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# Preliminary Discussions About Myocardial Injury and Rehabilitation

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**Abstract.** This paper demonstrated discussions about myocardial injury and rehabilitation, not only focus on one or two causes for myocardial injury to occur, but also on multi-pathogeny. It discusses how to recover the injured myocardial tissue through various medical methodologies. According to the literature review, understanding the diseases is based on their pathogenesis, as well as the etiology and occurrence of their related symptoms, as far as cardiomyocyte injury is concerned. Re-functioning cardiomyocytes when weakened or lost after cardiomyocyte injury has also been discussed. Not only the consequence of myocardial injury but also relative diseases.

**Keywords:** Myocardial injury, myocardial function, myocardial rehabilitation, cardiovascular disease, myocarditis.

## INTRODUCTION

Cardiovascular disease is a prominent social and medical problem all over the world nowadays. The prevalence continually rise and has become most frequent death all over the world [28].

The mortality rate of elderly diseases, especially cardiovascular diseases, increases with age, especially with the improvement of people's living standards and extension of life expectancy [21]. Worldwide, it was reported that the mortality rate of cardiovascular diseases increases significantly after age 40, as well as increasing about twice for every 10 years of age in people over 60 [21]. In China, the death rate from coronary heart disease (CHD) was more than half of the elderly [22]. Acute myocardial infarction (AMI) is one of the more serious types of CHD and can seriously affect the quality of life (QOL) of patients (65% are over 65 years old) due to impaired cardiac function and post-infarction angina pectoris.

Heart disease, whether classified by the formation mechanism or the degree of harm, is the most basic damage to the heart and causes different degrees of myocardial cell damage [27].

## DISEASES LEADING TO CARDIOMYOCYTE INJURY

### Ischemic Heart Disease (IHD)

Figure 1 shows multiple causes of myocardial injury. Not only discussion about current clinic circumstances, but also historic evaluations.

Ischemic heart disease (IHD), also known as coronary atherosclerotic heart disease (CHD), could lead to acute myocardial infarction (AMI) and heart failure (HF) [33]. The heart is a terminal differentiation organ and lacks sufficient regenerative capacity; after an AMI, cardiac function cannot be restored even with timely revascularization, and the necrotic myocardium cannot be reversed [33]. A large number of myocardial necrosis cases will lead to the occurrence of HF, and stem cell therapy has become a promising therapeutic method to restore the damaged cardiac function after AMI [33]. In the previous decade, a large number of animal and clinical studies have verified the effectiveness and safety of stem cell transplantation in the treatment of IHD; stem cells promote the recovery of damaged heart function through paracrine effect, immune

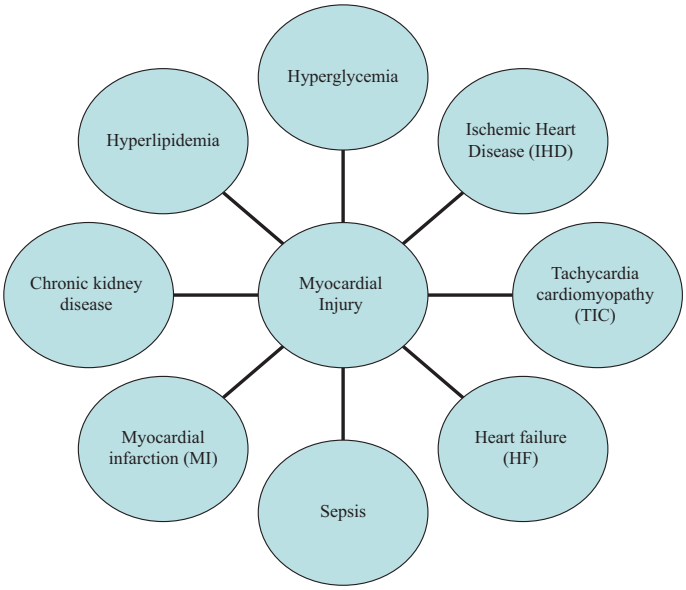


**Figure 1.** Causes myocardial injury.

regulation, proliferation, and differentiation and show a good clinical application prospect [33]. However, due to the low survival rate of transplanted stem cells, stem cell therapy is difficult to achieve sufficient effect, which limits its function, its efficacy is not enough to replace the lost cardiac function, and it is difficult to meet the clinical needs [33]. A large number of previous studies have shown that the survival rate of transplanted stem cells within 24 h is less than 10% regardless of intramyocardial injection, intracoronary injection or intravenous route, and the long-term colonization and survival are lower [33]. The main reasons leading to the low survival rate of transplanted stem cells include the following: the squeezing effect of blood flow and myocardial contraction and the loss and acute death of transplanted stem cells caused by needle injection; the local microenvironment such as necrotic tissue accumulation, acute inflammation, oxidative stress, ischemia and hypoxia, ischemia-reperfusion injury after myocardial infarction is not conducive to the colonization and survival of transplanted cells; the clearance effect of monocyte macrophage system on necrotic tissue and transplanted cells, etc. [33].

The related factors and occurrence sites of ventricular arrhythmia in patients with myocardial infarction recovery period (HMI) and the relationship between non-invasive detection indicators representing different pathological processes: heart rate variability (HRV), left ventricular ejection fraction (LVEF), early repolarization change (ERV), fragmented QRS wave (fQRS), and ventricular arrhythmia after myocardial infarction [24].

Ischemic postconditioning (IPO), as an endogenous protective mechanism, has been shown to effectively inhibit the apoptosis of cardiomyocytes, but whether it can also repair damaged cardiomyocytes by inducing cell proliferation is still unclear [26].

Coronary artery occlusion is caused by complete or incomplete occlusion of the lumen due to bleeding in the atherosclerotic plaque or subintimal of the diseased vessel, thrombosis in the lumen or persistent spasm of the artery, resulting in insufficient cardiac perfusion, ischemia of the heart tissue, and myocardial infarction caused by destruction of myocardial cells. Cardiomyocytes are essential for maintaining cardiac function. Mature cardiomyocytes are in the state of terminal differentiation, permanently exit the cell cycle, have little regenerative capacity, and cannot achieve self-healing after myocardial injury. After mammalian heart injury, the main reaction is myocardial fibrosis and the formation of non-contractile scar tissue, which will damage the heart function and eventually lead to heart failure. Therefore, promoting cardiomyocytes that exit the cell cycle to enter the cell cycle again and replenish the lost cardiomyocytes is the key to achieving myocardial regeneration [1]. At the early stage of the embryo, the proliferation level of mouse cardiomyocytes was very high, and the cell proliferation level began to decrease from the 10th to 12th day of the embryo. In the second week after birth, many cardiomyocytes undergo mitosis, or "cytoplasmic undifferentiation," to produce binucleate cardiomyocytes. After that, most cardiomyocytes were in a state of cell cycle arrest [2, 3]. In recent years, cardiomyocyte proliferation has been found in the hearts of adult mammals [4]. However, cardiomyocyte proliferation capacity is extremely limited and far from sufficient to restore cardiac function after injury [5, 6]. At present, the mechanism of how irreversible cell cycle arrest occurs in the postnatal stage is not clear. Therefore, by analyzing the difference between the cell cycle activities of neonatal and adult cardiomyocytes, understanding the basic mechanism of the cardiomyocyte cycle will help promote the proliferation of cardiomyocytes and recover the cardiac function damage caused by ischemic cardiovascular disease [34].

When the artery of the heart is narrowed and there is thrombosis at the stenosis, myocardial infarction will occur. Only a portion of the patients with myocardial infarction can survive and recover well. If the heart can survive this hurdle, the heart will have a recovery process. Of course, it is impossible to completely recover to the normal state [43]. After myocardial infarction, tissue ischemia, hypoxia, and necrosis lead to a multi-phase repair process. The damaged tissue is replaced by a fibrous scar produced by fibroblasts and myofibroblasts Reactive remodeling of non-infarcted ventricular wall, including interstitial and perivascular fibrosis, leads to changes in ventricular wall geometry, biomechanics, and biochemistry. Although the initial reparative fibrosis is essential to prevent ventricular wall rupture, an excessive fibrotic response leads to progressive impairment of cardiac function and ultimately leads to cardiac failure. In recent years, studies have shown

that the heart has plasticity, restoring damaged heart function and promoting the repair of infarcted myocardium are important targets for the treatment of cardiovascular diseases [56].

In recent years, myocardial tissue engineering has risen rapidly. Through the application of exogenous biomaterials to simulate extracellular matrix, damaged cardiomyocytes can be effectively repaired or reconstructed, which has great potential value in the treatment of ischemic heart diseases such as myocardial infarction [58].

Myocardial infarction (MI) is a serious threat to human health. In the field of treatment of cardiovascular diseases, the lack of self-healing ability of myocardial tissue is one of the current challenges. The existing clinical treatment methods can not restore the cardiac function of the lesions after MI Myocardial tissue engineering is an important potential way to repair damaged myocardial tissue. Tissue engineering materials can be used for the delivery of stem cells (bone marrow mesenchymal stem cells, and embryonic stem cells), growth factors (VEGF and IL-7) and simulated extracellular matrix [59].

With the extensive development of thrombolysis and emergency coronary intervention, the mortality of acute myocardial infarction (AMI) has been significantly reduced. However, ventricular remodeling and ischemic cardiomyopathy after myocardial infarction seriously affect the prognosis of patients. It is still a huge challenge to regenerate cardiomyocytes and even restore the function of the exhausted heart [68].

After reperfusion of the myocardium with abnormal blood flow perfusion, the myocardial segment with systolic dysfunction can gradually recover its function. The recoverable myocardium is called “viable myocardium”. Because myocardial revascularization can improve the damaged left ventricular function and improve the prognosis of patients, preoperative evaluation of viable myocardium is particularly important. As a non-invasive method, magnetic resonance imaging can evaluate the function, perfusion, myocardial cell membrane function and metabolism of myocardium; the presence or absence of viable myocardium was judged; quantify the number of viable myocardium; and it is expected to identify myocardial stunning and myocardial hibernation in viable myocardium [70].

Myocardial infarction is a serious threat to human health. Drug therapy and antithrombotic therapy can not restore cardiac blood flow and repair damaged cardiac tissue [77].

Myocardial infarction can lead to ventricular remodeling and heart failure Therefore, it is essential to restore the function of damaged myocardial tissue. Stem cell-based myocardial regeneration and repair therapy can ultimately avoid the process of ventricular remodeling and the occurrence of heart failure. Cell therapy for heart disease is a rapidly developing field, but there are still many difficult problems to solve [82].

Acute myocardial infarction (AMI) is a common and serious type of coronary heart disease. It will cause huge losses to patients, including myocardial damage and accompanying symptoms and signs such as cardiac insufficiency and arrhythmia. It will limit the physical labor and self-care ability of patients and cause serious physiological and psychological functional disorders such as anxiety and depression [89].

Ischemic heart disease is increasingly endangering people’s health, and the primary treatment to restore coronary blood flow often leads to myocardial ischemia-reperfusion injury (MIRI). Mitochondria provide a large amount of energy for cardiomyocytes, and participate in the regulation of apoptosis and signal transduction. In the ischemic and hypoxic environment, damaged mitochondria will undergo selective and programmed autophagy degradation to maintain the homeostasis of cardiomyocytes. This process is called mitochondrial autophagy and is closely related to the occurrence and development of Miri Mitophagy has dual characteristics. It is of great clinical significance to accurately and effectively intervene at this level to improve Miri. In recent years, Chinese medicine has played a multi-target and multi-channel therapeutic advantage in the prevention and treatment of cardiovascular diseases, and gradually attracted people’s attention. More and more studies have confirmed that Chinese medicine monomers, components, and compounds can save blood-deficient myocardium by protecting mitochondrial structure and function and regulating mitochondrial autophagy [40].

**Hyperglycemia or Hyperlipidemia**

Hyperglycemia or hyperlipidemia is a sign of diabetes, which can lead to increased myocardial vulnerability after myocardial ischemia/reperfusion (MI/R). When myocardial ischemia occurs in diabetes patients, the number of myocardial cell deaths, the degree of cardiac function damage, and no reflow after reperfusion are significantly increased compared with non-diabetes patients [30].

The expression of FoxO1 in myocardium of diabetes mice is higher than that of normal mice, while the expression of adiponectin and AdipoR1, which have protective effects on MI/R injury, is decreased, suggesting that FoxO1 may aggravate mi/R injury by downregulating adiponectin signal in diabetes myocardium; injection of FoxO1 siRNA into myocardium to inhibit FoxO1 expression can reduce the MI/R injury in diabetes, and the expression of adiponectin and AdipoR1 from myocardium can be restored, it is further suggested that the decrease of adiponectin caused by the overproduction of FoxO1 in diabetes mice is the cause of the aggravation of MI/R injury in diabetes mice; the experiment of high glucose cultured cardiomyocytes further clarified the relationship between high glucose induced overexpression of FoxO1 and decreased adiponectin and increased cardiomyocyte

apoptosis; injection of FoxO1 siRNA into the myocardium of APN-/-mice could effectively inhibit the level of myocardial FoxO1 mRNA but had no significant effect on the MI/R injury of APN-/-mice, the regulatory effect of FoxO1 on adiponectin signaling was confirmed again, the research suggests that controlling the overproduction of FoxO1 in diabetes mice is of great significance in alleviating mi/R injury in diabetes mice [30].

### Chronic Kidney Disease

Chronic kidney disease is a worldwide public health problem that is closely related to the incidence rate and mortality of cardiovascular disease. The harm of chronic kidney disease complicated with cardiovascular disease is becoming more and more serious. The incidence of myocardial infarction in patients with chronic kidney disease is twice that of normal people. Therefore, prevention and treatment of the cardiovascular system damage caused by chronic kidney disease is the primary issue that must be solved. Adiponectin is a 30 kDa cytokine derived from fat, which can reduce inflammatory reactions, promote energy metabolism, and protect the cardiovascular system. The main forms of adiponectin in blood are adiponectin trimer, full-length adiponectin, and high molecular weight adiponectin. In common diseases such as hypertension and coronary heart disease, the serum adiponectin level is low. Previous studies have confirmed that the level of adiponectin decreases after MI/R, and that adiponectin protects I/R myocardium by reducing oxidative/nitration stress. However, the plasma adiponectin level in patients with chronic renal insufficiency is increased, and the incidence rate and mortality of cardiovascular complications associated with renal insufficiency are also significantly increased. So far, the effect and mechanism of adiponectin on chronic kidney disease complicated by cardiovascular disease have not been clarified [38].

### Sepsis

Sepsis myocardial injury is an acute organ injury caused by sepsis or sepsis shock. It is characterized by impaired left (right) ventricular contraction and diastolic pump function Despite the active anti-infection, metabolic resuscitation, and multi-organ function maintenance, some sepsis patients still show low output and high resistance in hemodynamics after the recovery of cardiac preload (increased resistance of peripheral blood vessels and decreased cardiac output). In the final analysis, it may be because the damage to myocardial mitochondria has not been recovered. The dual role of microcirculation and mitochondria has now become a research hotspot for sepsis myocardial injury. As a central participant in the pathophysiology of myocardial injury in sepsis, the structure, quantity, and function of mitochondria are directly affected by the survival rate and prognosis of sepsis patients [67].

### Others

Tachycardia-induced cardiomyopathy (TIC) refers to a cardiomyopathy in which arrhythmia leads to reversible impairment of left ventricular function [53].

Cardiac troponin I increased significantly in patients with cardiopulmonary arrest at 1 h after recovery of spontaneous circulation, while CK, CK-MB, LDH, and AST began to increase at 2 h after recovery; the elevated level of cardiac troponin I was positively correlated with the degree of myocardial injury and the mortality of patients. Conclusion: (1) acute myocardial injury exists in patients with cardiopulmonary arrest and recovery of autonomic circulation and (2) the blood concentration of cardiac troponin I can reflect the severity of myocardial damage [3].

At present, there is a more unified view in traditional Chinese medicine on viral myocarditis that warming poison injures Yin. If it is treated incorrectly, Yin will damage Yang, and Yang Qi will also be injured. It can also be seen that warming poison is burnt, directly attacking the camp blood, and both qi and blood are injured, resulting in myocardial damage or heart failure.

The proportion of left ventricular enlargement and sustained decrease of LVEF in FM patients who survived and were discharged from the hospital was higher than that in NFM patients. A long recovery time of LVEF during hospitalization is an independent risk factor for impaired cardiac function after discharge in FM patients [1].

## MYOCARDIAL REHABILITATION

Table 1 shows multiple causes of myocardial rehabilitation. Not only discussion about clinic treatments, but also biological technique.

Cardiotrophin is a cytokine with myocardial protective functions [12].

Cell transplantation provides a new method for cell reconstruction of damaged hearts and functional recovery of failed hearts. Although animal experiments and clinical trials have confirmed that stem cell transplantation has an obvious therapeutic effect on myocardial infarction, the ischemia and inflammation in the myocardial infarction area constitute an “unfriendly” transplantation microenvironment, resulting in a large number of deaths of transplanted stem cells and seriously affecting the effect of cell therapy [73].

At present, the commonly used myocardial scaffold materials mainly include natural biomaterials (including collagen/matrigel, fibrin, chitosan, hyaluronic acid, and seaweed hydrochloric acid), synthetic materials (polyester synthetic materials and nanomaterials) and composite scaffold materials. Due to the complexity of the cardiac environment and cardiac function, the selection of scaffold materials should fully consider the biocompatibility, immunogenicity, conductivity, degradation rate, susceptibility to ischemia and hypoxia, and other factors [58].

**Table 1.** Causes myocardial rehabilitation.

| | | | |
|---|---|---|---|
| Cardiac nutrient | Cell transplantation | Myocardial stent | Auxiliary application: folic acid combined with VitB6 |
| Chinese medicine | Coenzyme Q10 and lysine inositol vitamin B12 oral liquid | Creatine phosphate sodium | Related compatibility of Pueraria lobata |
| Soluble rhct-1 | Artificial heart nano patch | Cardiac arrest (CA) Ca/ROSC restoration of spontaneous circulation (ROSC) | Ischemic postconditioning (IPO) |
| SPostC | Betulin | Imbalance of mitochondrial division and fusion in early stage after ca/rosc | Sodium nitroprusside |
| Hypoxia in MI state | let-7i | Inhibition of microrna-153-3p expression | Supplemental exogenous adiponectin |
| Combined application of vitamin C and immunoglobulin | Revascularization | Patch for myocardial recovery | High intensity interval exercise |
| Betaloc combined with spironolactone | SAB may regulate pkd1-hif-1 $\alpha$-VEGF pathway | Before PCI, half of urokinase was used for thrombolysis | Reperfusion |
| Bone marrow mesenchymal stem cells (BMSCs) | Keywords Polygonum cuspidatum; anthraquinone compound | Astragaloside iv | Others |

Although many scaffold designs have begun to meet the requirements, there are still various problems in clinical application [58]. I believe that with the further development of researchers and application tools, people can expect to create myocardial scaffolds that are close to the physiological function of the primary tissue, so that the cardiac function can be better restored [58].

The adjuvant application of folic acid and VitB6 in the treatment of acute myocardial infarction can reduce the level of myocardial damage factors after PCI, reduce the expression of serum Hcy and AMPK, improve the vascular endothelial function, be more conducive to the recovery of cardiac function, and reduce the risk of cardiovascular endpoint events [2]. The combination of coenzyme Q10 and l-inositol vitamin B12 oral liquid in the treatment of myocardial damage in rotavirus enteritis has a definite clinical effect, and the clinical symptoms of children have improved significantly, which is safe and reliable and worthy of clinical promotion [5].

Patients with viral myocarditis in convalescence often suffer from palpitations, night sweats, and other discomforts. The basic formula is Shengmai Powder; it can imitate Shenling Baizhu powder and Sijunzi Decoction to enhance the function of supplementing Qi and can also imitate Zengye Decoction to enhance the effect of nourishing yin [4]. Coptis chinensis and tangerine peel are commonly added and subtracted drugs. If the patient feels external evil again, add wind-cooling products, such as honeysuckle, forsythia, burdock, and pueraria [4]. Traditional Chinese medicine can inhibit oxygen free radicals, relieve $Ca^{2+}$ overload, regulate the energy metabolism of cardiomyocytes, repair microvascular damage, etc., restore the patient's body function, and provide protection against post-operative myocardial damage through multi-target and multi-layer therapy [85]. Baoxin decoction may regulate the expression of VEGF to maintain a suitable serum concentration to promote the establishment of collateral circulation in the ischemic area, protect the damaged myocardium, and promote the recovery of cardiac function [88]. The related compatibility of Pueraria lobata can positively regulate the myocardial mitochondrial structure and myocardial mitochondrial membrane potential, so as to recover the damage of myocardial mitochondria caused by diabetes cardiomyopathy. Among them, geqi shenlou prescription with the principle of supplementing Qi, eliminating stasis, and eliminating phlegm has the best improvement effect. It can be seen that the therapeutic mechanism may be through the synergistic effect of supplementing Qi, removing blood stasis, and eliminating phlegm, and positively regulating the quality of myocardial mitochondria to achieve the therapeutic purpose [7]. It is shown that the anthraquinone compound of Polygonum cuspidatum has certain therapeutic and protective effects on myocardial cell injury induced by ADM [69]. Astragaloside A can restore the reduced cardiac function indexes of rats with ischemia-reperfusion injury to a certain extent and reduce the elevated myocardial enzymes [72]. Astragalus injection has a strong protective effect on the damaged myocardium of sepsis patients and can recover the function of the inhibited myocardium early [79]. Liangge powder combined with interferon $\alpha$-2B can shorten the disappearance time of clinical symptoms in children with herpetic angina, protect the damaged myocardium, inhibit the inflammatory state of the body, and have small adverse reactions [74]. Shenmai injection combined with Danhong injection can effectively improve arrhythmia and reduce heart rate in patients with myocardial ischemia-reperfusion injury under cardiopulmonary

bypass and help to improve the oxidative stress state of patients, protect myocardial function, and promote the recovery of damaged myocardium [78].

The treatment of children with myocardial damage with sodium creatine phosphate is safe and effective [6]. The protective effect of weijianeng on myocardial damage can promote the recovery of myocardial tissue and cardiac function, improve the prognosis, and reduce the incidence of sequelae; it is worthy of clinical promotion [6]. The combination of sodium creatine phosphate and Xiyanping injection has achieved ideal results in the treatment of children with rotavirus-associated enteritis complicated by myocardial damage, effectively repairing damaged cardiomyocytes [54].

Soluble rhct-1 was obtained by renaturation; it has significant biological activity to promote the survival of damaged cardiomyocytes in vitro [12]. SAB may regulate pkd1-hif-1 $\alpha$-VEGF pathway and promote angiogenesis of myocardial tissue after myocardial infarction in rats [48]. The expression of SDF-1 and crcr4 in the injured tissue was upregulated, and the cardiac function was improved after local injection of hypoxic preconditioning MSCs or MSCs into the AMI area of SD rats The upregulation of SDF-1/crcr4 axis in the myocardial infarction area may be one of the important ways to improve cardiac function after hypoxic preconditioning MSCs or MSCs transplantation [25].

Engineers from Brown University in the United States and the Indian Institute of Technology have cooperated to create an artificial nano-patch for the heart to help restore the function of the damaged area caused by a heart attack [13]. This study was published in the recently published journal Biomaterials [13]. Surgical and bioengineering experts from the University of Pittsburgh in the United States designed a patch that can be placed in the damaged area of the heart muscle and can help the heart muscle recover, and conducted experiments on mice [43]. They found that this patch can make the heart muscle recover better [43]. After cardiac arrest (CA) and restoration of spontaneous circulation (ROSC) of Ca/ROSC, cardiac dysfunction occurs after resuscitation, but the damaged cardiac function can be completely recovered 48 h after ROSC. The reason may be related to the reversibility of myocardial injury and the gradual recovery of mitochondrial function and structure [18].

Spostc regulates HIF-1 $\alpha$/BNIP3 signaling pathway, promotes mitophagy, and alleviates hypoxia reoxygenation injury in cardiomyocytes. The main mechanism is that spostc can upregulate HIF-1 $\alpha$, further activate the downstream target gene BNIP3, promote BNIP3-mediated mitophagy, reduce LDH levels, inhibit cell apoptosis, and finally resist cell hypoxia reoxygenation injury by clearing autophagosomes and increasing cell viability [28]. In diabetes state, impaired HIF-1 can be activated by DFO pretreatment combined with spostc $\alpha$ and upregulate HIF-1 $\alpha$, and further promote HIF-1 $\alpha$/BNIP3-mediated mitophagy cleared damaged mitochondria in time, avoided the attack of mitochondria derived ROS on mitochondrial membrane potential, stabilized mitochondrial function, significantly reduced myocardial infarction area, and improved cardiac function [28]. Betulin inhibits the inflammatory pathway NF in ac16 cardiomyocytes by activating the STAT3 signaling pathway-$\kappa$ B and expression of inflammation-related genes [29]. The imbalance of mitochondrial division and fusion in the early stage after Ca/ROSC is involved in the pathological process of myocardial injury after resuscitation, and its mechanism may be related to the impairment of mitochondrial function [31]. Let-7i targets ccnd2 and E2F2, regulates the cell cycle of cardiomyocytes, regulates the proliferation of cardiomyocytes, and affects the recovery of cardiac function after myocardial infarction [34]. Inhibiting the expression of microRNA-153-3p can reduce the damage to cardiomyocyte mitochondrial structure in adriamycin-induced heart failure mice and promote the recovery of mitochondrial function in damaged cardiomyocytes [37].

The increased sensitivity of myocardial I/R injury in chronic renal insufficiency is related to the downregulation of APN/AdipoR1/AMPK signaling pathway and the increase of myocardial oxidative/nitration stress; exogenous adiponectin supplementation has a protective effect on myocardium with renal insufficiency [38].

The improved scheme of hyperbaric oxygen (HBO) has a better effect on the injured myocardium of patients with CO poisoning [15].

In the clinical treatment of viral myocarditis in children, the combined application of vitamin C and immunoglobulin can effectively reduce myocardial injury and inflammatory reaction, inhibit myocardial remodeling, and improve the treatment effect [39]. High-dose vitamin C is safe and convenient to treat HIE with myocardial damage, improve the clinical efficacy of HIE, promote its clinical recovery, and protect the damaged myocardium and promote its recovery [81].

The treatment of STEMI patients with half-dose urokinase before PCI can effectively improve the blood flow grade of the infarcted artery, promote the recovery of cardiac function, reduce the level of serum ALD and ICAM-1, reduce the myocardial inflammatory reaction and the degree of cardiac function damage, reduce the risk of adverse cardiovascular events, and improve the prognosis of patients [49]. The microvascular blood flow perfusion of stunned myocardium decreases, the microvascular endothelial function is damaged, and the vascular endothelial-dependent relaxation function is weakened [91]. Nitroglycerin has a delayed protective effect on the microcirculation and microvascular endothelial function of a stunned myocardium [91]. Reperfusion is the most effective method to treat acute myocardial infarction, early drug thrombolysis, interventional therapy, and

other methods are used to rebuild the blood supply of the ischemic myocardium [50]. However, the blood supply of the restored perfusion makes the myocardial tissue suffer secondary damage, i.e., myocardial ischemia-reperfusion injury. Experimental studies in recent years have shown that traditional Chinese medicine can slow down myocardial injury caused by reperfusion through drug pretreatment: by clearing excess oxygen free radicals, regulating the cell membrane pump activity of ATP, and improving the energy metabolism disorder of myocardium after reperfusion; by regulating the expression of apoptotic or inflammatory proteins of cardiomyocytes, it can inhibit apoptosis or inflammatory reaction and rescue dying cardiomyocytes; by inhibiting the expression of myocardial autophagy and regulating the expression of related gap junction proteins, it can reduce ventricular remodeling and achieve the purpose of protecting damaged myocardium [50]. The left ventricular systolic and diastolic functions of patients with acute viral myocarditis were damaged to varying degrees, and gradually recovered after treatment. Compared between the two groups, Shenmai injection has an obvious effect [96]. The increase of LVEDd and the decrease of LVEF are independent risk factors for post-infarction arrhythmia in patients with myocardial infarction in the recovery period. The severity of vascular disease affects the occurrence of arrhythmia after infarction by affecting cardiac function; in patients with ventricular arrhythmia after myocardial infarction, heart rate variability and left ventricular function were significantly decreased, and the incidence of early repolarization and fragmented QRS wave was significantly increased, moreover, the degree of impairment of heart rate variability and left ventricular function was related to the severity of ventricular arrhythmia after myocardial infarction; the ventricular arrhythmias in patients with anterior wall myocardial infarction tend to occur in the anterior part of the lateral wall and the apex of the heart, while those in patients with inferior wall myocardial infarction tend to occur in the lower part of the septum, which may be related to multiple blood supply and higher Purkinje fiber density in these parts [24]. In the state of MI, hypoxia can activate cellular ers, PBA can inhibit ers and its mediated inflammation and oxidative stress after MI, inhibit apoptosis, improve the survival rate of transplanted BMSCs, and improve the effect of BMSCs on promoting neovascularization, inhibiting myocardial fibrosis and improving cardiac function, cell experiments showed that PBA could inhibit ers activation of BMSCs under H/SD conditions, alleviate inflammation and oxidative stress, and improve the survival rate of BMSCs under H/SD conditions; the melatonin sustained-release cardiac patch prepared in this subject can stably release melatonin. The in vitro experiments show that the patch can reduce the inflammation and oxidative stress of BMSCs under H/SD conditions and improve the survival rate of BMSCs under hypoxia. Animal experiments have confirmed that melatonin sustained-release patch as a carrier of BMSCs can improve its survival rate and promote the recovery of cardiac function after MI [33]. Ischemic postconditioning (IPO) can effectively improve cardiac function; both immunohistochemical SABC method and immunofluorescence double labeling staining showed that IPO could induce the expression of cell proliferation specific indicators Ki67 and h3p, and the dividing cardiomyocytes were observed; IPO induced the expression of cyclina2 gene, which remained silent in adulthood, and then increased the expression of other cyclins and CDKs; IPO can initiate the expression of cardiac tissue related cyclins and CDKs, which may compensate or repair damaged cardiac tissue through the proliferation of cardiomyocytes [26]. The treatment of acute STEMI with sodium nitroprusside via coronary artery microcatheter can significantly improve the patient's symptoms, reduce myocardial damage, adjust the cardiac function to return to normal state, and reduce the occurrence of adverse events [32]. In patients with acute RVMI, even if the RCA trunk is successfully revascularized, the involvement of right ventricular branches is still closely related to hemodynamic changes such as hypotension and bradyarrhythmia with early impaired right heart function Strengthening the attention and protection of right ventricular branches during revascularization may help to stabilize hemodynamics in the acute phase and promote the recovery of right ventricular function [41].

The cardiac rehabilitation strategy of high-intensity intermittent exercise has high clinical application value in improving the long-term prognosis of patients with mild impairment of left ventricular function after PCI. The recovery effect of various physical indications of patients is good, which promotes the improvement of quality of life and is worthy of clinical promotion [45].

Betaloc combined with spironolactone in the treatment of heart failure in hypertrophic cardiomyopathy can significantly improve the patient's symptoms and recover the damaged cardiac function [46].

It is suggested that bone marrow mesenchymal stem cells (BMSCs) can participate in the reconstruction of damaged myocardium, improve left ventricular ejection fraction, reduce left ventricular end systolic and end diastolic volume, reduce infarct area, and improve left ventricular remodeling [1]. However, the long-term survival of BMSCs in infarcted myocardium has not been resolved; the change of microenvironment after myocardial infarction limits the repair ability of stem cells [68]. People are using BMSCs genetic engineering to solve this problem. Genetic engineering is the introduction of exogenous genes into target cells so that the cells have the ability to overexpress or silently express a certain gene [68].

"Myocardial No.1" can attenuate the inflammatory reaction, reduce the scope of myocardial fibrosis, enhance chemotaxis, promote the colonization of stem cells in the

damaged myocardium without affecting the formation of myocardial neovascularization, and finally regulate the inflammatory reaction after myocardial injury at a reasonable level conducive to recovery [76].

Adenosine preconditioning and ischemic preconditioning have similar cardioprotective effects on I/R myocardium Myocardial 99Tcm MIBI hemodynamic changes are closely related to myocardial viability and can be used as an effective index to evaluate myocardial adenosine preconditioning and its mechanism [80].

## MONITORING OF MYOCARDIAL FUNCTION

The myocardial perfusion level after PPCI in patients with AMI is related to myocardial systolic function, which can predict the recovery of myocardial systolic function [8].

Myocardial perfusion is closely related to myocardial systolic function; the application of MCE to evaluate the myocardial perfusion of patients before delayed PCI has predictive value for the recovery of myocardial systolic function after PCI [9].

The peak time of myocardial damage is 12–24 h after a crush injury. Ultrasonic strain rate imaging can accurately and objectively evaluate myocardial contractility [14].

For the treatment of patients with severe myocardial damage caused by acute CO poisoning, it is necessary to actively prevent and protect the cardiac damage caused by it, timely monitor the myocardial enzyme and ECG, and try to improve the cure rate and reduce the mortality [16].

Patients with COVID-19 often suffer from respiratory distress due to lung involvement, but acute cardiac insufficiency can also cause acute respiratory distress; the mechanism may be due to dysfunction of the immune response and damage of myocardial function caused by a cytokine storm. While paying attention to the lung injury of patients, we should consider the possibility of other organ damage; the improvement of myocardial enzyme, BNP, cardiac color ultrasound, and other examinations is helpful for differential diagnosis [52].

It is easy to misdiagnose and delay the treatment of preexcitation cardiomyopathy; the disease occurs in the right-side bypass [57]. The incidence of the right anterior wall bypass is the highest, and the composition is mostly in the right side wall and the right anterior wall after successful catheter ablation; the left ventricular function can be reversed and restored to normal [57]. The more severe the degree of LVEF damage, the longer the time for the recovery of cardiac function [57].

The change of the inverted T wave in the precordial lead is closely related to the improvement of RVD in patients with ape; the improvement of RVD can be predicted by the normalization of the inverted T wave in the precordial lead [64].

## CONCLUSION

A series of intracellular and extracellular miRNA changes are related to cardiovascular diseases such as myocardial infarction (MI), myocardial hypertrophy, cardiomyopathy, and arrhythmia [104]. Among them, MI can lead to insufficient blood supply and oxidative stress and then cause myocardial tissue necrosis, myocardial inflammatory reaction, pathological remodeling, and left heart dysfunction [102]. Although thrombolysis or surgical treatment can restore the blood supply in the ischemic area of MI, it can cause additional damage to the damaged myocardium, that is, myocardial ischemia-reperfusion injury (IRI) Myocardial remodeling induced by IRI includes cardiomyocyte death (necrosis and apoptosis), myocardial hypertrophy, angiogenesis, myocardial fibrosis, cardiac dysfunction, and so on. miRNAs play a specific role in the pathological process of myocardial remodeling induced by IRI This article reviews the role of miRNAs in myocardial cell death, myocardial inflammatory response, myocardial fibrosis, cardiac contractile dysfunction, and angiogenesis induced by ischemia-reperfusion (I / R) [102].

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