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Title (type in CAPITAL LETTERS, leave one blank line before the text):

BLADDER RESPONSE TO URETHRAL FLOW IN THE AWAKE EWE

AIMS OF THE STUDY

The flowing of fluid along the urethra can facilitate the bladder micturition contraction, a reflex appropriate to achieve complete bladder emptying (1). The reflex has been consistently observed in animals studied in the decerebrate, spinal, or anaesthetized intact state (1-2). By contrast it appears very difficult to demonstrate in awake healthy humans (3). The aim of this study was to investigate the bladder response to urethral flow in the awake intact state, using urodynamics in the ewe. The results might help to assess the place of the reflex in normal micturition, and to explain some of the observations in the humans.

METHODS

Experiments were performed on 15 adult healthy ewes. The animals lay on their right side, the shoulders and legs gently restrained with slings attached to the recording table. No sedative drug was used. The ewes, fully awake, remained quiet for about an hour in this situation, showing no sign of discomfort. The bladder was catheterized per urethram with a 8F four-lumen balloon catheter. One channel was used for bladder filling and emptying, another for independent bladder pressure recording. The balloon (20 ml) was gently snugged at the internal urethral meatus, and secured in this position by the pull of a weight. Urethral flows were obtained by slowly injecting 10 ml saline at the level of the proximal urethra (1 ml/s, channel opening 25 mm below the balloon) and letting the fluid flow back along the urethra. Body warm saline was used for all bladder infusions and urethral flows. Rectal pressure was continuously monitored together with the bladder pressure.

The bladder was filled by intermittent steps of 10-50 ml, until a maintained (>20 s) detrusor contraction occurred. When this micturition threshold volume (MTV) was reached, the bladder was immediatly emptied. Urethral flows were performed at various predetermined bladder volumes, during the filling sequences. As a control for the effects of any fluid leakage into the bladder each urethral flow was immediately preceded and followed by an identical infusion (10 ml in 10 s) into the bladder. Controls were also made by injecting the same amount of fluid into the vagina or pouring it on the perineum. In six animals lidocaine (hydrochloride gel 2%, 10 min in the urethra) was used to anaesthetize the urethral mucosa momentarily.

RESULTS

On bladder filling all animals exhibited a typical micturition reflex with MTVs ranging from 90 to 250 ml. At bladder volumes subliminal for this bladder-to-bladder micturition reflex, urethral flows consistently evoked a detrusor contraction. The response started less than 2 seconds after the beginning of the flow and its duration (28 ± 11 s) largely outlasted the stimulus. In the same situation, identical infusions into the bladder had little effect. Similar results were obtained in all animals.

The detrusor response to urethral flow had the characteristics of a typical micturition reflex. Its maximal amplitude (range 15-60 cm H_2O) equalled that of the micturition contraction in the same animal ($p=0.59$, Wilcoxon signed rank test). Like the latter it was accompanied by a series of brief perineal contractions and a raising of the tail. Some degree of bladder filling (mean 66±16 % of MTV) was always required to elicit the response. This dependence on background facilitation from bladder mechanoreceptors is also typical of the normal micturition reflex.

The response was clearly triggered by the flowing of the fluid along the urethra. Control infusions at other

sites were ineffective. Urethral anaesthesia selectively suppressed the response to urethral flow (down to 13% of its control amplitude, p<0.0001), indicating that the responsible afferents are in the urethra.

CONCLUSIONS

Small urethral flows appear to elicit typical large bladder micturition contractions in awake intact animals. The response increasing with the flow rate (4), the reflex effect should be quite significant during the actual normal micturition. The reflex is present in all studied species and has been seen in some neurological patients (5). It receives descending excitatory and inhibitory controls parallel to those of the bladder to bladder micturition reflex (4). Taken together, these data suggest that the negative results in normal awake human subjects are due to descending inhibitory controls rather than to the lack of the appropriate pathways.

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Title (*type in CAPITAL LETTERS, leave one blank line before the text*):

RELATION OF HUMAN BLADDER BLOOD FLOW TO VOLUME AND COMPLIANCE

Aims of Study: The mechanism by which the human bladder is able to maintain perfusion in the face of increasing distention has not been established. The following study was performed in order to characterize how progressive filling changes bladder blood flow and microcirculatory resistance in conscious patients. We also investigated the relationship between bladder compliance and overall bladder blood flow.

Methods: Seventeen awake patients underwent water cystometry followed by cystoscopy under local anesthesia with intramuscular placement of a laser Doppler flow probe into the posterior wall of the bladder. Simultaneous measurements of systemic blood pressure (SBP), bladder blood flow (BBF), and intravesical pressure (Pdet.) were obtained with the bladder filled with normal saline to 0% (empty), 25%, 50%, 75%, and 100% of awake Cystometric capacity (Cmax). Additional measurements were obtained immediately post bladder drainage.

Results: Mean BBF was lowest in the empty state and increased with bladder filling until its highest level occurred at 75% of Cmax. Conversely, mean bladder microcirculatory resistance (MCR; mean SBP/mean BBF) was highest in the empty state and decreased with filling up to 75% of Cmax. From 75% of Cmax to 100% of Cmax, mean Pdet. Increased by 73% (25.2 cmH20 --> 43.5 cmH20), resulting in a 72% increase in mean bladder MCR and a corresponding 36% decrease in mean BBF. Complete bladder drainage led to a drop in mean Pdet. to baseline (0% Cmax) levels while mean