Preservation of ejaculatory function by reconstruction of the canine hypogastric nerve

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Object. The hypogastric nerve (HGN) plays a crucial role in the primary functions of ejaculation: sperm transport through the vas deferens, secretion of prostatic fluid, and bladder neck closure. This study was undertaken to explore the possibility of restoring HGN function to the seminal tract and preserving its cross-innervation mechanism to the seminal tract after HGN–HGN reattachment.

Methods. Responses of the vas deferens/epididymis, prostate, and bladder neck to electrical stimulation of the lumbar splanchnic nerve (LSN) or the HGN and occurrence of antegrade ejaculation as a result of manual penile stimulation were examined in dogs that had undergone HGN–HGN reattachment. Eighteen months after the procedure had been performed bilaterally, 23 LSNs were electrically stimulated. In 17 LSNs this stimulation elicited elevation of vasal pressure (12 nerves bilaterally); in 18 LSNs, bladder neck pressure; and in 15 LSNs, prostate contraction. After resection of the right HGN in the dogs that had undergone HGN–HGN reattachment, 11 right-sided LSNs were stimulated; in seven LSNs, the stimulation elicited elevation of vasal pressure (five bilaterally), in seven bladder neck pressure, and in six prostate contraction. Twelve left-sided LSNs were stimulated; in seven LSNs, the stimulation elicited elevation of vasal pressure (four bilaterally), in six bladder neck pressure, and in six prostate contracton. Each of the 12 HGN stimulations made proximal to the site that had been sutured in dogs that had HGN–HGN reattachment caused responses of the three organs specified above that were comparable to those in control dogs. Manual penile stimulation elicited antegrade ejaculation in all three dogs examined.

Conclusions. The results of this study show that the function of the HGN in the seminal tract can be preserved after HGN–HGN reattachment and that restoration of its cross-innervation mechanism is possible.

Key Words • nerve reconstruction • hypogastric nerve • preservation of function • ejaculatory function • dog

The hypogastric nerve (HGN) is a peripheral sympathetic nerve that controls intrapelvic organs of the urinary, genital, and digestive tracts. In particular, the male genital tract requires sympathetic innervation via this nerve and lack of it causes significant disturbance in ejaculatory function, which leads to infertility.11,15,32,33,40

Ejaculation consists of three phenomena: seminal emission into the posterior urethra from the ejaculatory orifice and prostate; bladder neck closure to prevent retrograde ejaculation; and propulsion of seminal fluid out of the external urethral orifice. Disturbances of these processes have been caused by surgical injuries to the sympathetic pathway projecting to the seminal tract in the retroperitoneal and intrapelvic areas; these disturbances result in decreased ejaculation volume and dry ejaculation including emission loss and retrograde ejaculation.11,15,32,33,40

Previous studies have revealed that the aforementioned sympathetic efferent signals pass through the lumbar splanchnic nerve (LSN), caudal mesenteric plexus (superior hypogastric plexus in the human), hypogastric nerve, and pelvic plexus16,17,21,25,28 and that they cross to the other side of the body at the level of the caudal mesenteric plexus and the pelvic plexus (Fig. 1).16,17,21,26

On the other hand, the outgrowth of regenerating axons of a transected peripheral motor nerve is well investigated10,30,36 and reattachment of the motor nerve is performed clinically.3,5,55 The axonal sprouts cross the site of injury, reach the distal stump, and grow down the nerve to their peripheral terminations. However, application of these studies to the autonomic nerves innervating the genital tract has not been investigated until recently. The present study was undertaken to examine the possibility of surgical repair of the sympathetic efferent pathway projecting to the seminal tract. The aims of the experiments were twofold: 1) to examine restoration of the functions of the HGN in the seminal tract, including the vas deferens/epididymis, bladder neck, and prostate after transection and immediate microsurgical end-to-end suture of the HGN; and 2) to investigate preservation of the multiple cross-innervation mechanism of the sympathetic efferent pathway via the HGN (Fig. 1).

Materials and Methods

The design of our experiment was as follows. Transection and immediate microsurgical end-to-end suture of the HGN (HGN–HGN reattachment) was performed bilaterally. Eighteen months after the operation, we examined responses of the vas deferens/epi-
Reconstruction of the hypogastric nerve

**Animal Preparation**

Fourteen adult male mongrel dogs, each weighing 14 to 22 kg, were used in the experiment. Eight dogs served as control animals. Four dogs were neurally intact during the electrical stimulation experiment and the other four underwent unilateral transection of the HGN before the stimulation experiment. The remaining six dogs underwent transection of the HGN and HGN–HGN reattachment bilaterally 18 months before the experiment. Aseptic techniques and general anesthesia were used in this procedure. In the current study, the dogs were handled in the laboratory according to institutional guidelines for the care of laboratory animals as well as the guideline principles of the American Physiological Society, and with the approval of the institutional animal care and use committee.

**Exposure of the Sympathetic Nerve and Seminal Tract**

This experiment was performed in control animals and in animals that previously had undergone HGN–HGN reattachment. After intramuscular administration of ketamine hydrochloride (10 mg/kg body weight), each dog was placed supine. A vein in the forefoot was cannulated through which Ringer’s lactate solution was administered during the experiment. Immediately after intravenous injection of pentobarbital sodium (20 mg/kg body weight), the dog was intubated and ventilated by means of a Harvard respirator. Surgical anesthesia was maintained with repeated intravenous injection of pentobarbital sodium (5 mg/kg body weight, bolus), as necessary. After the abdominal cavity was opened via a midline incision, the caudal mesenteric plexus around the caudal mesenteric artery, and the LSN, HGN, bladder, and prostate were exposed as previously described. After the abdominal incision was extended to the scrotum to expose the testis, epididymis, and vas deferens.

**Transection of the HGN and HGN–HGN Reattachment**

Eleven months before the experiment began, six dogs underwent HGN transection and HGN–HGN reattachment. The HGN was identified near the ureter and a 2-cm portion was dissected with surrounding connective tissues and lifted upward using No. 1-0 silk thread. After transecting the lifted HGN completely at its midpoint with the surrounding connective tissues, end-to-end suturing was immediately performed, with the aid of an operating microscope, by using two No. 10-0 atraumatic nylon sutures. The procedure was applied to bilateral HGNs. Muscle, fascia, and skin were then sutured successively in layers. The animals recovered from anesthesia.

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**Fig. 1.** Diagram showing innervation of the canine seminal tract (A) and signals from each LSN to the vas deferens (B). The HGN projects from the caudal mesenteric plexus (CMP) to the pelvic plexus (PP) and includes multiple signals from each LSN to the seminal tract. These signals cross to the other side at the level of the CMP and the PP. Am = ampulla; CBPP = commissural branches between the right and left PPs; CMA = caudal mesenteric artery; CN = colonic nerve; CP = celiac plexus; CrMP = cranial mesenteric plexus; IMP = intermesenteric plexus; L = left; PN = pelvic nerve; Pro = prostate; r = right; RA = renal artery; SN = spermatic nerve; ST, sympathetic trunk.
under supervision and were checked regularly for several hours. Eighteen months later, at the time of the experiment, transection of the HGN was performed in four control animals and in some dogs that had undergone HGN–HGN reattachment.

**Conditions for Electrical Stimulation of the Nerve**

Because previous experiments have revealed that the LSNs from L-2 to L-4 mainly elicit responses of the vas deferens and bladder neck in the dog and rat, we investigated these nerves. Each of the LSNs (L-2–4) as well as each of the bilateral HGNs were stimulated. The LSNs were stimulated at a site approximately 10 mm from the sympathetic trunk and the HGNs at a site approximately 20 mm from the site sutured (HGNs). Prior to electrical stimulation, the nerve was transected centrally so that the peripheral autonomic pathway could be acutely decentralized and, thereby, the reflex input from the spinal cord could be eliminated. A stimulator was set for stimulus parameters of 8 V, 2 msec, and 10 Hz and applied to the distal end of the severed nerve with a pair of wire electrodes, as previously described. The parameter of 2 msec was selected because it provides maximum contraction of canine vas deferens. For measurement of contraction of the seminal tract, the stimulation was continued until responses reached a plateau or for 30 seconds if that did not occur. We confirmed that the parameters set by the stimulator were sufficient to cause elevation of the intraluminal pressure of the vas deferens and bladder neck, and contraction of the prostate by the stimulation of the distal end of the severed LSN, but not by the stimulation of its proximal end.

**Measurement of Intraluminal Pressure of the Vas Deferens and Bladder Neck, and Contraction of the Prostate**

Intraluminal pressure of the vas deferens was measured by inserting a 24-gauge elastic catheter into the lumen of the distal one-third portion of the vas deferens and directing the catheter toward the epididymis. Because this pressure is mainly caused by contraction of the cauda epididymis and pars epididymica of the vas deferens, it was designated as a response of the vas deferens/epididymis. Intraluminal pressure of the bladder neck was measured by placing an indwelling No. 10 French catheter with a pressure-sensitive rubber balloon into the bladder neck. Each catheter was connected to a pressure transducer. Contraction of the prostate was measured by suturing a force transducer to the surface of the prostate gland. The upper and lower edges of a transducer were sutured on the midanterior surface of the prostate in parallel with the long axis of the prostatic body. The force transducer detected contraction of the prostate at this site as a reduction in tension. Responses observed at the sites were simultaneously recorded in the present experiments.

**Manual Penile Stimulation in Dogs That Underwent HGN–HGN Reattachment**

Rhythmic manual stimulation of the penis was performed without anesthesia in three of the six dogs that underwent HGN–HGN suturing, as previously described. The ejaculate was examined microscopically.

**Macroscopic Findings of Splanchnic Nerves**

Immediately after the conclusion of electrical stimulation experiments, the dogs were kiled by administration of an excessive dose of pentobarbital sodium and were then fixed with 10% formalin solution to pursue by dissection the relationship of the LSN to the caudal mesenteric plexus.

**Sources of Supplies and Equipment**

Keisei produced the No. 10-0 atraumatic sutures used in the HGN–HGN reattachment. Electrical stimulation of the nerves was performed by using a model DPS-06 stimulator provided by Daiya Medical Systems. To measure the intraluminal pressure of the vas deferens and bladder neck and the contraction of the prostate, a force transducer was used the P10EZ Stratham pressure transducer obtained from Nihon Koden and the F-081S force transducer obtained from Star Medical Corp. All manufacturers of these supplies and equipment are located in Tokyo, Japan.

**Results**

**Anatomical Description**

The CMP, which is located around the caudal mesenteric artery, is composed of the LSNs, mainly from the L2–4 spinal levels, and the intermesenteric plexus, which connects the celiac and cranial mesenteric plexuses with the inferior mesenteric plexus (Fig. 1). The LSNs variably merge with each other before merging with the CMP. From the CMP, the lumbar colonic nerve projects to the descending colon and rectum and the right and left HGNs run caudally to the ipsilateral pelvic plexus, which is located near the lateral surface of the rectum. In dogs that underwent HGN–HGN reattachment, a variety of merging of LSNs (L2–4) was observed, as shown in Fig. 2. Electrical stimulation of the LSN was performed at the site at which the nerves merged together.

**Responses to Electrical Stimulation of LSNs and HGNs in Control Dogs**

Electrical stimulation of LSNs was performed in four neurally intact dogs. In these dogs 14 of 16 LSNs (L2–4) stimulated elicited elevation of intraluminal pressure of the vas deferens (11 bilaterally) and the remaining two...
Reconstruction of the hypogastric nerve

TABLE 1
Responses of vas deferens, bladder neck, and prostate to electrical stimulation of the LSN or HGN in control dogs and dogs that underwent HGN–HGN reattachment

<table>
<thead>
<tr>
<th>Site Transected</th>
<th>Nerve Stimulated</th>
<th>No. of Nerves Stimulated</th>
<th>Rt Vas (mm Hg)</th>
<th>Lt Vas (mm Hg)</th>
<th>BN (mm Hg)</th>
<th>Prostate (g)</th>
</tr>
</thead>
<tbody>
<tr>
<td>control animals</td>
<td>none</td>
<td>rt LSN 7</td>
<td>6 (104 ± 36)</td>
<td>5 (52 ± 19)</td>
<td>6 (31 ± 12)</td>
<td>6 (2.1 ± 0.4)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>lt LSN 9</td>
<td>6 (49 ± 18)</td>
<td>8 (113 ± 41)</td>
<td>7 (28 ± 13)</td>
<td>7 (2.3 ± 0.6)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>rt LSN 7</td>
<td>4 (19 ± 11)</td>
<td>5 (48 ± 21)</td>
<td>5 (22 ± 12)</td>
<td>5 (1.3 ± 0.4)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>lt LSN 9</td>
<td>6 (26 ± 14)</td>
<td>8 (109 ± 37)</td>
<td>8 (20 ± 11)</td>
<td>8 (1.9 ± 0.7)</td>
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<td></td>
<td></td>
<td>rt HGN 4</td>
<td>4 (162 ± 49)</td>
<td>4 (67 ± 27)</td>
<td>4 (38 ± 14)</td>
<td>4 (2.6 ± 1.0)</td>
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<tr>
<td></td>
<td></td>
<td>lt HGN 7</td>
<td>4 (59 ± 21)</td>
<td>4 (174 ± 44)</td>
<td>4 (37 ± 12)</td>
<td>4 (2.7 ± 1.2)</td>
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<td></td>
<td>lt LSN 9</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
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<td></td>
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<td>rt HGN 4</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
</tr>
<tr>
<td></td>
<td></td>
<td>rt HGN 6</td>
<td>8 (55 ± 38)</td>
<td>6 (32 ± 13)</td>
<td>8 (11 ± 4.8)</td>
<td>6 (1.8 ± 1.2)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>rt LSN 11</td>
<td>6 (34 ± 23)</td>
<td>9 (51 ± 37)</td>
<td>10 (10 ± 6.9)</td>
<td>9 (1.2 ± 0.9)</td>
</tr>
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<td></td>
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<td>rt LSN 12</td>
<td>5 (6.0 ± 1.7)</td>
<td>7 (25 ± 16)</td>
<td>7 (7.3 ± 3.2)</td>
<td>6 (1.2 ± 0.8)</td>
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<td></td>
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<td>rt LSN 12</td>
<td>4 (29 ± 27)</td>
<td>7 (48 ± 32)</td>
<td>6 (8.3 ± 5.4)</td>
<td>6 (1.1 ± 0.7)</td>
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<td>rt HGN 6</td>
<td>6 (108 ± 68)</td>
<td>6 (45 ± 21)</td>
<td>6 (21 ± 12)</td>
<td>6 (2.0 ± 1.3)</td>
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<td>lt LSN 9</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
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<td>lt HGN 12</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
</tr>
<tr>
<td></td>
<td></td>
<td>lt HGN 6</td>
<td>6 (42 ± 23)</td>
<td>6 (117 ± 63)</td>
<td>6 (22 ± 16)</td>
<td>6 (1.9 ± 0.9)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>lt HGN 6</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
</tr>
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</table>

* Transection (T) of the nerve was performed consecutively from T₁ to T₄ (see Figs. 3–6). Abbreviations: BN = bladder neck; ND = not detected; Vas = vas deferens.
† Number of nerves that showed responses followed (in parentheses) by mean ± standard deviation of the pressure or strength of contraction.

nerves elicited no response (Table 1). Thirteen of 16 LSNs stimulated elicited elevation of bladder neck pressure and prostate contraction, and the remaining three nerves elicited no response in either site.

Electrical stimulation of the LSNs was also performed in four other dogs in which the right HGN had just been transected. Five of seven right LSNs stimulated elicited elevation of vesical pressure (four bilaterally) and eight of nine left LSNs elicited elevation of vesical pressure (six bilaterally). Five of seven right LSNs stimulated elicited elevation of bladder neck pressure and prostate contraction and eight of nine left LSNs elicited elevation of bladder neck pressure and prostate contraction. All eight HGNs that were stimulated elicited elevation of vesical pressure, bladder neck pressure, and prostate contraction. Each LSN-induced response as well as each HGN-induced one was completely eliminated, respectively, by transecting the HGN bilaterally and by transecting the HGN at the site distal to the site stimulated (Table 1).

Responses in Dogs That had Undergone HGN–HGN Reattachment

Responses of the vas deferens/epididymis, bladder neck, and prostate to electrical stimulation of the LSN or HGN were tested in dogs that had undergone HGN–HGN reattachment. The responses follow.

Vas Deferens/Epididymis. Seventeen of 23 LSNs (L2–4) that were stimulated in six dogs elicited elevation of intraluminal pressure of the vas deferens (12 bilaterally and five ipsilaterally on the stimulated side) and the remaining six elicited no response (Table 1 and Fig. 3). When vesical pressure elevated bilaterally, the vesical pressure on the stimulated side was greater than that on the unstimulated side.

After the right HGN was transected, seven of 11 right LSNs that were stimulated elicited elevation of vesical pressure (five bilaterally and two contralaterally) and seven of 12 left LSNs stimulated elicited elevation of vesical pressure (four bilaterally and three ipsilaterally) (Table 1 and Fig. 4).

All of the 12 HGNs that were stimulated elicited elevation of vesical pressure. Each LSN-induced vesical pressure as well as each HGN-induced one was abolished, respectively, by transecting the HGN bilaterally and by transecting the HGN at the site distal to the site sutured (Table 1 and Fig. 4).

Bladder Neck. Eighteen of 23 LSNs (L2–4) that were stimulated elicited elevation of intraluminal pressure of the bladder neck (Table 1 and Fig. 5). After transection of the right HGN, seven of 11 right LSNs stimulated and six of 12 left LSNs stimulated elicited elevation of bladder neck pressure (Table 1 and Fig. 6).

All of the 12 HGNs stimulated elicited elevation of bladder neck pressure. Each LSN-induced bladder neck pressure as well as each HGN-induced one was abolished, respectively, by transecting the HGN bilaterally and by transecting the HGN at the site distal to the site sutured (Table 1 and Fig. 6).
Prostate. Fifteen of 23 LSNs (L2–4) that were stimulated elicited contraction of the prostate (Table 1 and Fig. 5). After transection of the right HGN, six of 11 right LSNs stimulated and six of 12 left LSNs stimulated elicited contraction of the prostate (Table 1 and Fig. 6). All of the 12 HGNs stimulated elicited contraction of the prostate. Each LSN-induced contraction as well as each HGN-induced one was eliminated, respectively, by transecting the HGN bilaterally and by transecting the HGN at the site distal to the site sutured (Table 1 and Fig. 6).

Antegrade Ejaculation by Manual Penile Stimulation

Manual penile stimulation elicited erection and antegrade ejaculation in all three dogs with HGN–HGN reattachment that were examined. Numerous motile spermatozoa (75 ± 26 × 10⁶/ml; motility 81 ± 12%) were observed microscopically in the ejaculate (1–1.8 ml).

Discussion

The present results indicate the following. 1) By suturing the HGN after transection, one can restore its functions in the vas deferens/epididymis, bladder neck, and prostate. The multiple signals emitted from the LSN to the seminal tract via the HGN are also able to be preserved (Fig. 7), namely: 1) to the ipsilateral vas deferens/epididymis without crossing to the other side; 2) to the contralateral vas deferens/epididymis by crossing to the other side at the CMP; 3) to the contralateral vas deferens/epididymis by crossing to the other side from the ipsilateral HGN at the commissural branches between the right and left pelvic plexuses (CBPP); 4) to the ipsilateral vas deferens/epididymis by crossing twice at the CMP to the other side and at the CBPP again from the contralateral HGN to the ipsilateral side; 5) to the bladder neck without crossing to the other side; 6) to the bladder neck by crossing to the other side at the CMP; 7) to the prostate without crossing to the other side; and 8) to the prostate by crossing to the other side at the CMP. The CMP in the dog coincides with both inferior mesenteric and superior hypogastric plexuses in the human. The superior hypogastric plexus consists mainly of LSNs (L2–4) and reaches the intrapelvic space as the HGN, whereas in humans the inferior mesenteric plexus is mainly formed by splanchnic nerves originating from the thoracic spinal cord, and its major portion reaches the colon and rectum. Therefore, the first site at which signals from the LSN to the seminal tract cross to the other side corresponds to the superior hypogastric plexus in humans.
The sympathetic efferent pathway projecting from an LSN to the vas deferens/epididymis includes more than four routes passing through two crosspoints to the other side in dogs and rats, and the pathway to the bladder neck includes more than eight routes passing through three crosspoints to the other side in rats. Although it is believed that a mismatching of transected axons cannot be avoided after subsequent regeneration within the nerve trunk, the present results indicate that many routes from an LSN to the seminal tract may be reconstructed by HGN–HGN reattachment.

Autonomic nerves are known to undergo regeneration following injury. Reinnervation of sweat glands in human skin flap, spontaneous return of human sexual function after pelvic fracture and urethral injury, return of spontaneous erection after sacral root transection in the baboon, and erection induced by stimulation of the rat pelvic nerve after its reattachment have been reported. The process of regeneration of transected axons is thought to be the following: the ruptured cell membranes of the proximal and distal portions of the transected axons are resealed 5 to 30 minutes after the transection. Thereafter, the proximal stump gradually regrows and the regenerating axons are guided by the Schwann cell pro-cesses. Growth of the proximal stump within the Schwann-cell basal lamina tubes is synchronous with the withdrawal and degeneration of the axonal remnants of the distal stump. When nerves, including sympathetic nerves, are injured, the distal axonal segment secretes nerve growth factor (NGF). In sympathetic nerves, NGF stimulates axonal growth, enhances regeneration of short adrenergic neurons specific to the genitourinary tract, stimulates outgrowth of sympathetic axons, and transforms immature adrenal chromaffin cells. In the current HGN–HGN reattachment experiment, NGF may stimulate axonal growth and the regenerating axonal sprouts may cross the site of injury, reach the distal stump, and grow down the nerve sheath to their peripheral terminations. The terminal site remains a subject for future study. One possibility is that the axonal sprouts make synapses with sympathetic postganglionic neurons innervating the seminal tract in the pelvic plexus because the HGN is composed primarily of preganglionic sympathetic neurons. The second possibility is that the axonal sprouts may terminate at the smooth muscle of the target organ.

We cannot state when reinnervation to the seminal tract is completed after HGN–HGN reattachment. Reinnervation of a sweat gland may take 11 months to several years in human skin flap, return of sexual function may occur between 6 and 19 months after pelvic and urethral
injuries, and erectile function in the baboon may return between 7 and 11 months after sacral root transection. Based on these findings, we selected 18 months after HGN–HGN reattachment to investigate reinnervation to the canine seminal tract. The current results can only indicate that reinnervation of the transected HGN to the canine seminal tract occurs within 18 months after HGN–HGN reattachment. As described by previous investigators, pharmacological treatment using NGF or nimodipine possibly accelerates axonal resprouting and may increase the speed of HGN reinnervation.

Because the completeness of the HGN transection significantly influences the results of the experiment, it was confirmed before we performed HGN–HGN reattachment in control dogs. The HGN transection we performed completely eliminated each of the LSN-induced responses in the seminal tract as well as each of the HGN-induced ones. Dogs that previously had undergone HGN–HGN reattachment were also tested after HGN transection showed evidence of complete transection of the HGN. Neuroplasticity after bilateral transections of the HGN may possibly influence results of the experiment. After the transections, compensatory signal via the lumbosacral sympathetic trunk, pelvic nerve, and/or sacral splanchnic nerve to the vas deferens can be accelerated in the dog. In addition, reorganization of the innervation of the vas deferens, sprouting of the pelvic preganglionic axons to reinnervate the decentralized vasal adrenergic ganglion cells, can be induced in the rat. Effects of these phenomena in the current study remain a problem for future investigation.

Clinically, dry ejaculation occurs when sympathetic pathways from the LSN to the seminal tract are injured bilaterally in the retroperitoneum or within the pelvis. In bilateral retroperitoneal lymph node dissection, most patients (74–89%) showed dry ejaculation and, in bilateral sympathectomy (L1–3), more than half (54%) of the patients demonstrated dry ejaculation. In unilateral injury to the LSN or the HGN, ejaculatory disturbance does not occur in most patients. In the animal experiments, all dogs whose HGN had been transected bilaterally demonstrated dry ejaculation. It is now possible to spare the LSN that controls ejaculation during surgery when disease is localized or not detected on the imaging study. However, there are more than a few patients in whom it is necessary to injure the sympathetic nerve pathway to the seminal tract bilaterally or the crosspoint of the nerves (such as the superior hypogastric plexus) according to the extent of disease. Such a situation can occur during operations for spondylolisthesis (anterior lumbar interbody fusion procedures), aneurysm of the abdominal aorta or iliac artery, and horseshoe kidney and colon cancer in addition to lymph node dissection or sympathectomy. No work has focused on procedures for restoring ejaculatory function in such patients to date. To our knowledge, research in cavernous nerve reconstruction for preservation of erectile function in the rodent is the only study reported on restoring sexual function. In the current study, function of the HGN after it had undergone end-to-end suture was nearly comparable to that of the control dogs, and dogs that had undergone the nerve reconstruction demonstrated antegrade ejaculation. This presents a clue about the kind of procedure necessary to preserve ejaculatory function in such patients. Immediate nerve reconstruction, which was used in the current study, may be more effective than delayed reconstruction because degenerative changes in the axons and fibrous alterations in the connective tissue around the nerve may make repair difficult without interposition of nerve graft. Methods providing the means for regenerating autonomic nerves would have great potential in such areas as surgery-related and traumatic nerve injuries.

Because the HGN transmits sympathetic signals to the bladder, penis, colon and rectum in addition to the seminal tract, sympathetic reinnervation may also occur in these organs after HGN–HGN reattachment. This was not the subject of the present study; however, it is now under investigation.

Conclusions

In conclusion, the present study shows that functions of the HGN in the seminal tract can be restored after transection by immediate suture and that sympathetic cross-innervation pathways via the HGN can also be preserved.

References

Reconstruction of the hypogastric nerve


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