EFFECTS ON SPINAL CORD AND CRANIAL NERVE BLOOD SUPPLY AFTER THE DECOMPRESSION OF SPINAL CORD INJURY ASSOCIATED WITH CERVICAL SPINAL STENOSIS

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ABSTRACT

Objective*: This paper attempts to study and analyze the effects on the spinal cord and cranial nerve blood supply after the decompression of the spinal cord injury associated with cervical spinal stenosis.*

Method*: With white rabbits as the subjects, the author conducted decompression on the subjects suffering from spinal cord injuries associated with cervical spinal stenosis and observed their somatosensory evoked potentials (SEPs) and serum myelin basic protein (MBP) concentrations.*

Results*: judged by the kinematic scores and SEPs, Group A, B, C, D and E were better than other groups in terms of neurological recovery, and Group F and G were better than Group H, I and J. There are no distinctive differences between Group H and I and Group J. According to the analysis of serum MBP concentration and histology, the longer the spinal cord is compressed, the more serious damages the spinal cord structure will suffer.*

Conclusion*: Early decompression can effectively maintain the spinal cord structure of the spinal cord injury patient and help recover the cranial nerve functions.*

Keywords*: Decompression of spinal cord injury associated with cervical spinal stenosis, Spinal cord structure, Cranial nerve blood supply.*

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Introduction

Spinal cord injury associated with cervical spinal stenosis (shown in Figure 1) originates from the cervical intervertebral disc, which can easily lead to decreased spinal cord storage space and serious injuries by external forces. Spinal cord injuries associated with cervical spinal stenosis without fractures or dislocations are very common in middle-aged and elderly people, and cervical spinal stenosis appear before the spinal cord injury. However, cervical spinal stenosis in the initial stage has no obvious symptom and imposes little impact on the human body, so the patient may suffer from paralysis once they encounter spinal cord injuries. In clinical treatment, non-surgical treatment is relatively limited, and most researchers conduct clinical practice on the decompression of the spinal cord injury associated with cervical spinal stenosis. The decompression treatment can be combined with other surgical methods to form an effective treatment, so it has a high application value in the treatment of the spinal cord injury with spinal stenosis[(1)]. In terms of this, foreign researchers have compared the effects of surgical treatment to see whether this treatment is superior. Some believe that the decompression surgery can effectively improve the spinal cord functions of the patients with spinal cord injury associated with cervical spinal stenosis, and promote the recovery of their cranial nerve blood supply, and that it has clinical application research value.

In recent years, the prevalence of spinal cord injury disease has been increasing year by year, which not only affects the daily life of patients, but also may cause certain fatalities, seriously affecting the patients' families.

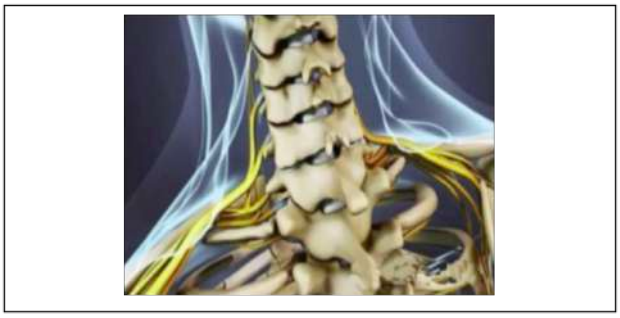

Figure 1: Spinal cord injury with cervical stenosis.

Acute spinal cord injuries can be divided into secondary injury and primary injury. The former is mainly caused by the mutual influences and gradual deterioration of the vascular mechanism and the neural biochemical mechanism, which can be prevented and controlled by some means. The latter is irreversible, in which case, the patients may experience problems such as penetrating injuries, spinal fractures, or spinal stretch[2]. According to the results of relevant clinical studies, the decompression surgery is one of the important and safe means to treat spinal cord injury diseases. During the decompression surgery, the surgeon needs to reconstruct the spinal sequence of the patient through surgical operations and correct the spine malformation. Although decompression has a certain effect in the treatment of spinal cord injury associated with cervical spinal stenosis, such effect may be reduced due to surgical operations, and as a result, the spinal cord and cranial nerve blood supply of the patient may not be recovered in time. In terms of the decompression surgery time, decompression surgeries of spinal cord injury associated with cervical spinal stenosis performed at different time have different treatment effects, resulting in differences in the recovery of the spinal cord and the cranial nerve blood supply[3]. Therefore, before the decompression surgery, we first need to conduct experimental study of the spinal cord injury disease through animal test, and then observe and compare the recovery of the animal bodies at different surgery time and select the optimal timing for decompression according to experimental results.

Based on the above analysis, it can be seen that, despite the many research results on the decompression of the spinal cord injury associated with cervical spinal stenosis, the effects of such treatment are still not stable and may easily affect the recovery of the patients' spinal cord and cranial nerve blood supply functions. Therefore, this paper, by taking white rabbits as the subjects, explores the effects of the surgery timing for decompression of spinal cord injury associated with cervical spinal stenosis on the patients' spinal cord and cranial nerve blood supply functions.

Methods

Experimental content

The author selected 95 New Zealand white rabbits of either gender, weighing between 2.5~3.0kg, from Jiangnan Experimental Animal Farm in Huishan, Wuxi, and fed them in the orthopedic animal laboratory of PLA 175 Hospital, with the room temperature controlled at 18~22°C and regular sterilization and ventilation.

The author divided the 95 white rabbits randomly into 10 groups - A, B, C, D, E, F, G, H, I and J, with 10 in each of these 9 groups - A, B, C, D, E, F, G, H, I and 5 in Group J. After molding, the author conducted anterior decompression to the subjects within appropriate time: Group A: 4h; Group B: 6h; Group C: 12h; Group D: 8h; Group E: 24h; Group F: 2d; Group G: 3d; Group H: 5d; Group I: 7d; and Group J: no compression in the control group.

Experiment reagents: PBS, PB, TriS-HCI phosphate buffer, TUNEL in-situ cell death detection kit (Wuhan Boster Biological Technology., Ltd.), import rabbit ELISA kit (Shanghai Westang Bio-Tech Co., Ltd.) and HE coloring agent (provided by Fuzhou Maixin Biotech. Co. Ltd.). Narcotic drugs: 2% pentobarbital sodium, lW0 chloral hydrate, 0.5% atropine sulphate injection. Animal drugs: gentamicin injection and penicillin powder injection (Yang et al., 2015). Specimen fixer: 4% paraformaldehyde.

Animal experimental table, microsurgical instruments, cryostat microtome, thermostatic water bath. Observation instruments: digital camera, OLYMPUS optical microscope, inverted fluorescence microscope OLYMPUS-BX6O, evoked potential recorder (Shanghai Haishen Medical Electronic Instrument Co., Ltd.), X-ray Machine, CT (Siemens), homemade modified A1Len.8 spinal cord impact damage device, which consists of an outer sleeve and a striker[4].

Experimental models

After giving intravenous anesthesia to a subject with 3% pentobarbital sodium at 30mg/kg, the author placed the subject on the operating table and let it lie on its back (Figure 2), performed skin preparation, sterilization and draping, and then incised the right side of the trachea longitudinally for about 3cm, retracted the musculus longus colli to the two sides, and exposed the CZ-C3 vertebral anterior (Figure 3).

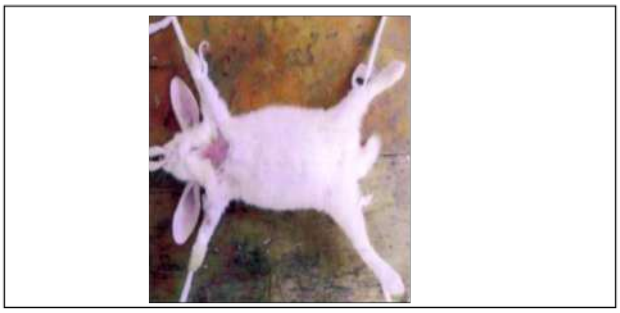

Figure 2: Animals fixed to the operating table.

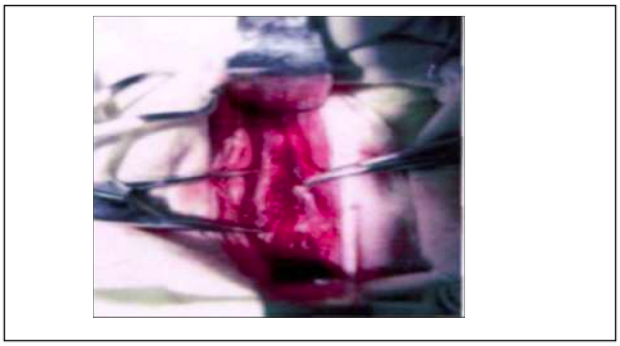

Figure 3: CZ-C3 vertebra exposed.

After identifying the CZ vertebral body, the author drilled the C3 vertebral body from the anterior wall to the back wall with a hand brace (Figure 3) to form a bone window with a diameter of 3.0lnm reaching the spinal dura mater, and placed a 150Inln-long round metal rod with a diameter of 2.5mm and a weight of 10.09 through a hollow tube with an inner diameter of 3.0lnm at 40mm above the spinal cord (through pre-test, the injury energy was determined to be 40.0gcm, as the external force that causes the incomplete injury of the spinal cord), and let the metal rod drop at a specified position freely to cause a spinal cord injury. The author observed whether there was any stress reflex in the lower limbs of the rabbit and evaluated the credibility of the spinal cord impact test[5].

The author placed a slightly-round-top titanium alloy screw with a diameter of 3. Sllun and a thread pitch of 1.Ilnln through the bone window. According to the spinal stenosis caused by compression, a crew with a length of Slllnl was placed. If any response was observed in the lower limbs of the rabbit, the author continued to screw it in by 1 turn (1 pitch) to complete the compression molding (30% of the diameter of the spinal canal). Then the author stitched the incision. After the surgery, the author performed CT scans and X-ray examination, and then removed the compressing screw at the designed time[6]. Within the 3 days following the surgery, the subject was given 200,000 units of Penicillin by intramuscular injection, twice a day. And it was also subject to manual bladder urination 3 times a day, respectively at 10:00, 16:00 and 22:00, until it could urinate on its own.

Indicator observation

Evaluation method: before the surgery, there was a 2-week adaptation period for the experimental animals. And they were observed for behaviors both before and after the surgery. The author released a single subject and placed food 1m away from it to measure the improved Tarlov score (Figure 4).

Figure 4: Determination of improved Tarlov score.

The observers were not the experimenters, but familiar with the scoring criteria. They gave the improved Tarlov scores of the injured animals 1h before the injury and 4h, 6h, 12h, 18h, 24h, Zd, 3d, 7d, 14d and 28d after it. The author used an evoked potentiometer for measurement. The stimulating electrode was a bipolar one, placed on the gastrocnemius skin; the recording electrode was a single-needle electrode, placed Zlnln after the coronal suture and 3Inm beneath the skin of the sagittal suture on the right side; the reference electrode was placed on the nose root, with a stimulation intensity of 2~12mA. With the significant jitter in the hind limbs as the criteria, the square wave was 0.3ms, the frequency ZHz, the cut-off frequency 20Hz~ZKHz, and the superposition performed for 500 times. The animals were measured for evoked potentials 1h before the injury and 4h, 6h, 12h, 18h, 24h, Zd, 3d, 7d, 14d and 28d after the injury[7].

Results analysis

4h after the injury, the author observed a small amount of focal hemorrhage, increase in the extracellular space of white matter and grey matter, mild edema, demyelination changes, enlargement of the space outside the axon, tunica vaginalis folds entering the axoplasm and axoplasm breaking.

The change is characterized by the dissolution of the cytoplasmic chromatin of the neurons and ischemia in the anterior horn cells of grey matter. At 6h, the author observed in the visible injured part an eccentric development of hemorrhage, which formed into a hemorrhage and necrosis region. The grey matter and white matter were markedly edematous, and the white matter was slightly degenerated and vacuolated. The motor neurons were slightly degenerated (Figure 5). 5-7 days after injury, most of the grey matter in the centre of the injury disintegrated, with some periventricular white matter, and cysts began to form. The boundaries were not fully clear. A large number of glial cell clusters were visible in the marginal areas, and part of the white matter region still had a normal structure[8-10].

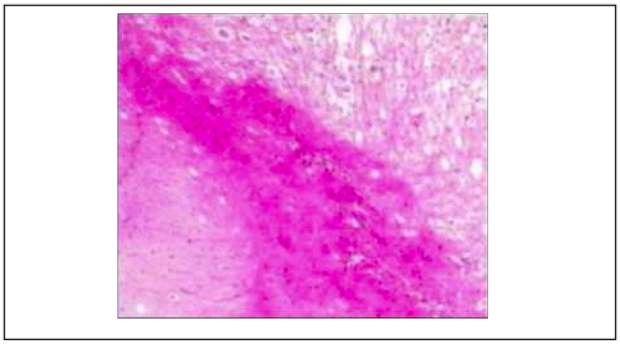

Figure 5: 6h HE stain x4.

The influences of the incubation time before and after the injury of each group are shown in Figure 4. As can be seen, the incubation time of each group was significantly prolonged after the injury ($P<0.05$), and there was no significant difference in the data of each group based on variance analysis ($P>0.05$). At 4h, the incubation time was significantly prolonged for each group. Within 18h, the incubation time of the decompression groups (A, B, C and D) had no significant change during 4-24h but began to decline after 24h. For Group E, the incubation time began to decrease after 2d. After 28d, Group A, B, C, D and E were close to normal. Compared with the control group and other groups, there was significant differences ($P<0.05$), but between these groups, there was no difference ($P>0.05$). After 24h, the incubation time for the decompression groups (E, F, G, H, I, and J) gradually prolonged during 4-24h and reached the peak at 24h. After that, the incubation time began to decrease[11-13]. The recovery of Group E, F, and G was slow. At 28d, the incubation time was slightly prolonged, but significantly better than that of the control group. There was no significant difference between these groups ($P<0.05$), but compared with Group A, B, C and D, there was significant differences ($P<0.01$). The recovery of Group H and I was very slow. On Day 28, the incubation time was shorter than that after the injury, but still a lot longer than that before the injury ($P<0.01$), and there was no significant difference compared with the control group ($P<0.05$). The incubation time of Group J (the control group) began to recover slowly on Day 14 and did not differ significantly from that after the injury[14,15].

Conclusions

According to SEP results, it can be seen that the decompression group had good recovery of the cranial nerve blood supply; According to the analysis of serum MBP concentration and histology, the longer the spinal cord is compressed, the more serious damages the spinal cord structure will suffer. Through the comparative experiment of the decompression of the spinal cord injury associated with cervical spinal stenosis on the white rabbits, it can be seen that, after the injury, the patients can achieve the best spinal cord structure and cranial nerve function recovery if they can receive decompression treatment within 18 days. If the compression time of the patient's spinal cord is prolonged, the recovery effect on the nerve functions may become poorer.

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