Neonatal CPR: room at the Top--A Mathematical Study of Optimal Chest Compression Frequency Versus Body Size.

Charles F Babbs

Andrew Meyer
Lehigh Valley Health Network, andrew_s.meyer@lvhn.org

Vinay Nadkarni

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Simulation and education

Neonatal CPR: Room at the top—A mathematical study of optimal chest compression frequency versus body size☆

Charles F. Babbs[a,b,*], Andrew Meyer[c], Vinay Nadkarni[d]

[a] Department of Basic Medical Sciences, Weldon School of Biomedical Engineering, Purdue University, 1426 Lynn Hall, West Lafayette, IN 47907-1246, USA
[b] Indiana University School of Medicine, USA
[c] Department of Neonatology, Children's Hospital of Philadelphia, USA
[d] Department of Anesthesia and Critical Care, Children's Hospital of Philadelphia, USA

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ABSTRACT

Objective: To explore in detail the expected magnitude of systemic perfusion pressure during standard CPR as a function of compression frequency for different sized people from neonate to adult.
Method: A 7-compartment mathematical model of the human cardiopulmonary system – upgraded to include inertance of blood columns in the aorta and vena cavae – was exercised with parameters scaled to reflect changes in body weight from 1 to 70 kg.
Results: Maximal systemic perfusion pressure occurs at chest compression rates near 60, 120, 180, and 250/min for subjects weighing 70, 10, 3, and 1 kg, respectively. Such maxima are predicted by analytical models describing the dependence of stroke volume on pump-filling time in the presence of blood inertia. This mathematical analysis reproduces earlier experimental results of Fitzgerald et al.[10] in 10 kg dogs.
Conclusions: Fundamental geometry and physics suggest that the most effective chest compression frequency in CPR depends upon body size and weight. In neonates there is room for improvement at the top of the compression frequency scale at rates >120/min. In adults there may be benefit from lower compression frequencies near 60/min.

1. Introduction

Without adjuncts such as interposed abdominal compression,[1,2] the major variables that can be adjusted during basic life support to improve artificial circulation include the chest compression depth and the chest compression frequency. In infants and neonates, current guidelines[3] recommend external compression to a depth of 33% of the chest diameter, which is relatively greater than that recommended for adults (about 20% of chest diameter). Further, our recent analysis of CT images of neonates (Online Supplement 4) reveals that with 33% compression depth one quarter of patients would experience maximal or over-compression of the mediastinum, completely flattening the heart within the anatomic space available between the sternum and the spine. If compression depth were increased further to 50% of chest diameter, there would be over-compression of the heart in nearly all neonates. It seems therefore that there is little room for improvement of neonatal CPR in the domain of compression depth.

The optimal rate or frequency of chest compression in neonates remains uncertain.[4–6] Since natural heart rates in neonates are in the range of 120–160 beats/min, we wondered if increasing chest compression frequency in neonatal patients might have the potential to boost artificial cardiac output compared to recommended compression frequencies, which are based largely on experimental work in animal models larger than neonates. In particular, we wondered if there might be a more sophisticated way to extrapolate animal and adult human data to patients of very small size, based on fundamental principles of mathematics, physics, and biology. This paper is dedicated to the proposition that such an extrapolation method is possible and indeed confirms the experiment of evolution for naturally beating hearts that faster rates are more effective and appropriate for smaller animals.

2. Theory

2.1. Scaling

Scaling rules in biology describe the structural and functional consequences of changes in body size among geometrically similar organisms.[7] Certain relationships between a representative linear

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* Corresponding author at: Department of Basic Medical Sciences, Weldon School of Biomedical Engineering, Purdue University, 1426 Lynn Hall, West Lafayette, IN 47907-1246, USA. Tel.: +1 765 496 2661; fax: +1 765 494 0781.
E-mail address: babbs@purdue.edu (C.F. Babbs).

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dimension of an animal, L, a particular cross-section, A, and body weight, W, are valid for animals of similar body shape. For example, lean body weight scales with the cube of any particular linear dimension, and the cross-sectional area of blood vessels scales with the square of the linear dimension.

To model effects of compression frequency in CPR over a wide range of body sizes, we need to know the scaling rules for relevant cardiovascular parameters. Vascular resistance is the ratio of perfusion pressure across a vascular bed to blood flow through the vascular bed. Normal arterial blood pressure and systemic perfusion pressure are relatively constant for humans of different body size from newborn to adult. Basal metabolic rate, and in turn oxygen consumption, scale approximately with body surface area—the predominate site of heat loss. The scaling of cardiac output with body surface area has lead to the concept of cardiac index, or forward blood flow per square meter of body surface area.[8] Since surface area is proportional to L^2 and weight is proportional to L,[3] the body surface area is proportional to the 2/3 power of body weight, $W^{2/3}$. In turn, blood flow or normal resting cardiac output is also proportional to $W^{2/3}$. Thus vascular resistance scales as pressure/flow, or $1/W^{2/3} = W^{-2/3}$, where pressure is constant across scales. So for vascular resistance, R, we can write for an appropriate constant, k_1,

$$R = k_1 W^{-2/3}. \tag{1a}$$

The compliance, C, of blood vessels is a function of vessel radius, $C = 2\pi L r^3/(Eh)$, where L is the length of the tube, r is the radius, E is Young's modulus of elasticity of the wall material, and h is the thickness of the tube.[9] For people of different size, r and h are proportional to L. This means that compliance scales as L^3 or the first power of body weight, W. Thus for vascular compliance we can write for an appropriate constant, k_2,

$$C = k_2 W. \tag{1b}$$

To change parameters from one body weight to another, suppose that we have a parameter, X, (such as L, R, or C) that scales with weight, W, according to the power function $X(W) = kW^a$, where k and a are constants and W is weight in kg. The reference adult body weight is 70 kg. Then $k = X(70)/70^a$, so the parameter value for any arbitrary weight, W, in terms of the value for a 70 kg adult is

$$X(W) = X(70)\left(\frac{W}{70}\right)^a. \tag{2}$$

In this way it is possible to scale theoretical models of the cardiovascular system for persons of different size from full sized adults to tiny babies, beginning with a standard textbook model for a 70 kg adult.

2.2. A working hypothesis

In a comprehensive study of the effects of compression frequency during cardiac arrest and CPR in medium sized dogs, Fitzgerald et al.[10] found that effects of compression timing variables are explained largely by limitations of pump filling and emptying, with the pump-filling terms being dominant. As long as the fraction of cycle time during which the pump input valve is open is greater than the time required for adequate pump filling, then there is cardiac output (compression rate × stroke volume) to be gained by increasing the compression frequency. However, as compression frequency increases further, time for pump-filling decreases. Then the pump fills only partially, so stroke volume decreases. In turn, cardiac output levels off and may even diminish at faster compression rates. This general principle was confirmed experimentally and applies either to the cardiac pump mechanism or the thoracic pump mechanism of CPR,[11] as well as to the naturally beating heart. Accordingly, to understand compression frequency effects in CPR, we decided to focus on pump filling.

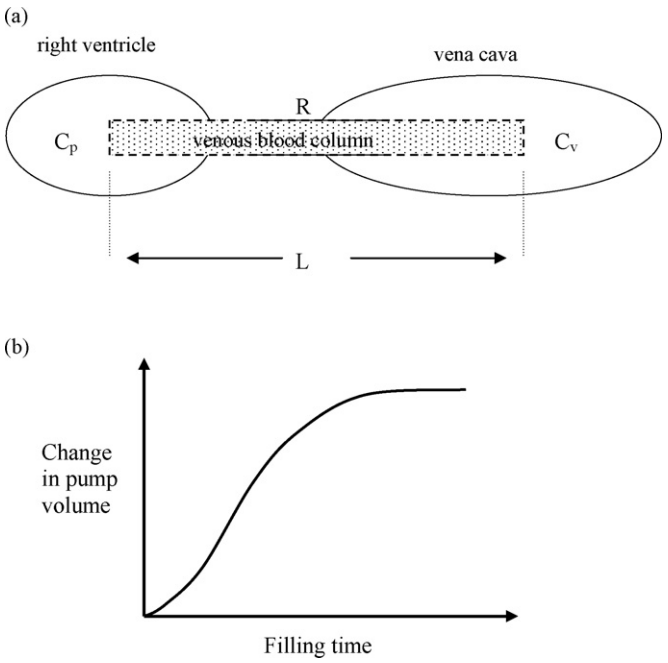

Fig. 1. (a) Two-compartment model of the great veins C_v (right) and chest pump C_p (left) during filling, connected by low resistance R of the open tricuspid valve. (b) Sketch of sigmoid pump volume increase as a function of time during filling.

To describe filling-related tradeoffs quantitatively, imagine an exceedingly simple two-compartment model shown in Fig. 1(a) in which the elastic central venous reservoir, C_v, denoting the venae cavae and right atrium, is connected to the input reservoir of the chest pump, C_p, denoting the right ventricle, by a low resistance pathway that includes the one-way tricuspid valve. A column of venous blood with mass density, ρ, cross-sectional area, A, and length, L, flows into the right ventricle (C_p) under the influence of axial pressure difference between C_v and C_p during a particular fraction, σ, of total compression cycle time, such as 1/3rd. For this simple model, as shown in Online Supplement 1, the compression frequency f_{max} for maximum forward flow is determined by Newton's second law of motion for the blood column flowing into the right ventricle, C_p, and can be approximated as

$$f_{max} \approx 0.41\sigma\sqrt{\frac{A}{\rho L}\left(\frac{1}{C_v}+\frac{1}{C_p}\right)}. \tag{3}$$

Eq. (3) implies a definite scaling rule for the optimal compression frequency as a function of body weight. The ratio A/L of the dimensions of the venous blood column scales in proportion to the linear dimension of the animal or $W^{1/3}$, which is the cube root of body weight. The compliance is directly proportional to body weight, W. Hence, the argument of the square root in (3) is proportional to $W^{-2/3}$. This means that f_{max} is proportional to $W^{-1/3}$. For this algebraic model of pump filling the frequency for maximum flow, f_{max}, scales inversely with the cube root of body weight. Using expression (2) for body weight, W, compared to a theoretical 70 kg adult

$$f_{max}(W) = f_{max}(70)\left(\frac{W}{70}\right)^{-1/3} \tag{4}$$

Compared to adults, smaller babies or children should have a distinctly higher maximally effective compression frequency, according to a simple pump-filling model based on Newton's second law.

Estimating the numerical values of venous compliances C_v and C_p in expression (3) for a normal adult human is relatively straightforward using textbook knowledge of physiology.[12] However, the

pressure vs. volume curves for veins are highly non-linear. Hence the dynamic compliances, dV/dP, of both the vena cavae and the right ventricle depend strongly on their degree of distension with blood.[13,14] During cardiac arrest (a most extreme form of acute congestive heart failure) venous volume increases and venous compliance decreases to a value about one half normal. As shown in Online Supplement 2, reasonable estimates for venous compliances in a 70 kg adult during conditions of cardiac arrest and CPR are C_v = 11.5 ml/mmHg and C_p = 9.0 ml/mmHg. Using these values, together with blood density 1 g/ml, A = 7 cm^2, L = 30 cm, filling time equal 1/3rd of cycle time, and 1 mmHg = 1333 g/(cm s^2), we can obtain the predicted compression rates for maximal blood flow given in Table 1.

The simple pump-filling hypothesis suggests that CPR compression rates should be distinctly higher for small infants than for large adults, not unlike the known differences in natural heart rates. The pump-filling model also suggests lower optimal compression rates for adults, compared to current guidelines. Values in Table 1 for subjects in the weight range of neonates or premature infants are substantially greater than 120/min, suggesting potential room for improvement, provided that humans or mechanical devices can sustain the higher rates for an adequate period of time.

To explore the scaling of optimal frequencies for chest compression with body weight in greater detail, we exercised computational models of the whole circulation, including both arterial and venous compliances, resistances of capillary beds, and inertia of blood columns in the aorta and venae cavae.

3. Computer models

For the present study we adapted the computational model shown in Fig. 2 and published previously for resuscitation research by one of us,[15] to include appropriate scaling of cardiovascular parameters with body size and also the effects of blood inertia in the aorta and vena cava, as described in Online Supplement 3. To simulate the redistribution of blood volume from arteries to veins during cardiac arrest and CPR, leading to increased arterial compliance and reduced venous compliance, we corrected standard normal values using the method of Online Supplement 2, so that aortic compliances for CPR are twice the values determined at normal arterial pressure and venous compliances are half the values determined at normal venous pressure. Vascular resistances in cardiac arrest may be either increased owing to endogenous epinephrine release or decreased, owing to effects of hypoxia, acidosis, and vascular collapse. Hence, we used the normal values of resistance in the computational model to explore the effects of compression rate and body size.

3.1. Cardiac and thoracic pump mechanisms

In our computational model the cardiac ventricles experience varying external pressure P_{chest} as a result of chest compression. The right atrium and thoracic aorta experience external driving pressure $f_{tp}P_{chest}$ for thoracic pump factor $0 \le f_{tp} \le 1$, depending on the degree to which the "thoracic pump" mechanism of CPR is working. In this way one can create a continuum of hybrid pump mechanisms ranging from pure cardiac pump (f_{tp} = 0) to pure thoracic pump (f_{tp} = 1), as described in reference.[11] When f_{tp} = 1 all mediastinal structures, including the great veins and thoracic aorta, experience a uniform "global" intrathoracic pressure rise, as originally conceived by Weisfeldt, Rudikoff and coworkers.[16] When f_{tp} = 0, only the right and left ventricles are pressurized, as in open chest CPR.[17–19] Intermediate values of the thoracic pump factor allow models approximating the consensus understanding,[20–22] in which both mechanisms are operative to some degree. In the present study we explored effects of body size and weight over a range of thoracic pump factors.

Table 1
Compression frequencies, f_{max}, for maximal blood flow according to a simple biomechanical model of chest pump filling, including blood inertia, which is described in detail in the Online Supplement 1.

Lean body weight, W (kg)	$(W/70)^{0.333}$	f_{max} (1/min)
70	1.00	64
20	0.65	99
10	0.52	124
5	0.41	157
3	0.35	184
2	0.30	214
1	0.24	267

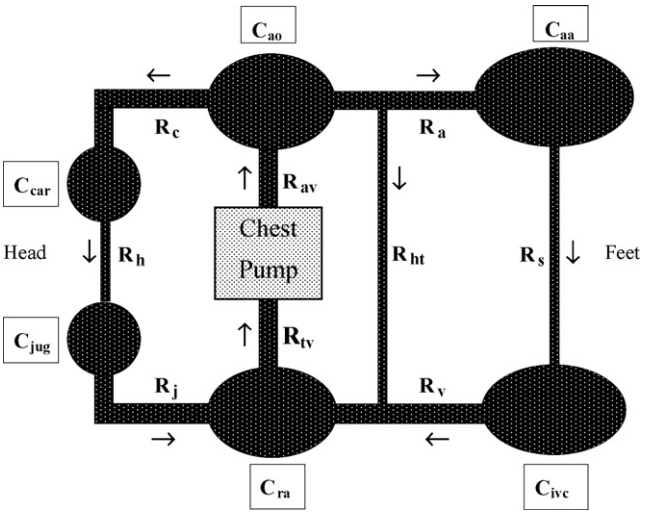

Fig. 2. Seven compartment model of the human circulatory system. Definitions of compliances, C, resistances, R, are as follows: carotid arteries (car), thoracic aorta (ao), abdominal aorta (aa), inferior vena cava (ivc), jugular veins (jug), right atrium (ra), and chest pump mechanism. Non-zero vascular resistances, R, connect the vascular compartments. The value of R_{ht} is large and represents the vascular resistance of the heart and miscellaneous tissues within chest. The value of R_s is intermediate and represents the vascular resistance of all tissue beds outside the chest, including arms, legs, and abdomen. The values of R_a, and R_v, are small and represent lumped in-line resistances of the great vessels between the chest and the abdomen, as are the values of R_c and R_j leading to the head and neck. The remaining small resistances represent outflow and inflow resistances of the cardiac valves, denoted by suitable anatomic subscripts.

4. Results

Fig. 3 shows results of simulations of CPR in the 7-compartment circulatory model for subjects of different body weight, using vascular compliance values appropriate for cardiac arrest and CPR. The quality of CPR is represented on each vertical axis in terms of systemic perfusion pressure. Systemic perfusion pressure, defined here as mean thoracic aortic pressure minus mean right atrial pressure, is a relatively stable predictor of resuscitation success in diverse animal models.[23–27] This figure of merit has been described by Otlewski as the most valid measure of coronary perfusion pressure.[28] Fig. 3(a)–(c) shows results for increasing thoracic pump factors. In all cases flow rises as a function of frequency to shoulder regions, and there is often a subtle peak in systemic perfusion as a function of compression frequency. The peaks are located at compression rates near 60, 120, 180, and 250/min for subjects weighing 70, 10, 3, and 1 kg, respectively. The peaks are more prominent when the thoracic pump factor is larger. These results are similar to those found previously in an analog model of the circulation during CPR including blood inertia.[29] Such peaks suggest that rate

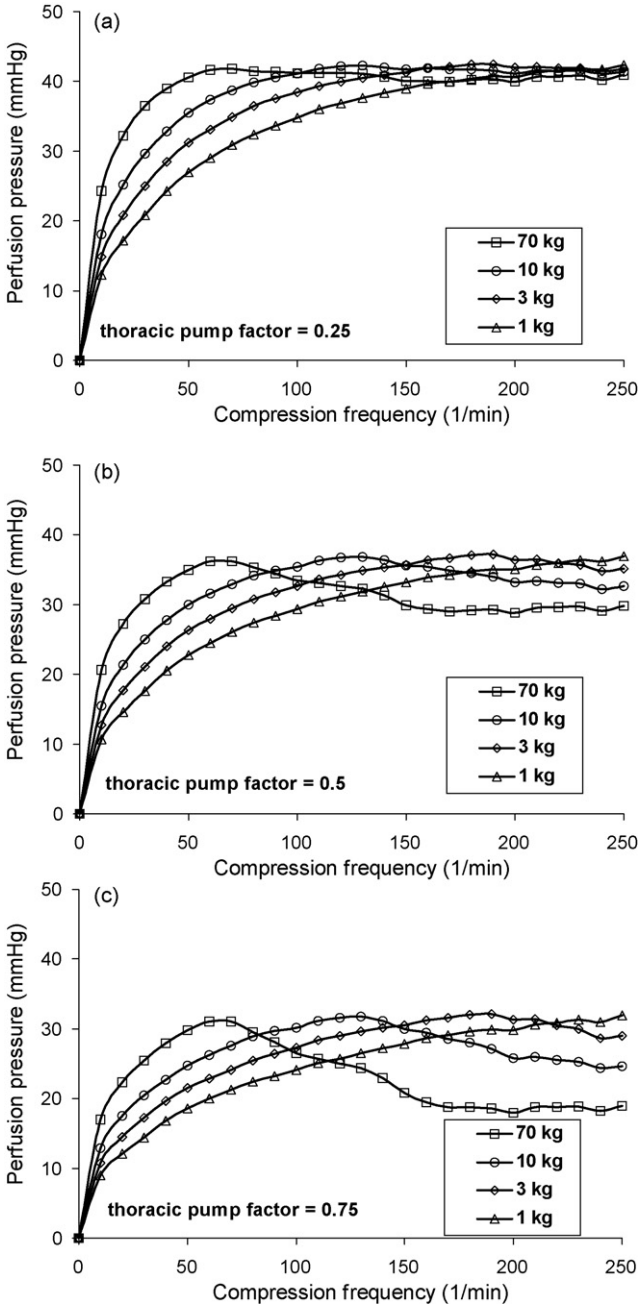

Fig. 3. Cardiopulmonary pressures in simulations of CPR for subjects of different body weight. Systemic perfusion pressure (mean thoracic aortic minus mean central venous pressure) is plotted as a function of chest compression frequency. Blood density is 1.05 g/ml. Thoracic pump factors are (a) 0.25, (b) 0.50, and (c) 0.75.

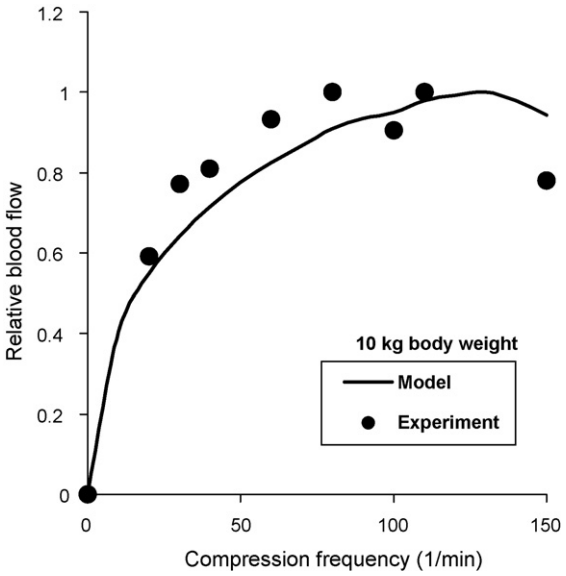

Fig. 4. Computational model vs. experimental data from Fitzgerald et al.[10] The fraction of maximal blood flow is plotted as a function of chest compression frequency. Thoracic pump factor for model calculations is 0.75. Corresponding curves (not shown) for thoracic pump factors of 0.25, 0.5, and 1.00 are very similar. Data points are means of experimental results.

effects are explained in large part by tradeoffs between compression frequency and the filling time for the chest pump, related to the inertia of blood columns in the vena cava (Online Supplement 1).

As validation of the computational model of Figs. 2 and 3 we compared its predictions, with the experimental results of Fitzgerald et al.,[10] who conducted a systematic study of compression rates varying from 20 to 150/min in anesthetized 10 kg dogs undergoing experimental CPR with a mechanical chest compressor (Thumper®). The comparison in Fig. 4 for 10 kg subjects shows that the present scaling model fits experimental data reasonably well.

5. Discussion

Given the unusual practical difficulties of working with human or animal models of cardiac arrest and CPR, theoretical and computer models have found a niche in resuscitation research.[2,30] Such models are independent of many confounding factors present in laboratory studies and in clinical trials. These include varying patient populations, cardiac arrest time, drug therapy, underlying disease, chest configuration, and body size, as well as varying rescuer size, skill, strength, consistency, prior training, and bias. In studies of multiple compression frequencies mathematical models avoid problems caused by progressive breakdown of the chest wall, depletion of endogenous hormones, and pulmonary contusion or liver laceration with repeated trials. Mathematical models also allow exact control of the dominant hemodynamic mechanism of CPR (thoracic pump vs. cardiac pump[11,31]).

With this approach, we found a clear effect of body size and weight on the optimal compression frequencies for CPR. The existence of plateaus and subtle peaks in the spectrum of systemic perfusion pressures versus frequency and their relative separation in the compression frequency domain are explained in part by tradeoffs between compression frequency and pump-filling time (Online Supplement 1). The overall amplitude of systemic perfusion pressure is strongly modulated by thoracic pump factor. The locations of the peaks in perfusion pressure along the frequency axis, however, are relatively independent of the thoracic pump factor but strongly dependent upon body size.

Our study is limited in that it uses simplified models of the circulation that do not reflect specific underlying disease states. We do not specifically model pre-existing congestive heart failure, which could decrease venous compliance by means of venous distension and in turn increase the optimal compression frequency. We also do not specifically model hemorrhage, which could have the opposite effect. We ignore increases in peripheral vascular resistance caused by endogenous or exogenous vasoconstrictors as well as decreases caused by hypoxia and acidosis.

The analytical model of pump-filling described in Online Supplement 1 does not account for the closed loop nature of an

intact circulation, in which, if pump filling is poor on one beat at high compression rates, venous pressure increases transiently and there is more filling on the next beat. This alternans-like phenomenon is quite evident in pressure read-outs from the closed loop computational model at high compression frequencies. The effect supports perfusion at higher compression rates to a degree greater than that predicted by the simple pump-filling model summarized in Eq. (3). However, the predictions of Eq. (3) regarding of the existence of peaks in perfusion and their locations in the frequency domain have been confirmed by more complex models (Fig. 3) and by experiment (Fig. 4).

6. Conclusions

There may be merit in exploring substantially slower compression frequencies for CPR in the world of adult medicine. Compression frequencies near 60/min (the original 1960 guideline recommendation) may be less tiring for rescuers and less traumatic for the patient than higher compression rates. Slower compressions would allow for better pump filling and also more complete chest recoil, as well as more attention to the optimal "rectangular shape" of the compression waveform,[32] which is quite difficult to achieve at higher frequencies. Slower compressions in adults may even be of psychological benefit to the resuscitation team, being less frenetic and hurried.

In smaller patients, especially infants and neonates, there are fundamental physical and mathematical reasons why improved blood flow may results from compression rates substantially faster than those effective in adult patients. These include the effects of the mass of venous blood columns entering the chest pump as described by Newton's second law of motion and the way in which mass, length, and area scale with body size.

In adult humans there may be some room for improvement at the bottom of the compression frequency scale. In neonates there is room for improvement at the top.

Conflict of interest statement

No conflicts of interest to declare.

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.resuscitation.2009.07.014.

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