Research Article

Prediction of Heart Failure in Children with Congenital Heart Disease Based on Multichannel LSTM

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Heart failure (HF) is a complicated clinical illness caused by a variety of primary and secondary causes, as well as increased infection pathways, that are associated with higher risk, illness, and costs. The overall incidence of congenital heart disease is approximately high and is the leading cause of death in infants and children. In this study, we present a novel computational model based on ECNN-LSTM for detecting congenital heart disease in real time and assessing its developing course objectively. The proposed model is a multiresolution singular value decomposition for congenital heart disease prediction in infants and children. Firstly, in the whole-life vibration time-domain signal of newborns and toddlers, the multiresolution singular value decomposition approach is employed to create approximation and detailed signals with different resolutions. Secondly, the health phases are split into the smooth running stage of newborns and toddlers, as well as the standard deviation of the earliest moments. Thirdly, the two-layer one-dimensional convolutional neural network structure divides the quick degradation stage, which can offer degradation information. Finally, the prediction of congenital heart disease is finished on LSTM utilizing the MSE loss function to unify the assessment scale. Validation with lifespan data demonstrates the feasibility and effectiveness of the proposed model. Moreover, the existing models have the insufficient ability for life span feature characterization in congenital heart disease prediction in infants and children.

1. Introduction

Congenital heart disease (CHD) is a structural or functional abnormality of the heart or blood arteries that is evident at birth [1]. Cardiac insufficiency, cyanosis of body endings, and poor development are the primary outcomes of CHD in children. CHDs are a major public health concern that affects human health [2]. They are complicated and varied and have catastrophic implications, typically leading to miscarriage, stillbirth, and neonatal death or impairment after delivery. Congenital heart disease was reported to be present in approximately 0.6 per 1000 live births worldwide from 1930 to 1934, and by 1995 onwards, it was present in approximately 9.1 per 1000 live births, accounting for nearly one-third of infant deaths related to congenital malformations [3]. According to World Health Organization (WHO) statistics, from 2014, roughly 1.5 million infants worldwide are born with congenital cardiac abnormalities. According to the Chinese Birth Defects Surveillance System findings, the incidence of CHD has increased to the first position in the birth defect parity since 2004 [4]. Patients with CHD are prone to serious complications such as heart failure, endocarditis, and pulmonary infections [5]. CHD can have a significant impact on patients’ physical and mental health, as well as the growth and development levels of the afflicted youngsters. Children with CHD frequently require long-term specialized medical care after birth, and health-care expenses are substantial, with the entire economic burden being significant. In China, the life cycle of new CHD reaches 12.6 billion yuan each year [6], and there are considerable geographical disparities in CHD incidence based on the manner of registration and means of diagnosis, as well as genetic, environmental, and socioeconomic variables.

In recent years, great strides have been achieved in the diagnosis and treatment of CHD, but because of the significant disease burden associated with premature heart disease, domestic and foreign researchers have increasingly turned their research focus to examining the genesis of CHD. Zhao
et al. performed neonatal screening for CHD and showed that the prevalence of CHD in children in Shanghai was 26.6% and showed that the prevalence of CHD in infants aged 0-1 year was 8.15% in Guangdong Province [7, 8]. The incidence of perinatal CHD in Gansu Province increased year by year from 12.66 per million in 2005 to 17.38 per million in 2010 [9, 10], and the ranking increased from third in 2005 to first in 2010. Currently, most of the findings suggest that CHD is caused by a combination of genetic and environmental factors [11]. Genetic factors play a major role in the pathogenesis of CHD [12], mainly including monogenic genetic defects [13], polygenic genetic defects [14], and chromosomal aberrations and translocations [7]. In a study of 36716 infants surviving from 2006 to 2008 in six counties (cities) of Shanxi Province, Perotin et al. and Ling et al. found that the risk of CHD in offspring of mothers with heart disease was 8.578 times higher than that of mothers without heart disease [6, 15]. By analyzing the ABO blood groups of 278 children with CHD, [16] found that the occurrence of precocious heart disease was associated with ABO blood group antigens, and the incidence of CHD was higher in children with blood groups A and AB containing A antigens than B and O. Environmental factors are mainly related to maternal intrauterine infections during pregnancy, smoking and alcohol consumption, exposure to specific substances before and during pregnancy, medications taken during pregnancy, illnesses during pregnancy, and nutrition [17, 18].

In this paper, a reliable and optimum classification strategy for the diagnosis of congenital heart disease in newborns and children is suggested, as well as a unique way to identify differentiable frequency component properties. The prevalence of CHD in children has been linked to maternal rubella virus infection, febrile illness or influenza during pregnancy, and medicines used during early pregnancy. The proposed study has shown that maternal use of progesterone hormones during pregnancy increases the risk of fatal cardiovascular defects as well as ventricular septal defects. Folic acid is a necessary ingredient for fetal growth and development, and if the mother is insufficient in the first trimester, problems in fetal neural tube development and abnormalities may occur. The first-line screening method for the identification of congenital cardiac disease is echocardiography, which is the most often used noninvasive test. Adult cardiac function is something that diagnostic echocardiographers are familiar with. When they apply the approach for measuring adult cardiac function to infants with congenital heart disease, however, they discover that the results of cardiac function evaluation using this method are inconsistent with clinical practice. Children’s heart function is assessed differently from adult cardiac function.

2. Related Work

A retrospective case-control study abroad showed that maternal supplementation with folic acid, multivitamins, and other nutrients in early pregnancy reduced the incidence of common trunk defects and limb malformations. In addition, foreign studies have reported that multiple pregnancies increase the risk of CHD; domestic studies have shown that maternal multiple births, passive smoking in early pregnancy, exposure to chemical reagents, living in an apartment renovated for less than 6 months during pregnancy, and living <50 m from the nearest major traffic artery are risk factors for CHD in the left-to-right shunt category. Studies of smoking and precordial disease have found that the first trimester of pregnancy increases the risk of fetal septal defects and that fetuses with >25 cigarettes per day have an increased risk of right-sided obstructive malformations [1].

Liu et al. observed a 6.8% incidence of congenital abnormalities, including 55 occurrences of CHD, in a prospective cohort analysis of 2580 HIV-uninfected US infants who were exposed to antiretroviral medications in early pregnancy. The mechanism of aberrant birthing history’s influence on CHD may be connected to changes in the intrauterine environment, as well as the psychological impact of abnormal childbirth history on a pregnant woman’s capacity to conceive again. According to a case-control research conducted in China, a mentally stimulating incident during pregnancy elevated the incidence of CHD in the child by 2.93 times. Epidemiological findings suggest that a high consumption of meat, eggs, soy, and dairy foods during pregnancy is a protective factor for the development of childhood precocious heart disease [19]. According to the literature, about 30% of CHDs can be prevented by changes in maternal behavioral patterns and adverse environmental factors [17]. Even if children with CHD are born with normal weight, their weight to height ratio decreases rapidly with age. The effect of several variables before, during, and after surgery can induce delayed development in children with CHD. It could be due to the child’s low body circulation volume; increased pulmonary circulation, which is prone to recurrent pulmonary infections; and insufficient nutritional intake due to feeding difficulties, all of which affect the child’s overall growth and development, as well as increased sympathetic excitability and increased energy expenditure. As a result, we assessed the growth and development of the children who satisfied the study’s inclusion and exclusion criteria, and suggested preventative interventions to enhance the children’s prognosis and quality of life [20].

Gansu Province’s geography is mostly hilly and plateau, with 86 counties (districts) and a population of 25,821,800 people. The province has 86 counties (districts) with a total population of 25,821,800 people, 52.57 percent of whom live in cities, indicating a low level of urbanization and a disparity between the province’s ecological environment, economy, society, humanities, science, and education and developed regions. The largest cardiac macro vascular surgery center in Gansu Region is the cardiovascular surgery department at Lanzhou University’s First Hospital, which handles the highest number of patients with congenital heart disease in the province each year. Similarly, a case-control research was conducted at Lanzhou University Hospital from August 26, 2017, to July 11, 2018. A questionnaire survey and analysis of the probable influencing variables of juvenile congenital heart disease were conducted to investigate the impact of CHD in parents before and during pregnancy and to offer a
scientific reference for the prevention of CHD and the development of relevant therapies [21], as well as to give a reference for preconception and prenatal health care.

3. Methodology

3.1. Heart Failure Causes. Physiologically, heart failure is a syndrome caused by cardiac insufficiency that occurs at rest or during stress and is characterized by inadequate pulmonary and/or systemic venous congestion and/or peripheral oxygenation. Heart failure is a progressive clinical syndrome of multiple etiologies with characteristic clinical signs and symptoms, a specific manifestation of pathophysiological abnormalities caused by complex interactions between the circulatory system, neurohormonal, and molecular abnormalities. Its etiology and clinical manifestations may vary considerably between children and adults, as well as among children of different ages. Cardiac insufficiency occurs in approximately 5% of patients with congenital heart disease in childhood, 10% to 20% after Fontan surgery, and up to 50% of patients with congenital heart disease in adults after Fontan surgery. Timely recognition and rational treatment are important to obtain a good prognosis [10, 11, 22].

The familiar New York Heart Association (NYHA) Heart Failure Classification does not apply to all children, and the behavioral model used to evaluate and adults cannot be used to measure infants and children who do not speak and do not exercise outdoors. It was modified to apply to children of all ages. The modified Ross classification incorporates symptoms of feeding difficulties, growth restriction, and exercise intolerance into a rating system comparable to the adult NYHA classification. The guidelines issued by the Cardiovascular Group of the Chinese Medical Association’s Pediatrics Branch in 2006 stated that school-age children can be referred to the NYHA grading scale and infants and children with heart failure to the modified Ross grading scale. The modified Ross classification assessment consists of a history and physical examination, in which the history is asked for any abnormal sweating and tachypnea, and the physical examination includes the presence of signs of dyspnea, breaths per minute, heart rate, and signs of hepatomegaly. In addition to questioning and physical examination, chest X-ray and echocardiography are important diagnostic aids [23]. Chest X-ray can show enlarged heart shadow, pulmonary stasis, and pulmonary edema, while echocardiography can help diagnose the cause and measure the systolic and diastolic function of the heart.

3.2. Multiresolution Singular Value Decomposition. In rotating machinery life prediction, the singularity signal is usually more important than the steady signal. The unique value of the signal might indicate the fault’s impact, oscillation, rapid change in speed, or structural deformation and fracture. As a result, this paper uses a multiresolution singular value decomposition technique for data preprocessing. The singularity signal is different from the signal obtained by singular value decomposition. A singular value signal or its derivative of some order changes abruptly at a certain point, causing the signal to have singularity at that point.

In the singular value decomposition process, the principle of SVD is that for a matrix signal \( \mathbf{H} \in \mathbb{R}^{m \times n} \), there must be an orthogonal matrix \( \mathbf{U} = (u_1, u_2, \ldots, u_m) \in \mathbb{R}^{m \times m} \) and an orthogonal matrix \( \mathbf{V} = (v_1, v_2, \ldots, v_n) \in \mathbb{R}^{n \times n} \), so that equation (1) holds:

\[
\mathbf{H} = \mathbf{USV}^T, \tag{1}
\]

where

\[
\mathbf{S} = \begin{cases} 
  (\text{diag} (\delta_1, \delta_2, \ldots, \delta_q), 0) & m \leq n, \\
  (\text{diag} (\delta_1, \delta_2, \ldots, \delta_q)^T, 0) & m > n,
\end{cases}
\tag{2}
\]

\( \mathbf{S} \in \mathbb{R}^{m \times n}, 0, 0 \) representing zero matrix, \( q = \min (m, n) \), and \( \delta_1 \geq \delta_2 \geq \ldots \geq \delta_q > 0, (i = 1, 2, \ldots, q); \) it is called the singular value of matrix \( \mathbf{H} \).

The purpose of multiresolution singular value decomposition is to obtain the approximate and detail signals of the \( j \)th decomposition of SVD. Therefore, equation (3) is expressed as follows by column vectors \( u_j \) and \( v_j \):

\[
\mathbf{H}_j = \delta_{u_j} \mathbf{u}_j \mathbf{v}_j^T + \delta_{v_j} \mathbf{u}_j \mathbf{v}_j^T, \tag{3}
\]

where \( \mathbf{u}_j \in \mathbb{R}^{q \times 1}, \mathbf{v}_j \in \mathbb{R}^{(N-1) \times 1}, i = 1, 2 \). Let \( \mathbf{H}_j = \delta_{u_j} \mathbf{u}_j \mathbf{v}_j^T \), then \( \mathbf{H}_j \in \mathbb{R}^{q \times (N-1)} \), which corresponds to a large singular value and reflects the main component of the signal, which is called an approximate matrix; \( \mathbf{H}_j = \delta_{v_j} \mathbf{u}_j \mathbf{v}_j^T \), then \( \mathbf{H}_j \in \mathbb{R}^{(N-1) \times (N-1)} \), which corresponds to the small singular value and reflects the detail component of the signal, which is called the detail matrix.

For any signal \( f(t) \in L^2 (\mathbb{R}) \), it can be decomposed into a series of approximate signals \( v_j \) and detail signals \( D_j \) by using the bisection recursive SVD decomposition algorithm. Let the subspace \( v_j \) belongs to \( s_j \), and the subspace \( D_j \) belongs to \( w_j \). The approximate signal is essentially obtained by using the approximate basis vector \( v_{j1} \), which also constitutes the base of subspace \( s_j \). Similarly, the detail basis vector with \( v_{j2} \) as \( D_j \) constitutes the base of subspace \( w_j \).

In the process of multiresolution singular value decomposition, the approximate basis vector and detail basis vector of the next layer are mainly obtained around the decomposition of the previous layer, and the relationship is as follows:

\[
\begin{align*}
\mathbf{v}_{j1}^T \mathbf{v}_{j2} &= 0, \\
\mathbf{v}_{j1}^T \mathbf{v}_{j1} &= 1, \tag{4}
\mathbf{v}_{j2}^T \mathbf{v}_{j2} &= 1.
\end{align*}
\]

As can be seen from equation (4), the approximate basis vector \( \mathbf{v}_{j1} \) and the detail basis vector \( \mathbf{v}_{j2} \) are orthogonal, so their subordinate subspaces \( w_j \) and \( S_j \) are also...
orthogonal, and the subspace $w_j$ is the orthogonal complement of $S_j$ in space $S_{j-1}$, that is,

$$
\begin{align*}
&\{w_j \perp S_{j-1}, \\
&w_j \oplus S_j = S_{j-1}, j \in \mathbb{Z}.
\end{align*}
$$

(5)

Equation (5) reveals the essence of bisection SVD; that is, a large space is decomposed into two orthogonal subspaces and direct sum. For any $S_{j-1}, j \in \mathbb{Z}$, then

$$
\bigoplus_{j=1}^{\infty} w_j = S_{j-1}, j \in \mathbb{Z}.
$$

(6)

Equations (5) and (6) show that SVD detail space $\{w_j | j \in \mathbb{Z}\}$ forms an orthogonal direct sum decomposition of space $L^2(\mathbb{R})$, which proves that the SVD decomposition process constitutes a multiresolution analysis, spaces $\{S_j | j \in \mathbb{Z}\}$ and $\{w_j | j \in \mathbb{Z}\}$.

3.3. Efficient Channel Attention Mechanism. The channel attention mechanism has been shown to offer a lot of potential for boosting deep convolutional neural network performance (CNNs). To enhance the gain, its representative approach senet learns the channel attention of each convolution block through two phases of squeeze and exception; however, this technique also increases the model complexity and computing cost. To capture local cross-channel interaction information, ECA primarily addresses the development of each channel and its $K$ neighboring points after convolution. It can alter the size of the convolution kernel adaptively and regulate the interactive coverage well. It can better complete the characterization of degradation index features when used with CNN to construct ECANM.

The aggregation feature $y \in \mathbb{R}^C$ without dimensionality reduction is given to learn channel attention

$$
\omega = \sigma(Wy),
$$

(7)

where $W$ is a $3 \times 3$ parameter matrix, processing $\omega$ column vector representation of

$$
\omega = \sigma \left( \sum_{j=1}^{K} w_j y_j \right), y_j \in \Omega_j.
$$

(8)

This strategy can be easily implemented by fast one-dimensional convolution with kernel size $K$.

$$
\omega = \sigma \left( C_1^{1D}(y) \right),
$$

(9)

where $C_1^{1D}$ represents one-dimensional convolution, involving only $K$ parameters. Because the kernel size $k$ under one-dimensional constraints is directly proportional to the channel dimension $C$, there may be a mapping between $K$ and $C$.

$$
C = \varnothing(k).
$$

(10)

Therefore, the above linear function is extended to nonlinear function, and a possible solution is introduced.

$$
C = \varnothing(k) = 2^{k-b}.
$$

(11)

Then, given the channel dimension $C$, the kernel size $k$ can be adaptively determined.

$$
k = \psi(C) = \left\lceil \frac{\log_2(C)}{\gamma} + \frac{b}{Y_{\text{odd}}} \right\rceil.
$$

(12)

Among them, the odd number of $|t|_{\text{odd}}$ represents the closest odd number of $T$. All experiments $\gamma$ and $B$, set to 2 and 1, respectively. Obviously, through the mapping sequence, the high-dimensional channels have longer interactions, while the low-dimensional channels have shorter interactions through nonlinear mapping. The schematic diagram is shown in Figure 1 which depicts the ECA module’s construction, where GAP represents global average pooling. The ECA module is a channel attention module that is both lightweight and efficient. First, given the input feature tensor $(H * W * C)$, GAP is used to determine the average value of all pixels on the feature map of each channel, reducing the number of parameters and calculations.

3.4. CNN-LSTM Network Structure. CNN, in conjunction with the ECA attention mechanism module, extracts features at the front end of the model, and the LSTM network uses the features collected by CNN to forecast. The ECNN-LSTM model is the name of this model. This model’s flow chart is depicted in full in Figure 2. Figure 2 depicts the ECNN-LSTM algorithm’s experimental flow.
kept and fed to the LSTM module to finish the prediction in Figure 2. After channel level global average pooling, ECA contributes to channel attention prediction by considering each channel and its K nearby nodes without diminishing the dimension. The interactive coverage (i.e., convolution kernel size k) is directly proportional to the channel dimension, and ECA may adaptively decide the value of K. The computation is straightforward, with few extra variables.

It is worth emphasizing that this is the overall network formed by the loss function with the same life prediction assessment scale, not the stacking between modules. Experiments demonstrate that the suggested strategy improves recognition accuracy [24].

### Table 1: Causes of hypercirculatory heart failure in children.

<table>
<thead>
<tr>
<th>Pathogeny</th>
<th>Corresponding diseases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Increased pulmonary blood flow</td>
<td>Left to right shunt (VSD, PDA, APW, AVSD)</td>
</tr>
<tr>
<td>Mixed lesion</td>
<td>TAPVC, TPA, SV</td>
</tr>
<tr>
<td>Parallel cycle</td>
<td>TGA</td>
</tr>
<tr>
<td>Increased cardiac output</td>
<td>Anemia, arteriovenous fistula, beriberi (vitamin B deficiency), etc.</td>
</tr>
<tr>
<td>Valve regurgitation</td>
<td>Mr, AI (congenital, rheumatic, infective endocarditis)</td>
</tr>
</tbody>
</table>

### Table 2: Causes of pump failure and heart failure in children.

<table>
<thead>
<tr>
<th>Pathogeny</th>
<th>Corresponding diseases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Congenital</td>
<td>Obstructive lesions (as, COA, PS, RVOTO); abnormal coronary artery originated from pulmonary artery; postoperative congenital heart disease</td>
</tr>
<tr>
<td>Acquired</td>
<td>Inflammation (viral myocarditis, HV related, etc.): dilated cardiomyopathy arrhythmia (tachycardia, atrioventricular block); others (left ventricular noncompaction, anthracycline toxicity)</td>
</tr>
</tbody>
</table>

Excessive circulation and pump failure are two pathophysiological abnormalities that can coexist in children with heart failure. Excessive circulation refers to a group of disorders that cause an abnormally high heart volume burden. There is a certain degree of pulmonary vein or pulmonary artery hypertension, and the left ventricular systolic function might be normal or high. Pump failure can be caused by worsened by heart failure can vary greatly depending on the kind of disease and the patient’s age. It may show evidence of malnutrition, growth restriction, or cachexia in addition to the normal symptoms of weariness, dyspnea, and activity intolerance. Long-term tolerance is possible in patients. There is no clear primary complaint despite the exercise limitation [5–7, 9]. The severity of the condition may be underestimated by the NYHA classification or the modified Ross classification, especially in individuals with complicated or cyanotic congenital heart disease. Heart failure is a clinical diagnosis, not an ultrasound report-based diagnosis.

3.5. Cases of Heart Failure in Patients with Congenital Heart Disease. The clinical symptoms of congenital heart disease worsened by heart failure can vary greatly depending on the kind of disease and the patient’s age. It may show evidence of malnutrition, growth restriction, or cachexia in addition to the normal symptoms of weariness, dyspnea, and activity intolerance. Long-term tolerance is possible in patients. There is no clear primary complaint despite the exercise limitation [5–7, 9]. The severity of the condition may be underestimated by the NYHA classification or the modified Ross classification, especially in individuals with complicated or cyanotic congenital heart disease. Heart failure is a clinical diagnosis, not an ultrasound report-based diagnosis.

Excessive circulation and pump failure are two pathophysiological abnormalities that can coexist in children with heart failure. Excessive circulation refers to a group of disorders that cause an abnormally high heart volume burden. There is a certain degree of pulmonary vein or pulmonary artery hypertension, and the left ventricular systolic function might be normal or high. Pump failure can be caused by
both congenital and acquired factors. Pump failure is defined by a decline in left ventricular or systemic ventricular function, and pulmonary venous hypertension affects the majority of children (Tables 1 and 2). When excessive circulation generated by an increased volume load causes heart failure symptoms, ventricular systolic performance is generally normal.

4. Experimental Result

If there is a small amount of reflux, the prognosis is good, and the best operation age is 1–2 years old; however, about 20% of patients have obvious reflux, pulmonary hypertension, and shunt, and symptoms occur in infancy. Those complicated with congestive heart failure or affecting growth

**Table 3: Hemodynamic manifestations and operation timing of ventricular septal defects with different sizes.**

<table>
<thead>
<tr>
<th>Large ventricular septal defect</th>
<th>QP/QS</th>
<th>VSD/aortic orifice diameter</th>
<th>Left ventricular size</th>
<th>Pulmonary artery pressure</th>
<th>Timing of operation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Small, restrictive</td>
<td>&lt;2</td>
<td>&lt;1</td>
<td>Normal</td>
<td>Normal</td>
<td>2-4 years old</td>
</tr>
<tr>
<td>All</td>
<td>≥2</td>
<td>&lt;1</td>
<td>Expand</td>
<td>Equal to body circulating pressure</td>
<td>1-2 years old</td>
</tr>
<tr>
<td>Large, nonlimiting</td>
<td>Left to right shunting depends on PVR</td>
<td>≥1</td>
<td>Expand</td>
<td>3-6 months or earlier after birth</td>
<td></td>
</tr>
</tbody>
</table>

Figure 3: Detection of different heart rates.

Figure 4: Ventricular horizontal shunt.
and development can be operated early. In the complete atrioventricular septal defect, 80% of the nonoperated patients died within 2 years old. The main causes of death were refractory heart failure, recurrent pulmonary infection, valve reflux, and pulmonary vascular disease. The incidence of vascular diseases was 30% within 1 year old, 80% within 2 years old, and 90% between 3 and 5 years old. Therefore, complete atrioventricular septal defect needs to be operated as soon as possible within 1 year old. If the general condition is good, it can be operated 3–6 months after birth. For patients with Down syndrome, the operation should be carried out early rather than late. The detection of different heart rates is shown in Figure 3.

Figure 4 depicts a ventricular horizontal shunt caused by a ventricular septal defect. With the extent of the defect, the hemodynamic symptoms and operation time are varied. Some per membranous defects heal on their own; 25% to 40% of them close before the age of two, and 90% of them close before the age of ten. As a result, monitoring and follow-up may be done on such youngsters without the need for immediate action. Surgical treatment is required for suberic, atrioventricular septal defects, and malocclusion defects, and defects larger than medium size should be addressed as soon as feasible.

Arterial horizontal shunt includes patent ductus arteriosus (PDA) and main pulmonary artery window. The shunt
flow of main pulmonary artery window is large and the patient's condition is serious. The child should be operated 6–8 weeks after birth because of early heart failure and pulmonary hypertension. The arterial catheter of 55% of infants was closed at 60 hours after birth, and there was little possibility of self-closure after 1 year old. Continuous open PDA is easy to cause infective endocarditis, heart failure, and pulmonary vascular disease. Premature infants are prone to PDA, and the incidence of continuous catheter opening is negatively correlated with gestational age at birth. If PDA is large and has obvious manifestations of cardiac insufficiency, early intervention treatment such as inflammatory pain or ibuprofen can be given to promote PDA closure. If drug treatment is ineffective, surgical ligation can be considered. PDA with severe pulmonary hypertension requires cardiac catheterization, including occlusion test, to comprehensively evaluate the nature of pulmonary hypertension before interventional therapy or surgery. For those with definite diagnosis of organic pulmonary hypertension (meaning severe pulmonary artery disease), the opportunity of operation will be lost, as shown in Table 3.

4.1. Obstructive Lesions. Left myocardial infarction: congenital mitral stenosis, supramitral stenosis ring, and tricuspid heart belong to supramitral and mitral level obstruction, and early intervention is required according to the condition. Stenosis of the aortic valve, supravalvular, and subvalvular can occur. In asymptomatic patients with aortic stenosis, usually, the maximum differential pressure ≥ 80 mmHg (1 mmHg = 0.133 kPa) and the average differential pressure ≥ 50 mmHg require surgery. The maximum differential pressure ≥ 50 mmHg with ST-1° changes requires surgery. Those with symptoms and heart failure should be operated immediately even if the differential pressure is not up to standard. This situation is common in newborns with severe aortic stenosis combined with heart failure and pulmonary hypertension. For aortic arch narrowing, the maximum differential pressure is generally >20 mmHg. Those with stable condition can be operated at a selected time and treated about 1 year old. Patients with heart failure need immediate intervention.

Right myocardial infarction: pulmonary valve stenosis with a crossvalve pressure difference of 60 mmHg requires intervention. Those with heart failure and right ventricular dysfunction caused by stenosis need emergency intervention. For right ventricular outflow tract obstruction, intervention measures shall be taken if the pressure difference is >40 mmHg, as shown in Figure 5.

Tetralogy of Fallot surgery often requires pulmonary valve crossing patch and right ventricular outflow tract reconstruction, which may lead to partial nonmovement or reverse movement of the right ventricle and affect ventricular contraction. [16] It was found that the right ventricular fac calculated by two-dimensional ultrasound was only slightly to moderately correlated with CMR in evaluating right ventricular function. Another study found that the right ventricular outflow tract ejection fraction calculated by CMR was >30% and dti-s was >8.4 cm/s, and the sensitivity and specificity of predicting right ventricular ejection fraction > 45% were better. CMR is the gold standard for postoperative cardiac function evaluation of complex congenital heart disease and congenital heart disease. However, due to the influence of price, accessibility, examination time, intracardiac device, and other factors, the prediction of non-concentricity rate is shown in Figure 6.

4.2. ECNN-LSTM Results. In this section, we present and discuss the results of the neural network topologies mentioned in Section 2, specifically the ECNN-LSTM. After preprocessing, the ECG signals were fed into both networks and, in the case of the CNN, after performing the GASF transformation. The findings show how much a CCN may enhance a classification task when compared to a normal feed-forward neural net. The performance of the nets was evaluated by repeating the entire training procedure ten times and evaluating after each time. In this way, we may evaluate the mean value of each neural network’s ultimate performance.
Figure 7 depicts the training set CCE error and accuracy across 100 training cycles for the run with the greatest prediction accuracy.

The evaluation of cardiac function in patients with congenital heart disease has its own characteristics. Especially, the evaluation of cardiac function in children is different from that in adults. It is a comprehensive clinical evaluation and cannot be limited to ultrasound images. Correct diagnosis and timely intervention are the key to treatment. The evaluation results affect the timing of treatment of congenital heart disease. Timely and successful intervention can reduce surgical complications, reduce mortality, and achieve a good long-term prognosis. New techniques such as strain imaging and 4D ultrasound are expected to play a greater role in the evaluation and prognosis of complex congenital heart disease.

5. Conclusions

A multiresolution singular value decomposition and ECNN-LSTM technique for child congenital heart disease prediction are presented to solve the problem of existing infant congenital heart disease. To show the probability and detailed features of the signal at different levels with multiresolution, the multiresolution singular value decomposition method is used to obtain approximate and detailed signals with different resolutions. The smooth running stage of newborns and toddlers is used to separate the health phases based on the standard deviation of the earliest moments. On LSTM, the prediction of congenital cardiac disease is finished. Validation with lifetime data demonstrates the feasibility and effectiveness of the suggested strategy.

Furthermore, the hospital’s ability to increase service quality in the future will be dependent on the support qualities of information services for epidemic-related websites [25]. We also want to use big data analytics platforms to employ parallel programming techniques to parallelize calculations over a large number of processing nodes [26–29].

Data Availability

The datasets used and analyzed during the current study are available from the corresponding author upon reasonable request.

Conflicts of Interest

The authors declare that they have no competing interests.

Authors’ Contributions

Jing Bai and Juan Fu are co-first authors with common contributions. The conception of the paper was completed by Jing Bai and Juan Fu, and the data processing was completed by Shihua Du, Xilong Chen, and Chengzu Zhang. All authors participated in the review of the paper. Jing Bai and Juan Fu have contributed equally to this work.

Acknowledgments

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