they will be discussed separately.

Our basic finding with respect to the target-reaching movements is the severe impairment of it after a dorsal C2 lesion while it was unimpaired after a corresponding C5 dorsal lesion. Surgery is certainly more difficult in C2 than in C5 but since no corresponding deficits were observed after the even more difficult ventral C2 lesion, it is concluded that the deficit is specifically due to the spinal cord lesion. We have considered the possibility that the differential effect of lesions in C2 and C5 might be related to the fact that the trapezius muscle is supplied from the C3–C4 segments but this fact does not seem to complicate with the interpretation of our results since complete denervation of the trapezius muscle in animals without spinal cord lesions does not influence motor performance in the tests (unpublished result). It is therefore highly likely that the severe defects after the dorsal C2 lesion is due to the interruption of the cortico- and/or rubrospinal input to the C3–C4 PNs. Conversely, it can be postulated that the command for precise target-reaching movement after a dorsal C5 lesion is mediated by the C3–C4 PNs. After the latter lesion the C3–C4 PNs retain their control not only from the cortico- and rubrospinal but also from the ventrally located tectospinal and reticulospinal tracts (Illert et al. 1977, 1978, 1981). The defects found after a dorsal C2 lesion show that the latter tracts cannot alone adequately govern the C3–C4 PNs. The persisting precise target-reaching movement after a combined C2 ventral and C5 dorsal lesion not only strongly support the view that the command is mediated by the C3–C4 PNs but also show that the cortico- and/or rubrospinal tracts alone may supply activation of them which is adequate for the particular movement investigated.

The findings after a ventral C5 lesion, presumably giving complete transection of the axons of the C3–C4 PNs (Illert et al. 1977, 1978), suggest that the

target-reaching movement may not depend exclusively on the C3–C4 PNs. Apparently, the cortico- and/or rubrospinal tracts have access to neuronal circuity within the forelimb segments that can transmit the command for lifting the limb. It was, however, a very striking finding that although the animals after a C5 ventral lesion could lift the limb with great ease, the aiming in this case lacked precision; as a result of successive corrections the animals displayed gross ataxia. So far we can only speculate about the reason for the ataxia. We assume that it is caused by the transection of the axons of the C3-C4 PNs since a corresponding C2 lesion rostral to these PNs has no effect. The most straightforward explanation would be to assume that the precise aiming in the normal target-reaching movement depends exclusively or very largely on the C3–C4 PNs and that the segmental neuronal circuity alone cannot appropriately substitute for them. Before accepting such a far reaching hypothesis it is necessary to exclude other mechanisms that may account for the ataxia. An interesting possibility emerges from the existence of the ascending collaterals from the C3–C4 PNs to the lateral reticular nucleus (Illert and Lundberg 1978, Alstermark et al. 1981). The activity in these collaterals mirrors that reaching the motoneurones via the C3–C4 PNs and it has been suggested that this ascending activity subserves the cerebellar feed-back control of the C3–C4 PNs. This hypothetical feedback does not seem to be required for the performance of the movement in the tests because the ventral C2 lesion which must have transected the ascending collaterals (Alstermark et al. 1981) had no observable effects on the movements (but note that the lesions were made after training). An interesting situation may arise when the descending branches from the C3–C4 PNs are transected in C5. If we assume that the ascending collaterals protect the PNs from retrograde degeneration, then the C3–C4 PNs might be activated when the brain commands a movement, and ascending activity will be evoked although no activity reaches the motoneurones. It is feasible that cerebellum on the basis of such misinformation will take false corrective measures causing ataxia. This working hypothesis has the advantage that it can be tested in lesion experiments.

The disappearance of the food-taking movement after a C5 dorsal lesion suggests that it depends entirely or largely on neuronal circuitry available to the cortico- and/or rubrospinal tracts within the forelimb segments, and that the C3–C4 PNs cannot alone mediate the command for this movement. The similar deficit after a dorsal lesion in C2 or C5 in fact suggests that the C3–C4 propriospinal system does not play any significant role for this movement. Conversely the persistance of the food-taking movement after transection of the propriospinal axons by a ventral C5 lesion shows that the cortico- and/or rubrospinal control of the neuronal circuitry in the forelimb segments suffices for the appropriate execution of this movement. It was a very striking finding that, although the gross ataxia made it difficult for the cats to reach the food with the paw, once this was achieved and the position of the paw stabilized by a special technique, then the animal had no difficulty in grasping the food and taking it to the mouth.

It remains to investigate whether or not toe flexors, wrist flexors or supinators of the paw can be effectively activated via the C3–C4 PNs. The deficit may well be that the combined pattern of activation which includes all the components of the food-taking movement is not represented in the connexions from higher centres to the C3–C4 PNs. There is no information regarding the segmental neuronal circuitry which mediates excitation of the appropriate motoneurones in the food-taking movement. However, tactile impulses from the paw are likely to contribute and it is of interest to note the commonly occurring convergence of cortico- and rubrospinal fibres on segmental interneuronal pathways from cutaneous afferents to motoneurones (Lundberg and Voorhoeve 1962; Lundberg et al. 1962; Engberg 1964; Illert et al. 1976b).

It might seem surprising that the target-reaching and the food-taking movements are subserved by different neuronal mechanisms since in the normal cat they are joined in one swift elegant movement. However, the regulatory demands on these movements are qualitatively very different, particularly when the animal is using the movements to obtain food in a more natural way than a more or less standardized test situation. Under natural conditions aiming will often be towards a moving target requiring a very high degree of spatial accuracy and probably also a fast regulatory mechanism by which an ongoing movement can be altered. It has already been hypothesized that the rich convergent connexions from subcortical motor centres onto the C3–C4 have evolved to subserve regulation of ongoing movements (Illert et al. 1977). By contrast the demand on the food-taking movement presumably is mainly with respect to the timing of grasping. It seems reasonable to suggest that this movement does not require such an elaborate command from higher centres and that the accurate timing of grasping may depend on the tactile impulses evoked by contact with the food. Denny-Brown (1966) has shown that powerful toe grasping ("clawing response") can be evoked in cats by a moving pressure on the distal paw. The time resolution of our measurements is not high enough to ascertain if grasping starts before contact and is reinforced by it or if it is triggered by the contact. A further investigation of this time relationship seems desirable, since food grasping is

an interesting model to elucidate the contribution of tactile information to motor commands.

As regards the long term recovery, it should be noted that supraspinal lesions were used in previous studies of the behavioural effects of transection of the cortico- and rubrospinal tracts (Tower 1935; Kuypers 1964; Lawrence and Kuypers 1968; Gorska and Sybirska 1980a, b). However, Kuypers (1964) briefly reported that transection of these pathways in C2 gave a similar motor deficit as the supraspinal lesions. A comparison of the present results with those of Gorska and Sybirska (1980a, b) suggests that a considerably faster recovery occurs after transection of the cortico- and rubrospinal tracts in C2 than after a combined transection of the pyramid and destruction of the red nucleus or after a transection of the pyramid alone. This holds true not only for the target-reaching but also, and in particular, for the food-taking movement, which only returned many months after pyramidal transection. In some of our cats fair food-taking movements were observed already in the 2nd month. At present we can only speculate about the reason for the faster recovery after spinal lesion. One difference between these two experimental series relates to the ipsilateral corticospinal fibres but the recovery is probably not due to these fibres alone since no trace of the food-taking movement recovered after a C2 hemisection. It might be argued that the spinal circuitry for food-taking gradually is sensitized after the lesions and that contact with food alone might evoke it. We do not favour this explanation since the food-taking movement is not evoked in other situations when there is accidental contact with the plantar surface of the skin; it rather seems to depend on a command timed to that of the target-reaching movement. The recovery appears to depend on ipsilateral (to the tested limb) ventral pathways but probably not via the C3–C4 PNs because there was hardly any recovery after combined ventral C2 and dorsal C5 lesions despite the fact that the rubro- and corticospinal tracts on which the food-taking movement normally depends retained their control of the C3–C4 PNs. It is also unlikely that pathways in the ventral funicle play an essential role in the long term recovery because the ventral funicle was spared in the C2 ventral and C5 dorsal lesioned cat. Since the critical lesion in the cats without long term recovery of the food-taking movement was in the ventral part of the lateral funicle (Table 1) it seems likely that the signal for food-taking movement after the dorsal lesions is carried to the forelimb segments by ipsilateral reticulospinal fibres. This route may not have been so easily available for recovery in the experiments by Gorska and Sybirska (1980b) who for fear of damage of the medial lemniscus transected the pyramid in the upper medulla oblongata at the level of corpus trapezoideus. The high pyramidotomy may have given an additional deficit in motor control by its interference with the corticofugal control of reticulospinal neurones and/or with the control of afferent neurones to cerebellum from the cerebral cortex.

Contrary to the food-taking movement the target-reaching movement ultimately recovered more or less completely after all the different spinal lesions (Table 1) but with some qualitative differences in the execution. It was a surprise that the recovery of the ability to lift the limb was faster after C2 hemisection than after the dorsal C2 lesion. However, the character of the lifting movement after these two lesions was clearly different. After the dorsal lesion the animal made rapid phasic movements while the movements after hemisection were slow and tonic. The normal rapid limb movement has an initial acceleratory and a later positional component (Ghez 1979). When the lifting movement returned after the dorsal C2 lesion it gave the appearance of a recovery of the initial acceleratory component of the normal movement with substantial loss of the positional response. These cats retained ipsilateral ventral pathways both to the C3–C4 PNs and the interneuronal circuitry in the forelimb segments. It is very tentatively suggested that these cats tried as far as possible to utilize signals in the remaining ipsilateral ventral pathways (probably reticulospinal) in order to activate the spinal neuronal circuitry normally used in target-reaching but that they had great difficulties in regaining the positional component of the lifting movement via this route. We also tentatively suggest that after an ipsilateral hemisection the higher motor centres lost access to the neuronal networks normally used in target-reaching and that they then successfully applied an entirely different motor strategy (perhaps utilizing postural neuronal mechanisms) which precluded rapid acceleration but was more effective in attaining an elevated limb position.

By necessity the present discussion has been centered around the contributions by individual neuronal pathways to movements. This should not be taken to indicate that we believe that the normal movements depend on one or the other pathway alone. The persistence of the food-taking movement after ventral lesions and the severe impairment of it after dorsal lesions merely shows that the cortico- and/or rubrospinal tracts alone can mediate the command and that ventral pathways alone cannot. This conclusion does not exclude a contribution from ventral pathways in the normal animal. Since a gradual recovery of the food-taking movement

occurs after the dorsal C2 lesion it cannot be excluded that ventral pathways also normally play some role for this movement, although too subtle to be revealed by the present tests in which the motor task is very simple compared with the natural situation in which a moving target often is the object. Correspondingly, the normal target-reaching observed after ventral C2 lesion cannot be taken to indicate that the ventral pathways known to converge on the C3–C4 PNs (Illert et al. 1977, 1978, 1981) do not contribute in the regulation of the normal movement. We also wish to emphasize that the target-reaching movement in the normal cat is unlikely to depend exclusively on the C3–C4 PNs, but as a first step towards understanding of the potential role of neuronal networks within the forelimb segments it is necessary to analyse the mechanism giving the ataxia after transection of the descending axons of the C3–C4 PNs. It must also be realized that transmission of a command to motoneurones may depend on pathways whose activity is not immediately related to the movement but nevertheless serve to keep up an appropriate excitability level. For example, our postulate that the virtually normal target-reaching found after the combined ventral C2 and dorsal C5 lesions depends on signals mediated by the C3–C4 PNs, does not imply that descending activity in the contralateral intact spinal half is without a role; it may well be important for the maintenance of the excitability required for activation of the motoneurones by impulses in the C3–C4 PNs.

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