Research of heart failure based on heart model and S1 complexity

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***Abstract*—To study the internal and external charac- -teristics of heart failure, a lumped-parameter cardiovascular simulation model is designed to analyze the internal characteristics, and a method is proposed that uses the complexity of the first heart sound (S1) amplitude sequence to analyze the external characteristics. State variable method is used to establish the mathematical expression of the cardiovascular model which includes three sub-models: systemic circulation sub-model, pulmonary circulation sub-model and heart sub-model. Based on this model the heart physiological feature is studied, especially under heart failure. Then, the complexity of S1 amplitude sequence is analyzed based on multiscale base-scale entropy algorithm. Simulation experiment shows that, one of the internal characteristics of heart failure is the reduction of myocardial elasticity coefficient, and one of the external characteristics is the reduction of the complexity of S1 amplitude sequence. Simulation result being in accord with clinical data indicates that the proposed approach has feasibility and practicability in heart failure diagnosis.**

Keywords—cardiovascular system model; heart sound; heart failure; base-scale entropy

I. INTRODUCTION

In recent years, the heart failure incidence has shown a gradual increasing trend and has been a main reason to cause death. The prevention and cure of heart failure is a hot spot in the field of international cardiovascular research. Study the mechanism of heart failure is conducive to its accurate diagnosis, prevention and treatment. Scholars have been using a variety of models to study the characteristics of the heart. In 2002, according to the theory of electric network and pulse intermittent method, Hu and other scholars established some model about the cardiac, pulmonary and systemic system to simulate the function of heart [1-3].

Heart sound is the external reflection of cardiovascular system and mechanical vibration of heart [4]. Hansen makes a study of S1 amplitude and myocardial contractility, of which the results prove that the change of S1 amplitude and the change of maximum rate of left ventricular pressure rising are positively correlated relationship[5]. Other scholars have proved that there is obvious relationship between the heart sound and cardiac contractility[6-9]. Therefore, we do some research about the change rule of the complexity of S1 amplitude sequence based on base-scale entropy, and get an important feature of heart failure. In this paper, we indicates that one of the inherent characteristics of heart failure is cardiac elasticity reducing, and one of the external characteristics is the complexity of S1 amplitude sequence reducing.

II. MODEL OF CARDIOVASCULAR SYSTEM AND SIMULATION EXPERIMENT

A. Model of cardiovascular system

The cardiovascular system is a "closed" pipeline system, which is mainly composed of heart and blood vessels. It is divided into two parts, the heart system and the vascular system. The vascular system is subdivided into systemic and pulmonary circulation. The heart can be divided into four chambers, which are left atrium, left ventricle, right atrium and right ventricle. The atrial septum divides left atrium and right atrium, and the ventricular septum divides left ventricle and right ventricle. The cardiovascular system model diagram is shown in Fig. 1.

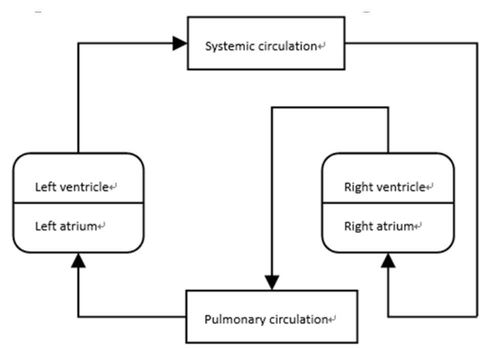

Fig. 1. The diagram model of cardiovascular system

Three basic principles to build the heart model: 1) Consistency principle: parameters or waveform obtained from the heart model should as far as possible consistent witn the measured. 2) Interpretability principle: The simulation model can explain the mechanism of cardiovascular and physiological mechanisms of heart sounds, and can be used to represent the state variables on cardiovascular physiology parameters, the physiological state of the heart can be observe by the state variable. 3) Controls principles: By changing one or more parameters of the model to simulate the relationship between changes in the various parameters and heart sounds and changes in

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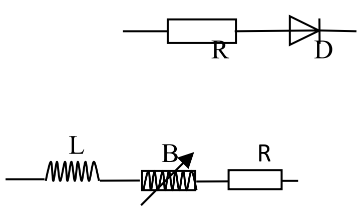
cardiovascular parameters of a healthy cardiovascular system or pathological situations, and research systems.

Cardiovascular system is a complex system, according to the cardiovascular dynamics principle and the related theory of fluid network; it is known that transmission equation in fluid and transmission equation in the grid have same mathematical form. And so are the equivalent network of fluid flow network and the equivalent circuit of power grid. So, the analogy could be gotten, between physiological parameters, fluid parameters and electrical parameters, as shown in table 1.

B. HEART SUB-MODEL

The main driving force of heart mainly comes from heart cyclical expansion and contraction movement. According to Olansen's five parts model: left ventricular free wall, right ventricular free wall, left atrial free wall, right atrial free wall and ventricular coupled wall, we can describe the cyclical expansion and contraction movement of the heart[10]. From paper [11], the role of the heart valve is to prevent the reversal of blood flow from artery to the heart or the reversal of blood flow from ventricle to atrium.

In order to comprehensively analyze the nonlinear characteristics of the valve and the relationship between blood flow and heart valve aperture, in this paper heart valve is simulated by three components: Bernoulli impedance (B), inertance of blood (L), and viscous resistance (R), as shown in Fig 2.



(a)Original model

(b) Modified model

Fig. 2. Model of heart valve

Then by this coupling of atrium and ventricle with modified heart valve, the modified heart sub model can be built up, as shown in Fig.3. This model includes atrium (ela, sla, era, sra), ventricle (elv, slv, erv, srv) and heart valve (Bmv, Lmv, Rmv, Bav, Lav, Rav, Btv, Ltv, Rtv, Bpv, Lpv, Rpv). Moreover, based on Sun's research, pericardial pressure (ppc) and thoracic internal pressure (pit) are taken into account [12].

C. SYSMTEMIC AND PULMONARY CIRCULATION MODEL

Referring to paper [11], this paper describes a systemic sub-model, which is formed by the coupling of the left heart system with the arterial system. In order to reflect the details of the systemic circulation, we take into account arterial compliance and peripheral resistance, as which has been shown in Figure 3. In this model, the time-varying elastance is used to simulate the left ventricular synchronous contraction. The artery system is represented by Cao, Lao, Rao, Cart and R1. The parameters Cao and Rao represent arterial compliance. Lao represents inertial effect of the artery blood. The parameter R1 represents peripheral resistance as show in Fig. 3.

D. CARDIOVASCULAR SYSTEM SIMULATIOM MODEL

Cardiovascular system simulation model includes the Heart sub-model and the Systemic and pulmonary circulation model. They are coupled based on the physiological characteristics of cardiovascular system, as shown in the Fig.3. Vascular system in systemic circulation has been translated into the specific aorta, arteries, capillaries, veins and inferior vena cava, so that they are closer to physiological structure of the vascular system.

III. SIMULATION EXPRERIMENTS

A. THE SIMULATION OF HEALTHY HEART

According to the parameter values shown in Fig.3, we can get a set of simulation results of heart under healthy conditions. Among them, Fig.4 shows the blood volume of ventricle and atrium, the blood flow through valve. Fig.5 shows the blood flow of systemic and pulmonary circulation. Fig.6 shows that the atrial and ventricular pressure changes over time.

Table 2 shows the simulation results including a set of characteristic parameter values. Where LVSP and LVDP means left ventricular systolic blood pressure and the left ventricular diastolic blood pressure respectively, AOSP and ADBP describes aortic systolic blood pressure and the aortic diastolic blood pressure , RVSP and RVDP demonstrates right ventricular systolic pressure and right ventricular diastolic pressure, PASP and PADP indicates pulmonary artery systolic pressure and pulmonary artery diastolic pressure.

In Fig. 3, it shows blood volume of ventricle and atrium, blood flow through valve. In Fig. 4, it is described blood flow of systemic and pulmonary circulation In Fig. 5, it is shown that left ventricle pressure and right atrium pressure change over time. The peak of plv curve represents LVSP, and it is 115.8 mmHg. The valley value means LVDP, and it is 4.28 mmHg. In Fig. 6, it shows the atrial pressure and ventricular pressure

B. THE SIMULATION OF HEART FAILURE

Reducing the myocardial elasticity could cause heart failure. Myocardial elasticity can be reflected by the elastance coefficient of ventricle and atrium in the sub-model, and the related coefficients include Elva, Elaa, Erva and Eraa. By setting the values of the above four parameters to 60% the original value, we can simulate cardiovascular model under heart failure which is caused by the decrease of myocardial elasticity. Setting the elastance in Fig.7 as one input of the entire cardiovascular model, we can get a set of simulation results under heart failure. Fig.8 shows left ventricle pressure and blood volume of left ventricle under heart failure and normal. Fig.9 shows the comparison of right ventricle blood pressures under normal and heart failure.

pressure decreases by 14.89%. Right ventricular systolic blood pressure decreases by 11.23%. Right ventricular diastolic blood pressure decreases by 82.86%. Cardiac output falls by 18.42%. Those parameters can reflect the clinical symptoms class I heart failure.

According to the simulation results, we can get the following conclusions: compared with the normal physiological condition, the left ventricular systolic blood pressure decreases by 24.17%. Left ventricular diastolic blood pressure decreased by 7.94%. The aortic systolic blood pressure decreases by 22.74%. The aortic diastolic blood pressure decreases by 14.89%. Right ventricular systolic blood pressure decreases by 11.23%. Right ventricular diastolic blood pressure decreases by 82.86%. Cardiac output falls by 18.42%. Those parameters can reflect the clinical symptoms class I heart failure.

TABLE I. THE ANALOGY OF THREE KINDS OF PARAMETERS

Physiological parameter	P (Blood pressure)	Q (Bloodstream)	V (Blood volume)	R (Bloodstream resistance)	L (Blood flow inertia)	C (Vascular compliance)
Liquid parameter	p (Press)	q (Flux)	v (Volume)	r (Glutinousness)	l (Inertia)	k (Elastic coefficient)
Electric parameter	U (Voltage)	I (Electric current)	Q (Electric charge)	R (Resistance)	L (Inductance)	C (Capacitance)

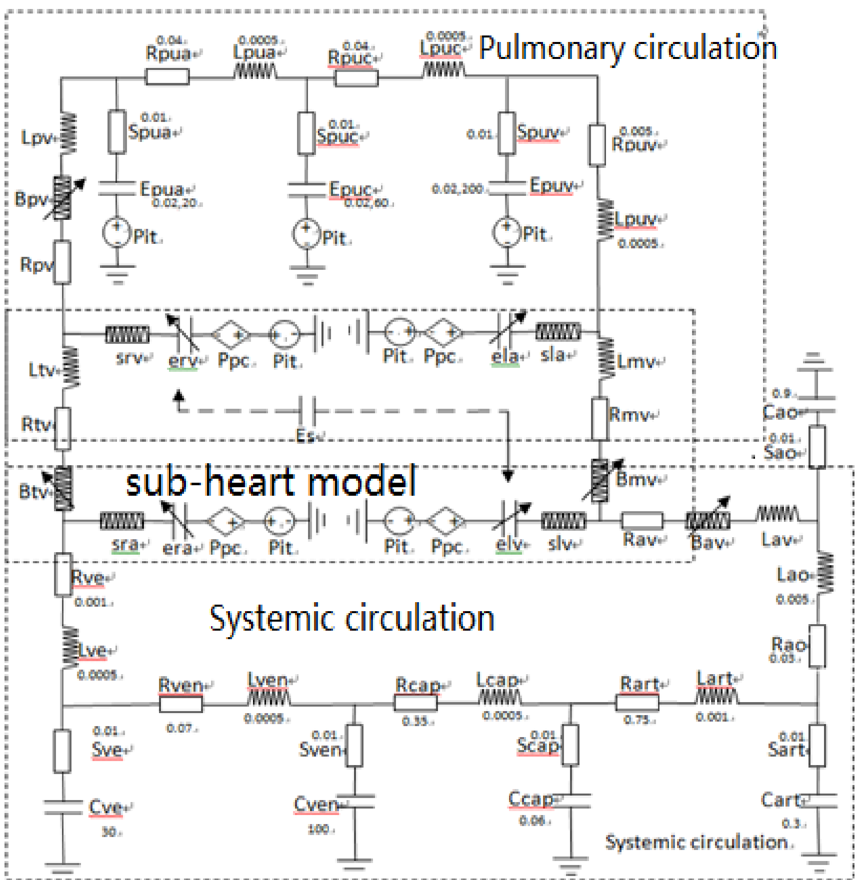


Fig. 3. Simulation model of the cardiovascular system

IV. S1 COMPLEXITY

A. HEART SOUND ACQUSITIO

In order to collect heart sounds for a long time, the shoulder belt heart sound sensor is adopted, which is invented by our research group. Reasonable structure makes it reliable in use. Heart sound can be collected by using it for a long time as shown in Fig.10. The device recommends a means of collecting heart sound. (PATENT NO: CN2013093000306700). The amplitude of S1 as shown in Fig.14.

Ventricular systole is the beginning of cardiac cycle. There are four different kinds of heart sounds in every cardiac cycle, generally, S1 and S2 can be heard[13]. Fig.6 reveals that S1 is mainly because of muscular contraction of left ventricle (elv, slv) and right ventricle (erv, srv), and the close of bicuspid valve （Bmv, Lmv, Rmv）and tricuspid valve (Btv, Ltv, Rtv). Besides, the vibration of the ventricular wall and the artery wall is also a part of S1, which is because of the open of aortic valve （Bav，Lav，Rav）and pulmonary valve (Bpv，Lpv，Rpv) and the ejection into the aorta （Cao, Sao, Lao, Rao）. The vibration frequency of S1 mainly ranges from 40～60Hz.

S1 amplitude has a close relationship to myocardial contractility, and the changes of myocardial contraction ability can be depicted by the fluctuation S1 amplitude [9].

The sequence can be obtained by extracting S1 amplitude, defined as x(i), $\{x(i): 1 \leq i \leq N\}$. The fluctuation diagram of the amplitude sequence of S1 is shown in Fig.11, and the horizontal axis shows the serial number of S1, and the vertical axis shows S1 amplitude. For clarity purposes, S1 amplitudes are shown only during 1000 cardiac cycles.

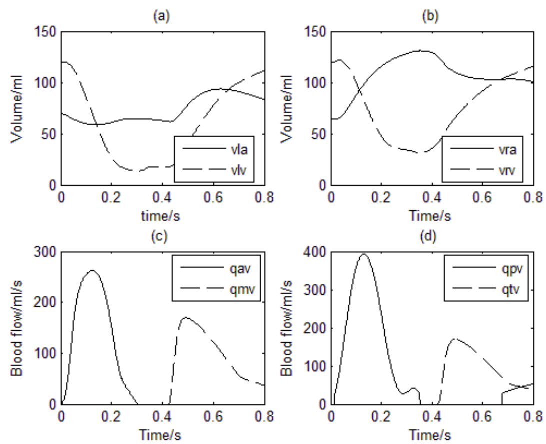


Fig. 4. Blood volume of ventricle and atrium, blood flow through valve

(a): Blood volume of left ventricle (vlv), blood volume of left atrium (vla). (b): Blood volume of right ventricle (vrv), blood volume ofright atrium (vra). (c): Blood flow of aortic valves (qav), blood flow of mitral valve (qmv). (d): Blood flow of pulmonary valve (qpv), blood flow of tricuspid valve (qtv).

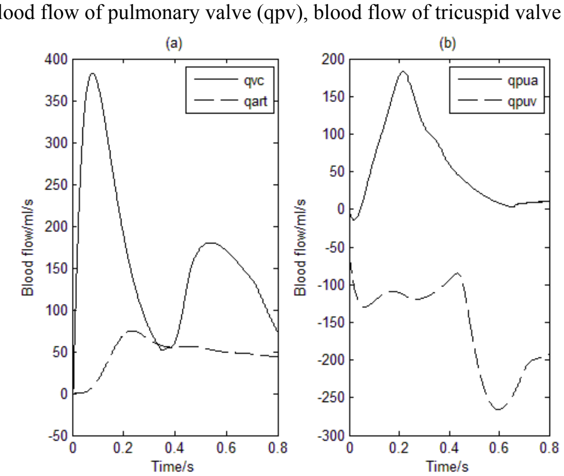


Fig. 5. Blood flow of systemic and pulmonary circulation

(a): Blood flow of vena cava (qvc), blood flow of artery (qart).
(b): Blood flow of pulmonary artery (qpua), blood flow of pulmonary vein (qpuv).

TABLE II. THE SIMULATION RESULTT

Characteristic parameters	simulation results	Normal value
LVSP	115.8mmHg	90-120mmHg
LVDP	4.28mmHg	0-10mmHg
AOSP	117mmHg	90-140mmHg
ADBP	64mmHg	60-90mmHg
RVSP	21mmHg	18-30mmHg
RVDP	2.1mmHg	-5-3mmHg
PASP	21mmHg	18-25mmHg
PADP	6.2mmHg	6-10mmHg
Cardiac output	5.695L/min	4.5-6L/min

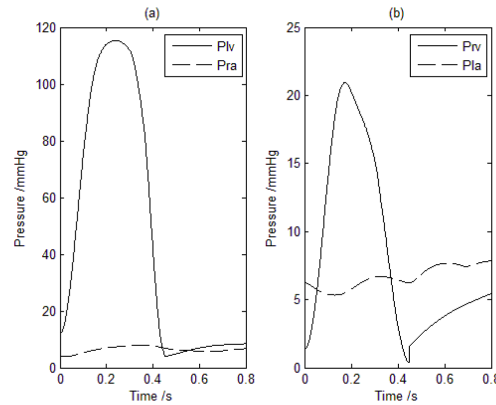
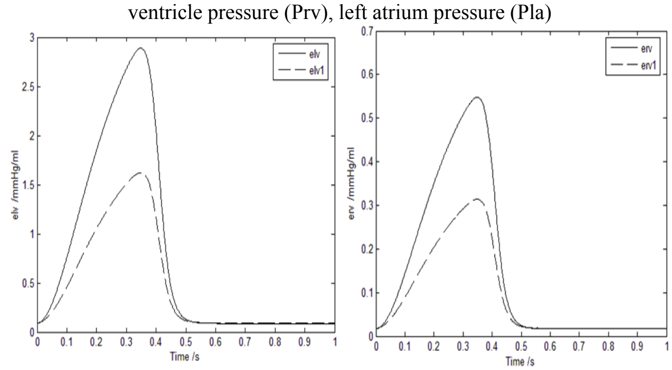


Fig. 6. The atrial pressure and ventricular pressure

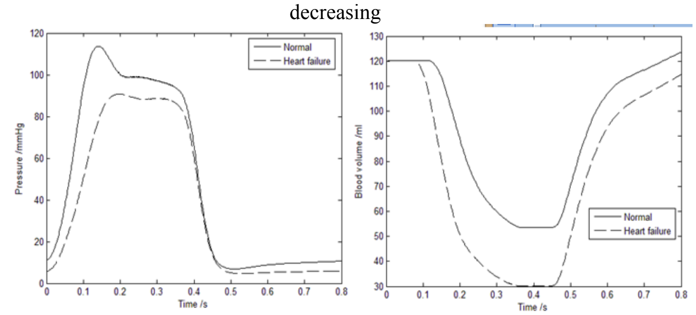
(a)Left ventricle pressure (Plv), right atrium pressure (Pra). (b)Right ventricle pressure (Prv), left atrium pressure (Pla)



(a) (b)

（a）Light ventricular elastance.（b）Right ventricular elastance

Fig. 7. Ventricular elastance under normal and myocardial elasticity decreasing



(a)Left ventricle pressure. (b) Blood volume of left ventricle

Fig. 8. Left ventricle pressure and blood volume of left ventricle under heart failure and normal

B. *MULTISCALE BASE-SCALE ENTROPY(MBE)*

The sequence x(i) is embeded into m-dimensional phase space,

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$$H(i)=\left[x(i),x(i+L),\cdots,x\left(i+(m-1)\cdot L\right)\right] \quad (1)$$

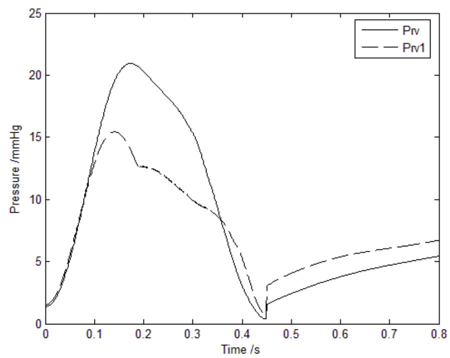

Fig. 9. The comparison of right ventricle blood pressures under normal and heart failure

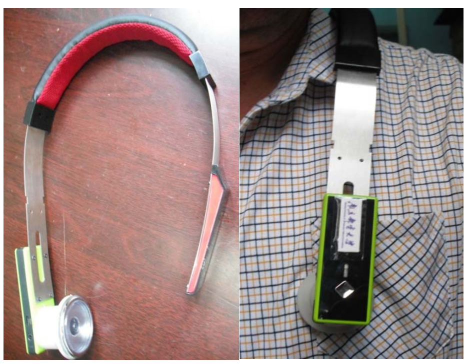

Fig. 10. Shoulder belt heart sound sensor

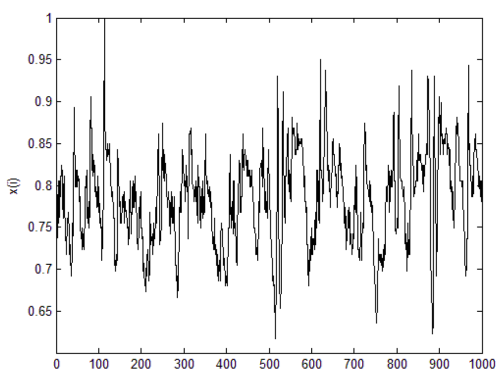

Fig. 11. Fluctuation diagram of the amplitude sequence of S1

Among it, m is embed dimension, L is delay time. Set L=1, and we have N-m+1 m-dimensional vector. For each H(i), define the basic scale is $RS(i)$, the mean is $u(i)$,

$$RS(i)=\sqrt{\frac{\sum_{j=1}^{m-1}\left[x(i+j)-x(i+j-1)\right]^2}{m-1}} \quad (2)$$

$$u(i)=\frac{\sum_{j=0}^{m-1}x(i+j)}{m}. \quad (3)$$

By means of the theory of symbolic dynamics [14], convert H(i) into m-dimensional vector symbol sequence: $S_i\big(H(i)\big)=[s(i),s(i+1),\dots,s(i+m-1)], s\epsilon\emptyset, \emptyset=\{1,2,3,4\}$,

$$s(i+j)=\begin{cases}1 & x(i+j)\le u(i)-\alpha\cdot RS(i)\\ 2 & u(i)-\alpha\cdot RS(i)<x(i+j)\le u(i)\\ 3 & u(i)<x(i+j)\le u(i)+\alpha\cdot RS(i)\\ 4 & u(i)+\alpha\cdot RS(i)<x(i+j)\end{cases} \quad (4)$$

Among them, i=1, 2, … , N-m+1; j=0, 1, … , m-1. 1, 2, 3, 4 represent a kind of symbol. α is scale parameter.

Count the number of times each symbolic vector occurs as NT(i), probability distribution as $f(i)$, so the base-scale entropy (BE) of $x(i)$ could be defined as follows [15]:

$$f(i)=\frac{NT(i)}{N-m+1} \qquad (i=1,2,\cdots,4^m) \quad (5)$$

$$BE=-\sum_{i=1}^{4^m} f(i)\cdot\log_2 f(i) \quad (6)$$

The uncertainty of symbolic vector can be quantified. The more symbol combination type, the bigger BE. From the definition of RS, it can be draw that RS can dynamically adapt to the change of $H(i)$. Our main purpose is to extract waveform vibration mode information, without thinking about amplitude information. This symbolic process is decided by $RS(i)$ and $u(i)$.

For the sequence $\{x(i),\ 1\le i\le n\}$ and time scale factor γ, introduce the multi-scale formula [16]:

$$y(j)_\gamma=\frac{1}{\gamma}\sum_{i=(j-1)\gamma+1}^{j\gamma}x(i) \quad \left(1\le j\le\frac{n}{\gamma}\right) \quad (7)$$

For different values of γ, calculate BE of different $y(j)_\gamma$. We can get the curve of BE changing over γ.

C. COMPLEXITY ANALYSIS OF S1AMPLITUDE SEQUENCE

The algorithm of MBE is applied on the heart sound signals, which include 30 healthy elders, 30 healthy young and 30 patients with heart failure. Concrete steps are: (a), Extract S1 amplitude sequence $x(i)$ based on the method introduced in section 3.1; (b), Dispose $x(i)$ based on formula (20), γ=1,2,…,15; (c), Set m=4, and calculate BE of each $y(j)_\gamma$.

For each scale factor γ, calculate the BE mean of three kinds of people. The mean entropy changing over scale factor can be obtained as shown in Fig.12.

From Fig. 12, it can be found that BE of patients with heart failure is significantly smaller than healthy elders, and BE of healthy elders is smaller than healthy young. We hold that S1 amplitude sequence signal of healthy young has more indeterminacy and great random characteristic. The myocardium of healthy young has abundant activities.

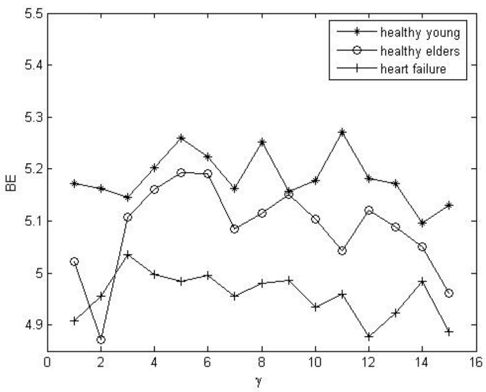

Fig. 12. The BE mean of three kinds of people

According to the theory of symbolic dynamics, the small BE show that vibration behaviors of this original dynamic process concentrate, and the big BE show that the vibration behaviors have more various vibration modes. Moreover, we can draw that ageing makes myocardial contraction function diminishes, and the effect of heart failure on myocardial contraction function is much stronger than factor of aging. The conclusion of the study is in line with the reality.

Set the mean of healthy elders and healthy young as the BE of healthy. When γ is equal to 5, the distinction is the biggest. At this point, BE of healthy is 5.2269, and BE of heart failure is 4.9848. The BE of S1 amplitude sequence of heart failure decreases by 4.6% than healthy.

V. Conclusion

According to the lumped-parameter cardiovascular model, when myocardial elastic decreases, the characteristics of heart are simulated by decreasing the elastance coefficient of ventricle and atrium in the sub-model. By setting the values of the four parameters Elva, Elaa, Erva, Eraa to 60% the original value, the simulation data correspond with the clinical data of heart failure. It indicates one of internal characteristics of heart failure is the decrease of myocardial elastic. When it is less than 60% of healthy heart, heart failure would occur.

Heart sound is one of the external manifestation of cardiac activity, contains lots of physiological information and pathological information[17]. The S1 amplitude has a relationship with cardiac contractility. The analysis of S1 amplitude is based on multiscale base-scale entropy. Experimental results show that the BE of S1 amplitude sequence of heart failure decreases by 4.6% than healthy.

Heart failure is studied from both inherent and external characteristic, which has never been done before. It is conducive to gaining an overall and deep understanding of heart failure, and provides a new method in heart failure diagnosis. Compare to the animal model of heart in the traditional researches of heart failure. The cardiovascular model in this paper can decrease the cost of experiment, and the experiment could be repeated. Moreover, it plays a guide role on animal experiments. Heart sounds can be collected for a long time without holding for the first time. The research of long heart sound signals is of great importance to make the best of heart sound and dig the great potential of heart sound.

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