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ORIGINAL PAPER

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# Perineal nerve stimulation for urinary sphincter control
## Experimental study

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**Abstract** The effect of electrostimulation of the perineal nerve on the external urethral sphincter (EUS) and urethral pressure was studied in 9 dogs. The nerve was displayed in the ischiorectal fossa through a para-anal incision and an electrode was applied to it. Perineal nerve stimulation effected an increase of the urethral pressure ($P < 0.01$) and EMG activity of the EUS ($P < 0.01$), but not of the vesical pressure ($P > 0.05$). The greater the stimulus frequency, the higher the rise of the urethral pressure and EUS activity up to 50 Hz beyond which no further increase occurred. The duration of response diminished with increased frequency, the shortest being at 80 Hz ($P < 0.001$). The latency of the response decreased with increased stimulus frequency ($P < 0.05$). In terms of force and speed of contraction, stimulus frequency of 50 Hz evokes the most adequate EUS contraction. An off-time of double the stimulation phase allows indefinite re-stimulation. Chronic electrostimulation of the perineal nerve may restore the sphincter tone so that perineal stimulation could be dispensed with.

**Key words** External urethral sphincter · Incontinence · Continence · Electrostimulation

Various techniques including electrostimulation have been used in the treatment of urinary incontinence. This is performed using either needle or surface electrodes [10, 14, 28] and plugs [7, 8, 13, 17, 25], electric pessaries [2, 9, 11], or by electrode implants into the pelvic floor muscles [1, 6] or sacral roots [12, 26, 27]. The aim of electrostimulation is to improve the activity of the external urethral sphincter and the pelvic floor musculature. This would result in an increase of urethral resistance [7, 8, 10, 13, 14, 17, 25, 28]. However, the overall clinical results of electrostimulation, especially in the long run, have been less satisfactory [12, 25].

The perineal nerve is the inferior and larger terminal branch of the pudendal nerve [22, 29]. It runs forwards below the internal pudendal artery which separates it from the dorsal nerve of the penis or clitoris. It then divides into posterior scrotal or labial and muscular branches. The perineal nerve, a branch of the pudendal nerve, supplies the external urethral sphincter. Direct stimulation of this nerve in sphincter incontinence was thought of to be an easier and direct approach in comparison to the sacral root approach, practiced by other investigators [12, 26, 27].

The aim of this study is to examine the effect of electric stimulation of the perineal branch of the pudendal nerve on the external urethral sphincter (EUS) and urethral closure pressure.

## Materials and methods

Perineal nerve stimulation was performed in 9 mongrel dogs (6 male, 3 female) with an average weight of 12.2 ± 4.4 SD kg (range from 10.5 to 16.5 kg). The bipolar electrodes used were of a cuff type with a surface area of 2 $mm^2$ each (Avery Laboratory, Farmingdale, N.Y.). Radiofrequency receivers (Avery) were implanted subcutaneously for activation via an antenna by an external adjustable stimulator.

The animals were given a 1-week period to acclimatize to the facilities prior to inclusion in the study. They were housed in cages and given access to water ad libitum. All dogs were maintained on a standard diet of meat dog chow each morning and were given free access to dry chow throughout the day.

The dog was anesthetized with pentobarbital sodium administered intravenously in a dose of 35 mg/kg body weight. The technique of pudendal nerve exposure was described elsewhere [19–21] and will be mentioned briefly. With the animal in the lateral position, a para-anal incision was made (1 cm from the anal orifice and at the base of the ischiorectal fossa) and the fossa was entered (Fig. 1 a, b). The inferior rectal nerve was identified crossing the base of the ischiorectal fossa latero-medially (Fig. 1 c). The nerve was hooked with the index finger and was followed to the pudendal nerve in the pudendal canal (Fig. 1 d, e). Pulling on the inferior rectal nerve with the index finger, stretched the pudendal nerve tight allowing

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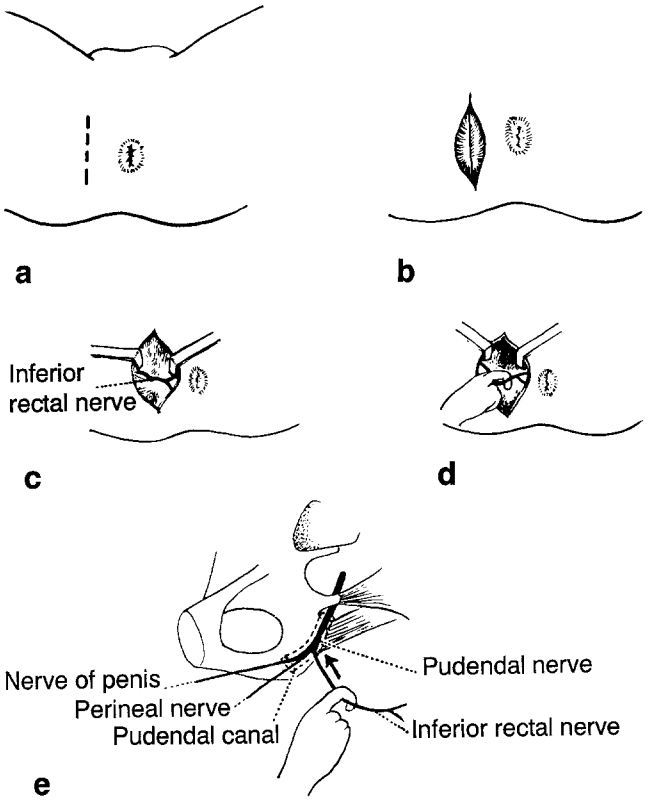


**Fig. 1a–e** Perineal nerve exposure. **a, b** Incision; **c** Inferior rectal nerve crossing ischiorectal fossa; **d** Inferior rectal nerve hooked with index finger; **e** Inferior rectal nerve followed to pudendal nerve

**Table 1** Urethral and vesical pressure response to different frequencies of perineal nerve stimulation (expressed as mean ± SD)

| Stimulus frequency (Hz) | Pressure (cm $H_2O$) | | | |
|---|---|---|---|---|
| | Urethral | | Vesical | |
| | Range | Mean | Range | Mean |
| 0 (basal pressure) | 65– 92 | 80.4 ± 10.7 | 13–19 | 16.6 ± 4.2 |
| 10 | 82–110 | 93.6 ± 10.8[b] | 12–18 | 16.2 ± 3.8[a] |
| 20 | 98–123 | 112.2 ± 15.3[b] | 12–19 | 15.9 ± 3.4[a] |
| 30 | 116–152 | 136.6 ± 20.6[b] | 13–18 | 16.4 ± 4.3[a] |
| 40 | 136–184 | 158.4 ± 36.4[c] | 11–17 | 15.8 ± 3.8[a] |
| 50 | 155–202 | 182.8 ± 38.2[c] | 11–19 | 16.2 ± 3.9[a] |
| 60 | 150–196 | 179.0 ± 35.3[c] | 12–18 | 16.2 ± 4.2[a] |
| 70 | 152–198 | 180.0 ± 38.8[c] | 13–19 | 15.9 ± 3.8[a] |

[a] $P > 0.05$; [b] $P < 0.05$; [c] $P < 0.01$

NB: The P value represents the level of significance when comparing basal urethral and vesical pressures with pressure changes occurring upon perineal nerve stimulation with different frequencies

easy identification. The fascia of the pudendal canal was slit open and the pudendal nerve was freed from the canal. The perineal nerve was then identified as the inferior and larger terminal branch of the pudendal nerve.

The electrode was applied to the perineal nerve in the ischiorectal fossa and the incision was closed. The radiofrequency receiver was implanted subcutaneously through a skin incision in the abdomen of the animal. A third-generation cephalosporin (Cefotaxime) was given parenterally for 3 post-operative days. Stimulation was applied

**Table 2** EMG activity of the external urethral sphincter on perineal nerve stimulation at different frequencies. The latency is also included (values given as mean ± SD)

| Stimulus frequency (Hz) | Potentials (μV) | | Latency (ms) | |
|---|---|---|---|---|
| | Range | Mean | Range | Mean |
| 0 (basal activity) | 62–108 | 90.4 ± 26.6 | – | – |
| 10 | 268–396 | 312.6 ± 46.7[a] | 2.1–2.9 | 2.5 ± 0.4 |
| 30 | 492–662 | 568.8 ± 58.2[b] | 1.8–2.5 | 2.1 ± 0.3[a] |
| 50 | 640–792 | 712.3 ± 60.4[b] | 1.2–1.6 | 1.4 ± 0.2[a] |
| 70 | 636–780 | 704.7 ± 62.2[b] | 1.2–1.6 | 1.4 ± 0.2[a] |

[a] $P < 0.05$; [b] $P < 0.01$

with a pulse width of 200 μs. The charge density applied to the nerve ranged between 2 and 6 μ C/cm$^2$/phase.

Two weeks after the operation, recordings were made under anesthesia using pentobarbital sodium administered intravenously. The vesical and urethral pressures were measured by a 10 F double-lumen catheter with side holes. The catheter was constantly perfused with saline at a rate of 2 ml/min and was connected to a strain gauge pressure transducer (Statham, 230b, Oxnard, Calif.). The response of the EUS to perineal nerve stimulation was recorded by a concentric needle electromyographic electrode (type 13 L 49 DISA, Copenhagen, Denmark) of 45 mm length and 0.65 mm diameter. In the male dog the needle electrode was introduced 0.5–1 cm in front of the anal orifice and (guided by a finger in the anal canal) was advanced until its tip lay just below the prostatic apex where the EUS is located [15]. A slight resistance was usually felt when the needle penetrated the muscle. In the bitch the needle electrode was introduced through the vaginal mucosa next to the external urethral orifice to a depth of approximately 0.75–1 cm.

The response recorded by the needle electrode was displayed on the oscilloscope of a standard EMG apparatus (type MES, Medelec, Woking, UK). Films of the potentials were taken on light sensitive paper (Linagraph, type 1895, Kodak) from which measurements of motor unit potential duration were made. The EMG signals were, in addition, stored on an FM tape recorder (type 7758 A, Hewlett Packard, Waltham, Mass.) for further analysis as required.

The correct position of the needle electrode into the sphincter was monitored by the burst of activity heard from the loudspeaker and visualized on the oscilloscopic screen of the electromyograph as the needle entered the sphincter. The latency of the external urethral sphincter response to perineal nerve stimulation at different frequencies was measured from the onset of the stimulus to the onset of the response. The aforementioned tests were performed at least twice to ensure reproducibility in the individual dog. The results were analysed statistically using ANOVA.

## Results

No complications were encountered from electrode application and all dogs tolerated the experiment. Perineal nerve stimulation resulted in the increase of both the urethral pressure ($P < 0.01$) and EMG activity of the EUS ($P < 0.01$); the degree of the increase was dependent on the frequency of the electrical stimulation (Tables 1, 2) (Figs. 2, 3, 4). With increasing stimulus frequency urethral pressure rose and sphincter EMG activity peaked at 50 Hz. Stimulation above this level affected no further increase in either the urethral pressure or the sphincter activity. In the meantime the vesical pressure showed insignificant changes upon perineal nerve stimulation, ($P > 0.05$) (Table 1).

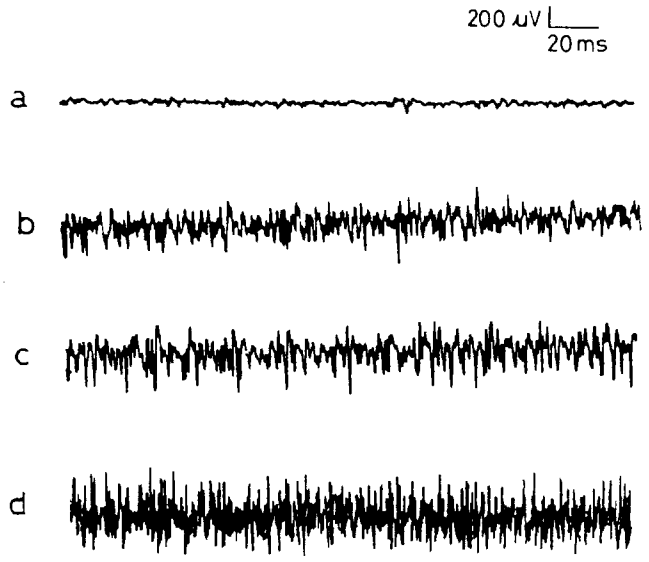


Fig. 2a–d EMG activity of the external urethral sphincter upon perineal nerve stimulation. The muscle activity increased with increased stimulus frequency. **a** Basal activity; **b** EMG activity upon stimulation frequency of 10 Hz; **c** EMG activity upon stimulation frequency of 30 Hz; **d** EMG activity upon stimulation frequency of 50 Hz

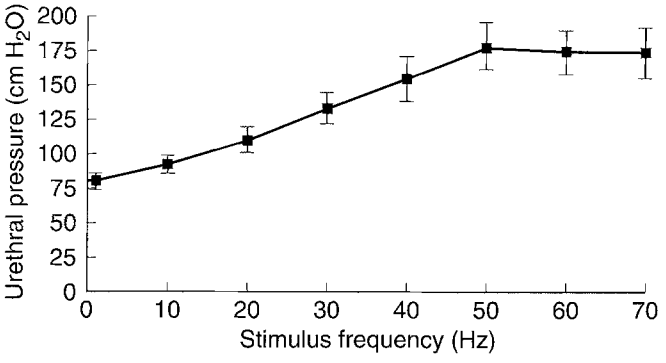


Fig. 3 Urethral pressure response to perineal nerve stimulation with different frequencies. The pressure increased with increased frequency up to 50 Hz, above which there was no significant pressure rise

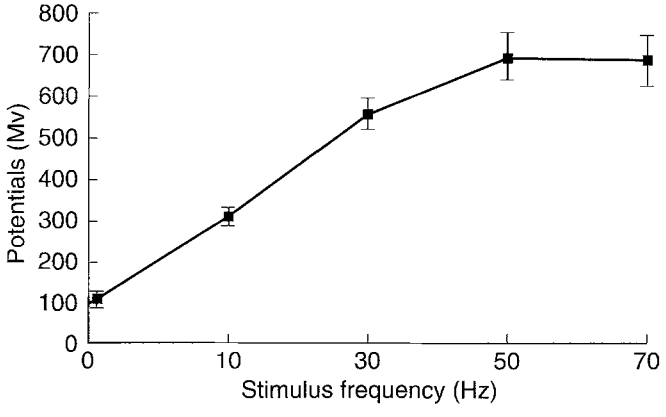


Fig. 4 Motor unit action potentials of the external urethral sphincter upon perineal nerve stimulation with different frequencies. The activity increased with increased frequency up to 50 Hz, above which there was no significant increase of activity

**Table 3** Relation of frequency of stimulus to stimulation phase (contraction time) expressed as mean ± SD

| Stimulus frequency (Hz) | Contraction time (s) | |
|---|---|---|
| | Range | Mean |
| 10 | 6–9 | 7.6 ± 1.2 |
| 20 | 6–8 | 7.0 ± 0.8[a] |
| 30 | 4–6 | 5.2 ± 0.9[b] |
| 40 | 4–6 | 4.5 ± 1.1[b] |
| 50 | 3–4 | 3.6 ± 0.8[b] |
| 60 | 1–3 | 2.4 ± 0.7[c] |
| 70 | 1–2 | 1.6 ± 0.6[d] |
| 80 | 1–2 | 1.4 ± 0.5[d] |

[a] $P > 0.05$; [b] $P < 0.05$; [c] $P < 0.01$; [d] $P < 0.001$

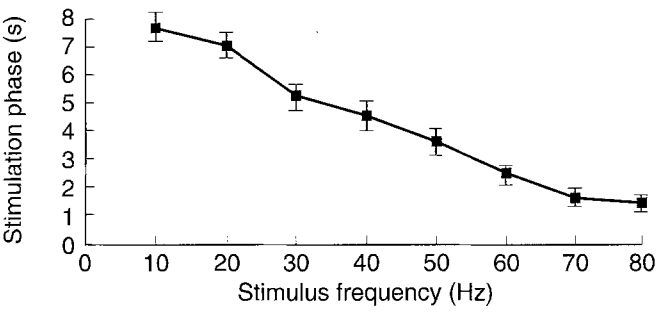


Fig. 5 The stimulation phase upon perineal nerve stimulation. It diminished with increased frequency up to 80 Hz, above which there was insignificant decrease

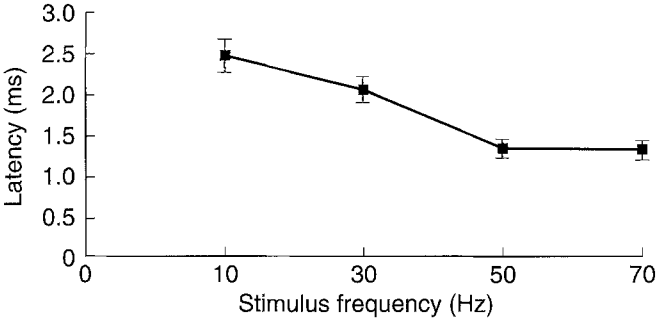


Fig. 6 The latency of the external urethral sphincter response to perineal nerve stimulation. It diminished with increased frequency up to 50 Hz, above which there was insignificant decrease

The duration of urethral pressure and EUS response varied according to the stimulus frequency. It diminished with increased frequency to reach the shortest duration at 80 Hz ($P < 0.001$) (Table 3) (Fig. 5) after which no further reduction occurred despite increases in the stimulus frequency. In addition the latency of the response varied with the stimulus frequency (Table 2) (Fig. 6). It decreased as the frequency increased ($P < 0.05$), but beyond a stimulation frequency of 50 Hz showed non-significant changes ($P > 0.05$).

After dropping to the baseline, and an off-time about twice as long as the stimulation phase the response


Fig. 7 EMG activity of the external urethral sphincter upon perineal nerve stimulation demonstrating the diminished activity if the muscle was re-stimulated after an off-time less than twice the stimulation phase: **a** EMG activity at stimulus frequencies of 10 Hz; **b** Re-stimulation after 5 s showing diminished EMG activity

resumed. If re-stimulation was induced before that time the response was weaker and the motor unit action potentials showed diminished amplitude and number of phases (Fig. 7). This was repeated several times with the same results. The response was reproducible indefinitely provided the period of off-time was double the stimulation phase. All results were reproducible at different stimulus frequencies (Fig. 8).

## Discussion

In the present study we directly stimulated the perineal nerve which innervates the EUS. The quality of sphincter contraction was dependent on stimulation frequency. The force of contraction increased and latency decreased with increased stimulation frequency up to 50 Hz, above which changes were insignificant. Moreover, the stimulation phase diminished with increasing stimulus frequency up to 80 Hz. The EUS thus fatigues earlier with increased frequency.

It appears from the present study that, in terms of force and speed of contraction, a stimulus frequency of 50 Hz evokes the most adequate EUS contraction and urethral pressure elevation. An off-time of double the stimulation phase allows re-stimulation indefinitely without fatigue.

Perineal nerve stimulation causes "induced" EUS contraction. It could be used for the treatment of urinary incontinence due to sphincteric dysfunction which is not accompanied with uninhibited detrusor contractions. If urinary incontinence is due to detrusor overactivity, we do not know whether perineal nerve stimulation and EUS contraction would inhibit this activity or not; this point needs to be investigated. It may be argued that the pain of nerve stimulation may limit the clinical applicability of the method. However, stimulation with the aforementioned intensities does not seem to induce pain when applied clinically as mentioned by other investigators [12, 30]. It is suggested that chronic stimulation may "restore" sphincter tone as a result of re-education and conditioning so that

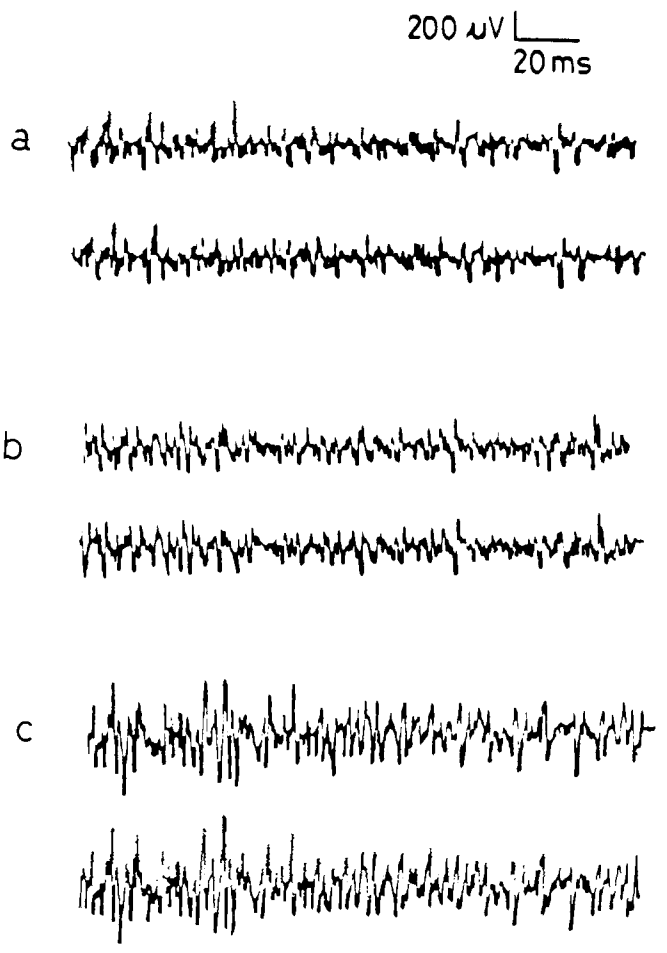


Fig. 8 EMG activity of the external urethral sphincter upon perineal nerve stimulation demonstrating the reproducibility of the response at different stimulus frequencies: **a** at 10 Hz; **b** at 30 Hz; **c** at 50 Hz

electric stimulation of the perineal nerve could eventually be dispensed with. Recent studies have demonstrated that striated muscles when under extended stress undergo change in fiber type and EMG activity and become a "compound muscle" which consists of a mixture of smooth and striated fibers [23, 24]. The striated fibers are fast twitch fibers which generate a high force but fatigue easily. The smooth fibers are slow twitch fibers which are capable of tonic activity at low levels of force with minimal fatigue [4, 5]. The "compound muscle", originally composed of fast twitch fibers, becomes more fatigue-resistant, but generates lower force of contraction than the original striated muscle [23, 24]. Furthermore, chronic electrostimulation of the striated muscles has effected changes in fiber type and enzymatic pattern so that the muscle becomes more tonic and fatigue-resistant [3, 16, 18, 30]. The histophysiologic effect of chronic electrostimulation of the perineal nerve on the EUS is an ongoing study and will be reported on later.

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