Investigation of the Spinal Cord Injury Caused by Endorphin A(1-17)
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To study the pathogenesis of spinal cord injury, its secondary injury and methods of treatment, we established the spinal cord injury animal model by injection of endorphin into the subarachnoid space of rats. With these models we found that injection of endogenous endorphin A(1-17), endorphin A with removal of tyrosine (2-17) and endorphin A with removal of 4 amino residues from the carboxyl terminal (1-13), into spinal cord subarachnoid space (i.th) all could cause hinderance of motor functions of hind limb in rats. At 24 hours time, pathological sections showed that there was obvious motor neuron injury. This proved that endogenous endorphin A could produce spinal cord injury. And its N-terminal tyrosine and C-terminal amino residues were all important in causing spinal cord injury. NMDA receptor antagonists APV, KYN and MK-801 had counteraction against the injury of endorphin A. All had definite therapeutic efficacy. However, APV and MK-801 themselves had certain toxicity, while KYN was a kind if endogenous substance, its toxicity was rather low. It might be developed into a type of effective anti-spinal cord injury drug. A type of calcium antagonists, verapamil, had the best effect in counteracting spinal cord injury. This drug was quick in producing effect and the effect was comparatively complete. Under the used dosage (50nmol, i.th) there was no toxic effect. At the same time these also showed that NMDA receptor and Ca²⁺ had important effect in causing spinal cord injury.