

Effect of Sports Training Based on Smart Medicine on the Nerve Function Recovery and Nerve Cell Apoptosis of Athletes with Hemorrhagic Brain Injury

Variun Verman*

Vytautas Magnus University, Lithuania

**corresponding author*

Keywords: Smart Medical, Exercise Training, Hemorrhagic Brain Injury, Recovery of Nerve Function, Nerve Cell apoptosis

Abstract: As an important part of the application of smart cities, the smart medical system is a powerful tool to achieve medical reform, improve medical conditions, and develop medical undertakings. It can provide urban residents with more convenient and smart medical services, and change the difficult and expensive plight of medical treatment. It plays an important role in urban development and solving urban problems. This article aims to observe and analyze the current situation and methods of athletes' brain trauma in the context of smart medical care. Analyze the impact of sports training on athletes' brain injuries. Understand the effects of exercise training on the recovery of nerve function and neuronal death. Compare with before and after exercise training, explore the effect of exercise training on neuronal function recovery and neuronal death of athletes with hemorrhagic brain injury. Using the research method of this article, we understand the influence of exercise training on the recovery of nerve function through the analysis of experimental data. And found that exercise training has an important role in restoring neurological function, combined with theoretical and experimental data to analyze the impact of exercise training on the neurological recovery of athletes with hemorrhagic stroke. The neuron mortality rate is up to 60%. Studies have shown that exercise training plays an important role in the rehabilitation of cerebral hemorrhage athletes, which is a good basis for clinical effective use.

1. Research Background

With the rapid development of my country's economy, major leaps have been made in infrastructure construction, people's living standards have been greatly improved, and the development of medical care is also extremely concerned. In addition to the various constraints of

medical technology and medical system, backward medical communication methods have also restricted The development of medical services. Crowded hospitals and lack of resources have caused problems such as expensive and difficult medical treatment, which have aroused widespread concern in the society. Interventions such as early drug treatment, physical therapy, occupational therapy, and speech therapy for stroke can help patients recover some of their impaired functions, but most patients still have varying degrees of sequelae, especially upper limb dysfunction [1]. Overcome acquired disuse and improve upper limb motor dysfunction after stroke [2-3].

Sports training therapy is mainly used to treat movement disorders after central nervous system injury [4]. In clinical practice, sports training therapy has proved to be very effective. In the middle and late stages, exercise therapy is effective for the recovery of nerve function after cerebral hemorrhage; with the prolongation of time, the curative effect becomes more and more obvious. In recent years, sports therapy has developed rapidly, mainly including sports training therapy [5-6].

The main basis for exercise training to promote the recovery of cerebral hemorrhage is neuroplasticity [7]. The specific mechanism is that the original parallel but not functional neural pathway can replace the damaged pathway, or the peripheral surviving neuron axon starts to germinate slowly, and finally form a new synaptic connection between nerve cells to replace the affected Damaged synaptic connections, a process that replaces cerebral hemorrhage with impaired nerve function, is also called functional reorganization [8].

This article uses experimental research methods to understand the role of nerve function recovery. And conducted a comparative study before and after use. Through theoretical analysis and experimental investigation through data, recording, sorting and calculation, drawing, analysis and processing, it is found that the impact of sports training on the brain injury of hemorrhage athletes. Through exercise training to simulate the recovery of nerve function of hemorrhagic brain injury and the simulation of neuron-related statistical data sets, combined with the empirical analysis of the influence of exercise training on nerves. Functional recovery and neuronal death combined with correct information to summarize and analyze the impact of sports training on neuronal function recovery and neuronal death of athletes with hemorrhagic brain injury. The results show that the recognition rate of this method reaches 25%.

2. Theoretical Basis

2.1. Hemorrhagic Brain Injury

CIMT mainly includes two aspects: restricting the health side; forcing the use of the affected body for a lot of repetitive, high-intensity training. After a stroke, there are often varying degrees of dysfunction. Rehabilitation training intervention can shorten the functional recovery process of patients and help patients return to society as soon as possible [9]. After cerebral hemorrhage, the blood flow of surrounding brain tissue drops to 20% -50% of normal blood flow. This low blood flow will not cause a large area of cerebral infarction. NGF mainly promotes the growth, development and maturation of neurons under physiological conditions. It can protect nerve cells in patients with cerebral hemorrhage. Inhibit delayed neuronal death, promote the normal proliferation of glial cells, and promote the formation of a large number of new blood vessels in the damaged brain tissue[10-11]. The motor dysfunction of many patients with cerebral hemorrhage is not caused by paralysis, but it is not timely intervention treatment through exercise training, which leads to postural spasm, joint deformity, language disorders, dysphagia, stool and urinary incontinence. As an important factor for brain function recovery, exercise training plays an important role in improving the degree of brain function recovery. Timely intervention of passive and active

movements is conducive to inducing the superficial and deep receptors of the skin, joints, and muscle tissue to produce a large amount of information transmission, and is also conducive to a large amount of output electrical activity in the brain center. NGF mainly plays a physiological role through p75 and TrkA receptors[12].

After intracerebral hemorrhage, microglia can be activated through different pathways within a few minutes. The traditional view is that CIMT mainly includes two aspects: restricting the health of the body; forcing the use of the affected body for a lot of repetitive, high-intensity training [13-14]. After a stroke, there are often varying degrees of dysfunction. Rehabilitation training intervention can shorten the functional recovery process of patients and help patients return to society as soon as possible.

Microglia interact with neighboring neurons and astrocytes through their branches and processes. Various pathological damages in brain tissue can cause the activation, morphology and function changes of microglia. The activated microglia are considered to have neurotoxicity and neuroprotective effects. The overall effect is determined by the pathological state and severity of brain injury. At present, it is believed that the occurrence of this phenomenon can be explained from the following aspects: mechanical compression of hematoma leads to the destruction of microcirculation function and structure, the volume of hematoma is proportional to the reduction of rCBF, damage to nerve conduction fibers leads to vascular regulatory dysfunction; local damage to the brain Tissue releases and absorbs vasoconstrictor active substances, which leads to vasoconstriction and further reduction of cerebral blood flow; the formation of cerebral edema and high intracranial pressure leads to automatic regulation of cerebral blood flow dysfunction; leukocyte adhesion and aggregation, some scholars have found that the reduction of rCBF After bleeding around the area, I was obviously accompanied by a large amount of white blood cell adhesion and aggregation. In addition, if the application of anti-white blood cell aggregation was applied before I could significantly increase the extent and degree of bleeding of the surrounding tissue, white blood cell adhesion and aggregation were also local. After playing a certain role in the mechanism of my blood flow decreased [15]. ICH mechanically compresses the surrounding tissue for more than 48 days to form a hematoma, which can cause irreversible damage to the nerve cells of the surrounding tissue. After the formation of cerebral hematoma, it can cause edema of brain tissue through space-occupying effect, which in turn leads to the generation of high intracranial pressure and the decrease of blood flow of brain tissue. It is generally believed that hematoma can expand within 8 hours after cerebral hemorrhage, but the expansion of hematoma does not represent the deterioration of certain clinical symptoms [16]. The deterioration of clinical symptoms may also be related to the severity of ischemia and hypoxia in peripheral tissues in the early stage of hematoma, compensatory ability of peripheral cranial cavity and bleeding rate; ultra-early thrombolysis can reduce the mechanical compression of hematoma in surrounding tissues and prevent the further development of vascular edema Development has important theoretical significance. Microglia are also considered to be the first cells to respond to cerebral hemorrhage. Erythrocytes can cause microglia to produce phagocytic functions in vitro and increase the expression of microglia proinflammatory genes; heme and fibrinogen released after rupture of red blood cells can also induce the activation of microglia through the toll-like receptor pathway. Promote the up-regulation of nuclear transfer factor expression and increase the expression of microglia proinflammatory genes. Glial cells can buffer K⁺, participate in neurotransmitter metabolism, ease the release of excitatory amino acids such as glutamic acid[17-18]. Intracranial hemorrhage secondary brain injury is a complex pathophysiological process involving many factors. The decomposition of red blood cells and other dead cells in the hematoma, the release of various

toxins, inflammation, and the release of thrombin will cause cell death. The cell death time is longer and the damage range is wider. The center and close relationship of most hematomas is cell necrosis and cell death around the hematoma. Hemorrhagic stroke seriously threatens the production and life of its residents.

After cerebral hemorrhage, there was an area around the hematoma, tissue damage gradually increased, and edema appeared. The ultrastructural changes in this area are reversible within a certain period of time. If appropriate treatment measures are taken during this time, the function of the damaged tissue can be restored. This area is called the penumbra or penumbra around the hematoma. Early cerebral hemorrhage. The swelling of astrocytes around the hematoma may be related to the stability of the neuronal environment [19]. Hemoglobin is lysed and released by red blood cells, which has a toxic effect on neurons; platelets can release serotonin and platelet-derived growth factors, increasing vascular permeability, and high thrombin concentration can produce cytotoxic effects on neurons and astrocytes Causes secondary damage.

2.2. Sports Training

We compared the efficacy of the three treatment options of rehabilitation training alone, acupuncture alone and the combination of the two, and explored a more effective rehabilitation program after stroke [20]. Rehabilitation training and acupuncture treatment are more cost-effective, or a combination of the two is more conducive to the recovery of the patient's function, and can also bring less economic burden to the patient. The functional reorganization of the cerebral cortex that occurs naturally after cerebral hemorrhage has certain limitations. As an important factor for brain function recovery, exercise training plays an important role in the degree of brain function recovery. It can promote the growth of axons and promote the functional reorganization and recovery of cerebral cortex. Exercise can enhance the role of neurotrophic factors, inhibit the expression of apoptotic genes, reduce the expansion of cerebral hemorrhage hematoma volume, and reasonable exercise training can improve the plasticity of the central nervous system. For example, the training method of neural manipulator.

Stroke is the main cause of limb motor dysfunction, which leads to abnormal motor function, speech swallowing, cognitive mood and behavior, which limits the patient's ability to move and social participation [21-22]. They need help from others to complete various related tasks, which seriously affects the patient's ability to participate in society. Neuroplasticity is related to the rehabilitation of muscle movement after stroke, including the establishment of new neural connections, the acquisition of new functions and the repair of injuries. Neuroplasticity is affected by injury to the hemisphere of the stroke [23]. Patients with stroke hemiplegia need to learn how to control trunk movement again. Through continuous learning, adaptation and adjustment of the differences in the spatial composition and time series of different muscles of the body, the energy consumption required by patients to complete sports goals is reduced as much as possible. Continuous sensory stimulation will continue to induce the recovery function of the motor cortex and enhance the excitability of the cortical spinal cord. The form of repetitive movement is the basis of motor learning and sports rehabilitation. The brain judges, analyzes and processes the incoming information, and effectively controls the muscles and skeletal nerves. Through continuous feedback and continuous adjustment of exercise modes, an optimized nerve remodeling mechanism is formed, which matches the strength, speed, and coordination of related muscles, thereby promoting and restoring various abilities. Repeated finger flexion and extension exercises can help flexor and extensor tendons to collect more motor units, improve nerve function and innervating muscle

function, and thus improve hand function and daily life activities. The waste of the affected hand and the compensation of the healthy side often result in the only exercise capacity of the affected side, rather than the actual motor function. When the upper limb function is restored, the muscle contracture of the affected limb greatly reduces the rehabilitation effect. Hand rehabilitation robots can generally be divided into two types, one is an auxiliary robot system, and the other is a therapeutic robot system, which can provide task-related rehabilitation training. The principle of treatment is to restore the plasticity of the central nervous system as the main standard. The clinical treatment cycle is long and the curative effect is slow. It is difficult to play a good role in improving finger pain, reducing finger muscle tension, and finger grip. The most important thing is that with the extension of the course of the disease, the patient's rehabilitation process gradually entered the platform stage. Facing repeated and boring rehabilitation training, they lost interest and confidence in rehabilitation.

Safe, high-intensity, more targeted, task-related training for brain injury patients, help clinical therapists work effectively in rehabilitation guidance, and promote patients to return to family and society to improve the recovery effect as soon as possible. Compared with traditional rehabilitation technology, robotic rehabilitation technology highlights the efficiency, fun and operability of training, and has been recognized by rehabilitation doctors at home and abroad, so that more and more rehabilitation robots are used in clinical rehabilitation. Mechanical arm training can better improve the upper limb motor function of stroke patients [24]. The robotic rehabilitation system uses a low-dimensional control scheme combined with EMG biofeedback to effectively restore the motor function of stroke patients. By measuring the thresholds set by the electrical signals of muscles on the body surface of different patients, different training programs can have different difficulty and difficulty, making the training more targeted and targeted, which is in line with the concept of precision medicine advocated by the world today. The most important point is that the manipulators set up in different training modes can set a large number of repetitive active sports training, and a large number of repeated rehabilitation training can improve the sensory input information and promote the establishment of synaptic connections for regenerated nerve axons that are guaranteed relationship.

2.3. Recovery of Nerve Function

The remodeling of nerve function is not just the process of axon regeneration and growth. The patient's final functional recovery indicates that this neural remodeling requires more precise control to rebuild the neural network. Six months after the onset, approximately 50% of patients have moderate to severe upper limb motor dysfunction, and 11 weeks after the onset of stroke, the recovery of upper limb motor function is stagnant, and independent life of upper limb motor function is particularly important [25]. The current clinical treatment is still relatively simple. Whether it is dehydration, intracranial pressure reduction or surgical minimally invasive surgery to remove hematoma, the prognosis is difficult to change. Neurotrophic factor refers to a large class of protein molecules produced by the body itself.

Neurotrophic factors not only exist in the nervous system of normal people, but also have multiple functions to protect damaged nerve tissue. Microglia is considered to be the first non-neural cell to respond to brain injury. It can regulate the survival and function of neuronal cells under pathological conditions of brain tissue. When the complement system is induced and over-activated for some reason, many fragments are produced. These fragments can induce the activation of related cells, release a large number of toxic molecules, further expand the

inflammatory response network, and destroy the body's own structure and tissues. The stress distribution changes with increasing load. The stress distribution generated by the hematoma compression reflects the degree of tissue damage and the direction of hematoma expansion. In this process, blood pressure and intracranial pressure correspond to load load and environmental load, respectively. Therefore, the physical damage of brain tissue is essentially the result of changes in stress and strain distribution of brain tissue after mechanical compression of hematoma. The hazard of stroke is serious neurological damage. At present, the high-risk population of stroke is no longer limited to the elderly, more and more middle-aged people, even young people have become a high-risk population of cerebrovascular events, which makes patients more urgent to improve various dysfunction after stroke And higher standards. Elderly patients hope to improve their motor function, achieve self-care, and reduce family burden, while young and middle-aged patients hope to return to work by improving their physical movement. The various exercise abilities of the upper limbs play an indispensable role in people's daily activities. Many basic activities in the daily life of hemiplegic patients are difficult to complete. At present, there is no clear clinical effect of various neuroprotective drugs, especially evidence-based medicine. The relationship between free radicals and brain tissue damage can be indirectly explained by the effectiveness of antioxidant therapy. In particular, as a free radical scavenger, metro amine can significantly reduce brain tissue damage after cerebral hemorrhage. The concept of intensive training rehabilitation therapy usually has two meanings: one is to increase training time, and the other is to increase training intensity. The former is mainly to increase the extra training dose or increase the total training time, the latter is usually to increase the training frequency.

The effect of exercise training on the recovery of exercise ability of patients with hemiplegia is ideal. In the process of rehabilitation, it helps patients to restore motor function to the greatest extent, and brings new hope to patients with stroke and hemiplegia. In addition, the effect of finger thinning on functional training for stroke hemiplegic patients needs to be improved. Improving the hand function of stroke patients with hemiplegia, thereby improving the life happiness index and helping them return to work, is a major challenge for rehabilitation medical workers. The mechanism of secondary nerve injury is complex. The possible mechanisms are the mass effect caused by hematoma expansion, the formation of cerebral edema around the hematoma, the abnormal metabolism around the hematoma, and the changes in the local cerebral blood flow around the hematoma.

3. Experiment

3.1. Research Object

Experimental animals and groups: 60 healthy male rats, weighing 260-300g, provided by the Experimental Animal Center of Hebei Medical College, clean grade. It is fed by standard feed and purified water. The feeding environment is a multi-layer flow rack in the animal room with constant temperature (20-26 °C). The first two groups used a model of collagenase to induce cerebral hemorrhage, and the operation group used physiological saline instead of collagenase. Each component is 24 hours, 72 hours, 7 days, 14 days, 21 days, 28 days, 6 post-execution points, 5 points in each group. Sports training equipment and methods: After 24 hours of cerebral hemorrhage in the sports group, the training was carried out by a special person according to the sports training plan. The running cage used for training is a thin cylindrical steel wire mesh cylinder with a circumference of 200 cm and a length of 50 cm. The base has a fixed frame and a handle at

one end to control the rotation speed. There are three compartments in the middle of the running cage, which can train 3 rats at the same time. It is used to train mice to grasp, spin and run.

3.2. Experimental Plan

Animal behavior evaluation: Bedson score: Lightly grasp the tail of the rat and lift it 10 cm above the desktop. The two front paws of the normal rat extend to the desktop. In rats with brain injury, the contralateral forelimb flexion and posture changes ranged from mild wrist flexion, elbow and shoulder extension to severe wrist and elbow flexion and shoulder rotation. Place the rat on a large, soft, elastic, and smooth recording paper for easy grasping. Then lift the tail and gently push the mouse's shoulder with your hand until the forelimb slides a few centimeters. This action is repeated several times in different directions. Rats with normal or mild dysfunction will produce the same thrust in different directions. 0 points: no neurological deficit; 1 point: any flexion of the forelimb, no other abnormalities; 2 points: decreased lateral thrust resistance; 3 points: same grade 2 behavior, Follow the spontaneous rotation (free movement to the paralyzed side circle). Balance beam walking test: measuring exercise coordination ability. The balance beam is 80 cm long, 2.5 cm wide, and 10 cm high from the ground. Scoring standard; 1: through the balance beam, the chance of falling is less than 50%; 2: through the balance beam, the chance of falling is greater than 50%; 3: through the balance beam, but the hind limbs of the affected side are paralyzed Move forward; 4 points: Can not pass the balance beam, but can sit on it; 5 points: Put the mouse on the balance beam, the mouse will fall down. Muscle strength test: A steel rope with a diameter of 0.15mm, a length of 46cm, is placed at a height of 70cm from the ground, and a foam box with a height of 3.5cm is placed. Put the two front paws of the mouse on the rope, loosen it, and record the time of the mouse on the rope. 0 o'clock: hanging on the rope for 0.2 seconds; 1: o'clock: hanging on the rope for 3-4 seconds; 2: o'clock: hanging on the rope for 5S; 3: o'clock: hanging on the rope for 5 minutes, placing the hind legs on the rope. Material and pathological section: after bleeding in each group, decapitate at different time points, carefully remove the skull, and remove the intact brain tissue. 4mm brain tissue in front of the forehead was taken, and a coronal incision was passed through the puncture point on the surface of the brain. Focal cerebral hemorrhage. Brain tissue in front of the puncture point was used to measure brain water content. The brain tissue about 4mm after the puncture point was taken and fixed with 4% polyoxymethylene solution, gradient alcohol dehydration, xylene transparent, wax dipping and embedding. Paraffin section, he staining, histological observation. Observation of cerebral edema: immediately prepare the same size and weight of aluminum foil paper to reduce brain tissue, weigh (wet weight) and record separately, then put it into a constant temperature drying oven, 100 °C, after 24 hours, remove the brain tissue to measure After returning to room temperature, (dry weight), re-weight and return to constant weight. Statistical method: use state8.0 statistical software to analyze the results on the computer, and record the test results with the average standard deviation. Weighing: Each rat is weighed at different training times, and the average value is taken as the weight of each group. Apoptosis detection: using TUNEL method. The specific steps were carried out according to the instructions of the kit, and the working concentration was 1: 100. Apoptotic cells are positive cells, and the nucleus is brownish yellow. Five fields were randomly selected to calculate the number of positive cells under high power (400X) microscope.

4. Experimental Results

4.1. Recovery of Nerve Function

(1) Walking test results

Table 1. Walking test results

Time	Trial Group	Control Group	Operation group
24h	3.5±0.45	3.5±0.45	0.0±0.0
72h	2.5±0.45	2.9±0.0	0.0±0.0
7Day	0.9	1.5±1.45	0
14Day	0	1±1.35	0
21Day	0	0	0
28Day	0	0	0

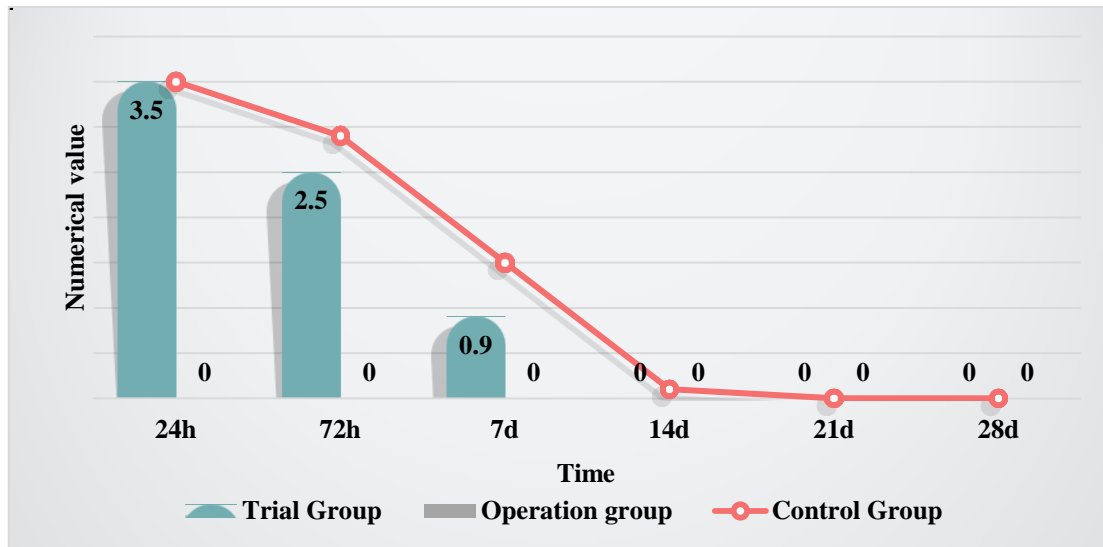


Figure 1. Walking test results

According to the data analysis, as shown in Figure 1 and Table 1, it is significantly different from the surgery group. After that, the exercise group began to improve, and improved obviously at 7 days; and returned to normal on the 14th day. However, the control group recovered slowly and the injury lasted until 21 days.

(2) Weight analysis

Table 2. Weight analysis

Time	Trial Group	Control Group	Operation group
24h	299±0.0	299±0.0	299±0.0
72h	284±9.9	289±12.04	299±0.0
7d	224.6±10.26	248±6.42	299±0.0
14d	203±4.48	252±5.71	319±4.10
21d	347±20.68	391±7.37	399±0.0
28d	361±15.43	391±7.37	399±0.0

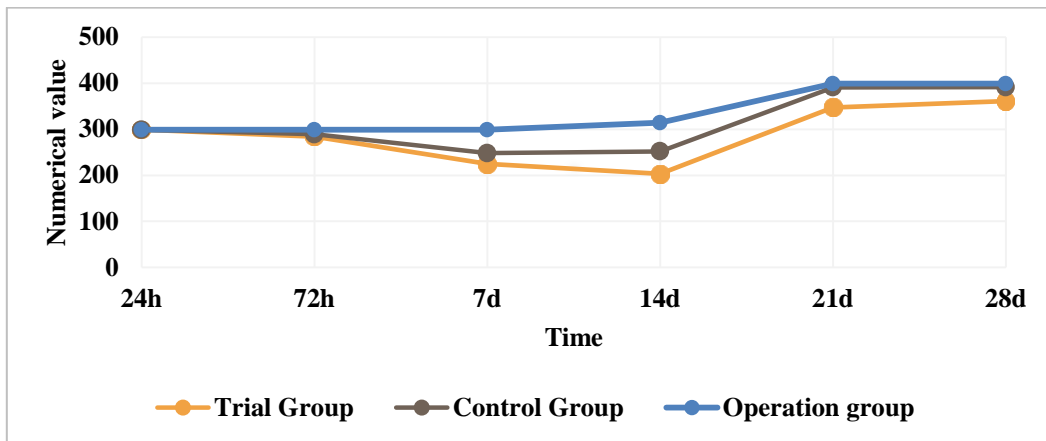


Figure 2. Weight analysis

According to the data analysis, as shown in Figure 2 and Table 2, compared with the control group, the rats in the experimental group significantly lost weight, especially 7 days after surgery. Compared with the operation group, the control group also experienced weight loss on the 7th and 14th days after surgery. The difference was statistically significant. On the 21st day, the body weight increased and basically recovered to the level of the operation group.

4.2. Apoptosis

(1) Grasp analysis

Table 3. Grip score analysis

Time	Trial Group	Control Group	Operation group
24h	0.3±0.45	0.3±0.45	2.9±0.0
72h	0.8±0.35	0.7±0.74	2.9±0.0
7Day	1.6	0.2	2.9
14Day	0	0.8	2.9
21Day	0	1.2	2.9
28Day	0	1.6	2.9

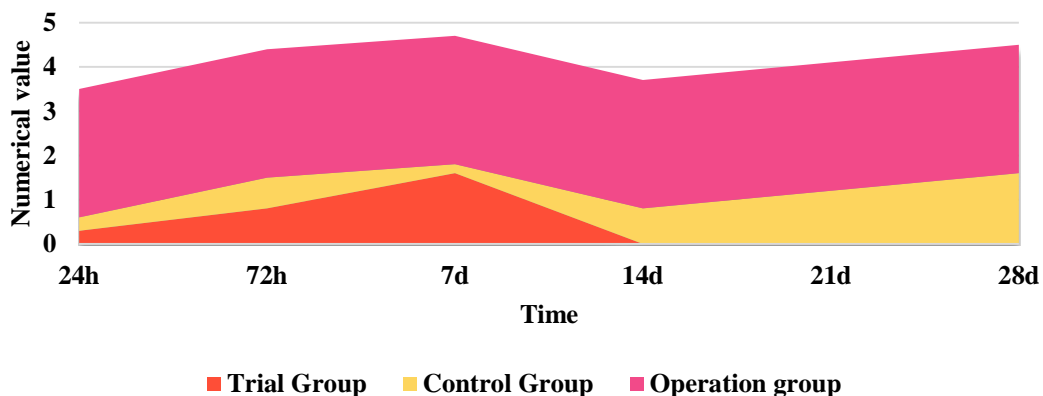


Figure 3. Grip score analysis

According to data analysis, as shown in Figure 3 and Table 3, the muscle strength of the exercise group began to recover at 72 hours after operation, and the muscle strength was significantly improved ; the control group returned to normal at 14 days after operation, but was still not fully restored at 28 days.

(2) Motion analysis

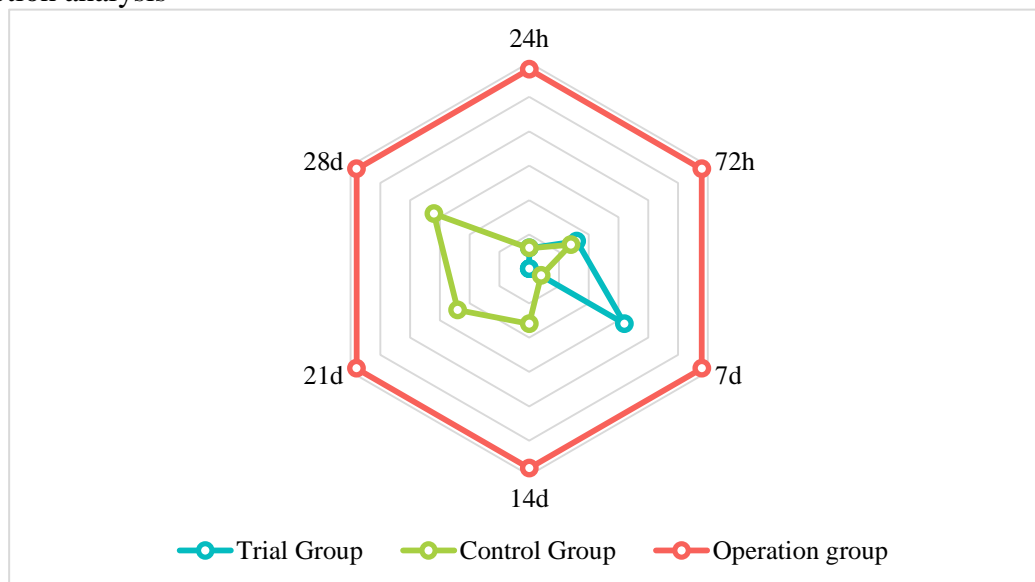


Figure 4. Kinematic analysis

According to data analysis, as shown in Figure 4, at 24h of cerebral hemorrhage. The expression of apoptosis in the two groups showed a downward trend on the 7th and 14th days after ICH, began to increase after 21 days, and showed a downward trend at 28 days.

5. Analysis and Discussion

5.1. Experimental Results Analysis

The weight of rats in the exercise group continued to decrease, and fell to the lowest level 14 days after surgery. The rats in the control group lost weight 7 to 14 days after ICH, while the rats in the surgery group did not gain weight within 7 days, and began to gain weight after 14 days. Rehabilitation treatment has a significant effect on the functional prognosis of cerebral infarction rats. Rehabilitation treatment mainly includes sports training, including passive sports and active sports. In the rehabilitation treatment of ischemic cerebrovascular disease, it is advocated to start the rehabilitation treatment of motor function as soon as possible after the onset. After 24 hours of intracerebral hemorrhage, the rats began to exercise, the function improved after 72 hours, and returned to normal after 14 and 21 days. However, the rats in the control group did not undergo exercise training, and their activities in the cage were limited. The recovery of nerve function was slower than that in the exercise group. This may be due to the limited activity of rats, which leads to the disuse of functions, which affects the recovery of their functions. The changes of brain tissue around the hematoma are similar to ischemic stroke, that is, low density ischemic focus and low perfusion around the necrotic area.

Brain injury after cerebral hemorrhage is secondary to ischemia, edema and inflammation caused

by hematoma, and eventually leads to nerve cell death. The pathological changes in the control group were mainly my performance, such as the disappearance of nerve cells in the early stage, leukocyte infiltration, the proliferation of glial cells and small blood vessels in the stage of economic recovery, and the gradual withdrawal of blood and damaged tissue in later stages. The defects were replaced by hyperplasia of glial cells and glial fibers, forming scars. Improve ischemic state and promote functional recovery. Brain water content change: The experimental group began to have cerebral edema at 6h after surgery, not only on the hemorrhagic side, but also on the opposite side, especially 24h and 72h after operation. On the 7th day, the edema was significantly alleviated, and on the 14th day, it returned to normal. Histopathological changes: Histopathological changes are consistent with the pathological changes of clinical cerebral hemorrhage. In the early stage of hemorrhage, the structure of nerve cells around the hematoma disappeared, and leukocytes infiltrated. During the recovery period, proliferation of glial cells, collagen fibers and small blood vessels appeared.

5.2. Discuss

There is no ideal animal model of cerebral hemorrhage. There are currently four animal models of cerebral hemorrhage commonly used in the world: collagenase inducible type, microcapsule inflatable type, spontaneous cerebral hemorrhage type and autologous blood injection type. The pathology is similar to hemorrhagic cerebral infarction, which is different from the incidence of clinical spontaneous cerebral hemorrhage. The mechanism is the formation of brain-occupying lesions. This mechanical injury is far from the actual pathophysiology and histological changes of cerebral hemorrhage, so it is rarely used except for studying the hematoma removal of cerebral hemorrhage. Spontaneous intracerebral hemorrhage model: Although the pathophysiology of the model is close to clinical intracerebral hemorrhage and vascular injury is similar to human hypertension arteriosclerosis, its application is very limited due to its hereditary vascular injury, uncertainty in model making and time required, Variability of lesion size and incompatible models. Because it is similar to human cerebral hemorrhage, it can simulate the space-occupying effect of hematoma formation and blood damage to extravascular brain tissue. Rehabilitation treatment has a significant effect on the functional prognosis of cerebral infarction rats. Exercise training can reduce the rapid development of primary hemorrhagic injury caused by the degeneration and degeneration of the contralateral neurons, thereby playing a role in protecting nerve cells.

6. Conclusion

Smart medical uses smart medical platforms and measurement terminals to achieve health management. Intelligent diagnosis and disease monitoring and monitoring can realize remote detection and analysis, diagnosis and treatment, so that users can understand the condition of the disease. Check and treat in time and in the early stages of the disease. Discovered from this research:

(1) Exercise can promote the recovery of limb function. Reducing nerve edema and increasing the number of nerves.

(2) Exercise can improve the nervous system function of rats with cerebral hemorrhage. The collagenase-induced intracranial hemorrhage model is very simple. High success rate and low mortality rate Stable and consistent hematopoiesis and obvious pathological and behavioral changes.

(3) Exercise training improves nerve function by inhibiting hematoma after cerebral hemorrhage

and apoptosis of nerve cells around hippocampus, promoting blood vessel regeneration. Collagenase-induced cerebral hemorrhage model is simple, with high success rate, low mortality, stable and consistent hematoma formation, pathological and behavioral changes.

Funding

This article is not supported by any foundation.

Data Availability

Data sharing is not applicable to this article as no new data were created or analysed in this study.

Conflict of Interest

The author states that this article has no conflict of interest.

References

- [1]Galvagno, S. M., Fox, E. E., Appana, S. N., Baraniuk, S., Bosarge, P. L., & Bulger, E. M., et al. (2017). *Outcomes after Concomitant Traumatic Brain Injury and Hemorrhagic Shock: a Secondary Analysis from the Pragmatic, Randomized Optimal Platelets and Plasma Ratios Trial*, *Journal of Trauma & Acute Care Surgery*, 83(4), pp.668-674. DOI:10.1097/TA.0000000000001584
- [2]Ma, L., Wen, X. H., Yang, H. B., Huang, J. H., & Chen, N.. (2016). *Influencing Factors for Brain Injury in Preterm Infants*, *Chinese Journal of Contemporary Pediatrics*, 18(6), pp.471-475. DOI:10.7499/j.issn.1008-8830.2016.06.001
- [3]Allison, R. Z., Nakagawa, K., Hayashi, M., Donovan, D. J., & Koenig, M. A.. (2016). *Derivation of a Predictive Score for Hemorrhagic Progression of Cerebral Contusions in Moderate and Severe Traumatic Brain Injury*, *Neurocritical Care*, 26(1), pp.80-86. DOI:10.1007/s12028-016-0303-5
- [4]Mobashsher, A. T., & Abbosh, A. M. (2016). *Compact 3-d Slot-Loaded Folded Dipole Antenna with Unidirectional Radiation and Low Impulse Distortion for Head Imaging Applications*, *IEEE Transactions on Antennas & Propagation*, 64(7), pp.3245-3250. DOI:10.1109/TAP.2016.2560909
- [5]Floccare, D. J., & Galvagno, S. M.. (2016). *Field Intubation for Hemorrhagic Shock: a Flawed Syllogism*, *Journal of Trauma & Acute Care Surgery*, 81(3),pp. 1.
- [6]Turliuc, D. M., Cucu, A. I., Dumitrescu, G. F., Sava, A., & Costea, C. F.. (2017). *Orbitocranial Penetrating Injury by a Metallic Foreign Body. Case Report and Anatomical Considerations*, *Nephron Clinical Practice*, 31(4), pp.437-443. DOI:10.1515/romneu-2017-0070
- [7]Leung, L. Y., Deng-Bryant, Y., Cardiff, K., Winter, M., & Shear, D.. (2016). *Neurochemical Changes Following Combined Hypoxemia and Hemorrhagic Shock in a Rat Model of Penetrating Ballistic-Like Brain Injury: a Microdialysis Study*, *Journal of Trauma & Acute Care Surgery*, 81(5),pp. 1. DOI:10.1097/TA.0000000000001206
- [8]Nikolian, V. C., Dennahy, I. S., Higgins, G. A., Williams, A. M., & Alam, H. B.. (2017). *Transcriptomic Changes Following Valproic Acid Treatment Promote Neurogenesis and*

- Minimize Secondary Brain Injury, Journal of Trauma and Acute Care Surgery*, 84(3), pp.1. DOI:10.1097/TA.0000000000001765
- [9]Dang B, Duan X, Wang Z, He W, & Chen G. (2017). *A Therapeutic Target of Cerebral Hemorrhagic Stroke: Matrix Metalloproteinase-9, current drug targets*, 18(999), pp.1-1. DOI:10.2174/1389450118666170427151657
- [10]Santiago Cepeda, Pedro A Gómez, Ana Mar ú CastañóLeon, Pablo M Munarriz, Igor Paredes, & Alfonso Lagares. (2016). *Contrecoup Traumatic Intracerebral Hemorrhage: a Geometric Study of the Impact Site and Association with Hemorrhagic Progression, Journal of Neurotrauma*, 33(11), pp.179-179. DOI:10.1089/neu.2015.4153
- [11]Gustav Folmer Gen á, Bentzer, P., Ostrowski, S. R., & Pär Ingemar Johansson. (2016). *Resuscitation with Pooled and Pathogen-Reduced Plasma Attenuates the Increase in Brain Water Content Following Traumatic Brain Injury and Hemorrhagic Shock in Rats, Journal of Neurotrauma*, 34(5), pp. 1054-1062.
- [12]Margolick, J., Dandurand, C., Duncan, K., Chen, W., & Hameed, S. M.. (2018). *A Systematic Review of the Risks and Benefits of Venous Thromboembolism Prophylaxis in Traumatic Brain Injury, The Canadian journal of neurological sciences. Le journal canadien des sciences neurologiques*, 45(4), pp.1-13. DOI:10.1017/cjn.2017.275
- [13]Sifat, A. E., Vaidya, B., & Abbruscato, T. J.. (2017). *Blood-Brain Barrier Protection as a Therapeutic Strategy for Acute Ischemic Stroke, The AAPS Journal*, 19(4), pp. 957-972.
- [14]Zhang, J., Shi, K., Li, Z., Li, M., & Shi, F. D.. (2017). *Organ- and Cell-Specific Immune Responses Are Associated with the Outcomes of Intracerebral Hemorrhage, Faseb Journal*, 32(1), pp.220-229. DOI:10.1096/fj.201700324R
- [15]Henninger, N., Compton, R. A., Khan, M. W., Carandang, R., & Muehlschlegel, S.. (2017). *Don't Lose Hope Early: Hemorrhagic Diffuse Axonal Injury on Head Ct Is Not Associated with Poor Outcome in Moderate-Severe Tbi Patients, Journal of Trauma & Acute Care Surgery*, 84(3), pp. 1. DOI:10.1097/TA.0000000000001733