A mechanism of retrograde degeneration and preservation of the facial nucleus following axotomy in brainstem

2005 Fiscal Year Final Research Report Summary

A mechanism of retrograde degeneration and preservation of the facial nucleus following axotomy in brainstem

Research Project

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16591434

Research Category

Grant-in-Aid for Scientific Research (C)

Allocation Type

Single-year Grants

Section

一般

Research Field

Cerebral neurosurgery

Research Institution

Kanazawa University

Principal Investigator

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Keywords

facial nucleus / retrograde degeneration / erythropoietin / nitrite oxide / brainstem / NADPH / facial nerve injury / Schwann cell

Research Abstract

This study was aimed to find out whether recombinant human erythropoietin (rhEPO) might have neuroprotective effect on the lesioned facial nucleus after axotomy of the central portion in brain stem. In the facial nerve transection in brain stem, compared with the control side, the survival ratio of the facial motoneurons was 25.0±2.4% (175±18.4/703±37.1) on day 14 and 2.8±1.4%(22.3±12.7/783±58.5) on day 28. Immunohistochemically, EPO were detected on astrocyte and EPO-R were detected on facial motoneurons in control. EPO expression was localized to reactive astrocytes in lesioned side on day 14. Nicotinamide adenine dinucleotide phosphate-diaphorase (NADPH-d) histochemistry to detect the neuronal nitric oxide synthase (nNOS) showed that the facial motoneurons were not positive for NADPH-d in control and on day 1. On the contrary, increased NADPH-d activity was seen in the facial motoneurons on day 4. The number of NADPH-d positive neurons and the intensity increased along with survival time, and nearly all survived motoneurons were stained on day 14. Administration of rhEPO (5000 U/kg) decreases the enzymatic expression of the number and staining intensity on day14(75.3±4.9%(481±128/636±144). The rats received rhEPO daily at dose of 5000 U/kg showed the 42.6±6.7% of the survival ratio of facial motoneurons on day 14, and 8.2±0.5%(57.3±11.2/703±145) on day 28. On the basis of these findings, it is suggested that rhEPO could act as one of the neuroprotective factors either by suppressing the nNOS activity or reducing the NO-mediated neurotoxicity in the lesioned motoneurons.