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Degeneration and Regeneration of Nerve and Muscle end Plates of Athletes after Peripheral Nerve Injury

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Abstract: With the development of sports in China, there are more and more peripheral nerve injuries, and the disability rate is extremely high. The treatment of this injury is a clinical problem to be solved. The purpose of this article is to analyze the effects of peripheral nerve injury and muscle end plate degeneration and regeneration by observing and analysing the effects of peripheral nerve injury on athletes, and to understand the effect of clinical and experimental research on the treatment of peripheral nerve injury in athletes. The comparison of nerve and muscle end plate degeneration and regeneration before and after, to study the driving path of peripheral nerve injury in athletes. Using the method of this paper, through the analysis of experimental data, we understand the role of nerve fibers after peripheral injury occurs, and find that nerve fiber regeneration plays an important role, such as nerve regeneration in the nerve group (4.65 ± 0.68) mm, which is combined with experimental data through theory,analyzed the degeneration and regeneration of nerve and muscle motor end plates are common in peripheral nerve injury and have an important role. The research results show that effective treatment in clinical and experimental research can delay the degeneration of motor end plate after peripheral nerve injury promote the regeneration of sports end plate, which can be used to prevent or slow down its atrophy to improve the recovery of its function after the growth of regenerated nerves.

1. Research Background

The main causes of peripheral nerve injury are impact injury, crush injury, crush injury, traction injury, compression injury and cutting injury [1]. There are many factors that affect regeneration, and the mechanism is complex. Peripheral nerve injury directly damages social labor resources and brings economic and ethical burden to social development and family life [2-4]. The functional recovery of different peripheral nerve injuries is different.

Peripheral nerve anatomical variation is common and individual differences are large, which has a great influence on the determination of EMG results [5]. Double innervation means that a muscle can have two innervations [6-7]. The proportion of functional recovery after ulnar nerve repair is much lower than that of median nerve and radial nerve. The role of the motor end plate in the motor system The motor end plate is the synaptic effect structure between the motor nerve terminal and the muscle fiber membrane, including the presynaptic membrane, the synaptic gap and the post-synaptic membrane [8-9].

The maintenance of the normal structure and function of the motor end plate depends on the complete connection with the central neuron, as well as the various nutrients, transfer factors and glycoproteins provided by the axoplasmic transport [10]. Under normal physiological conditions, motor neurons establish connections with skeletal muscle fibers through the motor end plate, and control skeletal muscles to perform various physiological functions [11-13].

This article uses the method of experimental research to understand the role of nerve fibers after the occurrence of peripheral injuries, and the comparative exploration before and after the application; through theoretical analysis and experimental exploration to find out the impact of athletes' peripheral nerve injuries; through data recording, collation and calculation , Drawing, analysis and processing of the data, through the simulation of the statistical data sets of clinical and experimental research related to the degeneration and regeneration of the nerve and muscle end plates after the peripheral nerve injury of athletes, combined with the data, empirically analyze the peripheral nerve injuries of athletes Combined with effective data, summarize and analyze the role of nerve and muscle end plate degeneration and regeneration in peripheral nerve injury. The results show that with this method, the recognition rate reaches 33%.

2. Theoretical Basis

2.1. Peripheral Nerve Injury

Abnormal innervation refers to the innervation of muscles without innervation, which can be seen in the small muscles of the hands and calf muscles. Abnormal innervation in nerve injury is very common. In normal electrophysiological examination, the smaller the distance between the stimulus point and the receptor point, the greater the radiation value of the compound action potential; otherwise, the smaller the radiation value of the compound action potential [14]. Because the farther away, the greater the power consumption, the smaller the radiation value that can be detected. In the repair of peripheral nerve injuries, although fine microsurgical techniques are used in clinics, the proximal end of each nerve fiber cannot be accurately connected with the corresponding distal end, resulting in motor consciousness and sensory motor disorders, resulting in loss of regional functions caused by these disordered nerve fiber growth. The ultimate goal of nerve repair and regeneration is to restore the function of the target organ, that is, to reconstruct the effective connection between the innervation and the target organ, and to reinstate the incoming and outgoing of sensory signals. However, the effectiveness of afferent and efferent depends not only on the number of regenerated nerve fibers, the quality of the corresponding nerve fibers and the number of effective contact with the target organ, but also the accuracy of the docking between the corresponding nerve fibers and the target organ. In other words, in addition to measuring the number of regenerated nerve fibers, the thickness of myelin sheath and the diameter of axons, the proportion of effective nerve fibers in regenerated nerve fibers should also be considered [15]. From a functional point of view, this phenomenon can be understood as the growth of sensory nerve fibers towards the promotor nerve, and the growth of motor nerve fibers towards the prosthetic nerve. There is no doubt that the incidence of connection errors will affect the effect of peripheral nerve regeneration. Therefore, it is incomplete to measure the effect of peripheral nerve repair by

the number and accuracy of regenerated nerve fibers. In addition to the behavioral and electrophysiological evaluation of repair quality, it is also necessary to determine the proportion of different nerve fibers in regenerated nerve fibers to further improve the evaluation of nerve regeneration effects, namely the evaluation of chemotaxis regeneration.

Brachial plexus root injury is an axon injury close to neurons. Its severity often makes the damaged neurons lose their regenerative ability, and even leads to the death of neurons. In addition, the inferior trunk of the brachial plexus is far away from the target organ, and nerve regeneration is often not achieved. Irreversible fibrosis of the muscles, especially the internal muscles of the hands, seriously affects the effect of nerve repair. The front thighs of the upper, middle and lower trunk form the lateral bundle, the front thighs of the lower trunk independently form the medial bundle, and the rear thighs of the upper, middle and lower trunk form the rear bundle. Peripheral nerve regeneration is a complex and coordinated process. There are two main theories, namely chemotaxis regeneration theory and contact orientation theory. Chemotaxis theory has been generally recognized by most scholars. The basic point of the theory of chemotaxis regeneration is that during nerve regeneration, chemicals released from nerve endings or target organs induce new axon directional growth. After nerve injury, a series of cell and molecular changes will occur at the far end, including the removal of axons and myelin sheaths and the proliferation of Schwann cells. Mature Schwann cells undergo dedifferentiation. These dedifferentiated Schwann cells form a binner zone in the basement membrane tube and produce a variety of neurochemokines and trophic factors. These molecules can guide and promote nerve regeneration. Therefore, nerve endings play an important role in chemotaxis regeneration. Regeneration after nerve injury often involves the interaction of multiple cells and factors, rather than being determined by single or multiple genes and proteins.

Neuronal degeneration and death also affect axon regeneration and functional recovery. After axon injury, in addition to the above-mentioned degeneration reactions, there are also regenerative changes, which are manifested in the formation of morphological growth cones, and the corresponding neuronal cell body and proximal axon anabolism are enhanced. Therefore, the survival of neuronal cell bodies and the functional state of neurons after peripheral nerve injury are one of the key factors for functional recovery. The number and size of muscle fibers in muscle tissue decreased significantly, and the proliferation capacity of satellite cells also decreased. In addition, when there are nerves in the remaining muscle tissue, they can reform into nerves. The synapse between muscles and the function of muscles cannot be fully restored. The phenomenon that nerve endings are affected by chronic axotomy and chronic denervation to inhibit nerve regeneration has attracted enough attention. The target tissue needs to form synapses with the axon Restore the number of nerve fibers lost. It is an effective method to translate and express the related proteins of neuron growth cone and generate the molecules needed for nerve growth.

2.2. Nerve Fibers

Extensive limb fixation will produce disused osteoporosis, joint capsule contracture, muscle elasticity loss and fibrosis, which will have a very adverse effect on muscle morphology and metabolism. Early use of various rehabilitation and physical therapy methods can make muscle contraction passive, at the same time, effectively prevent the stiffness of the relevant joints and promote the overall improvement of the effect of nerve repair [16]. It is no less important than simple surgical treatment. In particular, patients should be educated to establish awareness of rehabilitation treatment. After the peripheral nerve injury, in a similar situation, the recovery of muscle function is related to the distance between nerve and muscle. In theory, deltoid and biceps can also be recovered very well. Because the chemotaxis of nerve regeneration has three levels:

tissue specificity, topological anatomy specificity and terminal organ specificity, the regenerated nerve fibers will selectively regenerate to the distal nerves.

During nerve regeneration, the amplitude of EMG can reflect the degree of nerve regeneration from a certain angle. Acellular allogeneic nerves not only have a net-like internal structure, but also benefit the regeneration of nerve fibers. It not only destroys Schwann cells and myelin sheath, but also completely removes cell debris and myelin sheath residues, and better preserves the tubular structure formed by the outer membrane, the bundled membrane and the inner nerve membrane, and the Schwann cell basement membrane And laminin, that nerve has a good natural nerve branch structure after chemical extraction, which is conducive to nerve regeneration. Nerve injury may trigger the feedback regulation system of the tubulin gene through the dynamic balance of depolymerization and the polymerization of microtubules, leading to enhanced expression of neuronal tubulin genes. Growth-related protein is a neuron-specific protein, synthesized by the neuron cell body, and quickly flows to the terminal. During development, axons widely present in the central nervous system grow outward.

Nerve growth factor receptor (NGFR) has high-affinity and low-affinity receptors. NGFRmRNA is expressed in the early embryos and development of these motor neurons, but disappears in normal motor neurons of adult animals. Some people use neurotrophic factors, acupuncture, etc. to reduce neuronal degeneration and death, the effect is better. The protective effect of neurotrophic factors on neurons may be achieved in part by inhibiting the process of neuronal apoptosis, especially in the delayed apoptosis of neurons after injury. Gangliosides are normal components of the lipid layer of cell membranes. The application of exogenous gangliosides in the body can promote the regeneration of peripheral nerves. Tissue engineering includes two basic elements: cell scaffolds and seed cells. The cell scaffold should have a specific three-dimensional structure, which can accept the growth of regenerated axons and guide the axon machinery, which is conducive to the orderly distribution of seed cell scaffolds, maintains the active state, and can degenerate the regenerated nerves in time.

In the repair of peripheral nerve injury, important nerve regeneration genes can be used in the local area of the damaged area, thereby promoting nerve regeneration. The differentiation of fibroblasts plays an important role in the formation of neuromuscular joints. Acupuncture can improve the local blood supply, enhance the transport of metabolites, reduce the functional decline caused by inflammatory diseases, promote axon regeneration and repair to a certain extent, reduce the concentration of axon plasma, and promote the release of acetylcholine from vesicles, thereby stimulating bone Muscle movement. By enhancing the protein translation of the nerve growth cone, it can promote the outward growth of axons. Natural biological materials have inherent biological activity and can promote nerve regeneration, but their degradation rate and mechanical properties are difficult to control; on the contrary, synthetic polymer materials can control their degradation rate and mechanical properties, but their ability to promote nerve regeneration is significantly lower former. Other methods to improve nerve regeneration include low-frequency electrical stimulation. The best treatment plan after peripheral nerve injury should include the following aspects: maintaining long-term neuronal activity, promoting the speed of axon regeneration, conducive to the functional regeneration of neuromuscular junctions, and preventing muscle atrophy. It is impossible to achieve all of the above goals by affecting only one signal target, so some scholars have begun to study multi-target drugs. The nervous system, like other systems in the body, has the potential to regulate internal physiological mechanisms to cope with these changes, including promoting the survival of neurons, accelerating the regeneration of axons, and keeping denervated muscles in a healthy state.

2.3. Sports End Board

After denervation of skeletal muscle, capillaries degenerate and disappear faster than muscle fibers disappear. The reconstruction of vascular bed will affect oxygen metabolism and destroy microcirculation. Insufficient blood supply and large accumulation of collagen may be important reasons for preventing long-term denervation. Whether there is apoptosis in denervated skeletal muscle has attracted people's attention. When more and more cells undergo apoptosis, these muscle cells with early apoptosis characteristics cannot maintain their basic functions. After denervation, with the prolongation of denervation time, the number of skeletal muscle fossa cells decreased rapidly. However, in the early stages of denervation, the number of muscle fossa cells will increase. The mechanism of the increase is unclear and may be related to compensation after denervation. The maintenance of trabecular meshwork cell proliferation depends on innervation or muscle activity under innervation. The regeneration of muscle fossa cells under denervation forms a structure similar to myotubes, which belongs to non-neural regeneration. Its formation will not develop into mature skeletal muscle fibers, but will lead to the depletion of muscle fossa cells.

There is a complex relationship between motor neurons and muscle cells, and they are not affected by nerve impulses. The degeneration of the end plate after axonal injury is related to the concentration of certain substances at the distal end of the axon. When the concentration of these active substances drops above a certain threshold, the end plate changes. In normal nerve tissue, these substances can be synthesized by the cell body and transported to the axon. The speed at which these active substances are transported to the end and the equilibrium state in which they are consumed determines the time for the end plate to denature. Compared with ordinary vitamins, methylcobalamin is more easily absorbed by nerve tissue. Schwann cells can regenerate axons and provide necessary accessory interfaces, as well as regulate the growth of regenerated axons, promote axonal transport and axon regeneration. After denervation of skeletal muscle, the muscle fiber was stopped for a long time and the content of muscle protein decreased. The maintenance of the normal structure and function of muscle cells depends on the function of certain neurotrophic factors. The biggest change after denervation of skeletal muscle is the change of muscle morphology, structure, physiology and biochemistry caused by the lack of neurotrophic factors. Morphologically, the first manifestation is loss of sarcoplasm and reduction in diameter. The end plates of different types of skeletal muscle fibers have different morphological structures and enzyme activities. Due to the different types of muscle fibers, the shape, size and depth of joint folds of the endplate are also significantly different. The morphological structure of the sports end plate is related to the type and genus of animals.

Neuromuscular diseases often lead to changes in muscle fiber types and changes in motor end plates, resulting in the weakening and loss of muscle strength. Once Schwann cells proliferate and mature, they will not release or release these nutrients, resulting in rapid degradation of the sports end plate. Therefore, the repair of nerve injury should be carried out when the end plate of the sports end is not obvious, so as to achieve better results. The main purpose of this experiment is to observe the degeneration of the injured skeletal muscle after the right spinal cord is transected. If the myofasma is blocked, the ends are retracted more, the bleeding between the end spaces is obvious, the cells are infiltrated, and a large area of scars is formed in and between the muscle bundles to form fusiform neurofibroma. The changes of muscle and end plate after peripheral nerve injury are closely related to nerve injury and treatment. The factors that affect nerve regeneration will directly affect the changes of muscle and motor end plates: nerve injury plane: the higher the injury plane, the worse the recovery of sensory and motor functions; the nature and scope of nerve injury: the repair effect of sharp cutting injury is better than crushing Injury or torsional injury; operation and maintenance timing: early repair is better than late repair; surgical technique:

microsurgery is better than open-eye surgery; patient age: the younger the age, the better the recovery of nerve function. The degeneration of the motor endplate after denervation of skeletal muscle is related to the timing of nerve repair. Methods to promote nerve regeneration: drug treatment can promote axon anabolism and prevent axonal degeneration.

3. Subjects and Methods

3.1. Research Object

Case selection criteria: patients in a hospital orthopedic department meet the diagnosis of peripheral nerve injury and have complete hospitalization records and surgical records. The follow-up patients were their own or close relatives, with normal mental state and clear expression. A total of 171 patients with peripheral nerve injury diagnosed in our hospital from October 2018 to October 2019 were collected, of which 41 patients met the inclusion criteria. There were 38 males and 3 females. Follow-up time: 10 months to 9 years, 10 months. The patients were divided into three groups: non-operative group, neurolysis group and nerve repair group. Finally, evaluate the natural recovery of non-surgical patients and the efficacy of surgical patients.

Thirty healthy male rats, 8 weeks old, weighed 250g ± 20g. The experimental animals were anesthetized with 10% chloral hydrate injection, intraperitoneal injection (0.3ml / 100g), supine position, and the right inguinal incision was taken to expose the femoral nerve under sterile condition. According to different operations, they are randomly divided into three groups. Control group (10): The trunk was separated by 4mm upward, and the incision was closed layer by layer. Electrophysiological test group (10 rats): Based on the bifurcation of the femoral nerve, the trunk was cut 4mm, the ends were anastomosed, and the incision was closed layer by layer. The experimental group (10 rats) based on the bifurcation of the femoral nerve, cut off the trunk 4mm, and closed the incision layer by layer after anastomosis. After random numbering, the rats were divided into experimental group and control group, with 30 rats in each group.

3.2. Experimental Plan

(1) General behavior observation: observe the wound healing of experimental animals and postoperative complications. At 8 weeks after the operation, grab the tail of the experimental animal to make the forelimbs grasp the rod, and at the same time stretch the rod with two hind legs to observe the functional recovery of the hindlimb on the surgical side.

(2) Electrophysiological test: 8 weeks after the operation, the animal's electrophysiological test group was injected with 10% chloral hydrate injection and intraperitoneal injection anesthesia, and then the inguinal incision, the main and muscular branch of the femoral nerve were operational and normal Contact, and motor evoked potential measurement of the quadriceps.

(3) After the end of the electrophysiological test, the experimental animals were sacrificed and the nerve near the anastomosis of femoral nerve was taken. Immunohistochemical staining of longitudinal frozen sections. Observe the continuity of the anastomotic nerve.

(4) Retrograde tracer: prepare retrograde tracer into 5% absolute ethanol solution and store in 4 ℃ refrigerator.

(5) Statistical processing: The number of motor neurons is counted using image analysis software and expressed in the form of mean standard deviation. SPSS 17.0 software was used for paired t test; $P < 0.05$ was the standard of significant difference.

(6) Observation of exercise end plate: 12 weeks after surgery, the upper third of the gastrocnemius muscle was taken 3 mm thick, and small muscle tissue was fixed with gold chloride fixative. After translucency, blot dry with filter paper and put in 1% gold chloride solution for 15

minutes. After the tissue turns golden yellow, put it in 20% formic acid solution overnight. The next day, it was stored in a mixed solution of glycerin and alcohol. Take small muscle tissue and stir glycerin tablets. Observe the nerve endplate under light microscope and analyze the image.

4. Experimental Results

4.1. Peripheral Nerve Injury

(1) Comparison of surgical treatment

Table 1. Comparison of surgical treatment

Result	Transplant	Neurolysis	Total
Complete injury	33	36	69
Incomplete injury	11	27	38
Total	44	63	107

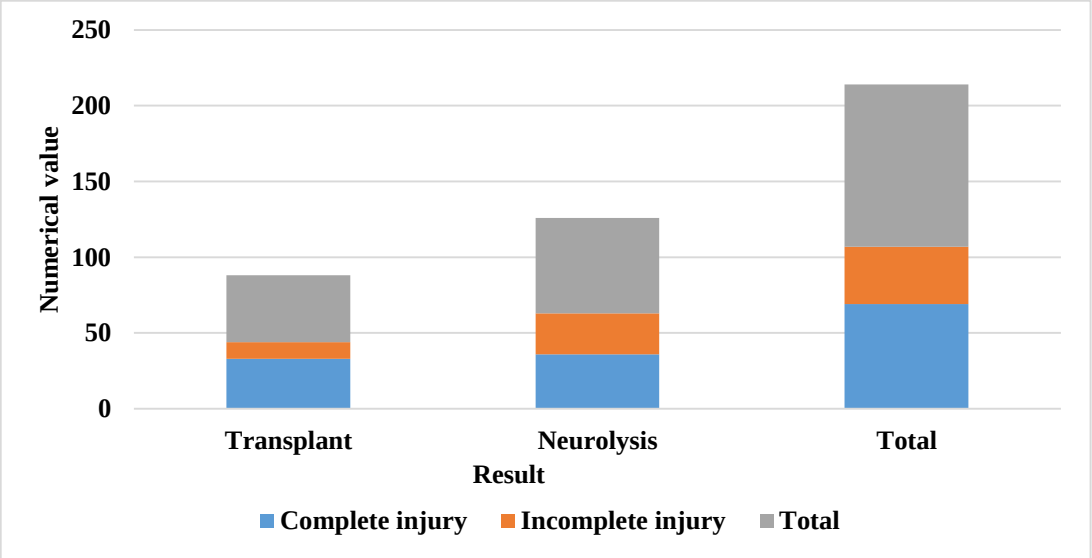

Figure 1. Comparison of surgical treatment

According to the statistical analysis of the data, as shown in Figure 1 and Table 1, compared with the results of EMG and surgical selection, 33 cases of complete injury during transplantation, 11 cases of incomplete injury, a total of 44 cases. There were 36 cases of total injuries and 27 cases of total injuries during neurolysis. The electrodes in the experimental group stimulated the proximal anastomosed nerve and the contralateral nerve respectively.

(2) Electrophysiological test results

Table 2. Electrophysiological test results

Group	Incubation period	Amplitude
Experience group	1.16±0.44	1.01±0.7
Control group	0.70±0.29	5.33±1.29

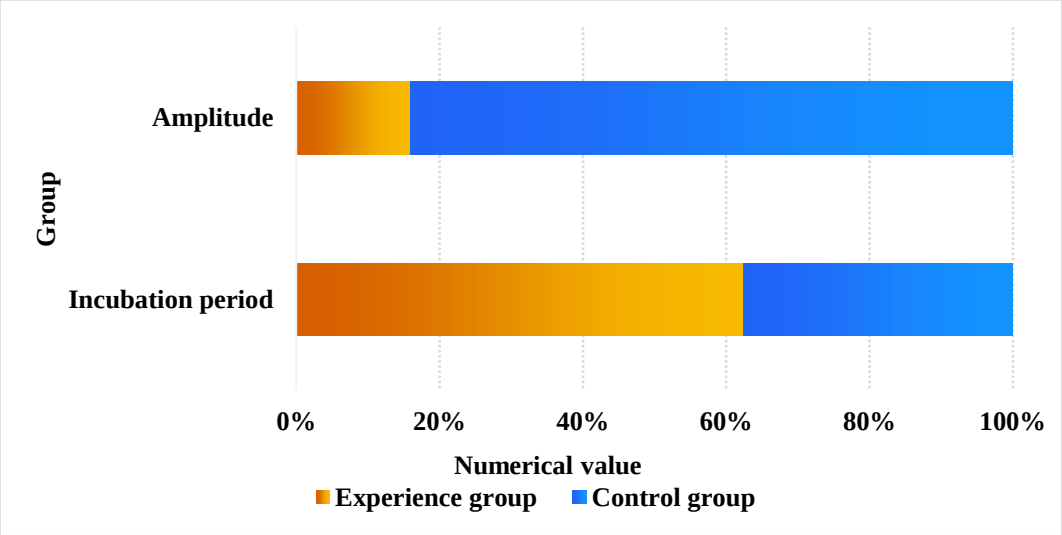

Figure 2. Electrophysiological test results

According to the statistical analysis of data, as shown in Figure 2 and Table 2, the incubation period of the experimental group was (1.16 ± 0.44) ms, and the incubation period of the control group was (0.70 ± 0.29) ms. Compared with the contralateral control group, the incubation period of the experimental group was prolonged and amplitude decline.

4.2. Sports End Plate

(1) Regeneration analysis

Table 3. Regeneration analysis

Group	Muscular branches	Nerve
Neurogroup	4.11±0.85	3.65±0.68
Duct group	4.77±0.41	3.81±0.57
Control group	6.56±0.72	6.38±0.78

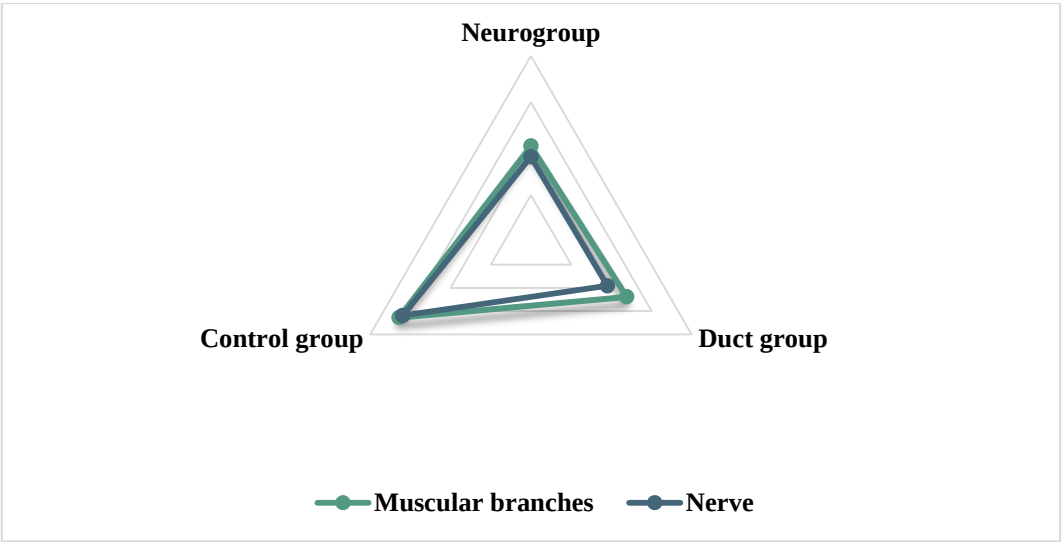

Figure 3. Regeneration analysis

According to the statistical analysis of data, as shown in Figure 3 and Table 3, the nerve groups were (4.11 ±0.85) mm, (3.65 ±0.68) mm, (4.77 ±0.41) mm, (3.81 ±0.57) mm, (6.56 ±0.72) mm and (6.38 ± 0.78) mm. The nerve anastomosis has good continuity, the adhesion around the anastomosis is slight, and the nerve conduit is atrophy. The staining showed that the regeneration of the main nerve fibers of the femoral nerve entered the proximal anastomosis of the graft. In the control group, the muscular branches and saphenous nerve axons have grown to the distal anastomosis of the graft, while the muscular branches and saphenous nerve axons of the nerve group and catheter group have not grown to the distal anastomosis. Compared with the control group, the nerve group and the catheter group were statistically significant, but there was no statistical difference between the nerve group and the catheter group.

(2)Sports end plate analysis

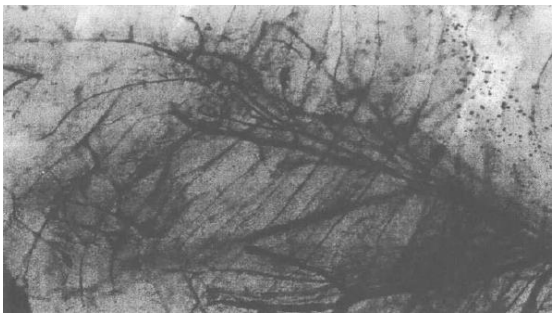

Figure 4. Exercise end plate of experimental group

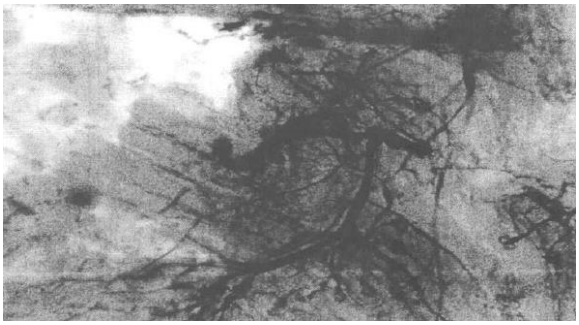

Figure 5. Motor endplate of control group

According to the statistical analysis of the data, as shown in Figures 4 and 5, at 12 weeks after the operation, the shape of the end plate of the experiment group was almost normal color, the number was complete and elliptical, and many thin branch muscles were not formed at the end of the axon Fiber, there are more end plate cores, the color and quantity of sports end plates are not significantly different from the normal group and the shape is mainly "claw-shaped image" or "chrysanthemum". The control group had fewer motor end plates, darker colors, less complete volume, less axon terminal formation, fewer end plate cell nuclei, and denser sarcoplasmic staining. The end plates were dumbbell-shaped, claw-shaped or daisy-shaped.

5. Analysis and Discussion

5.1. Experimental Results Analysis

The cause and degree of injury are different, and the response of the peripheral nervous system to the injury is completely different. Peripheral nerve injury can be divided into three types: nerve loss, axon rupture, and nerve trunk complete rupture. Peripheral nerve injury is divided into 5 levels:

Grade I nerve injury, that is, nerve loss. Axon continuity exists, but conduction is interrupted. Secondary nerve injury. That is, the axon is interrupted. The axon and myelin sheath were damaged, but the intima was not damaged. The lower part of the injury site was degenerated and the function was completely restored. Tertiary nerve injury refers to nerve fiber damage in the nerve bundle, including axon, myelin sheath and endometrium, but the nerve bundle membrane is intact. Part of the regenerated fibers cannot penetrate the scar and contact the distal organs, and the regeneration is incomplete. The fourth degree of nerve injury is nerve Cambodia injury, including axon injury, intramembrane injury and bundle membrane injury. Without surgical treatment, motor and sensory functions cannot be restored. V degree nerve injury. In other words, thousands of nerves collapsed. All nerves and adventitia in Cambodia were broken, the nerve trunk was completely destroyed, and continuity was lost. This kind of damage is the most serious one, usually with open damage. I, Ⅱ, and III degree nerve injury will naturally recover, and its effect is better than surgical suture or transplantation; grade IV and grade V injury must be repaired by surgery. In the treatment of nerve injury, morphological identification should be combined with electrophysiological detection. If the nerve trunk loses its normal shape, the pathological part is difficult and there is no electrophysiological conduction, so it is considered serious, so it needs to cut off the nerve segment and perform nerve suture or nerve transplantation. However, some scholars believe that if the neuromorphology is complete and continuous after internal release, no matter whether there is electrophysiological conduction, it should be retained and only internal and external release should be performed. There are neurotrophic factors in skeletal muscle, including nerve growth factor, brain-derived neurotrophic factor, neurotrophic factor and some unnamed neurotrophins. Proximal axon germination and elongation: Under appropriate microenvironment and necessary conditions, the proximal regenerated axon grows to the appropriate distal Schwann cell basement membrane tube in the nerve injury area and continues to grow toward the nerve endings. The best microenvironment for axon growth is the microenvironment of nerve endings. The regenerated axons and the corresponding terminal target organs rebuild synaptic connections. Recovery of target organs for nerve transplantation: to ensure that the function of target organs plays an effective role after nerve transplantation. If the target organ denervated for a long time undergoes irreversible changes, such as fibrosis, its function cannot be restored. The mortality rate of peripheral neurons in young animals is high, which may be due to the strong dependence of neurons of young animals on neurotrophic factors. At the same time, during the development and growth of young animals, neurons will inevitably die in a programmed manner. It is possible that the closer the injury site is, the fewer Schwann cells in the remaining nerve fibers and the less the synthesis of neurotrophic factors. At the same time, the fewer remaining lateral branches in the proximal section, the less nutrients are obtained from the target organ. The reduction of these organelles keeps neurons in a low-energy state and cannot synthesize enough nutrients for axon regeneration. Over time, the reversed axonal plasma flow is interrupted for a long period of time, leading to the loss of the nutritional function of the target organ neurons, as well as the interruption of the material and the transmission of information between the neurons and the body and its axon terminals, resulting in retrograde cytoplasmic neurons and neuron death are gradually increasing. Spinal motor neurons during nerve regeneration. Tubulin gene expression increased significantly.

5.2. Discuss

Neurotrophic refers to the ability of regenerated axons to grow and mature, and neurochemotactic refers to the ability to guide the direction of regenerated axons. Myelinated nerve fiber axons are surrounded by myelin sheath except for the beginning and end. In the developmental stage, Schwann cells develop from a common precursor cell into two cells with different antigens,

morphology and function: nerve sheath cells and non-neuricular sheath cells. The differentiation of these two cells is reversible. Skeletal muscle is the target organ of the peripheral nervous system. Its occurrence, function and structure are controlled and regulated by motor nerves. After the regenerated nerve endings enter the denervated muscles for a long time, the muscles atrophy and fibrosis, and the regenerative ability decreases. The number of regenerated muscle fibers decreases or cannot be regenerated, resulting in loss of muscle function. There are many reports on skeletal muscle atrophy, including protein metabolism, muscle enzyme activity, changes in muscle cell morphology, number and type of muscle fibers, electrophysiology, etc. The maintenance of the functional integrity of muscles requires the influence of many factors, and any one of them will affect the functional objective of economic recovery. Denervated muscles, and nerve regeneration that does not respond to severe atrophy are a common cause of poor clinical efficacy. The state of skeletal muscle as the target organ has a significant influence on the therapeutic effect of peripheral nerve repair.

After denervation, the terminal Schwann cells cover from the edge of the endplate to the neuromuscular junction and grow towards the muscle. Over time, these processes increase, become thicker, longer, and intertwined to form a network. Functional exercise is often advocated clinically, but the early fixation effect is not good. Loss of innervated muscles after peripheral nerve injury will lead to nutrient and wasted atrophy, which will exacerbate denervation expansion over time, eventually leading to irreversible muscle atrophy.

6. Conclusion

(1) The molecular level changes that occur in denervated muscles can maintain the ability to form new neuromuscular connections. Once nerves can be regenerated, these methods can also restore muscle innervation and normal function.

(2) Increase the content of acetylcholinesterase, delay the degradation of the end plate, promote the regeneration of the end plate and accelerate the recovery of kinetic energy. Effective treatment can delay the degeneration of motor endplate after peripheral nerve injury and promote the regeneration of motor endplate after peripheral nerve injury.

(3) After peripheral nerve injury, neurons rapidly change their transcriptome levels to form a regenerative phenotype. When the proximal peripheral nerve is damaged, the regenerated nerve takes a long time to reach the distal target tissue because the nerve regeneration distance is too long.

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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.

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