regeneration. There was no difference in RDLM $\exists_{II}$ tubulin mRNA grain density between nerve crush (0.09 $\forall$ 0.03 grains/:m) and control (0.09 $\forall$ 0.02 grains/:m) groups, indicating that the sciatic nerve was not injured and that RDLM grain densities serve as a good internal control.

CONCLUSIONS

Voiding behavior in the rat is highly variable and difficult to study. However, this research suggests that initiation of nerve regeneration occurs prior to normalization of voiding behavior after pudendal nerve crush. Urodynamic studies are needed to determine if pudendal nerve injury causes incontinence. This animal model could be useful for developing treatments to accelerate nerve regeneration and improve functional recovery after pudendal nerve injury.

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COMPLEX REPETITIVE DISCHARGES DURING URETHRAL SPHINCTER ELECTROMYOGRAPHY: CLINICAL CORRELATES

Aims of Study

Abnormal urethral EMG activity including complex repetitive discharges (CRDs) and decelerating bursts has been reported in patients with and without symptoms of lower urinary tract dysfunction and with a variety of neurologic disorders[1,2]. It has also been linked with a poor response to biofeedback techniques used to treat voiding dysfunction[3]. We sought to characterize patients with these EMG abnormalities in our referral urogynecolgy practice.

Methods

Needle EMG of the urethral sphincter is typically performed during multichannel urodynamic (MCUD) studies at our center. The Nicolet Viking IIE electrodiagnostic instrument processes the signal from a 30-gauge concentric EMG needle is placed approximately 5mm ventral to the urethral meatus. Optimal needle placement is confirmed by sound and by morphology of the motor unit action potential. Recording parameters include a bandwidth of 20-10,000 Hz, sensitivity of 50 μV and sweep speed of 10ms per division. EMG activity is continuously recorded during fill and void attempts with the patient seated at a 45 degree angle. Abnormal discharges at the time of needle placement or readjustment are discounted as likely artifact. Urodynamic diagnoses are made in accordance with the definitions of the International Continence Society. All patients with CRDs during any stage of their study were characterized by age, vaginal parity, history of neurologic disorder, lower urinary tract symptoms, and diagnoses during MCUD testing. The relationship between symptoms of strain voiding, and the finding of urethral CRDs was tested with a Chi-squared test of association and considered significant at the 1% level.

Results

The EMG data from 351 consecutive studies was reviewed. A subgroup of twenty-seven (8%) of the patients demonstrated CRDs during some phase of their study. The average age of this subgroup was 49.4 yrs (range 18-78) and mean vaginal parity was 2.3 (range 0-5). Whereas the majority of patients with CRDs were free of previously diagnosed or grossly detectable neurologic abnormalities, six (22%) of the patients had known or suspected neurologic disorders (4 spinal cord lesions, 1 with seizure disorder, 1 with possible multiple sclerosis). Strain voiding was reported in 13 (48%) of patients with CRDs, and in 18% of the entire population of 351 patients. A statistically significant association between the symptom of strain voiding and the finding of CRDs was present (χ^2=17.56, p<0.001). The mean spontaneous postvoid residual prior to urodynamic testing was 57cc (range 5-250). CRDs were recorded only at rest in 2 (7%) patients, only during bladder filling in 10 (37%) patients, only

during attempts to void in 5 (18%), during rest and filling in 2 (7%) patients, during filling and voiding in 6 (22%) patients, and throughout the study in 2 (7%) patients. Among the 23 patients with CRDs who reported any symptoms of incontinence, 19 (83%) had an incontinence disorder diagnosed by MCUD. 3 (11%) patients were unable to void during the study, and 10 (37%) patients voided with an interrupted urinary stream. Detrusor-sphincter-dyssynergia was present during attempts to void in 4 (15%) patients.

Conclusions

CRDs were recorded in 8% of our patients undergoing MCUD evaluation of their symptoms of urinary incontinence or retention. Slightly less than a quarter of this subgroup had a known relevant neurologic disorder. CRDs occur more commonly in patients with subjective voiding difficulty, but are not rare in women without voiding complaints. The relationship between these two entities remains to be explored. Based on the uncertain clinical relevance of the finding of CRDs, we believe it is premature to recommend alterations in the approach to patients with lower urinary tract dysfunction based on this finding.

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DIFFERENTIAL EFFECTS ON BLADDER AND EXTERNAL URETHRAL SPHINCTER OF CHEMICAL STIMULATION IN THE PERAQEDUCTAL GRAY OF THE ANESTHETIZED RAT

Aims of Study In rats, the pontine micturition center (PMC) is crucial for mediating coordinated micturition. It receives direct projections both from neurons in the sacral spinal cord [1] and from other areas of the brainstem including the periaqueductal gray (PAG)[2]. Because portions of the PAG receive direct projections from the spinal cord and contain cells that in turn project directly to the PMC, it has been proposed that the supraspinal portion of the micturition reflex path could have sequential connections in PAG and PMC [2, 3]. In urethane-anesthetized rats neurotransmission in the caudal part of the ventrolateral PAG was required to support micturition provoked by continuous cystometry [4]. We sought to determine whether discrete areas of the PAG, in addition to participating in the basic micturition pathway, had influences on bladder and urethra.

Methods Rats were anesthetized with urethane (1.2 mg/kg i.p.). A PE-60 tube was secured in the dome of the bladder for infusion and intravesical pressure measurements. Fine silver wire electromyography (EMG) electrodes were placed in the external urethral sphincter. The ureters were cannulated for urine drainage. With the rat's head secured in a stereotaxic apparatus, a minimal craniotomy was performed to expose the dorsal surface of the brain. Constant infusion saline cystometry (0.1 ml/min) was used to elicit repeated micturition contractions with bursting EMG activity and voiding. Measurements were also made under isovolumic