Scholar Publishing Group
International Journal of Health and Pharmaceutical Medicine
https://doi.org/10.38007/IJHPM.2022.030105
ISSN 2789-8393 Vol. 3, Issue 1: 48-59

Histopathological Morphology and Ultrastructure of the Heart Apparatus by Diving

Chadia Altrjmana and Rashare Almajede

Near East University, Turkey

Keywords: Diving Operation, Heart Organ, Histopathological Morphology, Ultrastructure Research

Abstract: To study the effect of diving on the histopathological morphology and ultrastructure of heart apparatus. 24 rats were randomly divided into control group, 6ata air group and 6ata normoxia high nitrogen group, 8 rats in each group. The control group took food at will under natural light. The experiment was carried out after one week of observation and quarantine. The other two groups were exposed to 4 ATA and 6 ATA with air and pure nitrogen respectively for 30 minutes. The effects of different high-pressure exposure schemes and time courses on the morphology and ultrastructure of rat heart were observed. The effects of different exposure schemes on myocardial histopathology and expression of myocardial ET-1 were observed. Meanwhile, the content of plasma ET-1 was measured, compared and statistically analyzed. In the heart rate comparison, the heart rate of air group decreased by 7% ($P < 0.01$) when it was pressurized to 4 ATA, 9% ($P < 0.01$) when it was pressurized to 6 ATA, and there was no significant change in the heart rate of normoxia and high nitrogen group when it was pressurized to 4 ATA and 6 ATA, and within 30 minutes after 6 ATA exposure. In the comparison of blood pressure, systolic blood pressure increased by 5% ($P < 0.05$), diastolic blood pressure increased by 11% ($P < 0.05$), and mean arterial pressure increased by 9% ($P < 0.05$). There was no significant change in pulse pressure. When the normoxia group was pressurized to 6ata, systolic blood pressure increased by 8% ($P < 0.01$) and pulse pressure increased by 19% ($P < 0.01$). No obvious pathological changes were found in the myocardium. On the expression of ET-1 in myocardium, the expression of ET-1 in vascular endothelium was significantly increased in normoxia and hyperhelium group and air group.

1. Research Background

From the development of diving medicine, participation has promoted the progress of diving technology. With the deepening of people's understanding of the underwater high pressure environment, the research of diving medical ergonomics is also in-depth and developing. The U.S.

Navy needs to test the ergonomics of its navy's experimental diving team. The French Navy also has institutions specializing in the ergonomic testing of diving equipment [1]. This paper expounds the importance of diving medical ergonomics in diving technology, especially in the training of diving operation ability. Physical strength is mainly manifested as physical load capacity, which is often evaluated by lung exercise test under normal pressure.

Although the maximum oxygen uptake of each person is different, the law of oxygen uptake increase is consistent with the increase of exercise load, and there is a certain limit [2]. The underwater environment has two main loads on the diver's body: one is the physiological and functional changes caused by the pressure itself, and the physiological effects caused by the changes of gas composition, such as the possible influence of hyperoxia inhalation on the way and mode of oxygen delivery [3].

During the diving operation, the pressure shall be equal to the ambient water pressure at the diving operation depth, so that the whole body is evenly exposed to the high-pressure environment inside and outside. In the process of operation, the inert gas (such as nitrogen in compressed air) in breathing gas gradually dissolves in various tissues of human body; the greater the depth (pressure), the longer the underwater working time, the more dissolved gas. Diving is a kind of work which is carried out by divers in special high-pressure underwater environment, with complex conditions and high technical requirements. The frequency of acute decompression sickness was lower in fishermen who strictly regulated decompression after diving.

2. Literature Review

Normal cardiac electrophysiological activity is caused by pacing of sinoatrial node, which produces excitation. High pressure exposure has a wide range of effects on the electrophysiological function of mammalian heart, which mainly affects the excitability, self-discipline and conductivity of cardiac myocytes. In the early stage, some foreign scholars used microsphere method to study the effects of high pressure exposure on the blood flow and heart function of various organs in rats [4-5]. Regnard first studied the heart rate response of frogs exposed to high pressure between 10MPa and 30MPa in 1884 [6]. Matei and other scholars used intracellular technology to record the electrical activity of sinoatrial node cell membrane, to explore the reasons for the slow down of autonomic heart rate and the interaction between pressure, membrane lipid and inert gas [7]. Hogan and Besch successfully imitated the effect of high pressure on the enhancement of myocardial contractility by using the famous sodium potassium ATPase pump blocker, calycoside C [8]. In 1985, parkon L.G. studied the relationship between high pressure and the decrease of membrane fluidity and the inhibition of sodium potassium pump function through fluorescence probe technology [9]. In 2002, michikokato et al. Used ultraviolet spectrophotometer and fluorescence spectrophotometer to explore the changes caused by the pressure of 100MPa or lower and higher pressure through the combination of enzyme-linked immunosorbent technology in the cell compartment under high pressure [10]. The impact of high pressure exposure on the body is on all systems of the body. As a part of the body, the heart cannot leave the overall impact of high pressure on the body. Tao hy CHCN i-ij was exposed to high pressure to study the change of ANP content; Rico DM was exposed to 2-3atal00% pure oxygen to explore the change curve of ANP, n-proanpl-98, proanp31-67 in blood [11]. Ping, Z and other scholars observed 96 divers' diastolic pressure, systolic pressure, stroke volume and output per minute by X-ray to study the specific changes of diastolic period and systolic period; in animal experiments and human experiments, they studied the high pressure under normal oxygen partial pressure, Changes and effects of cardiac output (C0), mean arterial pressure (MBP) and heart rate (HR); lafay observed changes of cardiac output, left ventricular ejection fraction and atrioventricular diameter through deep saturation diving

experiment [12]. In recent years, we have studied whether HBO pre adaptation can effectively prevent the ischemic injury of various organs in different animal models [13]. Under the adverse environmental conditions of diving site, the sick divers are often difficult to get timely and effective pressure treatment, and it is easy to miss the best treatment time, leaving paralysis, osteonecrosis and other sequelae, even death. The main source of myocardial energy is the aerobic oxidation of substances, so the myocardial oxygen consumption can be used as a good indicator of cardiac energy consumption [14]. The myocardial oxygen consumption mainly depends on the myocardial tension and the duration of tension. Therefore, HR and LVSP are commonly used to estimate the myocardial oxygen consumption. Gallaly, S.R. and other scholars use animal experiments and human experiments to study the increase of oxygen partial pressure when exposed to high pressure, The changes of myocardial oxygen consumption and the effects of hyperbaric exposure on cardiac pump function [15].

3. Experimental Research

3.1. Subjects

Male experimental rats, 10 weeks old, weighing 260-290g, were randomly divided into 24 groups: control group, 6ata air group, 6ata normoxia and high nitrogen group. The control group ate at will under natural light. The experiment was carried out after a week of observation and isolation. Before the experiment, intubation was performed in the femoral artery and left ventricle respectively. Air and pure nitrogen were pressurized to 6ata, respectively. Cardiac hemodynamic indexes of 4ata1min, 6ata1min, 15min and 30min were recorded. After the rats were killed, the heart was cut from the aorta 1:3 and divided into two parts along the long axis of the left ventricle, which were fixed with 10% paraformaldehyde solution. Plasma and serum samples were taken at the same time. He staining was used to observe the changes of myocardial tissue, immunohistochemical staining was used to observe the expression of ET-1 in left ventricular myocardium, and radioimmunoassay was used to measure the content of ET-1 in plasma.

3.2. Experimental Method

(1) The animals in the high-pressure exposure group were treated according to the routine treatment: the maximum depth of conventional air diving stipulated by various countries was 50-60m, and the suitable time for diving at different depths was also limited. The animals in the high-pressure exposure group were pressurized to the corresponding pressure according to 1ata / min, the high-pressure retention time was 60min, and the corresponding medium was used for ventilation for 10min. During the exposure period, the cabin temperature is 22-24 ℃, and the carbon dioxide concentration is less than 0.05%. The animals were exposed to the same regimen in the hut for three days. The normal control group was also placed in a pressure chamber, ventilated under normal pressure, and simulated other experimental processes and environmental conditions except pressure and oxygen concentration.

(2) Measurement of cardiac hemodynamic indexes in rats: the femoral artery catheter was made with two kinds of polyethylene catheter, pel0tube (0.30mrn, 0.50ram) and pes0tube (0.58ram, 0.96ram). When the butt joint is heated, a bulge is formed at the butt joint. Check whether the butt joint is firm and whether the conduit is unblocked for use. When used, heparin saline is injected into the catheter. Rats were anesthetized by intraperitoneal injection of pentobarbital sodium 45 mg / kg through left ventricular intubation. After anesthesia, the rats were lying on the operating table and the skin of the right neck was cut. After local lidocaine anesthesia, the right common carotid artery separates the longitudinal incision of the muscle on the inner side of the mastoid from the skin

intubation of the neck, and the ventral side of the prepared pes0 polyethylene pipe connects the pressure sensor to the inner and outer wire cabins. Fill the well with normal saline to discharge bubbles (small bubbles should also be discharged, otherwise the waveform is not accurate). At the end of the isolated right common carotid artery, a V-shaped orifice was opened, the prepared catheter was inserted slowly, a loose knot was made, the artery clamp was released, the catheter was sent to the left ventricle slowly, and the change of noise waveform was noticed. When the blood pressure waveform changes to a waveform of obvious diastolic and flat peak near the lower edge of 0 mm Hg, this indicates that the catheter enters the left ventricular cavity through the active leaves, and then the catheter is used to observe the change of the left ventricular steep pressure. The right thigh skin was cut along the direction of the femoral artery, and a section of femoral artery about 1.5cm long was separated. Before intubation, heparin saline was injected into artery intubation and pressure sensor to clear bubbles. The distal femoral artery was ligated, and the proximal artery was clamped, so as to reduce the artery wall in the distal ligation. The eye scissors were cut at a 45 degree angle, ready to insert the artery catheter into the proximal end about 0.5cm, release the artery clamp, insert the catheter into 1cm, and with the proximal ligation artery and catheter insertion thread, the blood pressure waveform of the femoral artery could be seen, and then the catheter was ligated and fixed with the distal ligation line, Observe the change of arterial blood pressure. At the same time, connect precordial area, right upper and lower limbs subcutaneously to the lead. Heart rate (HR), systolic pressure, end diastolic pressure and mean arterial pressure were recorded 15 minutes later. Close the cabin door, start to pressurize at a constant speed (1ata / min), stay at 4ata for 1min, record the indicators, and reach 6ata for 1min and 30min.

(3) Myocardial tissue optical examination: preparation of paraffin block: 10% paraformaldehyde (24-48 hours), water (overnight), 80% ethanol (4 hours), 95% (2 hours), 95% (2 hours), 100% (20 minutes), 1 wax (1.5 hours), II wax (1.5 hours), block. He staining: xylene I (10 minutes), xylene II (10 minutes), 100% alcohol (5 minutes), 95% alcohol I (5 minutes), 95% alcohol II (5 minutes), 80% alcohol (5 minutes), water hematoxylin (2.3 minutes), water HCl alcohol (2-3 times of brushing), 70% alcohol (5 minutes), water Yihong 80% alcohol (5 minutes), 95 alcohol I (10 minutes), 100% alcohol I (5 minutes), 100% alcohol II (5 minutes), xylene I (10 minutes), Xylene II (10 minutes).

(4) Immunohistochemical detection of myocardial tissue: the expression of ET-1 in three groups of different high-pressure exposure group and control group was detected by immunohistochemical method. Paraffin sections were dewaxed to water, washed with PBS for 3 × 3min, repaired with ph6.00.01mcb (microwave for 3 times, 20min), cooled naturally at room temperature, washed with PBS for 3 × 3min, inhibited endogenous peroxidase and pbs3 × 3min by 0.3% H202, incubated with 20% normal sheep serum at room temperature for 30min without washing, diluted with anti-1:100 Rabbit anti rat ET-1 antibody for 2h at 37 ℃; A rat anti-et-1 antibody was washed 3 × 3 with PBS minmin:Envision Washing with PBS for 3 × 3min; 10, pat for 8min; hematoxylin lining dyeing, hot water bluing; drying, resin sealing; microscopic observation, nuclear purple blue, positive brown yellow.

(5) Determination of ET-1 (radioimmunoassay): according to the principle of liquid phase competition inhibition, the sample was determined by non-equilibrium method. Firstly, the sample or standard to be tested is added with the limited antiserum for a period of time to react, and then the labeled antigen is added for competitive binding reaction. After the reaction, the antigen antibody complex was separated by adding the immune separation agent, the radioactivity of the complex was measured, the binding rate of each standard tube was calculated, the standard curve was made, and the sample concentration was calculated.

(6) Image analysis and statistical processing: immunohistochemistry semi quantitative analysis: use mv-cp410 camera, kontroncell image analysis system, swing display mirror, image acquisition card, digital camera or CD camera to collect digital images; optical display 100 times under the

microscope. The statistical data were expressed as mean. There was significant difference in paired t test before and after pressurization ($P < 0.05$). Use SPSS13.0 software for data processing.

4. Experimental Results

4.1. Basic Index Results of Heart

(1)Heart rate comparison

Table 1. Heart rate comparison

Classification	1ATA / 15min	4ATA / 1min	6ATA / 1min	6ATA / 15min	6ATA / 30min
Air group	347.4±24.6	326.1±22.9	319±22.5	318.2±21.2	316.2±26.4
Normal oxygen and high nitrogen group	362±32.6	357.4±20.1	350.5±32.6	363.8±33.3	362.7±24.6

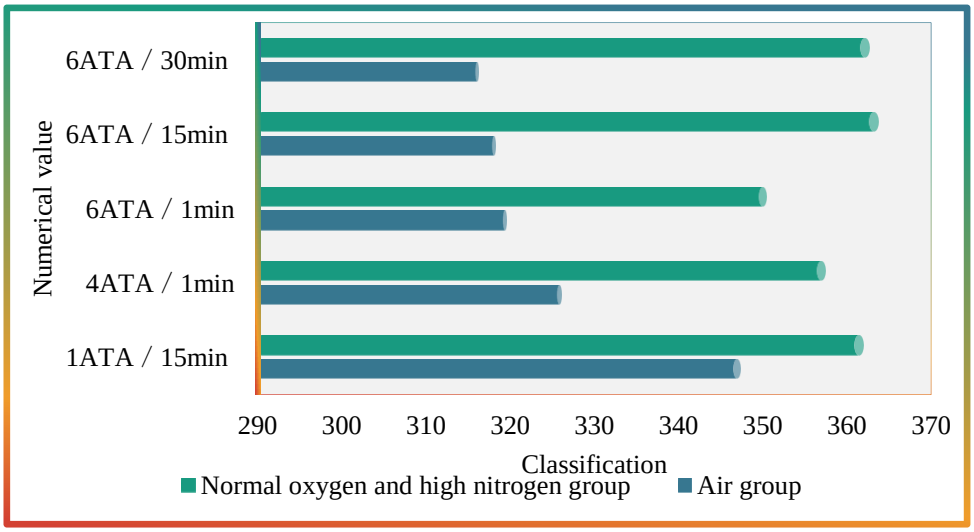

Figure 1. Heart rate comparison

According to the statistical analysis of the data, as shown in Figure 1 and table 1, the heart rate of the air group decreased by 7% ($p<0.01$) when pressurized to 4ATA, and 9% ($p<0.01$) when pressurized to 6ATA,. There was also significant difference between the two groups ($P < 0.01$). There was no significant change in heart rate in the normoxia and hypernitrogen groups when they were pressurized to 4 ATA and 6 ATA and exposed to 6 ATA for 30 minutes.

(2)Blood pressure comparison

Table 2. Blood pressure comparison

Classification	Blood pressure index	1ATA / 15min	4ATA / 1min	6ATA / 1min	6ATA / 15min	6ATA / 30min
Air group	SBP	107.1±5.7	110.7±2.4	114.8±6	110.2±6.6	112±5.3
	DBP	70.4±2.6	74.1±3.5	79.8±5.4	84.2±6.7	81.7±4.7
	MBP	82.6±2.6	86.3±2.2	91.5±4.8	92.9±5.9	91.8±4.5
Normal oxygen and high nitrogen group	SBP	112.1±5.7	123±5.6	123.8±7.4	129.7±5.6	120.8±7.9
	DBP	66.8±3.6	66±2.6	67.8±1.9	69.5±4.7	65.4±3.3
	MBP	81.9±1.7	85±2.4	86.5±1.8	88.9±3.9	83.9±1.6

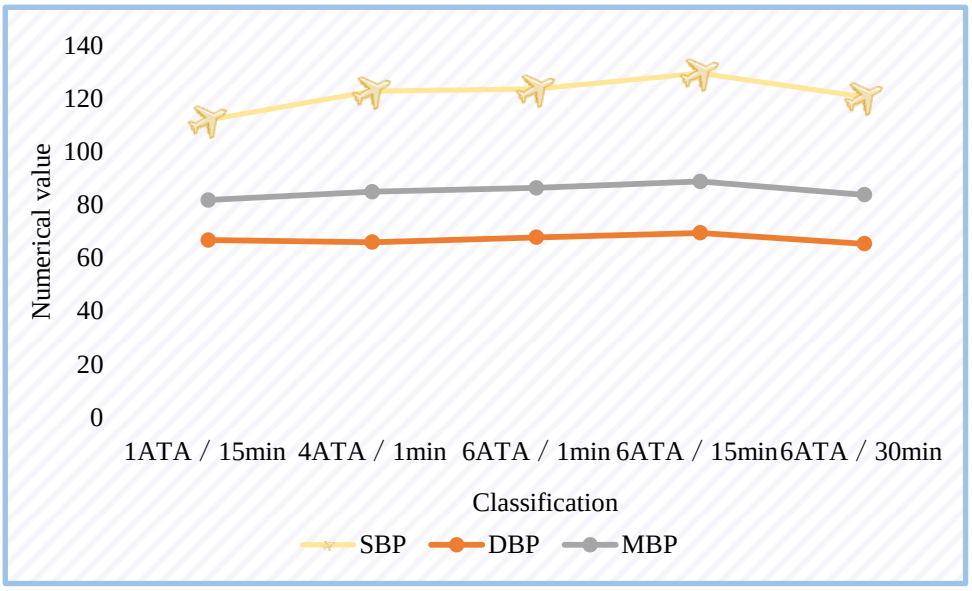

Figure 2. Blood pressure comparison

According to the statistical analysis of data, as shown in Figure 2 and table 2, when the pressure reaches 6ata, the systolic pressure increases by 5% ($P < 0.05$), the diastolic pressure increases by 11% ($P < 0.05$), the average arterial pressure increases by 9% ($P < 0.05$), and the pulse pressure has no significant change. Systolic and pulse pressures increased by 8% ($P < 0.01$) and 19% ($P < 0.01$).

4.2. Ultrastructural Test Results

(1)Comparison of histopathological morphology

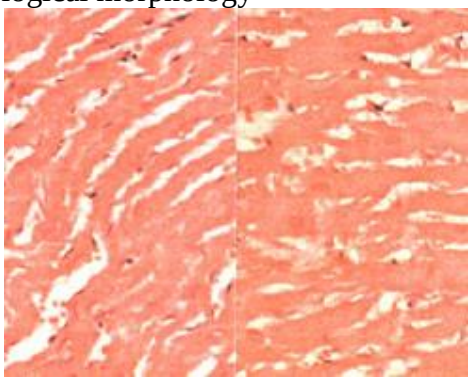

Figure 3. Comparison of histopathological morphology

According to the statistical analysis of data, as shown in Figure 3, myocardial cells have clear staining, clear bands, dense nuclei and no obvious pathological changes. The heart is the power device of blood circulation. There are some special morphological changes in the ultrastructure of heart tissue during heart failure. Under light microscope, the intercellular space of myocardial cells is large. The structure of myocardial cells is incomplete, the membrane is atrophic, the nuclear staining is uneven, and the light and dark stripes between myocardial cells are fuzzy. Under the electron microscope, the nucleus of myocardium was oval, the nuclear membrane structure was incomplete and concave, heterochromatin concentrated at the edge of nucleus, and chromatin concentrated in cytoplasm. Partial dissolution of myocardial fibers and dislocation of myofibrils. The structure of sarcomere is not clear, there are vacuoles between sarcomeres, the disc is loose and

the structure is irregular. Mitochondria have different shapes, incomplete membranes and even vacuolation. Lipofuscin can be seen. The disc is the cell boundary of the heart muscle. On the Z-line, there are two adjacent myocardial fibers, which are papillary processes. They are concave and convex, lacking each other. The resistance at the connection is very low. The excitation of muscle fibers can be transmitted from one cell to another, or even the whole heart muscle, which ensures the synchronous rhythmic contraction of myocardial fibers. The main function of mitochondria is to produce the energy needed for cell survival through aerobic oxidation. Myocardial mitochondria are very rich, accounting for about 30% of the cytoplasm of cardiomyocytes.

(2)Expression of ET-1 in myocardium

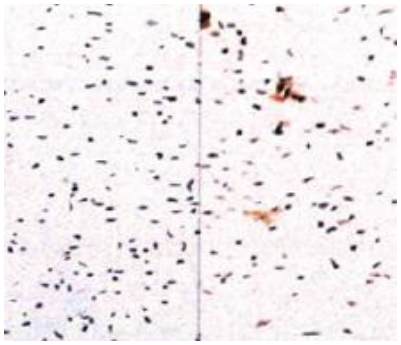

Figure 4. Expression of ET-1 in myocardium

According to the statistical analysis of data, as shown in Figure 4, brown staining showed that ET-1 was positive. The expression of ET-1 in control group, high pressure nitrogen group and air group was significantly increased. ET-1 was expressed in the cytoplasm and a small number of cardiac myocytes.

5. Analysis and Discussion

5.1. Analysis

The contractility of air group, normoxia group and high nitrogen group increased significantly at 4 ATA and 6 ATA. These results indicate that high-pressure exposure to simulated diving in a pressurized chamber can lead to an increase in myocardial contractility, regardless of oxygen partial pressure. As the heart rate of the normoxia group remained unchanged, it can be inferred that the increase of myocardial contractility was not related to sympathetic regulation. At the beginning of compression, the systolic force increased obviously, and then the increasing rate of systolic force decreased gradually, which may be related to the increase of respiratory gas density. End diastolic pressure is often used to reflect preload, and mean arterial pressure (MBP) is used to reflect afterload. You are influenced by many factors. When the air pressure reached 4 ATA and 6 ATA, the heart rate decreased and the diastolic pressure increased. When nitrogen and oxygen were added to 4 ATA and 6 ATA, there was no significant change in heart rate and diastolic pressure. It is suggested that high hydrostatic pressure has no effect on heart rate and peripheral resistance, while hyperbaric oxygen can reduce heart rate and increase peripheral resistance. The mechanism of heart rate slowing down under high pressure, the increase of blood oxygen tension, will lead to the adaptive response of cardiovascular system, thus reducing the stimulation of vascular chemoreceptor, and reducing its excitability, thus causing heart rate slowing or bradycardia, systolic pressure unchanged, increasing diastolic pressure and pulse pressure difference decreased. In our experiment, when the normoxia group was pressurized to 4 ATA and 6 ATA, the heart rate remained unchanged. High pressure exposure can delay the excitation and contraction coupling of

cardiomyocytes, prolong the plateau period and delay the repolarization of left ventricle, which may be one of the reasons for the increase of myocardial contractility, but there is no significant change in stroke volume. When the rats were repeatedly exposed to the mixture of 5 ATA air and normal oxygen partial pressure, the left ventricular hypertrophy was obvious, the coronary artery blood flow was increased, and the myocardial cell apoptosis was obvious. We speculate that high pressure exposure has a similar effect on human body. Repeated high-pressure exposure will cause continuous damage to the myocardium. The accumulation of this damage may lead to a heart attack based on the diver's original hidden heart attack. Therefore, we suggest that recreational divers and professional divers should have regular physical examination, pay special attention to the examination of heart function and cardiovascular system to avoid accidents.

The expression of ET-1 in left ventricular myocardium increased significantly in 6ata air and normoxic condition, while the content of ET-1 in plasma increased significantly in 6ata air and normoxic condition and high nitrogen condition ($P < 0.01$). High pressure exposure can stimulate the expression of ET-1 by combining some special regulatory sites of ET-1 gene. ET-1 can stimulate the proliferation and hypertrophy of myocardium and vascular smooth muscle, and plays an important role in the occurrence and development of congestive heart failure, ischemic heart disease, myocardial infarction and other cardiovascular diseases. The content of ET-1 in these patients' heart and plasma increases in varying degrees, This is closely related to the degree of heart failure and the duration of myocardial ischemia. Due to the increase of workload, contractility, ET-1 expression and ET-1 secretion, these factors may have short-term and long-term effects on the cardiac function of divers. Acute decompression sickness usually occurs within 1-3 hours after decompression. If a small amount of nitrogen bubbles embolism for a long time, it will lead to ischemic necrosis of bones and joints, difficulty in limb movement or lameness. Osteonecrosis of decompression sickness is one of the late complications and sequelae of decompression sickness. It is the manifestation of serious damage of decompression sickness of bone and joint system. Regular physical examination should be carried out before employment and diving. Every year, the bones and joints, especially the large joints of limbs, are examined by X-ray until four years after the cessation of high-pressure operation. Serious illness, frailty, long-term fractures, alcoholism and obesity are also taboos. We should pay attention to science in diving. When the safety of the diver is threatened by the water surface or underwater environment, diving is prohibited. It depends on the diving depth, labor intensity, water temperature, flow rate, operation mode and personal conditions. Strictly control the underwater operation time, correctly select the appropriate decompression scheme, and strictly abide by the safety operation rules of diving operation. Treatment principle: acute decompression sickness must be treated with pressure as soon as possible. According to clinical manifestations, comprehensive auxiliary treatment should be given in time. At this time, patients with symptoms due to the failure of timely or correct stress treatment should still actively carry out stress treatment.

5.2. Discussion

In modern society, society is changing every day. Modern society needs more interesting and fresh leisure ways. The existing is far from meeting people's needs. New things need to appear to meet their physical and psychological needs. Many leisure ways in the market are not satisfactory. Most people know only one, not the other. The emergence of leisure diving is to break people's inherent thinking and concept. People are eager for a new way of traveling. Facts show that it is difficult to meet people's needs only by relying on the natural scenery of big rivers and rivers. The same landscape, the same face, people gradually appear aesthetic fatigue, people no longer want this simple tourism. With the development of economy, tourism related matters become more and

more important. People have more time and money, so they naturally want to accept more and better leisure activities. Compared with ordinary leisure sports, diving has its own characteristics. Diving exploration is carried out underwater, which is very different from the activities on the ground. Recreational diving activities will see many relatively unfamiliar scenes on the ground, because there are fewer participants, so the scenery you see is also well preserved. The unspoiled underwater world is also unforgettable, which is the fun of leisure diving. However, the specialty of leisure diving lies in the existence of certain risks, because different from land, underwater is facing the threat from the sea and many creatures, it naturally needs professional talents, and it is urgent to cultivate professional talents. When we are underwater, we also need to master a series of survival technologies, so as to maximize our game effect and our life safety. In the undersea world, we can start from a new angle and a new feeling, which is a huge spiritual enjoyment that cannot be said.

With the rapid development of social economy, the demand for energy is growing, and land resources are becoming increasingly scarce. In addition to resource development, maintaining maritime sovereignty and territorial integrity is also a strategic issue facing China. With the rapid development of submarine forces, higher requirements are put forward for submarine support and rescue technology. Improving diving technology and corresponding medical security technology is the key link of rescue work. The key problem of diving medical security is to prevent and treat decompression sickness. If the air pressure around the body is rapidly and greatly reduced (this may happen in diving operations, submarine escape and rescue, etc.), the physiological inert gases (such as nitrogen, helium, etc.) dissolved in the body can not be discharged through the respiratory and circulatory system in a state of dissolving, but in the organization form a bubble overflow, it may cause rashes, spasms, joint muscle pain. Respiratory and circulatory disorders, neurological dysfunction and even death. Generally speaking, the physiological effect of high pressure and its impact on the body are temporary and reversible, but the impact is serious or long-term. Improper recovery will also lead to long-term irreversible pathological changes. When people enter the water, they have to breathe the same gas as the surrounding water pressure. Environmental factors in prevention include temperature, hydrological conditions and underwater characteristics. During diving, the risk of decompression sickness will increase if there are factors that can increase dissolved nitrogen in the body (rapids, soft substrate, low visibility, etc.) or reduce nitrogen emission during decompression (low water temperature, etc.). Personal rhythms: vigorous exercise before diving can affect rhythms for hours (which can make the body need more decompression gas). It increases the nitrogen in the blood after diving. Strenuous exercise can make the diver tired and increase the burden of diving (the increase of heart rate increases the frequency of breathing). This can cause muscle damage and increase the risk of infection. Vigorous diving (swimming against the current) also increases heart rate and breathing rate. The breathing rate will be three to four times that of a simple dive. Psychological stress, panic and fear also speed up the heart rate, which is equivalent to strenuous exercise. Health is also important. A strong diver has a lower heart rate and less blood is produced through the heart and lungs, so he needs less gas than a fat diver. Any diver with an infectious disease increases the risk of decompression sickness. They are usually tired, irregular and prone to air bubbles. Smokers are more likely to produce bubbles in the blood than nonsmokers. It may be caused by chemical changes in the blood or lung damage caused by smoking. Smokers are in a sub-health state. Older divers are more likely to develop decompression sickness than younger divers. Old divers are not as healthy as young divers in theory. In theory, older divers have more fat in their bodies than younger divers. According to the theory of decompression sickness, women are twice as likely to have decompression sickness as men. In theory, women have more fat, less muscle and are more likely to get sick than men. Of course, if compared with other factors, other factors will be more important. The operational factors include technical level, violation of diving rules, etc. The faults of diving support system include: gas

supply system fault, diving equipment fault, artificial water supply system fault, decompression system fault, surface underwater communication system fault, etc. Due to the operation or calculation error of the surface support personnel, the safety problem of the diver's air supply or outlet is caused. With the increase of depth, the influence of living gas environment on human function is obviously different. Including the changes of physiological and physiological functions caused by pressure itself, such as the effects of hyperbaric neurosis, i.e. pressure load; physiological effects caused by the changes of gasification components. In high pressure environment, it is necessary to closely monitor the physical functions of divers to ensure that divers can dive in a safe range. The longer the diving time, the higher the incidence of acute decompression sickness. Due to the hydrostatic pressure of water, divers must breathe high pressure compressed gas when working in water.

It is very necessary to train the diving personnel in working knowledge, understand the basic theory and decompression principle of diving, and prevent the occurrence of acute decompression sickness. Standardized decompression is also one of the important measures to reduce acute decompression sickness. After the completion of diving, to enter the state of decompression, adequate decompression is necessary. Insufficient decompression can cause decompression sickness and seriously endanger the health and life safety of workers. Therefore, the diving operator should reduce the pressure according to the decompression table. The decompression schemes of all decompression tables, including the standard air decompression tables of the Chinese Navy, the standard air decompression tables of the Soviet Union and the French diving decompression tables, all have a depth of 9 meters, and the stay time under water is generally 10 minutes, but the combination of the actual diving depth and time is rarely one of the decompression schemes fully in line with the use. However, the decompression scheme selected is always treated according to the conservative principle; The depth of the selected decompression scheme is always equal to the actual diving depth or the next level depth is slightly greater than the actual diving depth; the underwater retention time of the selected decompression scheme is always equal to the actual underwater retention time or the next level time slightly greater than the actual water retention time. If ground decompression is required, the cabin shall be operated on site by a lifeguard or qualified diving supervisor. In general, repeated diving is prohibited. If repeated diving is required, decompression shall be carried out according to special regulations. Diving is also an important risk factor for acute decompression sickness. Heavy diving is more likely to cause acute decompression sickness than light diving. It is not allowed to work in the suspended state. The risk of floating (i.e. too much air in the diving suit, suddenly rising rapidly after losing control) is very large. Preventive measures and Countermeasures for acute decompression sickness: establish and improve relevant laws and regulations, improve the management organization, and strengthen the supervision of labor law enforcement and other relevant departments. Health supervision and disease prevention and control institutions shall, in accordance with the relevant provisions of occupational health laws and regulations, actively strengthen supervision and monitoring, crack down on illegal employment, and strengthen the pre employment health inspection and post employment health monitoring management of diving operators. Strictly select diving personnel, and conduct physical examination and technical training for diving operators. Only those who pass the examination can be employed. Establish strict inspection system. Check whether the diving personnel of the working department have the work license and illegal diving operation, organize the local medical and health department and the occupational disease prevention department, and regularly conduct the diving medical knowledge and diving safety protection training for the diving personnel. Improve the rescue system of decompression sickness, establish a green first-aid channel for decompression sickness in the hospitals around the diving and fishing intensive sea areas, and equip full-time medical personnel to ensure that the diving personnel with acute decompression sickness are treated in a

timely and effective manner. The employer shall strengthen the safety management system for diving personnel. Improve the emergency rescue plan for accidents caused by decompression sickness, and be able to handle emergencies accurately and timely. Divers shall receive formal technical training and obtain corresponding qualification certificates before work. Actively learn the medical prevention and treatment knowledge of decompression sickness, and be familiar with the causes and prevention methods of decompression sickness for divers. Regular physical examination and stress exercise. Those who are not healthy can't dive.

6. Summary

The main risk factors of acute decompression sickness are diving depth and diving style. The deeper the diving depth, the more prone to acute decompression sickness. The change of heart function of divers may be the result of repeated high pressure exposure, so the examination of heart function should be paid attention to during physical examination. Hyperbaric oxygen exposure can cause the increase of systolic and diastolic blood pressure. Diving has little effect on cardiac histopathology.

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] C, H. S. S. A., D, A. E. K. E. C., B, T. M. M., C, H. M. R. B., & E, E. S. B. (2016)“ Histopathological and Immunohistochemical Study of the Protective Effect of Triptorelin on the Neurocytes of the Hippocampus and the Cerebral Cortex of Male Albino Rats after Short-Term Exposure to Cyclophosphamide”, Journal of Microscopy and Ultrastructure, 4(3),pp. 123-132. https://doi.org/10.1016/j.jmau.2015.12.002

[2] Tabassum, K., Shaiba, H., Essa, N.A., and Elbadie, H.A. (2022). An Efficient Emergency Patient Monitoring based on Mobile Ad Hoc Networks. Journal of Organizational and End User Computing(forthcoming). https://doi.org/10.4018/JOEUC.289435

[3] Zhu, Y. D., Hu, L., Li, P., Zhang, M., & Liu, Y. Q. (2018)“ Effects of Celastrus Orbiculatus on Epithelial Mesenchymal Transition in Gastric Mucosal Epithelial Cells by Inhibiting Lgr5 Expression From Rats with Gastric Precancerous Lesions”, American Journal of Chinese Medicine, 46(05), pp.1129-1143. https://doi.org/10.1142/S0192415X18500593

[4] Nogarol, L. R., Brossi-Garcia, A. L., De Souza, R. B., & Fontanetti, C. S. (2016)“ Histopathological Effects of the Herbicide Atrazine on Gills of the Brazilian Endemic Bivalve Diplodon Expansus”, International Journal of Environmental Analytical Chemistry, 96(1-5),pp. 387-403. https://doi.org/10.1080/03067319.2016.1150461

[5] Qin, Q., Yang, X., Zhang, R., Liu, M., and Ma, Y. (2022). An Application of Deep Belief

Networks in Early Warning for Cerebrovascular Disease Risk. Journal of Organizational and End User Computing(forthcoming). https://doi.org/10.4018/JOEUC.287574

[6] Isabelle Labonté, Michel Laviolette, Ron Olivenstein, Jamila Chakir, LouisPhilippe Boulet, & Qutayba Hamid. (2016)" Quality of Bronchial Biopsies for Morphology Study and Cell Sampling: a Comparison of Asthmatic and Healthy Subjects", Canadian Respiratory Journal Journal of The Canadian Thoracic Society, 15(8),pp. 431. https://doi.org/10.1155/2008/202615

[7] Gherman, C. M., Deak, G., Matei, I. A., Ionic?, A. M., D'Amico, Gianluca, & Taulescu, M., et al. (2016)" A Rare Cardiopulmonary Parasite of the European Badger, Meles Meles: First Description of the Larvae, Ultrastructure, Pathological Changes and Molecular Identification of Angiostrongylus Daskalovi Janchev & Genov 1988", Parasites & Vectors,9(1),pp. 423. https://doi.org/10.1186/s13071-016-1718-8

[8] Das, T., & Goswami, M. (2018)" Histopathological and Ultrastructural Changes in the Gill and Liver of Fresh Water Fish Channa Punctatus Exposed to Sodium Arsenite", Bioence Biotechnology Research Communications, 11(3), pp. 434-441. https://doi.org/10.21786/bbrc/11.3/12

[9] Badr, E. D. F. M. M., Nabil, I. M., & Dwedar, F. I. (2017)" The Effect of Tributyltin on Thyroid Follicular Cells of Adult Male Albino Rats and the Possible Protective Role of Green Tea: a Toxicological, Histological and Biochemical Study", Egyptian Journal of Forensic Sciences, 7(1), pp.7. https://doi.org/10.1186/s41935-017-0012-z

[10] Memaria, E., Hosseinianb, M. A., Mirkheshtia, A., Arhami-Dolatabadig, A., & Shiriand, S. (2016)" Comparison of Histopathological Effects of Perineural Administration of Bupivacaine and Bupivacaine-Dexmedetomidine in Rat Sciatic Nerve", Experimental and Toxicologic Pathology, 4(10), pp.559-564. https://doi.org/10.1016/j.etp.2016.09.001

[11] Khafaga, A. F., Abu-Ahmed, H. M., El-Khamary, A. N., Elmehasseb, I. M., & Shaheen, H. M. (2017)" Enhancement of Equid Distal Limb Wounds Healing by Topical Application of Silver Nanoparticles", Journal of Equine Veterinary Science, 61(2018),pp. 76-87. https://doi.org/10.1016/j.jevs.2017.11.013

[12] Jun, L., Xiaole, H. E., Ping, Z., Shenghu, Z., & Xusheng, L. I. (2016)" Protective Effect of Diosgenin on Chondrocytes Mediated by Jak2/Stat3 Signaling Pathway in Mice with Osteoarthritis", Journal of Zhejiang University(Medical ences), 45(5),pp. 453.

[13] Beshay, E. V. N., Rady, A. A., Afifi, A. F., & Mohamed, A. H. (2019)" Schistosomicidal, Antifibrotic and Antioxidant Effects of Cucurbita Pepo l. Seed Oil and Praziquantel Combined Treatment for Schistosoma Mansoni Infection in a Mouse Model", Journal of Helminthology, 93(3), pp.286-294. https://doi.org/10.1017/S0022149X18000317

[14] Choi, G. H., Ko, H., Pedrycz, W., Singh, A. K., & Pan, S. B. (2020). Recognition system using fusion normalization based on morphological features of post-exercise ecg for intelligent biometrics. Sensors, 20(24), 7130. https://doi.org/10.3390/s20247130

[15] Abdul-Hamid, M., Abdella, E. M., Galaly, S. R., & Ahmed, R. H. (2016)" Protective Effect of Ellagic Acid against Cyclosporine a-Induced Histopathological, Ultrastructural Changes, Oxidative Stress, and Cytogenotoxicity in Albino Rats", Ultrastructural Pathology, 40(4), pp.205-221. https://doi.org/10.1080/01913123.2016.1203854