QUANTITATIVE ANATOMICAL AND BEHAVIORAL ANALYSES OF REGENERATION AND COLLATERAL SPROUTING FOLLOWING SPINAL CORD TRANSECTION IN THE NURSE SHARK (GINGLYMOSTOMA CIRRATUM)

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Abstract. The spinal cord was transected at the mid-thoracic level in 32 nurse sharks. Four animals per group were sacrificed at intervals of 10, 20, 30, 40, 60 and 90 days postoperative. Two groups of fish underwent a subsequent spinal cord retransection at the same site at 90 days and were sacrificed 10 and 20 days later. Three sections of spinal cord were removed from each shark for histological analysis. Behaviorally, timed trials for swimming speed and a strength test for axial musculature contraction caudal to the lesion site were performed at 5 day postoperative intervals. Histological analysis showed little regeneration (9–13%) of two descending tracts 90 days following the lesion and no return of rostrally controlled movements caudal to the lesion. However, synaptic readjustment did occur caudal to the lesion. This phenomenon was attributed to local segmental sprouting of adjacent, intact nerve fibers. A close correlation was shown between this synaptic readjustment and the strength of uncontrollable undulatory movements seen caudal to the lesion site following spinal cord transection. The relationship of regeneration and collateral sprouting to quantitative behavioral changes is discussed.

INTRODUCTION

The shark possesses a unique nervous system among the vertebrates. This uniqueness is apparent both behaviorally and anatomically. In contrast to other vertebrates in which spinal cord transection results
in paralysis caudal to the lesion, sharks have exhibited coordinated undulatory movements following either ablation of the medulla (17, 18) or spinal cord transection (12, 13, 34–36, 46, 47, 51). Ten Cate and Ten Cate Kazejawa (51) further reported that spinal sharks were able to swim using coordinated undulatory movements between those portions of the body rostral and caudal to the lesion site. Anatomically, one trait which is particularly unique to the elasmobranchs is the reported absence of intermuncial cells (Golgi type II) in the spinal cord (1, 30, 40, 52, 53). Presumably, the vast majority of descending tracts end directly on motor horn cells without intermediary neurons to intercede or modulate information from higher centers. This anatomical trait lends itself well to the quantitative study of synaptic contacts on motor horn cells via descending spinal tracts following spinal cord transection. In addition, although lesion studies have been done, no regeneration studies have been attempted on elasmobranchs to date. It is the uniqueness of their central nervous system and the lack of regeneration studies that have prompted their use as an experimental animal in this study. An attempt was made in this series of experiments to quantitatively analyze and correlate the anatomical and behavioral aspects of the regenerative process in the shark following spinal cord transection.

MATERIALS AND METHODS

Subjects. Thirty-six male and female nurse sharks (*Ginglymostoma cirratum*), approximately two feet in length, were used in this study.

Environment. All fish were kept at Marineland of Florida in an outside, circular, salt water tank 15 feet in diameter and six feet in depth. A constantly circulating salt water system was used to insure proper oxygenation, salinity and water temperature. Sharks were fed to satiation daily on cut up fish.

Operative procedures. All operated fish were anesthetized with Tricaine methanesulfonate (MS-222, 1:4000, Finquel, Ayerst Laboratories), then placed on an operating board. A dorsal longitudinal incision was made at the midline in the thoracic region at the level of the trailing edge of the pectoral fins, and the musculature dissected away to expose the spinal column. A laminectomy was performed and the spinal cord transected with a scalpel. To prevent infection, the wound was sutured and powdered sulfathiazole-sulfonilamide was applied to the suture line, followed by a 0.1 cc intramuscular injection of Longicil. Animals were tagged for identification by attaching a numbered clamp.
and colored streamers to the anterior dorsal fin. All sharks, including four normals, were separated into nine groups (four animals per group). One group was killed at 10, 20, 30, 40, 60 and 90 days by anesthetization followed by perfusion with 10% buffered formalin. Two groups of animals underwent a subsequent retranssection of the spinal cord at the same site at 90 days and one group was killed by the above fixation method at 10 and 20 days following the retranssection.

**Histology.** Three pieces of spinal cord were removed from each shark; a 2 cm section at the site of lesion, a second section (1 cm in length) six spinal segments caudal to the site of lesion and a 1 cm section immediately caudal to the second section (Fig. 1).

![Fig. 1. Location of spinal cord sections removed for histological analysis. A: section taken for Bodian stain; B, section for Naka stain; C, section for Rasmussen stain.](image)

The 2 cm section of spinal cord at the site of lesion was cut horizontally at 15 μm and stained using the Bodian silver technique (6) counterstained with cresyl-violet and eosin.

The section of spinal cord six spinal segments caudal to the lesion was serially sectioned horizontally at 30 μm and stained using a modified Nauta technique (10) for degenerating nerve fibers. The sections from all postoperative groups were screened to assess the optimal time required to visualize individual degenerating, argyrophilic axons. Degenerating, descending nerve fibers were counted in the ventral cerebello-spinal tract and the combined tecto-thalamospinal tracts. The locations of the tracts were determined from the original description by Kappers (29) which has since been reviewed and described by others (1, 30, 32). The degenerative pattern within the spinal cord was plotted by drawing a composite cross-sectional diagram made by serially examining each horizontal section. The histological sections corresponding to the anatomical locations of the given descending tracts were selected and the number of degenerating fibers counted in each tract. These data were compared to the number of degenerating nerve fibers found following the subsequent retranssection (Fig. 2).
The third section of spinal cord was used for a quantitative analysis of the synaptic terminals on the perikaryon and primary dendrites of ventral motor horn cells following spinal cord transection. This 1 cm section of spinal cord was removed immediately caudal to the section

![Diagram](image)

Fig. 2. Cross sectional diagram of spinal cord showing the location of the combined tectospinal-thalamospinal tract (TeS-ThST) and the ventral cerebello-spinal tract (VCST).

used for fiber tract analysis so that number of regenerating nerve fibers and the synaptic profiles could be compared. The spinal cord was sectioned coronally at 10 µm and impregnated using the Rasmussen stain (45) for the light microscopic demonstration of bouton terminaux, then counterstained with cresyl-violet and eosin. Synaptic counts were made on only those motor horn cells in which a prominent nucleolus and primary dendrite could be seen in a given section. Sixteen motor horn cells were counted per shark, utilizing eight cells on the left side and eight cells on the right side for a total of 576 motor horn cells. All counts were made on coded slides to insure unbiased results. The resultant data were decoded and the levels of significance determined for intra- and intergroup interactions by using a computer program for multivariate analysis of variance.

**Behavior.** Operated sharks were observed daily while swimming in the tank and compared to normal sharks with respect to swimming ability. In addition, two quantitative tests were performed on all sharks, both preoperatively and at five day postoperative intervals.

The first test consisted of removing each shark from the tank and strapping it to a board with the body rostral to the lesion firmly held in place. That portion of the body caudal to the lesion remained un-
restrained with the exception of the caudal peduncle to which a hose clamp was attached. The clamp was connected with a screw to a Statham load cell assembly (Model UL-4) which was in turn mounted on a Statham Universal Force Transducer (Model UC-3). The entire transducer assembly was securely mounted on the test board. The output of the force transducer was fed into a two channel Grass polygraph recorder. Two electrodes were then attached to the paired barbels located on the underside of the snout of the shark (Fig. 3). The shark was stimulated using a constant current stimulator producing a 10 mA pulse of 50 ms duration. The strength in kilograms of the response of the caudal body musculature following stimulation was recorded on the polygraph and compared to preoperative and normal data. A minimum of five responses was recorded for each shark on a given trial day.

The second behavioral test consisted of timed swimming trials. Each shark was placed in the water at one end of a 7'×3'×2' tank and the

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time required for the shark to swim the length of the tank was recorded. Two consecutive timed trials were conducted on each shark on a given trial day, and only those trials were counted in which swimming was uninterrupted over the entire distance. The postoperative data for both behavioral tests were compared to normal data in addition to intra- and intergroup interactions among postoperative groups utilizing a computer program for analysis of variance.

In addition to statistical comparisons between groups for a given set of behavioral data, statistical correlation test (49) were utilized to compare quantitative anatomical and behavioral data.

RESULTS

Histological

Site of lesion. Histological analysis at the site of lesion at 10 days showed a dense scar separating the cut ends of the spinal cord (Fig. 4A). The diameter of the spinal cord at the scar was approximately 75% of normal and composed of neuroglial, ependymal and pial cells. Blood cells and phagocytes were also present in abundance throughout the scar, but no blood vessels were seen here at this time. A cistern lined with ependymal cells was found within the site of lesion at the rostral stump of spinal cord. This cistern appeared to be a terminal dilatation of the central canal and was present in all histological preparations throughout the postoperative period. No nerve fibers were found within the scar at 10 days. In fact, many large nerve fiber tips were found as far as 5 mm from both cut ends of spinal cord. These large nerve fibers were beaded at their terminals and ended in large spherical globules (Fig. 5A). This phenomenon also persisted throughout the postoperative period.

At 20 days, the scar looked much the same as it did at ten days. However, nerve fibers were now seen immediately adjacent to the scar with some fibers beginning their intrusion into the scar from both stumps of cord (Fig. 5B).

By 30 days (Fig. 4B), the cistern lined with ependymal cells had increased considerably in size while the diameter of the cord at the scar was further reduced (55% of normal). Nerve fibers were seen penetrating the scar from both stumps of cord but none were seen at the center of the scar.

At 40 days, there was little change in the appearance of the scar or the density and intrusion of nerve fibers into the scar area.

The lesion sites at 60 and 90 days appeared comparable. The scar was a loose cellular matrix made up of neuroglial and ependymal cells (Fig. 4C). Small blood vessels were also present within the scar and
Fig. 4. Low power view of horizontal sections of spinal cord at the site of lesion. The rostral stump of spinal cord is on the left in each micrograph. Bodian silver stain ×12.8. A, 10 days postoperative, showing a small cistern (X) immediately rostral to the scar; B, 30 days postoperative. The cistern (X) has increased even further in volume; C, 90 days postoperative. Ventral root nerve fibers (arrows) are seen growing caudally along the edge of the spinal cord and into the lesion site.

were predominately arranged parallel with the long axis of the spinal cord. The ependyma-lined cistern at the rostral stump of spinal cord had increased still more in size, but no further reduction was observed
Fig. 5. A, large severed nerve fibers near the site of lesion at 60 days postoperative, showing beaded configurations which end in large spherical globules (arrows). Bodian silver stain × 270; B, regenerating nerve fibers (arrows) at 20 days postoperative, beginning their intrusion into the rostral portion of the scar (S). Bodian silver stain × 670. C, normal motor horn cell showing synaptic contacts in the form of black dots on the cell membrane as revealed by the Rasmussen technique for synaptic endings (oil immersion, × 960); D, motor horn cell caudal to the lesion site 10 days following spinal cord transection, showing a reduced number of synaptic contacts. Rasmussen stain (oil immersion, × 960).
Fig. 6. High power views of horizontal sections through the ventral cerebello-spinal tract. Nauta stain × 960. A, 10 days following initial transection; no significant degeneration is seen; B, 20 days following initial transection; both coarse (double arrows) and fine (single arrows) degenerating, but relatively intact, nerve fibers are seen. Animals sacrificed at this time were used for nerve fiber counts; C, 90 days following initial transection; tract contains only diffuse axonal debris; D, 20 days following retranssection; small caliber degenerating nerve fibers (arrows) can be seen among the debris caused by the initial transection.
in the diameter of the scar. There was also a large increase in the number of nerve fibers within the site of lesion at 60 and 90 days. These nerve fibers were of small caliber and appeared to completely traverse the scar by following neuroglial bridges and blood vessels. Ventral root nerve fibers were also seen growing caudally along the ventrolateral surface of the cord until they reached the lesion site where they grew into the scar and the caudal stump of spinal cord (Fig. 4C).

**Descending fiber tract analysis.** There was no evidence of degeneration with the Nauta technique at ten days following both the first transection and the subsequent retranssection (Fig. 6A). This agreed with lesion studies in the shark utilizing the Nauta technique where degenerating, argyrophilic axons were not observed until approximately 14 days following the lesions (11, 23, 24). Those sharks sacrificed at 30, 40, and 60 days showed severe degeneration of the tracts such that individual degenerating axons could not be identified. At 90 days, degeneration was characterized by diffuse axonal debris (Fig. 5C). In contrast, 20 day animals showed darkening and irregular beading, typical of degenerating nerve fibers and were thus used to make the nerve fiber counts (Fig. 6B). Ascending tracts showed no retrograde degeneration six spinal segments caudal to the lesion.

Nerve fiber counts in the ventral cerebellospinal tract and the combined tectospinal-thalamospinal tracts are summarized in Table I. De-

<table>
<thead>
<tr>
<th>Tract</th>
<th>Numbers of fibers</th>
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<tr>
<td></td>
<td>Normal (Mean ± Sx)</td>
<td>Regenerated 90 days (Mean ± Sx)</td>
<td>Regenerated 90 days %</td>
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<tr>
<td>Ventral cerebellospinal</td>
<td>774 ± 35</td>
<td>72 ± 16</td>
<td>9.3</td>
</tr>
<tr>
<td>Tectospinal-thalamospinal</td>
<td>1584 ± 127</td>
<td>213 ± 49</td>
<td>13.4</td>
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generating nerve fibers counted 20 days following the first transection were the normal complement of axons in each of the respective tracts. Those degenerating nerve fibers counted 20 days following the retranssection represented the number of regenerated and/or sprouted descending axons that originated rostral to the lesion site. The appearance of new axons in these descending tracts was minimal 90 days after the initial spinal cord transection. The number of degenerating nerve fibers found within the two descending tracts following the second transection
represented only 9.3% (ventral cerebellospinal tract) and 13.4% (combined tectospinal-thalamospinal tract) of degenerating nerve fibers seen in these tracts following the initial transection. In addition, the degenerating nerve fibers seen following the retranssection were considerably smaller in diameter than those found following the initial lesion and could therefore be easily identified among the diffuse axonal debris caused by the first transection (Fig. 6D).

Synaptic profile analysis. The results of the synaptic terminal counts on motor horn cells caudal to the lesion are summarized in Fig. 7. The average number of boutons represented was combined from the data on both the left and right sides of the spinal cord, since there were no statistically significant differences between the right and left counts. There was a high correlation throughout the postoperative period ($r = 0.97$), between bouton counts on cell bodies and primary dendrites.

The number of boutons on cells bodies was statistically less than normal ($P < 0.05$) throughout the entire postoperative period (Fig. 5C, D), including the retranssected groups. There was, however, a statistically significant increase in boutons as a function of time ($P < 0.05$) from 10 days (45% of normal) to 60 days (90% of normal). No significant differences occurred between days 60 and 90, and the two retranssected groups.

The number of boutons per 10 $\mu$m primary dendrite closely paralleled somatic data. Following the initial transection, the bouton count on primary dendrites dropped to 62.2% of normal and was significantly greater ($P < 0.05$) with increased time until 90 days at which time they were 91.4% of normal and statistically indistinguishable from normal.

![Fig. 7. Results of bouton counts as revealed by the Rasmussen stain. A high correlation is shown ($r = 0.97$) between counts on motor horn cell bodies and primary dendrites.](image-url)
There were no statistically significant differences between the 90 day animals and the two retransected groups.

Behavior

Swimming ability. Immediately upon recovery from anesthesia, all operated sharks exhibited almost continuous undulatory movements caudal to the site of spinal cord transection while at rest. This phenomenon persisted throughout the postoperative period and was never observed in normal animals. In contrast, there appeared to be no swimming movements caudal to the lesion when the animals attempted to swim. Forward movement was accomplished by "walking" along the bottom of the tank using the pectoral fins or by rapid side to side movements of the head while dragging the caudal portion of the body. Turning could only be realized by paddling movements of the pectoral fins.

After approximately 30 days, undulatory movements became strong enough to propel the sharks forward. The animals did not appear able to control these undulatory movements, which resulted in further impairment of their swimming ability. Stimulation caudal to the lesion site by gentle prodding or by an inadvertant touch by another shark caused an increase in undulatory amplitude which either flipped the shark over on its back, using the snout as a pivotal point, or pushed the animal into a wall despite its efforts to prevent this by using the pectoral fins to "backpedal" away from the wall. This inability to effectively use the caudal portion of the body persisted for the duration of the postoperative period.

Quantitative tests. The results of the strength tests and timed swimming trials are summarized in Fig. 8. The strength tests showed two types of responses. The first response was elicited following stimulation and was in the form of a single, sharp flexure of the axial musculature. A second, consecutive stimulation usually elicited the same type of response in the opposite direction (Fig. 9B). This "response following stimulation" was present in both normal and operated animals, although considerably reduced in the operated animals. The second type of response was the previously mentioned undulatory movements which required no stimulation and occurred only in operated animals (Fig. 9A).

The "response following stimulation" was significantly less than normal throughout both postoperative periods ($P < 0.001$) with no statistically significant differences occurring between any postoperative groups (Fig. 8A).

Undulatory movements (0.25–0.5 cycles/s) were weak during the initial phase of the first postoperative period, but a statistically significant increase in strength occurred with increased time from 20 to 60
days ($P < 0.05$). From 60 days to 20 days following the retransmission, there were no significant differences between the postoperative groups. In addition, the strength attained by undulatory movements at 60 days was statistically indistinguishable from the strength of the "response following stimulation" seen in normal animals. Thus, although return of axial musculature strength to normal levels did occur following spinal cord transection, this return of strength was exhibited only in the form

![Graph A](image1)

**Fig. 8.** Results of quantitative behavioral tests. **A,** results of strength test following spinal cord transection; **B,** results of timed swimming trials following spinal cord transection.
of uncontrollable undulatory movements caudal to the lesion site. In contrast, movements following stimulation rostral to the lesion were greatly reduced immediately following spinal cord transection and remained at reduced levels for the duration of the postoperative period (Fig. 8A).

A significant increase was observed in the time required to swim 7 feet following spinal cord transection ($P < 0.001$). Normal animals required an average of 1.48 s, whereas the mean value for transected animals was 8.72 s. There was no improvement in swimming times during the initial postoperative period and the second spinal cord transection had no further effect (Fig. 8B).

**Comparison of quantitative anatomical and behavioral data**

The postoperative changes in undulatory strength were compared to the postoperative increase in the synaptic complement on motor horn cells caudal to the lesion and the resultant data were plotted in graph form (Fig. 10).

Figure 10A shows the combined plot of undulatory strength and the number of boutons on motor horn cell bodies during the postoperative period. Both phenomena showed an increase with time up to 60 days. From 60 days to 20 days following the second transection, there were no statistically significant differences in either parameter. A statistical comparison between these data showed a correlation coefficient of $r = 0.93$.

The combined data for undulatory movements and synaptic complement on motor horn cell primary dendrites (fig. 10B) closely resembled the results shown in Fig. 10A. Both synaptic complement and undulatory strength showed a statistically significant increase ($P < 0.05$) beginning at approximately 30 days and leveling off by 60 days with
no significant changes occurring in either parameter following the second transection. Statistical comparison of these two phenomena resulted in a correlation coefficient of $r = 0.91$.

![Graph A](image)

![Graph B](image)

Fig. 10. Combined results of quantitative anatomical and behavioral data. A, comparison between synaptic count on motor horn cell bodies caudal to the lesion and undulatory strength ($r = 0.93$); B, comparison between synaptic count on motor horn cell primary dendrites caudal to the lesion and undulatory strength ($r = 0.91$).

**DISCUSSION**

The results of this experiment show that regeneration of nerve fibers across the site of lesion does occur in the shark spinal cord. However, regeneration of descending nerve fibers to an area six spinal segments
caudal to the lesion is minimal and has no discernable influence on behavioral patterns during the postoperative period. This poor regeneration and lack of functional return in sharks is somewhat unexpected. It is well established that primitive vertebrates show more vigorous central nervous system regeneration than higher forms (25,44). One reason given is that more primitive animals reportedly possess an abundant supply of pleuripotent cells capable of differentiation into neural elements (31). Teleosts demonstrate regeneration and return of function ranging from four days in guppies (26,27) to approximately 35 days in goldfish (2, 4, 5). Regeneration and return of function in various parts of the central nervous system of reptiles (7), amphibia (9) and birds have also been reported.

There are three visible factors at the lesion site which may impede growth of nerve fibers through this area. The first is the formation of a scar between the cut ends of spinal cord similar to that seen in both mammals and teleosts. In mammals, the scar is thought by Windle and his associates (54) to form a mechanical barrier which prevents regeneration. However, in teleosts, it does not apparently effect the vigorous regeneration and return of function seen in these animals (4). The second factor is the cistern which forms immediately rostral to the scar. A similar phenomenon is seen in mammals (44) with the exception that there is no ependymal lining (14, 15). Kao and his associates (28) implicate lysosomal enzyme activity as a causative factor in mammals, but in sharks, the cistern may be caused by the scar preventing the rostro-caudal flow of cerebrospinal fluid. This blockage might, in turn, cause increased internal pressure with a resultant increase in the size of the cistern, concomitantly reducing the available cross-sectional area of spinal cord at the lesion site. Consequently, those nerve fibers which do regenerate through the lesion are forced to the periphery of the cord. The third factor is the 45% reduction in the diameter of the spinal cord at the lesion site, resulting in a further depletion of the neuropil in this area.

Despite the fact that nerve fibers do not grow through the lesion until 40–60 days, the number of synapses on motor horn cells six spinal segments caudal to the lesion show an increase beginning at 20 days. Synaptic return occurring before the return of rostral input indicates that both somatic and dendritic synaptic phenomena have similar origins which must be caudal to the site of lesion in the form of local, segmental sprouting. This hypothesis is further supported by data following the retranssection, i.e., there is no significant change in the number of boutons on motor horn cell bodies or primary dendrites following retranssection.
Since there is no return of swimming ability and no rostral control of axial musculature strength, the functional significance of this sprouting phenomenon and the return of synaptic contacts must be addressed. Perhaps the answer to this question lies in the unique undulatory movements seen in spinal sharks, which in the present experiment begin immediately upon recovery from anesthesia and increase in strength up to 60 days, after which time they remain relatively constant. Comparison of this data with the number of boutons on motor horn cells reveals a very high correlation. From this data, it seems reasonable to propose that local sprouting may be responsible for synaptic return and the increase in the strength of undulatory activity. In further support of this hypothesis, undulatory movements were without control from rostral centers since retranssection had a minimal effect on both synaptic complement and undulatory movements (Fig. 10). If undulatory strength and synaptic return were due to regenerated descending tracts, a second spinal cord transection should have resulted in a substantial loss of these boutons with a concomitant reduction in undulatory strength. Since neither of these phenomena occurred, it is highly unlikely that the small amount of descending tract regeneration could have been responsible for either synaptic return or the closely correlated increase in undulatory strength.

It should also be noted, that although nerve fibers were found in the caudal portion of the lesioned spinal cord in the areas normally occupied by specific descending tracts, the origin of the new nerve fibers may simply be by way of collateral sprouts from indigenous spinal neurons and/or dorsal roots located immediately rostral to the lesion, rather than from the original neurons located in higher centers. Similarly, regenerating axons may not synapse on their original termination sites.

The present experiment also contributes some information concerning the anatomical basis for undulatory movements in spinal sharks. There are at least two major hypotheses relating to this phenomenon. Ten Cate and Ten Cate Kazejawa (51) propose that undulatory movements are propagated over the lesion site by tensile stimuli applied to posterior musculature when an active contraction occurs in the head region, implying the activity of a chain of peripherally controlled reflexes. Gray and Sand (17, 18) disagree. They show that coordinated responses do not occur if two regions of the body of a dogfish are isolated from one another by a second spinal cord transection; each isolated section of the body exhibiting spontaneous, independent undulatory activity. Gray and Sand attribute this to an inherent undulatory discharge rhythm within the spinal cord. In support of Ten Cate and Ten Cate Kazejawa
Lissman (35, 36) and Roberts (46, 47) showed that dorsal root input is mandatory for the maintenance of undulatory movements. However, the hypothesis of Ten Cate and Ten Cate Kazejawa must be partially rejected, since in the present experiment, undulatory movements were most prevalent when the shark was at rest and were not observed in normal attempts to swim. The two major theories need not be mutually exclusive, however. It appears from previous and present data that the spinal cord of a shark has an inherent undulatory discharge pattern modulated by local sensory input and input from brain centers. If the predominant influence of rostral centers on this undulatory discharge pattern is inhibitory, and the major effect of dorsal root input is excitatory, transecting the spinal cord will release the caudal section of the spinal cord from rostral inhibitory influences, thus allowing the inherent discharge pattern to be exhibited in the form of undulatory movements caudal to the lesion site. The synaptic sites left vacant by spinal cord transection may then be replaced with excitatory synapses from dorsal root fibers or indigenous spinal tracts by way of collateral sprouting, causing an increase in the discharge pattern with a resultant increase in the strength of undulatory movements.

Perhaps the most unique aspect of this series of experiments is the demonstration of a possible functional correlation between the return of synapses on motor horn cells caudal to the lesion and the increase in undulatory strength during the postoperative period. Since Liu and Chambers (33) showed that anatomical sprouting does occur in the vertebrate spinal cord, others have shown that it can occur in many locations throughout the central nervous system (CNS) (19, 38, 42, 43). Functional correlates have been suggested for collateral sprouting in the CNS by McCouch et al (37) in which spasticity was thought to be a result of sprouting and by Schneider (48), who described visual sparing in hamsters following sprouting in the visual system. More recently, (16, 39) it has been shown that following deafferentation, behavioral modifications occur in cats after collateral sprouting in the spinal cord. Numerous publications have recently appeared reviewing plasticity of various parts of the CNS following lesions (3, 8, 19-22, 42, 50, 55) and thus will not be repeated here. There is general agreement that the functional role of collateral sprouting following lesions in the spinal cord is unclear. However, it has been well established that this phenomenon plays an important role in the reestablishment of lost synaptic contacts, and there is increasing evidence that it is implicated in behavioral changes. The results presented in this paper add further credence to the existing evidence which suggests that there
is indeed a behavioral correlation to sprouting in the central nervous system.

I would like to thank the people at Marineland of Florida, in particular Mr. Cecil Walker without whose help this project could not have been accomplished. In addition, I would like to thank Dr. J. J. Bernstein for his help and guidance, Mrs. Linda Burroughs for her assistance with the histology, and Dr. J. W. Wagner for his help with the manuscript.

This research was supported by the Center for Neurobiological Sciences, J. Hillis Miller Medical Center, University of Florida, Gainesville, Florida, NIH grant No. 25-P-30268/6-02 and Edward G. Schlieder Foundation grant No. 410-11-6121.

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Accepted 1 February 1979

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