Functional Coupling Between Motor and Sensory Nerves Through Contraction of Sphincters in the Pudendal Area of the Female Cat

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Lagunes-Córdoba R, Hernández PR, Raya JG, Muñoz-Martínez EJ. Functional coupling between motor and sensory nerves through contraction of sphincters in the pudendal area of the female cat. J Neurophysiol 103: 74–82, 2010. First published October 21, 2009; doi:10.1152/jn.00712.2009. The question of whether skin receptors might help in the perception of muscle contraction and body movement has not been settled. The present study gives direct evidence of skin receptor firing in close coincidence with the contraction of the vaginal and anal sphincters. The distal stump of the sectioned motor pudendal nerve was stimulated. Single shocks induced a wavelike increase in the lumen pressure of the distal vagina and the anal canal, as well as constriction of the vaginal introitus and the anus. The constriction pulls on and moves the surrounding skin, which was initially detected visually. In the present experiments, a thin strain gauge that pressed on the skin surface detected its displacement. Single shocks to the motor nerve induced a wave of skin movement with maximal amplitude at 5 mm from the anus and propagated with decrement beyond 35 mm. The peripheral terminals of the sensory pudendal nerve and the posterior femoral nerve supply the skin that moves. Sensory axons from both nerves fired in response to both tactile stimulation and the skin movement produced by the constriction of the orifices (motor–sensory coupling). In cats with all nerves intact, a single shock to the sensory nerves induced reflex waves of skin movement and lumen pressure (sensory–motor coupling). Both couplings provide evidence for a feedforward action that might help to maintain the female posture during mating and to the perception of muscle contraction.

INTRODUCTION

The pressure in the lumen of the distal vagina increases and the vaginal and anal openings constrict by applying shocks to the distal stump of the cut motor pudendal nerve (MPN; Lagunes-Córdoba et al. 2009). The constriction of the openings apparently pulled on the surrounding skin. This was directly visualized as reported in our former study. We inferred that skin movement might excite the skin receptors sensitive to stretch. We aimed to measure the skin movement, to find the contribution of the vaginal and anal constrictions to that movement, and to test whether the skin movement excites stretch receptors of the moving skin (motor–sensory coupling). In addition, we tested whether the sensory nerves that innervate the moving skin induce reflex contraction of the anal and the vaginal sphincters (sensory–motor coupling). This was found to occur in the present study. Both couplings might be the basis of a positive feedback loop and perception of movement.

METHODS

Preparation

We studied 57 female cats weighing 2.7 to 3.6 kg. The Internal Committee for Care of Laboratory Animals (CICUAL) of the CINVESTAV approved the protocols based on the observance of the Mexican Norm for the Use of Laboratory Animals (NOM-062-ZOO-1999). CICUAL supplied the cats, which were anesthetized by intraperitoneal injection of pentobarbital (35 mg/kg) plus additional intravenous (iv) doses of 10 mg as needed. During the experiments, the cats did not show changes in pupil diameter, heart frequency, or withdraw reflex in response to noxious stimulation.

The iliac bone was fixed with the cat in prone decubitus (see Cueva-Rolón et al. 1993). Not only the pudendal nerve trunk but also the cutaneous posterior femoral nerve (PFN; Burgess and Perl 1973; Langley and Anderson 1896; McMahon et al. 1982) were exposed in the ischiadic fossa and cut on both sides of the body near the sciatic notch. The pudendal nerve trunk divides into the sensory pudendal nerve (SPN) and the motor pudendal nerve (MPN). The posterior femoral nerve (PFN), which is only cutaneous, lies lateral to SPN and divides into two branches (medial and lateral). The motor nerve divides into a urethral branch (ub; Fig. 1A), which was sectioned as far distal as possible, and a lateral, thicker branch. According to most authors, the latter branch innervates the external anal sphincter (see, e.g., Krier 1985; Paroschy and Shefchik 2000) but, in addition, also innervates the constrictor sphincter and the ischiocavernous muscle (Lagunes-Córdoba et al. 2009). A schema of all these nerves in the ischiadic cavity is shown in Fig. 1A. The left SPN, MPN, and the PFN branches were used for either bipolar stimulation or recording (Ag electrodes) against the killed nerve end. In seven experiments, the right MPN was stimulated and the electric activity of the left MPN was recorded. In five cats with intact nerves, the sensory nerves were stimulated to search for reflex responses.

Recording of pressure in the vaginal vestibule and the anal canal

The pressure in the lumen of the vaginal vestibule and of the anal canal was measured as previously described (Lagunes-Córdoba et al. 2009). Briefly, a bulb filled with water and tightly plugged to a Statham transducer was introduced 16–21 mm into the vagina and 20–30 mm into the anal canal (Fig. 1B). The axial length of the vestibule is about 20 mm (Crouch and Lackey 1969; Lagunes-Córdoba et al. 2009; Watson and Glover 1993; Zambelli and Cunto 2005). Stimulating MPN with single shocks induced waves of anal pressure (Pac) and vaginal pressure (Pv). Pv and Pac could not be recorded at the same time in the same cat. The output of the transducer fed a preamplifier (model 7P1F, Grass Technologies). As previously shown, the latency of the twitch of the soleus muscle, which was recorded using this preamplifier, was longer than the output of the twitch recorded from the output of a system using an optocoupler strain gauge (OCsg) and a regulated current supply, which was directly connected to the recording instrument (Delgado-Lezama et al. 2005).
In the present experiments, the recorded Pv wave that was induced by stimulation of the motor nerve was preamplified. Thus Pv and Pac might also be delayed, which is important in the present study. To test for a possible delay of the pressure waves, we compared Pv with the tension twitch (Tv) of the vaginal constrictor sphincter (VCS; Lagunes-Córdoba et al. 2009). The system recording tension with OCsg has no delay. Tv was recorded by introducing into the vestibule the short (1.5 cm) arm of a rigid L-shaped rod 2 mm wide, the vertical long arm being attached to the OCsg (Fig. 1B). OCsg was raised 1–2 mm to produce about 20 g tension on a small part of the wall of the vaginal vestibule. Tv and Pv were recorded at the same time. Tv appears distorted and represents only an unknown fraction of the total tension developed by VCS, although the relevant issue here is the delayed latency (≈7 ms) of Pv with respect to the latency of Tv (averaged traces in Fig. 1B; n = 30). We do not know whether in addition to the preamplifier, the pressure transducer might also contribute to the Pv delay.

The similar latency of the nerve-elicited Pac allows the inference of a similar delay with respect to the twitch of the external anal sphincter.

**Skin displacement**

A strain gauge composed of an all-turn resistance (≈300 \( \Omega \)), included in a rectangular wafer case (the cantilever; 6.5 \( \times \) 3.9 \( \times \) 0.1 mm [length \( \times \) width \( \times \) thickness]), detected the movement of the skin (displacement detector [DD]; strain gauge, EA-05-06 2AQ-350LE; Vishay Micro-Measurements, Wendell, NC; Fig. 1C). DD formed part of a Wheatstone bridge. Two other resistances in the bridge had the same value and the fourth resistance was a 0- to 500-W potentiometer, to adjust the bridge output at rest to 0 V if needed. A regulated power supply (homemade) fed the bridge with 9 V; an operational amplifier amplified the bridge output 1,000-fold. One of the short (3.9 mm) edges of the cantilever was fixed to a rigid pole attached to a Zeiss micromanipulator (M1); the other edge pressed perpendicularly on the skin surface. DD was advanced to produce a skin indentation (Fig. 1C) which prevented the cantilever from sliding when the skin moved. The depth of the indentation (≈1 mm) was fixed when the wave of skin displacement (Ds) induced by MPN stimulation was maximal (Fig. 1D). This DD edge moved with the skin and the cantilever displacement, which then generated a bridge output voltage. In different trials, the DD plane was adjusted to different angles with respect to the vertical (sagittal) plane to optimally detect the direction of skin movement (see RESULTS).

**Calibration of the displacement detector**

When the skin moves, DD bends and generates a bridge voltage. Even if the skin displacement follows a straight line, DD describes an arc that cannot be known. An empirical method to calibrate the detector was used by moving by known distances a piece of a 3-mm-thick gel foam material that mimicked the skin; DD indented 0.5 mm the gel foam surface using M1 (see earlier text). The gel foam was fixed to another manipulator (M2). For each distance traveled by M2, the DD voltage was read. Figure 1D shows a plot of the results, the best-fitting function (parabola), and the regression line (\( R = 0.9966 \)). Within the range of the plotted distances (1,000 \( \mu m \)), a linear function describes the distribution of points nearly as well as the parabola, but the two functions depart from each other for larger distances. The parabola might result from recoiling of DD. Significant recoil begins at 1,000 \( \mu m \); thus we used shorter distances. The main disadvantage of this method is that the elastic properties of the gel foam and the skin are not precisely the same.

DD has two faces. One faced the direction of the gel foam movement adopting a slight concavity and the other adopted a symmetric convexity. Moving M2 in the opposite direction, the latter face adopts concavity. The voltage sign reversed and its absolute magnitude for a given distance was reduced. The face giving the larger absolute voltage was selected to face the direction of movement (see RESULTS). The math function must be determined for each DD. Although the wafer case is durable, we recalibrated every two mounts.

**Quantification of nerve discharge**

In six experiments, the electric activity of SPN and of the two branches of PFN were rectified and integrated at intervals of 2–10 ms using the Origin software program (MicroCal, Northampton, MA). The integrated area is an index of the intensity of nerve activity.

**Statistical analysis**

Ten to 500 successive traces were averaged using the Axotape software program (Axon Instruments, Foster City, CA). Mean average
here is the average of the averaged samples in each cat. The deviation
from the mean of the averaged samples in each cat was <10% for
10–30 samples and <5% for 100–500 samples. Averages were taken
as samples. Statistical significance of difference between averages
was estimated using the t-test.

At the end of the experiments, the cats were killed by an overdose
of an iv injection of pentobarbitone. Additional details are given in the
appropriate section of RESULTS.

RESULTS

In 40 studied cats, the stimulation with single shocks of the
distal stump of the motor pudendal nerve (MPN) induced three
waves: 1) a wave (twitch) of pressure in the vaginal vestibule
as described previously (Pv; Lagunes-Córdoba et al. 2009); 2) a similar but smaller wave of rectal pressure (Pac), and 3) a
wave of skin displacement (Ds) in the perineum and in the
posterior side of the thigh (Fig. 2A). Shock intensity 1.2- to
1.3-fold the threshold (2.9–3.2 V, duration of 50 μs) to MPN,
either left or right, induced maximal Pv, Pac, and Ds. The
latency of the pressure waves was longer than the Ds latency
due to instrumental delay (see METHODS).

The mean peak pressure and timing of Pv [42.19 ± 0.73 cm
of H2O and 47 ± 5.03 ms (SD) were similar to those reported
previously (Lagunes-Córdoba et al. 2009)]. Pv and Pac could
not be recorded at the same time. Either Pv or Pac was
recorded in 30 cats, but both were recorded at different mo-
ments in another 10 cats.

To our knowledge, there is no previous report of Pac and only
one report of the axial force developed by the external anal
sphincter in response to single shocks to MPN (Krier et al. 1988).
The peak pressure of Pac (13.5 ± 0.92 cm of H2O; SE; 20 cats)
was only 32%, on mean average of the Pv peak amplitude. Pv and
Pac from the same cat were compared (t-test, two-tailed, paired
samples; n = 10); the difference was significant statistically (P =
0.0006). The time to peak of the three waves did not differ
significantly, but the Ds wave decayed more slowly than Pv and
Pac, possibly due to the viscoelastic properties of the skin. This
slow decay might determine the long duration of the sensory
discharge (see following text). The timing and the profile of the
decay varied at different recording sites.

Decrease of Ds with distance from the anus

Ds was selected at different distances from the anus at 5-mm
steps along a line traced on the skin from the center of the anus
to the lateral and lower part of the posterior side of the thigh at
45° to the middle line. The reason for choosing this line is given
later. DD was positioned at 90° from this line, facing the anus
(see the inset in Fig. 2B). As the distance from the midline
increased, the Ds amplitude decreased (Fig. 2B) and the latency
increased (Fig. 2D). The average amplitude (200 samples per
average) was maximal (600–900 μm). The latency was 5.05 ±
0.27 ms at 5 mm from the anus. The Ds wave propagated >35
mm from the anus. At 35 mm, the Ds latency was 9.95 ± 0.6
ms. The difference of latency at 5 versus 35 mm was signifi-
cant (n = 7; P = 0.00037). The average velocity of Ds
propagation along the line was 7.1 m/s.

Vector analyses of Ds

Ds was recorded at several sites on the perineum and the
posterior surface of the thigh with DD positioned parallel to
either the ground plane (0°) or sagittal plane (90°). Taking as
vectors the peak amplitude of Ds at 0 and at 90° in each strain
gauge position, and adding the vectors (graphic method; or-

FIG. 2. A: averaged waves (n = 30) of pressure in the anal canal (Pac),
Pv, and Ds at 10 mm from the anus. The inset in B is a diagram of the sites
of Ds recording at 5-mm steps from the anus to the external and lower part
of the thigh, with DD facing the anus. Ds and Pv were recorded at the same
time (see text) and Pac was recorded at a different time in the same cat. Pv
and Pac have the same instrumental delay (=7 ms) with respect to Ds.
Discounting the instrumen
tal delay, Pv and Pac have approximately the same latency as that of Ds. B: averaged Ds recordings (n = 30 traces in
each average) at increasing distances from the anus. C and D: the decrease
in amplitude and the increase in latency (7 preparations), respectively, of
Ds at different distances; the lines join the averages.
thogonal rule; insets in Fig. 3, A and B), all resultant vectors pointed to an area surrounding the anus even if DD was positioned closer to the vaginal introitus (Fig. 3C). A Ds wave was also recorded with DD positioned at other angles, but all resultant vectors pointed to the anal area. Accordingly, the main force pulling on the skin of the perineum and the posterior surface of the thigh appears to be produced by the constriction of the anus; however, \(Pv > Pac\). The low Pac amplitude compared with the PV amplitude might result from the longitudinal arrangement of the muscle fibers of the external anal sphincter (EAS; see DISCUSSION).

**Discharge of sensory nerves during Ds**

It was inferred that the displacement of the skin induced by MPN stimulation might excite receptors sensitive to stretch. SPN and PFN send their peripheral endings to the displaced skin (see following text). Stimulation of MPN with single shocks induced in these nerves a discharge as prolonged as Ds (Fig. 4). After accounting for the delay in nerve conduction (METHODS), the earliest nerve discharge occurred either in SPN or in the media branch of PFN but in close coincidence with the onset of Ds. In addition, the maximum intensity of the discharge coincided with the greatest slope of Ds (Figs. 5 and 6).

Thus at least some skin receptors are sensitive to acceleration (Cueva-Rolón et al. 1994).

The mean averaged latency (30 samples per average) of the discharges in SPN and in the two branches of the PFN was measured in 19 cats. In 17 of these cats, the latency was \(5 \pm 0.08\) (SD) in SPN, \(6.48 \pm 0.9\) ms in the medial branch of PFN, placed at 2–2.5 cm from the nerve entry to skin.

The length of the sensory axons between the electrode and the receptors was not known, but a conduction delay is expected. Using pin electrodes to separately stimulate the vulva and the skin that surrounds the anus induced action potentials in SPN and in the medial branch of PFN. No potentials were induced in the lateral branch of PFN. Single shocks to the vulva induced potentials in both SPN and the medial branch of PFN. The delay was 0.47–0.8 ms in SPN and 0.87–0.93 ms in PFN (three cats). The shock to the perianal skin induced potentials only in the medial branch of PFN, at a latency of 1.1–1.25 ms. It appears that SPN innervates the skin surrounding both the introitus and the anus and PFN innervates only the skin that surrounds the anus.

Discounting the nerve conduction delay, the differences in the latency of the discharges induced by MPN were significant between SPN and the medial branch of PFN \((P < 0.001; t\)-test,

![FIG. 3.](image-url) A and B illustrate the method used to find the resultant vector of averaged \((n = 30)\) Ds waves that were recorded at 0° (parallel to the ground plane) and at 90° in the sagittal. The averaged recordings in A were taken at 5 mm from the anus (top right point in C). Ds was considerably larger recording at 90° than at 0°, but the larger Ds in B was recorded with DD at 0° and 5 mm lateral to the vaginal orifice (bottom right point in C). C schematizes the resultant vectors in 9 sites in the same cat. All vectors point at an area surrounding the anus. See text for further details.

![FIG. 4.](image-url) Ds and Pac, and activity of the SPN and of the 2 branches of the PFN (average of 30 superimposed traces.). Single-shock stimulation of MPN elicited the waves and the nerve discharge. Similar findings were made in all studied cats \((n = 41)\). Abbreviations as in Figs. 1 and 3.
paired samples) and the significance was greater between SPN and in the lateral branch of PFN ($P < 0.0001$). The longer latency in the lateral branch might depend on the longer distance (15–25 mm) between the orifices and the closer nerve terminals (Fig. 5). In 2 of 19 cats, however, the latency of the discharge in both branches of PFN was similar (6.2 and 6.8 ms; Fig. 5B). Both the intensity of the discharge and the size of the receptive field of the lateral branch of PFN were unusually large; the lateral and the medial PFN fields overlapped. In addition, the area of the SPN field was unusually small and the intensity of its discharge was smaller than that in both PFN branches. It appears that when the SPN field is small, PFN fields are large. These findings suggest that the discharge intensity might correlate with the size of the field area.

To estimate the discharge intensity, the electroneurogram (ENG) of each nerve was rectified and integrated at 10-ms intervals ($n = 6$; Fig. 6). The area of the integrated discharges may be greater either in SPN or in the medial branch of PFN. The discharge of the lateral branch of PFN was less intense in 17 of 19 cats (Fig. 6A), but in the two cats with a large receptive field the discharge was more intense (Fig. 5B).

It was previously reported (three cats) that the field of SPN is a small skin area that extends no more than 5 mm from the vulva (Cueva-Rolón et al. 1994). In the present study, the size of the SPN field was explored in a larger number of cats ($n = 16$). Although the area of the SPN field varied substantially, two observations were consistent. First, only SPN supplies the skin of both the clitoral sheath and the vulva; in two cats, the SPN field was restricted to these structures. Second, the SPN field did not extend above a transverse line that divided the vulva into halves and about 5 mm lateral to the vulva. The greatest variation was found in the length of a 1- to 3-mm-wide strip of skin that may extend as much as 25 mm below the clitoral sheath following the edge between the posterior and the medial surface of the thigh (Fig. 5A).

The fields of the posterior femoral nerve (PFN) branches also varied substantially. In most cats, the medial branch innervates about 10–20 cm$^2$ of the skin on the posterior surface of the thigh, including the skin surrounding the anus, except its rostral part. The medial PFN field overlapped partially or totally with the SPN field, except for the skin of the clitoral sheath and caudal to the sheath.

FIG. 5. A and B illustrate results from 2 different cats. The diagrams at the left are the receptive fields of the recorded nerves; at the middle are the electroneurograms (ENGs; 30 superimposed traces). Single shocks to MPN induced the Ds that were recorded 5 mm from the anus, which is shown at the right together with the integrated nerve activity at 10-ms intervals (single traces from each nerve chosen at random). A: represents the results of 14 of 16 cats. The data in B were obtained from one of 2 cats with the SPN field restricted to the clitoral sheath and maximum PFN-I field. Abbreviations as in Fig. 1A. See text for further details.

FIG. 6. Pv, Ds, and ENGs of SPN and MPN of the left side (averages of 30 superimposed traces). The right MPN was stimulated with single shocks. The MPN ENG shows that at least one spike discharged synchronously after the twitch (dots) in the successive trials. B: Ds, Pac, and SPN discharge in response to stimulation of ipsilateral MPN with single shocks (left), and with brief (320 ms) tetanus at 60 Hz (right). See text for further details. Abbreviations as in Figs. 1, 3, and 5.
In all cats, the field of the lateral PFN branch surrounded and was larger than the field of the medial PFN branch with variable overlap. The field of the lateral branch reached the lateral surface of the thigh. In two cats, the lateral field of PFN completely overlapped the medial field. Given the variability in the size of the skin fields, it is not possible to present a general scheme.

**Afferent discharge in the motor pudendal nerve**

In a prior study, probing the vaginal vestibule with brief (<20 ms) controlled strokes (intact nerves) induced in MPN a short-latency (<2 ms) discharge that preceded the reflex response, with a latency of about 8 ms (Lagunes-Córdoba et al. 2009). It was postulated that this short latency might result from the firing of muscle afferents. In six of the present preparations with intact nerves, the right MPN was stimulated and the ENG of the left MPN showed a discharge with a latency as short as the SPN discharge (≈5 ms; Fig. 6A). Thus the discharge in MPN suggests that it might originate in afferent fibers from muscle. Furthermore, in three cats, at least one axon spike fired at regular intervals before the stimulus and after the sphincters twitched, the spikes were synchronized in the successive trials (dots in Fig. 6A). We do not know in which muscle this discharge might originate. This type of afferent firing is shown by Ia fibers from extensor muscles of the cat leg (Binder and Stuart 1980).

**Sensory discharge during tetanus**

During tetanic stimulation (350-ms duration at 50 Hz to MPN; five cats) not only the rectal and vaginal pressures (not shown) but also the skin displacement increased and reached a plateau (Fig. 6B). The ENG of SPN showed intense firing during the tetanic rising phase, which was more intense than that during the rise of the single twitch. The ENG response decreased during the plateau, to increase again, but with less intensity than that during the reduction in tetanus intensity. It appears that at least some skin receptors are more sensitive to dynamic displacement than to static stretch.

**Discharge of single axons**

In seven cats with intact nerves, the S2 dorsal root was transected as close as possible to the spinal cord. Single unitary spikes with short, fixed latency (1.2–1.7 ms; n = 21) were induced in dorsal root filaments by single shocks to SPN, PFN, or MPN (direct responses; Figs. 8A and 9A).

**AXONS FROM THE SKIN.** Seventeen axons (five cats) fired with short latency to nerve stimulation (<1.5 ms; Fig. 7A); 10 axons fired in response to PFN and 7 to stimulation of SPN. The axons showed a burst of spikes in response to tactile stimulation produced by a puff of air (Fig. 7A; see METHODS). A burst of spikes was also induced by MPN stimulation (Fig. 7C).

**MUSCLE AFFERENTS.** In four cats, a single shock to MPN elicited a short-latency spike, suggesting that the spikes were generated in muscle afferents. An air puff directed exclusively to the clitoral sheath induced two spikes in one axon (Fig. 8B); the other three axons did not respond to external stimulation. A burst of spikes followed the direct response to MPN stimulation (Fig. 8, C and D).


**Reflex Ds, Pv, and Pac**

In five cats with intact spinal roots and nerves of the left side and sectioned nerves in the right side, separate, single-shock stimulation of the sensory nerves induced Ds, Pv, and Pac. To evaluate the efficacy of transmission from the sensory nerves to the motoneurons, peak amplitudes of the reflex waves (Ds, Pv, and Pac) were compared with the amplitude of the direct waves induced from MPN. No reflex waves were elicited by the lateral PFN branch.

The average peak amplitude (30 samples per average) of the direct waves was the largest, and it was taken of 100%, and the amplitude of the T-reflex waves was taken as the percentage fraction of the direct waves (Fig. 9). The absolute amplitude of the waves and the position of DD varied among cats, which is irrelevant here because the DD position was constant in each experiment. The reflex waves elicited by SPN were the largest in the five cats and no response or waves <6% were elicited from the lateral branch of PFN. Thus the entire trunk of PFN was stimulated instead of their branches. The latency of the reflex waves was longer than the latency of the direct waves due to the reflex delay (Fig. 9).

The efficacy of generating the reflex waves was always stimulating SPN than that stimulating PFN (Table 1). Thus it appears that SPN axons might induce a larger proportion of the MPN motoneurons to fire than PFN axons. The amplitude of both the direct and the reflex Pac is low compared with the amplitude of Pv. The direction of the muscle fibers in the external anal sphincter might produce the low amplitude of Pac (see DISCUSSION). However, the efficacy of the reflex transmission from both SPN and PFN afferents to MPN efferents was higher for those that generate Pac than that for those that generate Pv (Fig. 9; Table 1).

**DISCUSSION**

**Motor–sensory coupling**

The constriction of the pelvic orifices, mainly the anus, pulled on the surrounding skin; then, skin receptors that are sensitive to light tactile stimulus fired. Thus the receptors are stimulated by the skin stretch produced from both the outside...
and the inside. The stretch from the inside evidences a motor–sensory coupling, which enriches the function of skin receptors. In addition, afferent fibers from muscle also fired. We cannot discern whether these afferents originated in the anal or the vaginal sphincter, or in the ischiocavernosus muscle (see Lagunes-Córdoba et al. 2009). It appears that muscle contraction and nerve response in the overlying skin is a single but compound action. Similar coupling might be present in other vertebrates and areas of the body and contribute to human kinesthesia. This issue is a matter of past and present debate. Some authors inferred that skin and joint receptors do not participate in the sense of position (e.g., of knee and fingers) and that this sense depends only on muscle receptors (see, e.g., Clark et al. 1985); however, other authors believe that skin receptors participate (see, e.g., Moberg 1983). However, in psychophysics studies, the firing of receptors assumed to be involved was not recorded. We do not refute the possible role of muscle receptors in the perception of movement. Rather, the present recordings provide evidence that skin receptors, which are moved passively by underlying and adjacent striated muscle contraction, can provide afferent activity that corresponds to the muscle contraction. The firing that is induced by muscle contraction might help not only to kinesthesia, but also to make us aware of the completion of a motor command sent by the CNS. We are not saying that skin receptors sensitive to stretch participate in all muscular sensations. Muscle pain, for example, could involve receptors in the muscle.

FIG. 8. Responses of a muscle afferent from S2 dorsal root filament (30 superimposed traces in A and C); In A, direct response to stimulation of the left motor nerve. In B, response to strong air puff directed onto the clitoral sheath (single trace). In C, the same direct spike (arrow) shown in A but at slower sweep speed. The direct superimposed spike is followed by spikes elicited by the motor phenomena exemplified here by the increase in vaginal pressure. D is a single trace of those traces superimposed in C to show that 3 other spikes (dots) were activated.

FIG. 9. Ds, Pv, and Pac elicited in the same cat by single shocks to the nerves indicated at the right of the traces. See details in text.

and the inside. The stretch from the inside evidences a motor–sensory coupling, which enriches the function of skin receptors. In addition, afferent fibers from muscle also fired. We cannot discern whether these afferents originated in the anal or the vaginal sphincter, or in the ischiocavernosus muscle (see Lagunes-Córdoba et al. 2009). It appears that muscle contraction and nerve response in the overlying skin is a single but compound action. Similar coupling might be present in other vertebrates and areas of the body and contribute to human kinesthesia. This issue is a matter of past and present debate. Some authors inferred that skin and joint receptors do not participate in the sense of position (e.g., of knee and fingers) and that this sense depends only on muscle receptors (see, e.g., Clark et al. 1985); however, other authors believe that skin receptors participate (see, e.g., Moberg 1983). However, in psychophysics studies, the firing of receptors assumed to be involved was not recorded. We do not refute the possible role of muscle receptors in the perception of movement. Rather, the present recordings provide evidence that skin receptors, which are moved passively by underlying and adjacent striated muscle contraction, can provide afferent activity that corresponds to the muscle contraction. The firing that is induced by muscle contraction might help not only to kinesthesia, but also to make us aware of the completion of a motor command sent by the CNS. We are not saying that skin receptors sensitive to stretch participate in all muscular sensations. Muscle pain, for example, could involve receptors in the muscle.

It was expected that the skin twitch would precede the nerve discharge, but the latencies of Ds and the nerve discharge were about the same (\(\pm 5\) ms). We tentatively conclude that skin receptors are as sensitive as— or more sensitive than—the strain gauge to detect instant acceleration of small skin displacements.

TABLE 1. Efficacy of transmission (%) to produce Ds, Pv, and Pac by stimulation of SPN and PFN

<table>
<thead>
<tr>
<th>Stimulated Nerve</th>
<th>Ds</th>
<th>Pv</th>
<th>Pac</th>
<th>Experiment 1</th>
<th>Experiment 2</th>
<th>Experiment 3</th>
<th>Experiment 4</th>
<th>Experiment 5</th>
<th>Average</th>
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<tr>
<td>SPN Ds</td>
<td>75</td>
<td>77</td>
<td>74</td>
<td>62</td>
<td>69</td>
<td>71.60</td>
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<td>26</td>
<td>22</td>
<td>39.80</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SPN Pac</td>
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<td>99</td>
<td>89</td>
<td>100</td>
<td>85.9</td>
<td>83.97</td>
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<td></td>
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<tr>
<td>PFN Ds</td>
<td>36</td>
<td>36</td>
<td>40</td>
<td>70</td>
<td>25</td>
<td>41.40</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PFN Pv</td>
<td>6</td>
<td>13</td>
<td>38</td>
<td>26</td>
<td>20</td>
<td>20.60</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PFN Pac</td>
<td>31.2</td>
<td>31</td>
<td>85</td>
<td>100</td>
<td>82</td>
<td>65.84</td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>

Ds, skin displacement; Pv, duct pressure; Pac, pressure in the anal canal.

Differential actions of the anal and the vaginal sphincters

We were initially biased to look only for the role of the vaginal sphincter on skin movement. The role of the more powerful anal constriction was not expected. We speculate that the introitus constriction itself plays a role by pulling on the skin. We do not know whether in normal conditions the vaginal and the anal openings constrict independently of each other. The amplitude of the pressure wave was smaller in the anal canal than that in the vagina, but the latencies of Ds and the nerve discharge were about the same (~5 ms). We tentatively conclude that skin receptors are as sensitive as—or more sensitive than—the strain gauge to detect instant acceleration of small skin displacements.

Regardless of which sensory nerve was stimulated, the efficacy to induce Pac and Ds was similar \((P > 0.05)\), sug-
gesting that Pac and Ds are both produced by the external anal sphincter.

**Muscle afferents**

In male and female cats, reflex MPN spikes were evoked by stimulation of the contralateral SPN (reflex transmission; Krier 1985; McMahon et al. 1982). The spike latency was 3–20 ms. In our laboratory, the latency of the pudendal reflex was 8 ± 1.2 ms (Lagunes-Córdoba et al. 2009). Thus the shorter latencies reported earlier appear to be too short and the longer latencies too long. We wonder whether the short-latency spikes that were assumed to be a reflex response might suggest they were produced by muscle afferents. The external anal sphincter has muscle spindles (Chennels et al. 1960; Krier et al. 1988). The so-called motor nerves also contain a sensory component.

**Area of the sensory fields**

The area of the skin sensory fields reflects the extension of the axon terminals. In the female rat, the area of the SPN field relates to the hormonal status (Komisaruk et al. 1972). In the female cat the hormonal status, possibly linked to seasonal change, might also induce changes in the length of the skin sensory axons. The small SPN field found in two cats might reflect a retraction of axon terminals.

**Basis for a positive feedback loop**

Afferent fibers from the cat SPN activate motoneurons in the lumbar and sacral cord (Bradley and Teague 1977; Cueva-Rolón et al. 1993, 2002; Fedirchuk et al. 1992; Raya 2004), which might induce a reflex constriction of the vaginal introitus, the anus, or both. This might then stimulate additional activation of receptors in a positive feedback. The loop proposed here would involve skin afferents and, possibly, muscle afferents. In support of this proposal is the fact that probing the vagina does trigger skin and muscle receptors and MPN after-discharge (Lagunes-Córdoba et al. 2009). Muscle afferents, spinal interneurons, and gamma-motoneurons might also be part of an additional feedback loop similar to that reported previously (Cueva-Rolón et al. 2002; Muñoz-Martínez and Delgado-Lezama 2007; Raya et al. 2004; see also Appelberg et al. 1983; Hulliger 1984; Jankowska and Gladden 1999). The firing of bistable motoneurons might produce the MPN after-discharge reported recently (Lagunes-Córdoba et al. 2009), but only if the membrane potential of the cell body is depolarized at 2–2.5 mV below the firing threshold (Cueva-Rolón et al. 2002; Hounsgaard et al. 1984, 1988). Thus bistability might generate the sustained MPN firing, but only if the membrane potential of the motoneuron rises to a critical level in response to the afferent firing (Cueva-Rolón et al. 2002). Thus the cell property (bistability) and the circuit might cooperate. In addition to the conditioning voltage, metabotropic transmitters (or modulators) from sensory fibers to motoneurons might increase the excitability of motoneurons (Delgado-Lezama and Hounsgaard 1999).

**References**


McMahon SB, Morrison JFB, Spillane K. Afferent fibers from the cat SPN activate motoneurons in the lumbar and sacral cord (Bradley and Teague 1977; Cueva-Rolón et al. 1993, 2002; Fedirchuk et al. 1992; Raya 2004), which might induce a reflex constriction of the vaginal introitus, the anus, or both. This might then stimulate additional activation of receptors in a positive feedback. The loop proposed here would involve skin afferents and, possibly, muscle afferents. In support of this proposal is the fact that probing the vagina does trigger skin and muscle receptors and MPN after-discharge (Lagunes-Córdoba et al. 2009). Muscle afferents, spinal interneurons, and gamma-motoneurons might also be part of an additional feedback loop similar to that reported previously (Cueva-Rolón et al. 2002; Muñoz-Martínez and Delgado-Lezama 2007; Raya et al. 2004; see also Appelberg et al. 1983; Hulliger 1984; Jankowska and Gladden 1999). The firing of bistable motoneurons might produce the MPN after-discharge reported recently (Lagunes-Córdoba et al. 2009), but only if the membrane potential of the cell body is depolarized at 2–2.5 mV below the firing threshold (Cueva-Rolón et al. 2002; Hounsgaard et al. 1984, 1988). Thus bistability might generate the sustained MPN firing, but only if the membrane potential of the motoneuron rises to a critical level in response to the afferent firing (Cueva-Rolón et al. 2002). Thus the cell property (bistability) and the circuit might cooperate. In addition to the conditioning voltage, metabotropic transmitters (or modulators) from sensory fibers to motoneurons might increase the excitability of motoneurons (Delgado-Lezama and Hounsgaard 1999).

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