Modeling and Simulation of Hemodynamic Effects During Different Cardiopulmonary Resuscitation (CPR) Technologies

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Abstract: Cardiopulmonary Resuscitation (CPR) is a simple and effective medical treatment for sudden cardiac arrest patients. Because of those existing interferences in the experiments, most of the animal experiments and the CPRs in vivo studies get contradictory conclusions. In this report, according to the current theories of CPR, we got a CPR model; simulated various CPR technologies, and compared their hemodynamic variables. Compared the simulation results and animal experimental data, this report showed that when the External counterpulsation technique applied in CPR, the cardiac output, as well as the diastolic and the aortic pressure would be improved, which was consistent with the results of animal experiments.

Keywords: Cardiopulmonary resuscitationt; CPR techniques; CPR model; Hemodynamic simulation

1. Introduction

Cardiopulmonary Resuscitation (CPR) is a simple and effective medical treatment for sudden cardiac arrest patients. Standard Cardiopulmonary Resuscitation (SCPR) was put forward by Kouwenhoven in 1960. Since then, numerous CPR techniques have been proposed to generate better forward blood flow, increase coronary perfusion pressure (CPP), and finally to improve survival rates. Most of these techniques have been abandoned due to poor laboratory and clinical outcomes. In 2010's International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations, only several CPR techniques are reviewed [1]. Among them are the well-known Active Chest Compression Decompression CPR (ACD-CPR) and Interposed Abdominal Compression CPR (IAC-CPR).

External counter pulsation (ECP) and enhanced external counter pulsation (EECP) are circulatory assistance techniques. There are many animal experiments and human trials to show these measures may improve hemodynamics [2,3].

In China, Hengxin Yuan [4] has proposed a new CPR technique named Active Chest Compression Decompression with Enhanced External Counter Pulsation and the Inspiratory Impedance Threshold Valve (AEI-CPR), which can be simply understood as: AEI-CPR=ACD-CPR+ EECP+ITV (which is the same as ACD-CPR+ITV except that EECP is applied sequentially to lower limbs). Yuan has done animal experiments to show that it may increase CPP, increase diastolic pressure of aortic aorta and improve coronary perfusion.

In 2007, Yannopolus [5] has done a similar work. He shows that lower extremity counter pulsation during the decompression phase of CPR improves hemodynamics.

There are many human trials and animal experiments to be done to compare hemodynamics and outcomes to apply different CPR techniques. Yet, these results are conflicting. To date, there is no conclusive evidence to support or refute the use of any alternative CPR technique.

One of the most important reasons leading to conflicting results is that there are many confounding factors (such as varying patient populations, downtime, drug therapy, central venous pressure, peripheral vascular resistance, underlying disease, chest configuration, and body size, as well as varying rescuer size, skill, strength, consistency, prior training, and bias) present in human trials and animal experiments, it is hard to compare experimental data obtained from different experiments.

Some researchers follow another line of CPR research. They construct mathematical models [6,7] based on basic cardiovascular physiology, and simulate hemodynamic effects of different CPR techniques. This method has several benefits [6,7]: it's possible and easy to compare hemodynamic effects of different CPR techniques under same conditions; it allows exact control of the hemodynamic mechanisms of CPR (such as thoracic pump mechanism or cardiac pump mechanism); simulations can be done many times for different control parameters (such as the magnitude of the compression pressure applied) to find optimal ones, which are very costly, if possible, by doing animal or human experiments.

Babbs has done a lot of work to follow this line of research. Our group has extended Babbs' work [8,9,10] to simulate and compare hemodynamics of AEI-CPR, ACD-CPR and IAC-CPR.

But we find that the cardiac output our group get from simulations is too good and doesn't agree very well with experimental data. We hypothesize it's because the spring and damper model used to simulate the thoracic pressure generated by compression is problematic. So in this paper, we decide to remove the spring and damper model, and simply use a half sinusoidal pressure source to apply to the blood vessels within the thoracic chamber.

In this paper, we simulate and compare hemodynamic effects of SCPR, ACD-CPR and IAC-CPR. Our

simulation results show that the simulation results of the revised model better corresponds with experiments. To investigate whether ECP may improve hemodynamics during CPR, we also simulate SCPR+ECP (which is the same as SCPR except that during chest relaxation phase, ECP is applied to lower limbs) and ACD-CPR+ECP (which is the same as SCPR except that during chest relaxation phase, ECP is applied to lower limb).

As a first attempt, we do not simulate ITV and EECP in this paper. As our future work, we may incorporate these effects in our simulations.

2. Method

The structure of the revised model is similar with that appears in [6,7,8,9,10] and is drawn as follows:

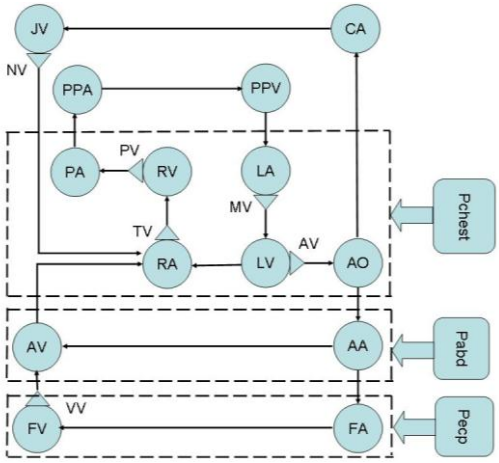

Figure 1 Model Diagram

In Figure 1, 14 circles represent 14 circulatory compartments (LA: left atrium; LV: left ventricle; RA: right atrium; RV: right ventricle; AO: aortic aorta; AA: abdominal aorta; AV: abdominal vein; FA: femoral artery; FV: femoral vein; CA: carotid artery; JV: jugular vein; PA: pulmonary artery; PPA: peripheral pulmonary artery; PPV: peripheral pulmonary vein). Triangles represent valves (MV: mitral valve; AV: aortic valve; TV: tricuspid valve, PV: pulmonary valve, NV: Niemann's valve; VV: venous valve). Round corner rectangles represent pressures applied to blood vessels when using different CPR techniques. Blood vessels contained in dashed rectangles are influenced by the corresponding pressures. In particular, Pchest is the pressure applied to blood vessels contained in the heart chamber; Pabd is the pressure applied to abdomen blood vessels when IAC-CPR is employed and Pecp is the pressure applied to femoral blood vessels when ECP is employed.

2.1 Circulatory System Model

The circulatory system model is the same as in [7]. The model consists of 14 compartments as the circles shown in Figure 1. Model parameters are the same as in [7], except that in [7] Cppa=0.0042 and we conclude from context that it is a clerical error and the intending value should be Cppa=0.00042.

2.2 External pressure applied

When abdominal compression is applied, Pabd is applied to abdominal artery and abdominal vein.

When external counter pulsation is applied, Pecp is applied to femoral artery and femoral vein.

When chest compression and (or) decompression is applied, Pchest is applied to all heart chambers and thoracic vessels that are contained in the dashed rectangle pointed to by Pchest, as shown in Figure 1, which include: left atrium, left ventricle, right atrium, right ventricle, pulmonary artery, pulmonary vein, peripheral pulmonary artery and aortic aorta.

2.3 Pump Mechanisms

There are two dominant mechanisms hypothesized which generate forward blood flow during CPR: thoracic pump mechanism and cardiac pump mechanism.

As in [6,7], we use a control parameter Tpfactor which stands for thoracic pump mechanism factor. When Tpfactor=0, the pump mechanism is purely cardiac, and Pchest is only applied to left ventricle and right ventricle. When Tpfacotr=1, the pump mechanism is purely thoracic, Pchest is applied to all heart chambers and great vessels in the thoracic chamber. When 0<Tpfactor<1, it means hybrid mechanism, and we simulate this by applying Pchest to left ventricle and right ventricle, and applying Pchest*Tpfactor to the remaining heart chambers and great thoracic vessels.

In this paper, we set Tpfactor=0.75 when simulating CPR techniques.

2.4 Simulation Aspects

In this paper, we simulate five CPR techniques: SCPR, ACD-CPR, IAC-CPR, ACD-CPR+ECP and SCPR+ECP.

For all CPR techniques, we set compression frequency f=100/min, duty cycle=0.5 (duty cycle is the fraction of chest compression within a chest compression/relaxation cycle) according to 2010 International Consensus.

For all CPR techniques, Pchest is in effect. During the chest compression phase, Pchest is a half sinusoidal curve with a peak value of 45mmhg, which is according to the literature that during CPR, the generated thoracic pressure is approximately 45mmhg.

For ACD-CPR, during the chest relaxation phase, Pchest is a half sinusoidal curve with a peak value of -20mmhg.

For IAC-CPR, Pchest and Pabd are in effect. During the chest relaxation phase, Pabd is a half sinusoidal curve with a peak value of 100mmhg.

For ACD-CPR+ECP and SCPR+ECP, Pchest and Pecp are in effect. During the chest relaxation phase, Pecp is a half sinusoidal curve with a peak value of 150mmhg.

2.5 Computational Aspects

The model equations are similar to those appear in [6,7] with adoptions described above. These equations are programmed in Matlab and solved by using a fixed step size (0.001s) fourth-order Runga Kutta algorithm. All simulations run 1 minute and all simulated pressures and blood flows are stable after 30 seconds.

3. Results

3.1 Model Validation

As in [7], we validate the model by running simulations under normal physiological conditions.

Under normal conditions heart rate is 75/min, heart period is 0.8s, durations of ventricle contraction is approximate 0.3s. So we set compression frequency f=75, and duty cycle=3/8=0.375.

We apply Pchest to only left ventricle and right ventricle. For left ventricle, Pchest is a half sinusoidal curve with a peak value of 120mmhg; for right ventricle, Pchest is a half sinusoidal curve with a peak value of 20mmhg.

To use these parameters, we may simulate hemodynamics under normal conditions. We get a cardiac output of 4.87L/min, which is close to textbook value 5L/min; cerebral blood flow is 0.94L/min, which lies in the normal range of 15%-20% of cardiac output; aortic aorta pressure is 117/74mmhg, which is close to textbook value 120/80mmhg, coronary blood flow is: 0.34L/min, which is higher than normal value 0.25L/min, this may due to the reason that the coronary circulation model is too simple.

3.2 Simulations Results

We simulate five CPR techniques: SCPR, ACD-CPR, IAC-CPR, SCPR+ECP and ACD-CPR+ECP in this paper. After 30 seconds, all simulated pressures and flows are stable, and we calculate cardiac output, cerebral blood flow, coronary blood flow and mean CPP after stabilization. The results are listed in Table I.

TABLE I. SIMULATION RESULTS

CPR Technique	**Cardiac output (L/min)**	***Cerebral Blood Flow (L/min)***	***Coronary Blood Flow (L/min)***	***Mean CPP (mmhg)***	***Mean Systolic aortic pressure (mmhg)***
SCPR	1.58	0.41	0.10	27	41
ACD-CPR	2.13	0.56	0.14	36	48
IAC-CPR	2.86	0.75	0.17	44	55
SCPR +ECP	2.14	0.42	0.10	27	45
ACD-CPR +ECP	2.69	0.56	0.14	37	53

To investigate the benefits of other CPR techniques over SCPR, we draw Figure 2.

The cardiac output of 1.58L/min during SCPR is a little bit higher than 1L/min, which is typically reported in the literature. It may be due to the reason that in our model, CPR quality is ideal, which cannot be reached in experiments.

In reference [11], it reports that IAC-CPR increases CPP by 5 ± 15.1mmhg comparing to SCPR which agrees well with our simulated 7mmhg.

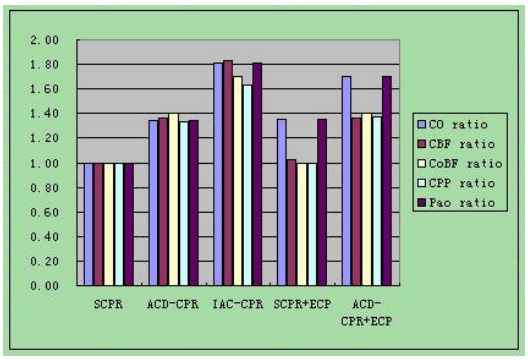

Figure 2 Ratios of other CPR techniques to SCPR. CO: cariac output; CBF:cerebral blood flow; CoBF: coronary blood flow; CPP: coronary perfusion pressure,Pao: pressure of aortic root

In reference [12], it reposts that systolic aortic pressure is 39.02 ± 21mmhg during SCPR, and simulation gives 41; systolic aortic pressure during IAC-CPR is 63.6 ± 21mmhg, our simulation gives 55mmhg.

In reference [13], it reports that IAC-CPR increases cardiac output by 75% compared to SCPR, our simulation gives 81%.

In reference [14], it reports that systolic arterial pressure during ACD-CPR is 88.9 ± 24.7mmhg, simulation gives 48mmhg.

In reference [15], it reports that SCPR+ECP generates CPP of 25 ± 1mmhg, which is close to our 27mmhg; ACD-CPR+ITV+ECP generates CPP of 43 ± 9mmhg, in our simulation of ACD-CPR+ECP, CPP generated is 37.

From all these comparisons between experimental data and simulation results, we may see that in general our simulation results agree quite well with experimental data.

From Table I and Figure 2, we see that the hemodynamics of ACD-CPR and IAC-CPR is better than SCPR, and IAC-CPR is the best among these three techniques.

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When ECP is applied during SCPR or ACD-CPR, it can improve cardiac output. But it does not improve CPP, cerebral blood flow or coronary blood flow.

4. Conclusion

In this paper, we adapt the previous CPR models and simulate five different CPR techniques: SCPR, ACD-CPR, IAC-CPA, SCPR+ECP, ACD-CPR+ECP. As with all models, our revised model has some limitations. But compared with experimental data, we find that our simulation results agree quite well with experimental data. Given the benefits of models and simulations, our model may be a useful tool in CPR research.

From simulation results, we see that hemodynamics of ACD-CPR and IAC-CPR is better than that of SCPR, which agree with many experimental work.

When ECP is applied during ACD-CPR and IAC-CPR, it can improve cardiac output. Yet ECP is not widely used during CPR, we suppose that ECP is worthy for further research.

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