training system was assessed by Hand grip force, Medical Research Council, Range of motion, Upper extremity Fugl-Meyer Assessment, and Motor Activity Log. In addition, we measured functional magnetic Resonance Imaging (fMRI) analysis for cortical reorganization during both hand movement. Participants received 60 min, 20 sessions, five days a week, for four consecutive weeks. The assessments were done before and after 20 training sessions

Results: Robot-assisted wrist training system was built with the bilateral wrist flexion/extension and pronation/supination performance that provides a repetitive active-active, active-passive, and passive-passive mode. Muscle strength, Motion of wrist, and motor function were enhanced after 4-week training of wrist. And the cortical activity change was associated with inducing reorganization of motor cortex networks

Conclusion: We developed bilateral wrist robotic device and robot-assisted wrist training system. Robot-assisted wrist training system showed improvement of upper limb function after training of wrist. Our system might be efficient robot-assisted wrist training for improving muscle strength, motor performance, and cortical reorganization in patients with chronic hemiplegic stroke and contribute to the development of translational research in rehabilitation robot.

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Development of an "In Situ" renal perfusion system to study the origin of urinary biomarkers in a nephrotoxicity model induced by gentamicin

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Abstract

Background and Aims: Gentamicin is an aminoglycoside antibiotic widely used for the treatment of many infectious diseases. Its main side effect is its nephrotoxicity, which occurs in 10-25% of therapeutic courses, despite proper monitoring and hydration of patients, can lead to acute kidney injury (AKI). We have previously demonstrated that urinary damage markers like albuminuria increased as a consequence of the addition of gentamicin. In the present work we aimed at specifically studying the renal handling of albuminuria in a nephrotoxicity model, through in situ renal perfusion experiments.

Methods: Male Wistar rats were administrated by a single dose of gentamicin (150 mg/kg), or not. After 5 days, rats were anesthetized and an extracorporeal circuit for kidney perfusion was set up. The renal artery, vein and ureter of the right kidney were ligated. The renal artery of the left kidney and the urinary bladder were canulated. A catheter was placed in the right carotid artery and connected directly to the renal artery. Urine was continuously collected from a catheter placed in the urinary bladder at 10 min intervals. After 1 h of renal perfusion with blood from the carotid artery, oxygenated and warm (37 °C) Krebs-dextran (40 g/L of dextran) was perfused through the renal artery at 3 mL/min, and was discarded through the renal vein. Albuminuria was measured in the different urine fractions.

Results: From the second day after gentamicin administration, albuminuria was significantly increased, as compared to control rats, in which urinary markers were undetectable. When exogenous Krebs solution prefunding the kidney, neither gentamicin rats nor control rats excreted albuminuria. As a control of the perfusion experiments, urinary markers still appeared in the urine in gentamicin rats whose kidney was perfused with its own blood. However, albuminuria was undetectable in control rats.

Conclusions: Our results support the idea that excess albumin found in the urine (albuminuria) as a consequence of treatment with gentamicin comes from the blood ultrafiltrate reaching Bowman's capsule and not from the renal parenchyma. More interestingly, our results provide and in situ method to test the origin of urinary biomarkers in different conditions.

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