RESULT: In all patients, the pressure/flow plots obtained before operation showed a normal pattern which is characterized by nearly horizontal slope, low Pdet.max and high Qmax, indicating that the patients had neither outlet obstruction nor weak detrusor. After suburethral operation, all urodynamic parameters showed the statistically significant changes, i.e., Qmax was decrease while Pdet.max, Pdet.clos and linPURR were increased (Table 1). Among the parameters, linPURR showed the greatest change (5-fold increase). Of 10 patients, four had outlet obstruction symptoms such as frequency, urgency and urge incontinence after the operation, although bladder emptying was complete. The remaining six showed a good surgical outcome.

In correlation analysis between symptoms and urodynamic parameters, a decrease in Qmax showed a high SENS, high NPV, low SP and low PPV for symptoms. However, those were not statistically significant. Although elevation of Pdet.max and Pdet.clos over 10cmH2O showed a relatively high SENS, SP, PPV and PNV, no statistical significant correlation with the presence of symptoms was demonstrated. The increase of linPURR over $0.2cm/ml \bullet sec^{-1}$ was the only urodynamic parameter showing the significant correlation with symptoms of bladder outlet obstruction (Table 2).

CONCLUSION: The present study demonstrates urodynamic evidence of outlet obstruction after suburethral sling surgery. The postoperative decrease in Qmax as well as the postoperative increase in Pdet.max, Pdet.clos and linPURR suggest that this procedure inevitably induces bladder outlet obstruction, although a degree of obstruction may differ with each individual patient. Therefore, from a clinical point of view, it is important to determine whether outlet obstruction is clinically significant.

The present study also shows that the increase in linPURR alone can be correlated to symptoms secondary to obstruction. This may suggest that the clinical significance of obstruction should be diagnosed based on the changes in the slope of the pressure/flow plot after the operation.

Table 1. Changes of urodynamic values in Qmax, Pdet.max, Pdet.clos and linPURR

	Qmax (ml/sec)	Pdet.max (cmH_2O)	Pdet.clos (cmH_2O)	LinPURR ($cmH_2O/ml*sec^{-1}$)
Pre op mean (SD)	29.2 (14.2)	15.5 (9.18)	6.28 (4.20)	0.12 (0.08)
Post op mean (SD)	20.3 (10.5)	29.8 (12.3)	16.1 (10.1)	0.62 (0.78)
P value	<0.05	<0.05	<0.05	<0.05

Table 2. Correlation between symptoms and the change of urodynamic parameters

	ΔQmax<-15	ΔPdet.max>20	ΔPdet.clos>10	ΔlinPURR>0.2*
SENS(%)	50	100	80	100
SP(%)	66.7	50	60	83.3
PPV(%)	50	57.1	66.7	80
NPV(%)	66.7	100	75	100

Δ = postoperative Value - preoperative Value
* p < 0.05

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GABA-ERGIC CONTRIBUTION TO RAT BLADDER OVERACTIVITY FOLLOWING MIDDLE CEREBRAL ARTERY OCCLUSION

Aims of Study

The inhibitory effects of γ-aminobutyric acid (GABA) on bladder motility have been investigated in animal models, and in the micturition reflex pathway this agent appears

to have an inhibitory function at both spinal and supraspinal synapses (1). To evaluate the contributions of $GABA_A$ and $GABA_B$ mechanisms to bladder overactivity following cerebral infarction (2), GABAergic drugs were administered intracerebroventricularly to unanesthetized female rats.

Methods

To occlude blood flow in the left middle cerebral artery (MCA), a 4-0 monofilament nylon thread was inserted into the left internal carotid artery under halothane anesthesia. After recovery from anesthesia, rats were placed in a restraining cage. Coscious cystometric recordings were obtained via cystostomy catheter implanted into the bladder dome. Under urethane anesthesia urethral activity was measured as urethral perfusion pressure using a double-lumen catheter with its tip embedded in a cone-shaped plug, which was wedged in the bladder neck. External urethral sphincter activity were monitored by electromyogram using 2 wire electrode. After these recordings, infarction volumes were measured by perfusion staining with the use of 2% 2,3,5,-triphenyltetrazolium chloride.

Results

Intracerebroventricular administration of both muscimol ($GABA_A$ receptor agonist; 0.1 - 10 nmol) and baclofen ($GABA_B$ receptor agonist; 0.1 - 3 nmol) produced dose-dependent inhibitions of micturition with increases in bladder capacity (BC). The effects of high doses (1 - 10 nmol) were similar in sham operated (SO) and cerebral infarcted (CI) rats. However, lower doses of muscimol (0.1, 0.3 nmol) and baclofen (0.1 nmol) reduced BC in CI rats. After bicuculline ($GABA_A$ receptor antagonist; 1, 3 nmol) administration, BC in both SO and CI rats first decreased and subsequently increased. An increase in urethral pressure was observed after administration of bicuculline (3 nmol), but not with either muscimol or baclofen. Infarct volumes in muscimol-, bicuculline- or baclofen-treated rats were not significantly different from those of vehicle-treated rats.

Conclusions

These results suggest that $GABA_A$ and $GABA_B$ receptor appear to be involved in supraspinal inhibitory mechanisms in the micturition reflex. Cerebral infarction may change GABAergic mechanisms in the pontine micturition or storage center, resulting in increasing sensitivity to GABA agonists. Therefore, therapeutic potential of GABAergic agents for the overactive bladder should be carefully evaluated.

References

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