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RECONSTRUCTION OF BLADDER FUNCTION AND PREVENT RENAL DETERIORATE THROUGH END-TO-SIDE NEURORRHAPHY IN RATS WITH NEUROGENIC BLADDER

Hypothesis / aims of study

To investigate the feasibility of restoring bladder function and preventing renal deterioration by L6 ventral root (L6VR) and L4 ventral root (L4VR) end-to-side neurorrhaphy in rats with neurogenic bladder (NB).

Study design, materials and methods

Forty-two rats were assigned to the end-to-side coaptation (ECG, n=16), no coaptation (NCG, n=16), or control groups (CG, n=10). The ventral and dorsal roots of left L6 and S1 spinal nerves were transected in ECG, and the distal stump of L6VR was sutured to the lateral face of the L4VR. In NCG, the ventral and dorsal roots of left L6 and S1 spinal nerves were transected, but the distal stump of L6VR was not coapted; in CG, no operative procedure was performed. Nerve regeneration, bladder function, and renal function were evaluated with fluorogold (FG) retrograde tract-tracing, cystometry, electrical stimulation, histology and [serum biochemistry](#) measurements. The data were analyzed using one-way analysis of variance.

Results

In ECG, the FG-labeled neurons were observed in the left ventral horn of the L4 spinal cord. Maximum cystometric capacity, post-void residual urine, and bladder compliance in ECG were less than in NCG rats, but significantly greater than in CG. There was no significant difference in maximum voiding detrusor pressure between ECG and CG, but both were greater than NCG ($P<0.001$). ECG rats showed a significant increase in intravesical pressure when the left L4 VR proximal to the coaptation was stimulated, but no change was observed in NCG rats. The bladder weight of ECG rats was significantly lighter than in NCG rats. Serum creatinine, blood urea nitrogen, and the fibrotic area of bladder and kidney were decreased in ECG compared with NCG ($P<0.001$).

Interpretation of results

The presence of FG-labeled neurons in the left ventral horn of the L4 spinal cord segment in ECG rats indicates that a new [pathway](#) was established through end-to-side neurorrhaphy between L6VR and L4VR. MBC, PVR, and BC in ECG were fewer than those in NCG but greater than in CG. There was no difference in maximum detrusor pressure between ECG and CG, but both were higher than in NCG rats. When the left L4 VR proximal to the coaptation was stimulated, there was an increase in IVP in the ECG rats. These results further indicate that a new [neural reflex pathway](#) innervated the bladder. Chronic overfilling of the bladder can cause bladder wall hypertrophy, increasing bladder weight. With the transection of L6 and S1 spinal nerves, the bladder lost innervations and contractility. Since the rat could not micturate voluntarily, this resulted in chronically high pressure, causing structural damage and fibrosis and increasing bladder weights in the transected groups. However, ECG rats' bladder weights were lighter than NCG rats', evidence that end-to-side neurorrhaphy can mitigate bladder damage. Histology indicated that transection of L6 and S1 spinal nerves damaged the bladder wall, causing fibrosis. However, the end-to-side neurorrhaphy partially prevented this: the damage and [fibrosis](#) of the bladder wall was significantly less in the ECG rats than NCG rats. This indicates that the bladder was re-innervated by the nerve and the bladder function was partially restored by end-to-side neurorrhaphy. Transection of L6 and S1 spinal nerves cause urine retention, leading to renal fibrosis and renal dysfunction. H&E and Masson trichrome staining confirmed that renal structure was damaged in the NCG and ECG rats, but that the extent was less in the ECG. In contrast, kidney weights and macroscopic appearances showed no obvious differences among the three groups. The presence of microscopic damage in the absence of macroscopic changes may be because the experiment was too short to have caused serious kidney damage.

Concluding message

End-to-side neurorrhaphy is a useful method to restore bladder function and protect renal function in NB.

References

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Disclosures

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