corded in the soma of the alligator Purkinje cell after local stimulation (*7*).

On several occasions, all-or-nothing extracellular action potentials were recorded near the surface of the cortex (Fig. 2, E–I). These action potentials were in most instances negative and had a very prolonged time course with a slow, notched falling phase which suggests noncontinuous conduction. At the soma, single or repetitive positive-negative action potentials could be observed in the same experimental conditions. These have a short duration and are in every way similar to those recorded in cat or frog Purkinje cells (*9, 10*).

We conclude, therefore, that local stimulation of the surface of the alligator cerebellum evokes action potentials near the tip of the dendrites. These action potentials are conducted downward toward the soma. The conduction velocity is not uniform throughout the length of the dendrite, being rather slow at the surface and fastest at the level of the single dendritic stem and the axon. Similar potentials have been recorded in the pigeon cerebellum (*7*). In the pigeon, there is also the tendency for Purkinje cells to have only a single mainstem dendrite (*1, 3, 7*).

The blockage of the DAC is postulated to be produced by inhibitory action of stellate cells on the dendrites of Purkinje cells. Golgi and electron-microscopical studies of the alligator cerebellar cortex show that the axonal synapses of such stellate cells cover most of the dendrites from a depth of 150 μm to near the soma of the Purkinje cell (*5*). These interneurons, apparently forerunners of the basket cells of birds and mammals, can inhibit the dendritic spikes of Purkinje cells for a distance of up to 400 μm lateral to an excited beam of parallel fibers (*16*). This corresponds very well to the lateral extent of their axons.

From analyses of these and previous studies on the comparative aspects of cerebellar function, it seems probable that the cerebellum has undergone a gradual series of changes with evolution. In primitive cordates such as anura, which lack stellate and basket cells in the molecular layer, no long-lasting inhibition of Purkinje cells is demonstrable (*10*). In reptilia, aves and mammalia, on the other hand, this inhibition is present (*7*), but in the case of the reptiles, it seems to be restricted to the dendrites. Finally, the inhibitory synaptic system reaches its maximum action only in the more advanced forms, as the stellate cell terminals migrate giving rise to the "baskets" which cover the entire soma and axons of the Purkinje cells.

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Neonatal Castration: Influence on Neural Organization of Sexual Reflexes in Male Rats

Abstract. *Most male rats castrated 4 days after birth and given exogenous testosterone in adulthood were sexually motivated but incapable of completing the mating sequence with an ejaculatory response. When tested for sexual reflexes after spinal transection, these animals displayed impairment of genital responses. Similarly treated 12-day castrates exhibited a complete mating sequence and had normal sexual reflexes. Thus neonatal testicular androgen appears to have an organizational influence at the spinal level on neural tissue mediating sexual reflexes.*

It is believed that gonadal androgen has two roles in influencing sexual behavior of male mammals (*1*): one concerns the influence of androgen in the prenatal or early postnatal organization of neural tissue that mediates sexual behavior; the second concerns the postpubertal activation of previously organized neural tissue, resulting in the onset of mature patterns of sexual behavior. In support of this concept, several experiments (*1*, *2*) have demonstrated that most male rats, castrated on or before the 4th or 5th day after birth and given exogenous testosterone in adulthood, show a high degree of sexual motivation when tested with receptive females (indicated by the frequent occurrence of mounts and copulatory intromissions), but apparently cannot complete the mating sequence, as is shown by the absence of an ejaculatory pattern. Most male rats castrated after 10 days of age exhibit the complete mating pattern, characterized by several copulatory intromissions (normally five to 15) followed by an ejaculation. I was concerned with localization of the part of the central nervous system that is apparently organized by the neonatal androgen environment and irreversibly altered by neonatal castration.

The subjects were 17 Long-Evans male rats castrated 4 days after birth and 12 male rats castrated 12 days after birth. At 30 days of age the subjects were weaned and housed singly. Starting at 85 to 90 days of age, the subjects were daily injected subcutaneously with about 60 μg of testosterone propionate in oil per 100 g of body weight; injections were continued until the end of the experiment. In neonatal castrates given testosterone in adulthood, a frenulum is retained extending between the ventral surface of the penis and the preputial sheath. Therefore, in order to allow subjects to penetrate the females completely during intromission, the penile frenula of both 4-day and 12-day castrates were cut (ether anesthesia) 10 to 24 days after initiation of the hormone injections.

Table 1. Mean numbers of genital responses during four 30-minute tests for sexual reflexes. Numbers of rats appear in parentheses. Erections (Erec.), quick flips (QF), and long flips (LF) were significantly fewer for 4-day than for 12-day castrates ($P < .01$, two-tailed Mann-Whitney U-test). Only two 4-day castrates had quick flips. RC, response clusters.

Castrates	RC	Erec.	QF	LF
4-Day (6)	11.1	22.9	2.3	0
12-Day (9)	13.9	37.9	27.5	8.6

Fourteen to 28 days after initiation of the injections, subjects were given a series of three mating tests with receptive females; the tests lasted 30 minutes and were 1 to 3 days apart. The numbers of mounts without intromission, mounts with intromission, and mounts with ejaculation were recorded during each test; testing was discontinued when an ejaculatory pattern was observed. If a subject intromitted but did not ejaculate during the three tests, he was subsequently given a 60-minute mating test (in which a new receptive female was introduced after 30 minutes) to determine whether ejaculation would occur during extended testing. If an animal only mounted during the 30-minute tests, it was considered very unlikely that he would ejaculate during the extended test.

Eight of the 12-day castrates ejaculated during one of the 30-minute tests and three of the other four ejaculated during the 60-minute test. On the other hand, only three of the 17 4-day castrates ejaculated during either a 30-minute test or the 60-minute test. Of the 14 4-day castrates that never ejaculated, all mounted during at least one of the 30-minute tests (mean, 11 mounts per test; range of individual means, between one and 38) and ten intromitted during at least one of the 30-minute tests (mean, 14 intromissions per test; range of individual means, between two and 32 for animals that did intromit).

Since the occurrence of spontaneous seminal emissions (ejaculation) has been reported for normal male rats, on the basis of spontaneous appearance of seminal plugs when male rats are prevented from grooming the genital region (*3*), I wished to determine whether the 4-day castrates that did not ejaculate during the mating tests had spontaneous seminal emissions. Elizabethan collars, which prevented grooming of the genital area, were placed on eight of the 4-day castrates that did not ejaculate and on eight of the 12-day castrates that had ejaculated. The animals were then placed in cages floored with 1.2-cm wire mesh so that seminal plugs falling through the mesh could be observed and counted. The collars were worn for 48 hours, and the subjects were checked twice daily for the presence of a seminal plug in the preputial sheath or on the paper below the cage. Each subject expelled at least one plug; during the 48 hours the means were 3.25 plugs per 4-day castrate and 3.13 plugs per 12-day castrate. These data suggested that the reason why the 4-day castrates could not ejaculate was not impairment of autonomic control of seminal expulsion.

Since evidence reported (*4*) indicates that at least some somatic motor aspects of the ejaculatory behavior are mediated in the spinal cord, the next step was to test the possibility that the 4-day castrates did not have a normally organized neural pattern of sexual reflexes at the spinal level. Spinal transection was performed on the 4-day castrates that had not, and on the 12-day castrates that had ejaculated; the surgical procedure and postoperative care were essentially the same as those reported (*4*). Six of the 4-day castrates and nine of the 12-day castrates survived the surgery and postoperative period in satisfactory condition and showed strong somatic reflexes.

Subjects were allowed 20 days for recovery from spinal shock before testing for sexual reflexes by a reported method (*4*). Briefly, the method consists in placing the subject on its back in a glass cylinder for partial restraint, pushing the preputial sheath behind the glans penis, and holding it so for 30-minutes. With this type of stimulation, postpubertally castrated, spinal male rats receiving exogenous testosterone propionate exhibit clusters of genital responses intermittently every 2 to 3 minutes throughout the 30-minute period (*4*). Clusters of genital responses usually begin with three to four brief (1-second) erections of the glans, followed by two to three quick dorsal flips of the glans. The quick flips last less than 1 second and are accompanied by no pelvic movement or ventral flection of the body. After the series of quick flips, one to three extended or long dorsal flips of the glans usually occur, lasting 1 to 2 seconds. The long-flip responses, although not accompanied by seminal expulsion, do include strong ventral flection of the pelvis and appear to represent ejaculatory responses.

The spinal subjects received four 30-minute tests at 2-day intervals (Table 1). The spinal 12-day castrates had all three kinds of genital responses, but none of the spinal 4-day castrates had long-flip responses and only two of them had any quick flips. In number of response clusters, consisting mostly of erections, the 4-day castrates were not significantly different from the 12-day castrates ($P > .10$, two-tailed Mann-Whitney U-test).

It appears that the 4-day castrates could not ejaculate because of lack of organization of the reflex neural substrate related to motor components of ejaculatory behavior. The long-flip and possibly quick-flip responses appear to be manifestations of this reflex substrate. It is interesting that the organization of the spinal timing mechanism responsible for the occurrence of genital response clusters was not impaired.

It has been shown that gonadal androgen has an activational influence on sexual reflexes of male rats (*5*). It is now apparent that the early postnatal androgen environment plays a role in the neural organization of sexual responses at the spinal level. In fact the high degree of sexual motivation in the 4-day castrates, as indicated by mounting activity, suggests that perhaps the main difference between the 4-day and 12-day castrates reflects neural organization at the spinal level.

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