

ELECTROMYOGRAPHIC STUDIES OF URETHRA AND PUDENDAL NERVES IN FEMALE PATIENTS WITH TYPE III STRESS INCONTINENCE.

Aims

Some studies were accomplished involving the pelvic innervations in incontinent women, not being taken into consideration the grade of incontinence (1,2,3). The most serious degree of incontinence (type III) seems to have intrinsic urethral lesion as an aggravating fact, in which the neurological compromise is presumable but yet speculative. The limited information in this sense, were obtained by means of electromyographic needle evaluations (4). In this study, we intend to investigate urethral neurophysiological alterations in women with type III incontinence through surface electrodes, more accepted by the patients.

Methods

The tests employed were the urethro-anal reflex latency, pudendal somatosensory evoked potential (SSEP) and pudendal nerve terminal motor latency (PNTML), together with the urethral and clitorian sensory threshold determination. Seven patients with clinical and urodynamic diagnosis of type III urinary incontinence were studied. Thirty-eight normal volunteers with similar age formed the control group. The urethral sensory threshold and the latency of the urethro-anal reflex were accomplished by means of transurethral ring electrode (Dantec 9021L0111) stimulation and peri-anal surface electrodes for registration. Bilateral paraclitorian stimulation was used for the recording of the cortical evoked potential and sensory threshold determination. The recordings were obtained in the midline of the scalp (points Cz' and Fz of the 10-20 International System). For the registration of the distal latency of the pudendal nerve, we used the St. Mark's electrode (Dantec 9013L4401), taking into consideration the age of the patients and controls (5).

Results

The urethral sensory threshold and the urethro-anal reflex latency were recorded in 6 incontinent women and were absent in 1, and are exposed in Table 1 and Table2. The SSEP (P1 latency) after right and left stimulation were, respectively, 38.87 ± 1.04 ms e 38.80 ± 1.93 ms for the incontinent group and 38.04 ± 2.80 ms e 38.30 ± 2.15 ms for the control group ($p > 0,05$). The right and left clitorian sensory threshold presented values of 4.60 ± 1.60 e 3.63 ± 1.47 mA for the incontinent group and 4.12 ± 1.60 e 3.70 ± 0.99 mA for the control group ($p > 0,05$). The pudendal nerve terminal motor latency (PNTML) at the right side, was recorded in 5 patients, absent in 1 and not searched in another due to vaginal stenosis. The results are exposed in Table 3.

TABLE 1: Urethral sensory threshold X Control group.

	patients	
	Type III incontinence	control
SAMPLE	6	28
AVERAGE	5,80	3,47
SD	2,49	1,25
t = 3,36	p=0,002	gl =30

TABLE 2: Urethral-anal latency X Control group.

	Latency (ms)	
	Type III incontinence	control
SAMPLE	6	26
AVERAGE	62,80	55,53
S.D	8,97	5,24
t = 2,70	p=0,01	gl = 32

TABLE 3: Pudendal nerve terminal motor latency X Control group

	Terminal motor latency	
	Type III incontinence	control
SAMPLE	5	14
AVERAGE	2,70	1,83
SD	0,58	0,38
t = 3,861	p=0,001	gl = 17

Conclusions

These results suggest alterations on the urethral innervations of type III incontinent women. The consequences of this fact regarding treatment and the physiopathologic details involved, need further studies.

References

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