

ACUTE TIBIAL NERVE STIMULATION TO SUPPRESS BLADDER CONTRACTIONS IN THE CAT

Hypothesis / aims of study

Percutaneous Tibial Nerve Stimulation (PTNS) is an experimental therapy for patients with overactive bladder syndrome (urgency/frequency syndrome and urge urinary incontinence) who do not respond to conservative treatment and are not ready for surgical treatment. The precise mechanism of action of PTNS has not yet been clarified. The tibial nerve (L4-S3) is a mixed nerve and consists of motor and sensory nerve fibres. It is supposed that PTNS modulates signals to and from the bladder (S2-S4) by afferent stimulation through the sacral plexus. Urodynamic evaluation of patients with refractory overactive bladder syndrome successfully treated with PTNS has shown that PTNS does not suppress the detrusor overactivity, but delays its onset. Acute tibial nerve stimulation is not effective during urodynamic evaluation in these patients. This study is designed to investigate the acute effects of tibial nerve stimulation on bladder contractions in the cat.

Study design, materials and methods

One male cat (3.7 kg) was anaesthetised with ketamine (30 mg/kg i.m.) and alpha-chloralose (65 mg/kg i.v.; maintained with 15 mg/kg i.v. as needed). A transurethral (5F) catheter was placed in the bladder for bladder filling, and connected to an infusion pump. The bladder and ureters were exposed through a midline incision, and the ureters were ligated, transected and drained externally. A catheter (3.5F) was placed through the bladder dome, fixed, and connected to a pressure transducer for pressure measurements. The bladder pressure was displayed on urodynamic equipment, on a digital chart recorder, and stored on digital tape for off line analyses. A monopolar stimulation cuff electrode was placed around the left tibial nerve and a subcutaneous needle served as the return. Saline at room temperature was used for cystometry and the bladder was filled at 1 cc/minute until robust bladder contractions occurred, then acute tibial nerve stimulation was started (amplitude: 0.25-1 mA, pulse width: 100 microsec, frequency: 1-40 Hz, duration: ~ 10 s).

Results

Distention-evoked reflex bladder contractions with amplitudes up to 60 cmH₂O occurred at bladder volumes of 15-20 cc. These bladder contractions were acutely suppressed by tibial nerve stimulation in 11/23 trials. Frequencies \geq 5 Hz appeared more effective (suppression in 10/19 trials) than 1-2 Hz (suppression in 1/4 trials). Suppression was achieved only with stimulus amplitudes that resulted in toe flexion and fanning in the stimulated lower leg, and not when stimulation was below the threshold of the motor response. Suppression of bladder contractions was reversible, and there was a carry-over effect of 1-2 minutes between cessation of stimulation and the recurrence of bladder contractions.

Interpretation of results

Acute tibial nerve stimulation in a cat model appears to be effective in suppressing bladder contractions. Stimulation exhibited a carry-over effect since the bladder contractions did not recur immediately after stopping stimulation. These results suggest an explanation why *acute* PTNS is not effective in patients. In patients, PTNS is performed at an amplitude below the threshold of the motor response; this level of stimulation did not result in suppression in these experiments. Therefore, it is likely that in patients stimulation intensities are too low for acute alterations in the cystometric response. However, PTNS at the intensity required for a motor response is usually not tolerable by patients.

Concluding message

Acute stimulation of the tibial nerve in a cat model appears to be effective in suppressing bladder contractions when stimulation amplitude is above the threshold for a motor response.

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