Spinal nerve root injury induced by chronic compression and nucleus pulposus

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Spinal nerve roots are often subjected to various degrees of both mechanical deformation and biochemical influence in different spinal disorders, and spinal nerve root injury is considered to be related to clinical symptoms such as pain and neurologic deficit. The overall aim of the investigations was to establish a model for analyses of underlying pathophysiological mechanisms in spinal nerve root injury and thus also provide a tool for experimental evaluation of different therapeutic interventions.

Methods: 1) Model: The first sacral nerve roots were exposed and an ameroid-constrictor was applied on the left S1-root just cranial to the dorsal root ganglion. The ameroid material absorbs water and, due to a rigid outer metal shell, the inner diameter of the constrictor is gradually reduced. After 1 or 4 weeks, EMG-measurements were performed and tissue samples were harvested for histological evaluation. The gross anatomy and the vascular supply of the pigs spinal nerve roots were studied by dissection and inkinjections. 2) Neuropeptides in compressed nerve roots; Concentrations of substance P and Vasoactive Intestinal Polypeptide (VIP), in spinal nerve roots and dorsal root ganglia following chronic compression, were analyzed by radioimmunoassay (RIA). 3) Neuropeptides after nucleus pulposus application: Concentrations of substance P and VIP, following application of autologous nucleus pulposus, were analyzed by RIA. 4) Mechanical and biochemical injury: Nucleus pulposus from a lumbar disc in the same animal was applied epidurally around a sacral nerve root, with or without an ameroid constrictor, and neurophysiologic and histologic evaluations were performed. 5) Methylprednisolone and nerve root injury: The effects of intervention by intravenous injection of methyl-prednisolone to reduce the nerve root injury after epidural application of autologous nucleus pulposus were assessed neurophysiologically and histologically.

Results: 1) Model: A significant decrease of the nerve conduction velocity in compressed compared to non-compressed spinal nerve roots was found after both 1 and 4 weeks. Histologically, the ameroid-constrictors induced nerve fiber damage, endoneural hyperemia, bleeding and inflammation at the compression zone. 2) Neuropeptides in compressed nerve roots: There was a statistically significant increase in substance P concentrations in the compressed dorsal root ganglia at both 1 and 4 weeks. The substance P concentration was also significantly increased in nerve roots after 1, but not after 4 weeks. The VIP levels were not significantly changed in either tissue. 3) Neuropeptides after nucleus pulposus application: There were no statistically significant differences in substance P or VIP concentrations in the exposed nerve roots or dorsal root ganglia compared to control nerve roots and dorsal root ganglia, exposed to retroperitoneal fat, after 1 or 4 weeks. 4) Mechanical and biochemical injury: There was a significant reduction of nerve conduction velocity for all exposed nerve roots as well as for control nerve roots when nucleus pulposus had been applied, but there were no statistically significant differences between the nerve conduction velocities following the combined application of nucleus pulposus and compression or nucleus pulposus alone. Light microscopic evaluation demonstrated axonal changes in nerve roots exposed to the constrictor. 5) Methyl-prednisolone and nerve roote injury: The nucleus pulposus-induced effects on nerve function were reduced if high-dose methylprednisolone was administrated intravenously within 48 hours after nucleus pulposus application.

In conclusion, a model for controlled, partial nerve root injury using a gradual compression-onset constrictor is presented. Changes in nerve root function, morphology and neuropeptides were found after induction of a controlled, graded chronic nerve root injury.