Medical Engineering and Physics 000 (2017) 1–7

Contents lists available at ScienceDirect

Medical Engineering and Physics

journal homepage: www.elsevier.com/locate/medengphy

Development and validation of an improved mechanical thorax for simulating cardiopulmonary resuscitation with adjustable chest stiffness and simulated blood flow

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ARTICLE INFO

Article history:
Received 4 August 2016
Revised 6 February 2017
Accepted 6 February 2017
Available online xxx

Keywords:
Mechanical thorax model
Resuscitation
Thorax stiffness
Blood flow

ABSTRACT

Investigations of compressive frequency, duty cycle, or waveform during CPR are typically rooted in animal research or computer simulations. Our goal was to generate a mechanical model incorporating alternate stiffness settings and an integrated blood flow system, enabling defined, reproducible comparisons of CPR efficacy. Based on thoracic stiffness data measured in human cadavers, such a model was constructed using valve-controlled pneumatic pistons and an artificial heart. This model offers two realistic levels of chest elasticity, with a blood flow apparatus that reflects compressive depth and waveform changes. We conducted CPR at opposing levels of physiologic stiffness, using a LUCAS device, a motor-driven plunger, and a group of volunteers. In high-stiffness mode, blood flow generated by volunteers was significantly less after just 2 min of CPR, whereas flow generated by LUCAS device was superior by comparison. Optimal blood flow was obtained via motor-driven plunger, with trapezoidal waveform.

1. Introduction

During the past few decades, guidelines for cardiopulmonary resuscitation (CPR) have been modified periodically in terms of chest compression, medication protocol, airway management, and defibrillation criteria to improve patient outcomes. However, reported rates for survival to discharge after cardiac arrest remain low, averaging only 16% in hospitals and 10–12% in non-clinical settings [1–3]. A host of factors contribute to such poor outcomes, primarily treatment delays, training deficiencies in those administering CPR, and prolonged periods before instituting advanced life support (ACLS), including defibrillation. Also implicated is the inconsistent quality of chest compressions (frequency, depth) during resuscitation. Certain studies have exposed inadequacies in frequency and/or depth of compressions during CPR, even if done by well-trained personnel [4,5]. Those engaged are apt to show fatigue in short order, delivering fewer and less vigorous compressive efforts over time. The result is a substantial reduction in circulatory blood flow. Mechanisation of chest compression may offer a better means of sustaining circulation during CPR, potentially improving outcomes.

Two physiologic mechanisms responsible for blood flow during resuscitation are discussed widely in the literature: intrathoracic pump effect [6–9] and direct cardiac compression [10–13]. In theory, circulation may be enhanced by increasing compressive frequency (direct compression) or by modifying duration of compression or duty cycle (intrathoracic pump). A number of mechanical devices for CPR use, developed since the early 1970's, have shown success in combatting physical fatigue by bolstering thoracic compression. Previous experimental and clinical study data have also underscored the potential advantages of mechanical (vs. manual)

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http://dx.doi.org/10.1016/j.medengphy.2017.02.005
1350-4533/

CPR [14–20], typically focusing on the impact made by duty cycle or compressive waveforms [21–25]. Despite these efforts, no long-term survival benefits have emerged from large-scale investigations into cardiac arrest suffered out-of-hospital; and some devices have even been linked with injuries that are quite severe [26–28].

Due to medical and ethical issues, studies of compressive frequency, duty cycle, or waveform during CPR are often limited to animal experimentation or computer simulations [21,29–31]. However, this knowledge is an indispensable prerequisite for developing automated mechanical devices to guarantee optimal perfusion during CPR. Furthermore, CPR training is flawed, given the unrealistic and static thoracic rigidity of dummies used. Chest rigidity and recoil are generated by springs in commercially available phantoms for CPR training, and rigidity is fixed in these models [32,33].

A number of researchers have explored the variability of thoracic stiffness during resuscitation. Nysaether et al. [33] measured compressive forces and damping characteristics during CPR in 59 patients. A wide range (50–500 N) of compressive force was documented, which varied by depth of compression (20 mm: 50–150 N; 30 mm: 100–400 N; 50 mm: 200–500 N); and damping factor (150–240 Ns/m) varied as well (20 mm: 150 Ns/m; 30 mm: 240 Ns/m). Bankman et al. [34] also recorded stiffness in a healthy volunteer (20 mm: 150–200 N) as a function of compressive velocity. In a study by Tomlinson et al., [35] average stiffness and force-compressive depth values were determined in 91 patients. Compressive force ranged from 50–500 N (20 mm: 50–180 N; 30 mm: 100–280 N; 40 mm: 150–500 N). Average stiffness (calculated: $F_{38\ mm}/2F_{19\ mm}$) was 1.41 ± 0.25 N/mm. Another study by Neurauter et al. [36] involved 90 patients suffering cardiac arrest. Average chest stiffness (calculated) ranged from 4.5–7.7 N/mm, again varying by depth of compression (15 mm: 4.5 N/mm; 30 mm: 5.7 N/mm; 50 mm: 7.7 N/mm). Using a modified Thumper device (Michigan Instruments, Grand Rapids, MI, USA) on 11 patients, Tsitlik et al. [37] determined an average chest elasticity of 54.9 ± 29.1 N/cm, an average maximum compressive force of 414 ± 73 N, and an average maximum compressive depth of 4.34 ± 0.82 cm. Finally, Gruben et al. [38] measured average maximum compressive force (431 ± 30 N), average maximum compressive depth (3.83 ± 0.56 cm), and average stiffness (37.4–282 N/cm, dependent on compressive depth) during 16 clinical resuscitation attempts. Of note, no standardised examinations, implemented according to accepted guidelines for effective CPR (frequency, 100/min; compressive depth, 50 mm) and using a defined testing format, could be found in the literature.

Our intent, therefore, was to generate a mechanical model of the thorax, capable of realistic variation in chest stiffness, with an integrated blood flow system reflecting the two main physiologic influences discussed in the literature. Through suitable experimental investigations, the ability to mechanically reproduce physiologic parameters of CPR could thereby be proven.

2. Materials and methods

2.1. Experimental testing

To define chest stiffness in the model, data in the literature were reviewed (see Section 1). However, most determinations were based on manual chest compression or involved compressive frequencies/depths that deviated from actual resuscitation guidelines (frequency, 100/min; depth, 50 mm); so our own measurements were obtained from 20 fresh cadavers. This investigation was approved by the local Ethics Committee (#141/14, TUM). Chest elasticity measurements in cadavers (male, 65%; female, 35%; age: 74 ± 13 years; height, 1.7 ± 0.11 m; weight, 80.15 ± 15 kg; body mass index [BMI], 27.9 ± 4.3; time after death, 70 ± 22 h) were carried out using a rigid load frame (Fig. 1) equipped with a combined motor-driven plunger (#148,826, 250 W; Maxon Motors AG, Sachseln, Switzerland) and linear module (KK 0,6005; HIWIN Corp, Offenburg, Germany). To measure displacement curve force, the plunger was equipped with a load cell (KM-40; 2 kN; ME-Meßsysteme, Hennigsdorf, Germany) and a way transducer (MLO-POT-100-LWG; Festo AG, Esslingen, Germany). The plunger, initially run for 10 cycles in sinusoidal waveform (frequency, 10/min; compression depth, 30 mm) to condition the chest, was boosted thereafter (frequency, 100/min; compression depth, 50 mm). Load displacement curves during simulated CPR were electronically recorded (PowerLab; AD Instruments, Oxford, UK). Setup of the plunger is also shown in Fig. 1.

Chest stiffness was defined by linear regression of the force displacement curve from 30–50 mm. The average of 15 cycles was recorded. Statistical computations relied on standard software (SPSS v22; SPSS Inc, Chicago, IL, USA), applying Pearson's correlation test to assess any relationship between chest stiffness and age, gender, postmortem time, or BMI.

2.2. Construction of mechanical thorax

Our model of thorax incorporates a mechanical element, enabling variable rigidity, and a blood-flow simulation unit. A detailed model overview is provided in Fig. 2. Based on published data and our own measurements, the model range for stiffness is 6–16 N/mm, for a maximum force of 300–800 N at compressive depth of 50 mm. The mechanical aspect was constructed using a pneumatic system. Three pneumatic pistons (ADN-32-100-A-P-A-S11; Festo AG), regularly fed compressed air and connected to an adjustable motor-controlled valve (ST4118L0804; Nanotec, Munich, Germany) offer variable chest resistance, combining three springs (0X-DF2091, C = 0.57; Febrotec, Halver, Germany) for faster chest recoil after compression. Both pistons and springs are arranged radially on a base plate, surrounding the blood-flow unit. As haptic interface, a rubber-coated compression plate was attached to the cylinders, bearing a way transducer (WS 31; ASM GmbH, Moosinning, Germany) for detecting depth and number of compressions. The flow-generating unit itself consists of a manufactured one-chamber silicone heart (Silastic T4; Ebalta, Rothenberg, Germany), with a filling volume of 130 ml. Inlet and outlet of the heart are separated by two mechanical heart valves (Tekna, 22 mm; Edwards, Irvine, CA); and a fluid reservoir, with a bubble trap (D729 Microtrap; LivaNova, London, UK), sits in between to simulate variable cardiac preload via height of the hydrostatic head. The heart resides in an airproof chamber made of polymethylmethacrylate (PMMA) that is connected to the mechanical component, with a gaiter containing an adjustable-length plunger. Upon compression plate movements, the gaiter volume declines, pressure inside the chamber rises, and the heart is sequentially compressed, simulating intrathoracic pressure fluctuations. The silicone heart model is also compressed by the variable plunger, accounting for cardiac compression during CPR. Through valve-controlled venting of the airproof chamber, an isolated heart compression (due to plunger alone) is also feasible.

The pressure regulatory valves in the pneumatic pistons (and thus simulated chest pressure), as well as the positioning mechanism of the compressive plunger and the way transducer (relaying both cycle count and compressive depth), are manipulated by a personal computer that communicates with an internal microcontroller (ATmega 328; Atmel Corp, San Jose, CA, USA). Setting variations for each component are visualised in a display panel for direct modification via button interface or by personal computer. Stiffness of the model can be changed automatically, depending on the number of compressions that have been performed, or

Please cite this article as: S. Eichhorn et al., Development and validation of an improved mechanical thorax for simulating cardiopulmonary resuscitation with adjustable chest stiffness and simulated blood flow, Medical Engineering and Physics (2017), http://dx.doi.org/10.1016/j.medengphy.2017.02.005