On Peripheral Nerves Controlling Ejaculation

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Effects of section and stimulation of the hypogastric, pudendal and pelvic nerves on emission of the seminal fluid into the posterior urethra and on ejaculation from there were observed recording the posterior urethral pressure, using mongrel dogs. Bipolar electric stimulation was given continuously to the nerves under the conditions of 0.5–2 V, 0.5–2 msec, and 5–40 cps. For recording the posterior urethral pressure, a 0.6 polyethylene tube was inserted into the posterior urethra. On recording, communication between the posterior urethra and the urinary bladder was blocked to prevent regurgitation of the seminal fluid into the bladder and the external urethral orifice was ligated to produce a similar effect to an increased resistance in the anterior urethra by erection. The results obtained were as follows: Among the above-mentioned nerves, emission occurred by stimulation of only the hypogastric nerve. The emission caused a gradual rise in the posterior urethral pressure. When it reached the maximum, rhythmic alterations occurred. The rhythmic alterations were concluded to be the same phenomenon as ejaculation. It was noted that for occurrence of rhythmic alterations, both the hypogastric nerve and the pudendal nerve were concerned and that the pelvic nerve as well as the hypogastric nerve was involved in seminal emission.

It is generally accepted that the mechanism of so-called “ejaculation” which includes two phenomena, seminal emission and ejaculation, depends on normal nervous functions. The available literature (Munro et al. 1948, Learmonth 1931, Rieser 1961, Retief 1950) has indicated that the spinal center of “ejaculation” exists between the lower thoracic and superior lumbar segments of the spinal cord. The spinal center is supposed to be excited by the impulses ascending through the pudendal nerve and its centrifugal stimulation is delivered through the thoracolumbar sympathetic trunk. By this centrifugal stimulation spermatozoa are transferred from the epididymis to the vas deferens, and on attaining to orgasm they are excreted to the posterior urethra together with secretions from the seminal vesicle, prostate and periurethral glands (emission). Subsequently, stimulation from the sacral cord through the pudendal nerve induces contraction of the perirectal and perineal musculature, resulting in rhythmic ejections of the seminal fluid from the external urethral orifice (ejaculation) (Hotchkiss and Fernandez-Leal 1957, Potts 1957). The “ejaculation” mechanism described above has been,
however, by no means confirmed experimentally or clinically.

In the present paper, the relationship of the emission and ejaculation with the peripheral nerves which are supposed to control them was investigated experimentally, using a newly devised method, posterior urethrogram.

**Materials and Methods**

For the present study 22 adolescent male mongrel dogs weighing around 10 kg were used.

1) **Exposure of nerves**

The experimental animals were subjected to light anesthesia by intravenous injection of pentobarbital, and then the hypogastric, pelvic and pudendal nerves were exposed by the following methods.

**Hypogastric nerve:** After fixing the subjects in a dorsal position, the abdomen was opened by lower midline incision. The bilateral hypogastric nerves were exposed at the lateral sides of the sigmoid colon for section.

**Pelvic nerve:** Laparotomy was performed as mentioned above. The bilateral pelvic nerves were exposed at the lateral sides of the urinary bladder and cut in the portion immediately before entering the pelvic plexus.

**Pudendal nerve:** The pudendal plexus was exposed by longitudinal incision at the lateral side of the tail. The bilateral pudendal nerves were cut in the vicinity of the plexus, together with the internal anal nerve.

2) **Stimulation of nerves**

Using silver wire of 200µ in diameter as a stimulating electrode, a bipolar electric stimulation was given to the peripheral cut end of the nerves. The stimulation was made continuously with square wave of 1–5 V in amplitude, 0.5–2 msec in duration and 5–40 cps in frequency. The stimulator used was an Electronic Stimulator, MSE-3R (Nihon Koden Co.).

3) **Measurement of seminal emission and recording of the posterior urethral pressure (posterior urethrogram)**

For measurement of the amount of seminal emission, a No. 6 polyethylene tube was inserted into the urethra through the external urethral orifice, so that the tip of the tube might be positioned in the prostatic region of the urethra. In this experiment a clamp was fixed at the neck of the urinary bladder to prevent regurgitation of the seminal fluid into the bladder, and the external urethral orifice was lightly ligated for adding a resistance to the anterior urethra.

The gross observation was carried out on states of excretion of the semen from the posterior urethra and the amount of seminal fluid excreted was measured for 3 minutes.

The alteration of the posterior urethral pressure was recorded by connecting the tube to an electric transducer. For recording of the pressure change a Multipurpose Recorder, twin, with Carrier Amplifier RP-2 (Nihon Koden Co.), connected to the electric transducer was used. Recording was made by means of heat-writing at a speed of 1 mm/sec and for amplitude, 5 mm of the recording paper as 10 cm H₂O.

**Results**

1) **Section and stimulation of the hypogastric nerve**

Stimulation of the bilateral hypogastric nerves within the range of aforementioned stimulating conditions caused outflow of the seminal fluid. Since the largest
volume of the fluid was obtained by continuous stimulation of 2 V, 10 cps, 2 msec, stimulation of the hypogastric nerve to cause seminal emission was made under these conditions in the subsequent experiments.

In 3-5 minutes after onset of the stimulation, an outflow of the fluid from the posterior urethra occurred. This fluid was transparent and serous at first, followed by a gradual increase in its concentration, assuming semen-like appearance. A microscopic examination showed to contain innumerable spermatozoa in the fluid. After having confirmed an outflow of the seminal fluid, the changes in the posterior urethral pressure under hypogastric nerve stimulation was recorded. When the bilateral hypogastric nerves were stimulated, the posterior urethral pressure rose gradually as seen in Fig. 1. As the pressure augmented, however, the rate of rising was reduced to reach the maximum. When the maximum was attained, rapid rhythmic alterations of the intraluminal pressure occurred as shown in Fig. 1. At this time, rhythmic trembles of the penis and rhythmic contractions of the musculature surrounding the penis were noted. These pressure alterations disappeared by incision of the periurethral musculature.

The rhythmic alterations of the intraurethral pressure started at the end of 2'42"-14'40" (average 6'49") after the onset of the hypogastric nerve stimulation, and the pressure at this moment was 30-88 cm H₂O (average 53.0 cm H₂O) as shown in Table 1. The rhythmic pressure alterations occurred in a cycle ranging from 14-20 cycles/5 sec with an amplitude of 8-72 cm H₂O.

When the ligature at the urethral orifice was loosened while the posterior urethral pressure was showing rhythmic alterations, the seminal fluid was ejaculated outside, synchronizing with the rhythmic movements. These ejaculatory movements occurred in the same strength and frequency as those produced by manual stimulation of the penis in an unanesthetized dog. When the seminal fluid was not allowed to be ejaculated, the rhythmic alterations persisted for a while after a cessation of hypogastric nerve stimulation.

In order to clarify whether the rhythmic alterations in the posterior urethra was caused directly by the stimulation of the hypogastric nerve or indirectly by the rise in the posterior urethral pressure, the following experiments were made. Another No. 3 polyethylene tube was inserted into the posterior urethra. When the pressure was lowered during the rhythmic contractions by removing the fluid through this tube, the internal pressure began to decrease. The rhythmic pressure alterations diminished in amplitude and disappeared when the internal pressure was reduced to 4-32 cm H₂O, a mean of 21 cm H₂O (Fig. 2). And then, physiologic saline was instilled into the posterior urethra through the second tube to raise the intraurethral pressure during the stimulation. When it reached the above-mentioned maximum, rhythmic alterations in the internal pressure was noted to reappear (Fig. 3). On the other hand, when hypogastric nerve stimulation was discontinued no rhythmic alteration occurred on raising the internal pressure by introducing physiologic saline into the posterior urethra (Fig. 4).
When stimulation of the bilateral hypogastric nerves was started, the posterior urethral pressure rose gradually. As the pressure rose, the rate of rising was reduced to reach the maximum. Then, rapid rhythmic alterations of the pressure began to occur, as seen in the figure.

Amplitude: 5 mm = 10 cm H₂O  Recording speed: 1 mm/sec

Rhythmic alterations disappeared as the internal pressure was lowered by removing the seminal fluid.

Amplitude: 5 mm = 10 cm H₂O  Recording speed: 1 mm/sec
Fig. 3. Posterior urethrogram of normal dog (3).
When the posterior urethral pressure was raised by an instillation of physiologic saline under hypogastric nerve stimulation rhythmic alterations occurred. As the pressure was lowered, they disappeared.
Amplitude: 5 mm = 10 cm H₂O
Recording speed: 1 mm/sec

Fig. 4. Posterior urethrogram of normal dog (4).
Unless hypogastric nerve stimulation was given, no rhythmic alteration occurred, when the posterior urethral pressure was raised by an instillation of physiologic saline.
Amplitude: 5 mm = 10 cm H₂O
Recording speed: 1 mm/sec
Fig. 5. Effect of section of bilateral pudendal nerves on posterior urethrogram.
A) Before section of the pudendal nerve.
B) After section of the both pudendal nerves. Note that the posterior urethral pressure rose by seminal emission as before the section.
C) After section of the both pudendal nerves. The posterior urethral pressure was raised by instillation of physiologic saline over the maximal value obtained prior to the section, but no rhythmic alteration appeared.
A), B) and C) were recorded during continuous hypogastric nerve stimulation.
Amplitude: 5 mm=10 cm H₂O  Recording speed: 1 mm/sec
2) **Stimulation of the bilateral pudendal and pelvic nerves**

When the bilateral pudendal nerves were stimulated, no discharge of the seminal fluid was observed. Similarly, when the bilateral pelvic nerves were stimulated, no discharge of the seminal fluid was noted.

3) **Effects of section and stimulation of the pudendal and pelvic nerves on the posterior urethral pressure curve recorded during hypogastric nerve stimulation**

   a) **Pudendal nerve**: After confirming a rise and rhythmic alterations to occur in the posterior urethral pressure, the bilateral pudendal nerves were cut and the electrodes were fitted to the peripheral end.

   When the bilateral hypogastric nerves were stimulated, the posterior urethral pressure rose just like before section of the both pudendal nerves (Fig. 5). However, when this rise reached the maximum which had produced rhythmic alterations prior to the section, no rhythmicity appeared. Furthermore, when physiologic saline was instilled into the posterior urethra through the second tube, no rhythmic alteration in the posterior urethral pressure appeared even after the pressure had reached a value twice higher than the maximum obtained before the section (Fig. 5).

   Then, the electric stimulation under the conditions of 5 V, 30 cps and 1 msec with stimulating period of 1 to several seconds was given to the peripheral cut end of the pudendal nerve. When the stimuli were delivered, a rapid rise in the posterior urethral pressure which was more than 30 cm H$_2$O was noted as seen in Fig. 6. In spite of this pressure rise, rhythmic alterations did not occur. When the stimulation was repeated with the external urethral orifice opened, discharge of the seminal fluid was observed concurrently with the rise of the urethral pressure.

   b) **Pelvic nerve**: The seminal emission caused by the hypogastric nerve stimulation was noted to persist after section of the pelvic nerve. However, whereas the volume of the seminal fluid before the section of the nerve was 1.6 ml/min on the average, it was 0.3 ml/min after the section, showing a considerable decrease. When the posterior urethral pressure was recorded during the hypogastric nerve

![Fig. 6. Effect of stimulation of bilateral pudendal nerves on posterior urethrogram.](image)

When the internal pressure was 30 cm H$_2$O, the bilateral pudendal nerves were stimulated. The stimulation caused a rapid rise of the pressure and ejection of the seminal fluid shown by the arrows.

Amplitude: 5 mm = 10 cm H$_2$O  Recording speed: 1 mm/sec
Fig. 7. Effect of section of bilateral pelvic nerves on posterior urethrogram.
A) Before section of the pelvic nerve.
B) After section of the bilateral pelvic nerves. Hypogastric nerve stimulation did not cause elevation of the posterior urethral pressure and no rhythmic alteration occurred.
C) After section of the bilateral pelvic nerves. When the posterior urethral pressure was raised by instillation of physiologic saline, rhythmic alterations occurred just like before the section.
A), B) and C) were recorded during continuous hypogastric nerve stimulation.
Amplitude: 5 mm=10 cm H₂O  Recording speed: 1 mm/sec
stimulation, its elevation caused by seminal emission was so slight that the maximum did not exceed 20 cm H$_2$O. No rhythmic alteration occurred. (Fig. 7). On the other hand, when the posterior urethral pressure was raised by an instillation of physiologic saline during continuous hypogastric nerve stimulation, rhythmic alterations occurred in the same way as before section of the pelvic nerve.

When the posterior urethral pressure was 20 cm H$_2$O, the peripheral cut end of the pelvic nerve was stimulated with the same stimulating conditions as that of the pudendal nerve, but there hardly occurred any change in the pressure.

**DISCUSSION**

Histologically the male accessory sexual organs, including the epididymis, seminal vesicle, vas deferens and prostate gland are subject to the distribution of the nerve fibers which come out from the lower thoracic, lumbar and sacral nerves (Fukuyama 1954, Okuno 1958, Kimura 1960). Therefore, the peripheral nerves associated with ejaculation are thought to consist of three nerves, namely the hypogastric, pudendal and pelvic nerves.

As to the relation of the hypogastric nerve with seminal emission and ejaculation, it is generally accepted that the movement of the male accessory sexual organs can be caused by the hypogastric nerve stimulation (Learmonth 1931, Hukovic 1961, Smith and Lebeaux 1970, Farrel and Lyman 1937, Naimzadda 1966, Sexana 1970). However, this acceptance is derived from the results of the studies of functions of each individual organ. Only a few reports have been issued on the integral function including transport of spermatozoa, emission of the seminal fluid into the posterior urethra, and ejaculation of the fluid from the posterior urethra through the urethral orifice.

In 1931 Learmonth conducted an electric stimulation to the hypogastric nerve of humans during surgical operation, and observed the change in the posterior urethra with a cystoscope. He reported that hypogastric nerve stimulation produced: 1) contraction of the urethral orifice, 2) increased tonus of the trigone of the bladder, 3) contraction of the urethral orifice, 4) contraction of the prostatic smooth muscle, 5) contraction of the vas deferens and ejaculatory duct, 6) vasoconstriction of the trigone of the bladder and 7) emission of the seminal fluid. Similar results were reported by Semans and Langworthy (1938) who made experiments on cat, although they stated that no contraction of the internal orifice of the urethra was observed in their study.

Concerning the effect of section of the hypogastric nerve on "ejaculation" it is a well known fact that an ejaculatory disturbance occurs after the damage or section of the thoraco-lumbar sympathetic pathways, such as thoraco-lumbar sympathectomy (Retief 1950, Bacq 1931, Rose 1953, Whitelaw and Smithwick 1951), Cotte's operation (Shishito et al. 1960) and the damage to the sympathetic nerve during the surgery in the pelvic cavity (Rieser 1961, Goligher 1951, Bruch 1955, Shellen 1960). Based on these facts, in addition to manifestation of an ejaculatory disturbance due to spinal injury, it is presumed that the human spinal
The disturbance in ejaculation due to a damage in the sympathetic nervous system is separated as elimination of seminal emission and retrograde ejaculation, and in general the former seems to be more frequent (Retief 1950). It has not been elucidated up to the present by which part of injury of the sympathetic nervous system the elimination of emission or retrograde ejaculation occurs.

In the present study, in order to eliminate the influence from the center, section of the hypogastric nerve was performed to stimulate only its peripheral end. When the hypogastric nerve was cut and its peripheral end was stimulated, seminal emission occurred, but the seminal fluid was regurgitated into the urinary bladder, resulting in retrograde ejaculation. Then in order to observe emission and ejaculation in detail, connection between the urinary bladder and the posterior urethra was blocked with a clamp. Since erection of the penis was not observed in this experiment, the external urethral orifice was ligated to produce a similar effect to an increased resistance in the anterior urethra by erection. Then, a catheter was inserted into the posterior urethra, and time course of the pressure change in the posterior urethra caused by the hypogastric nerve stimulation was recorded. This record was tentatively nominated as the posterior urethrogram.

When the hypogastric nerve was stimulated the posterior urethral pressure

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<th>No. of dogs &amp; No. of tests</th>
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<th>Rhythmic movement of posterior urethral pressure</th>
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Mean 53.0 6'49" 27.9 15,8/5

center of "ejaculation" is located in the lower thoracic-lumbar region of the spinal cord.

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<th>TABLE 1. Hypogastric nerve stimulation and posterior urethral pressure</th>
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| Mean 53.0 6'49" 27.9 15,8/5 |
Nervous Control of Ejaculation

...gradually rose due to seminal emission. When the pressure reached 30–88 cm H$_2$O, a mean of 53.0 cm H$_2$O, rhythmic alterations suddenly occurred in the posterior urethral pressure (Fig. 1). The rhythmic alterations were accompanied with rhythmic movements of the penis, and loosening of the ligation of the external urethral orifice resulted in shooting out of the content, synchronizing with these rhythmic movements. Since the rhythmic alterations of the posterior urethral pressure disappeared by section of the periurethral musculature, the alterations were certainly due to contraction of the musculature. From the above-mentioned reasons the rhythmic alterations were supposed to be the same or at least similar phenomenon to ejaculation.

Further, it was noted that rhythmic alterations did not occur on raising of the internal pressure by instillation of physiologic saline without the hypogastric nerve stimulation, and that the alterations appeared when the internal pressure was raised during the hypogastric nerve stimulation and they disappeared as the internal pressure was lowered even during hypogastric nerve stimulation.

These findings indicate the necessity of not only an elevation of the posterior urethral pressure but also continuous hypogastric nerve stimulation in the rhythmic alterations of the posterior urethral pressure. And also these suggest that the hypogastric nerve is actively involved in the mechanism of ejaculation.

As to the relation of the pudendal nerve with seminal emission and ejaculation, the seminal fluid emitted into the posterior urethra is supposed to be ejaculated by contraction of the periurethral musculature by centrifugal stimuli passing through the pudendal nerve (Hotchkiss and Fernandez-Leal 1957, Potts 1957, Semans and Langworthy 1938). In addition to the periurethral musculature, the muscles in the perineal region are considered to participate in the contraction (Greene et al. 1963).

According to Sugita (1964), the pudendal nerve contains constituents of the autonomic nerve in view of the histological constitution, and Semans and Langworthy (1938) and Whitelaw and Smithwick (1951) insisted that the nerve fibers associated with ejaculation were the parasympathetic passing through this nerve.

It was experimentally and clinically confirmed in our department that section of the pudendal nerve decreased urethral resistance in comparison with that before the section (Funyu 1961, Rikimaru 1966). Semans and Langworthy (1938) observed that stimulation of this nerve produced rhythmic contractions of the periurethral musculature, by which ejaculation of the intraurethral fluid occurred, and Bors (1952) reported the occurrence of contraction of the internal urethral orifice. These reports suggest that this nerve is closely linked with ejaculation.

In the present experiments, when the bilateral pudendal nerves were stimulated, there occurred no seminal emission at all. When the both pudendal nerves were cut, seminal emission caused by the hypogastric nerve stimulation was hardly influenced by the section, and the elevation of the pressure did not change so much from that seen before the section. However, it was found that even though the
posterior urethral pressure rose, no rhythmic alteration of the pressure, nor rhythmic tremble of the penis occurred. A similar result was obtained in the artificial elevation of the posterior urethral pressure by instillation of physiologic saline under continuous hypogastric nerve stimulation.

While cutting of the pudendal nerve abolished the rhythmic alterations, stimulation of the peripheral end of the pudendal nerve augmented the internal pressure abruptly, causing ejection of the content fluid from the urethral orifice.

Accordingly, the rhythmic alterations of the posterior urethral pressure can be regarded as a reflex mediated by both the pudendal nerve and the hypogastric nerve, when the above-mentioned participation of the hypogastric nerve in the rhythmic alterations was taken into the consideration.

It was clearly shown by Kimura (1960) that the pelvic nerve sends medullated fibers to the accessory sexual organs, although Langley and Anderson (1895–1896) stated that there was no efferent fiber going from the sacral nerve toward these organs.

In regard to the previous papers on the relationship between the urethra and the pelvic nerve, there are not a few reports (von Zeissl 1983, Elliott 1907, Barrington 1931) suggesting the existence of some obscure interrelationship between them.

Regarding seminal emission and ejaculation, none of the previous literature has elucidated its relation to the pelvic nerve. Retief (1950) reported on the 11 cases which received the extensive thoraco-lumbar sympathectomy. There was ejaculatory disturbance in 7 cases, 3 of which showed retrograde ejaculation. Since seminal emission persisted after the operation, although it became retrograde ejaculation, he inferred that motor fibers of the epididymis, vas deferens and seminal vesicle for emission of the fluid might be parasympathetic nerve. Mitchell (1938) also stated that since emission continued after section of the hypogastric nerve, “ejaculation” might depend on the integrity of parasympathetic nerve, rather than that of sympathetic nerve.

In the present study stimulation of the pelvic nerve produced no emission of the seminal fluid into the posterior urethra evidencing that ejaculation did not occur by stimulation of this nerve alone.

Regarding emission of the seminal fluid into the posterior urethra by the hypogastric nerve stimulation and the posterior urethrogram, after section of the pelvic nerve, the seminal emission was considerably decreased and the posterior urethral pressure did not rise, as seen in Fig. 7. When the posterior urethral pressure was artificially raised under the hypogastric nerve stimulation, rhythmic alterations of the posterior urethral pressure were obtained again. However, stimulation of the pelvic nerve did not produce alteration of the posterior urethral pressure.

These facts seem to indicate that the pelvic nerve is involved in emission of the seminal fluid into the posterior urethra as well as the hypogastric nerve, but not related to ejaculation.
Appendix

This study was carried out from March to October, since the seminal emission caused by hypogastric nerve stimulation was noted to decrease in the winter season.

Acknowledgment

I wish to express my grateful appreciation to Professor Sentaro Shishito for his instruction through this study.

References


