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Experimental paper

Comparison of mechanical characteristics of the human and porcine chest during cardiopulmonary resuscitation☆

Andreas Neurauter[a], Jon Nysæther[c], Jo Kramer-Johansen[d], Joar Eilevstjønn[c], Peter Paal[a], Helge Myklebust[c], Volker Wenzel[a], Karl H. Lindner[a], Werner Schmölz[b], Morten Pytte[d], Petter A. Steen[d], Hans-Ulrich Strohmenger[a,*]

[a] Department of Anesthesiology and Critical Care Medicine, Innsbruck Medical University, Anichstrasse 35, 6020 Innsbruck, Austria
[b] Department of Trauma Surgery and Sports Medicine, Innsbruck Medical University, Innsbruck, Austria
[c] Laerdal Medical AS, Stavanger, Norway
[d] Institute for Experimental Medical Research, Department of Anaesthesiology and Pre-hospital Division, Ulleval University Hospital and University of Oslo, Oslo, Norway

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ABSTRACT

Background: Most studies investigating cardiopulmonary resuscitation (CPR) interventions or functionality of mechanical CPR devices have been performed using porcine models. The purpose of this study was to identify differences between mechanical characteristics of the human and porcine chest during CPR.
Material and methods: CPR data of 90 cardiac arrest patients was compared to data of 14 porcine from two animal studies. Chest stiffness k and viscosity μ were calculated from acceleration and pressure data recorded using a Laerdal Heartstart 4000SP defibrillator during CPR. K and μ were calculated at chest compression depths of 15, 30 and 50 mm for three different time periods.
Results: At a depth of 15 mm porcine chest stiffness was comparable to human chest stiffness at the beginning of resuscitation (4.8 vs. 4.5 N/mm) and clearly lower after 200 chest compressions (2.9 vs. 4.5 N/mm) ($p < 0.05$). At 30 and 50 mm porcine chest stiffness was higher at the beginning and comparable to human chest stiffness after 200 chest compressions. After 200 chest compressions porcine chest viscosity was similar to human chest viscosity at 15 mm (108 vs. 110 Ns/m), higher for 30 mm (240 vs. 188 Ns/m) and clearly higher for 50 mm chest compression depth (672 vs. 339 Ns/m) ($p < 0.05$).
Conclusion: In conclusion, human and porcine chest behave relatively similarly during CPR with respect to chest stiffness, but differences in chest viscosity at medium and deep chest compression depth should at least be kept in mind when extrapolating porcine results to humans.

1. Introduction

During cardiopulmonary resuscitation (CPR), closed chest compressions are performed to maintain coronary and cerebral perfusion, and several animal studies have shown that quality and performance of CPR correlate directly with survival from cardiac arrest.[1–3]

CPR guidelines recommend a chest compression depth of 4 to 5 cm for adults independently of sex, body size, and physical status.[4] In the past, many CPR experiments were performed in canine models, but porcine models are currently more popular. Also, rodent models are good screening tools, but they are too small in scale to allow direct extrapolation to humans. Thus, most experimental CPR studies in the last 20 years have been performed using porcine models with extrapolation to human CPR irrespective of comparability between the human and porcine body. Unfortunately, many interventions that were highly effective in CPR laboratories have failed in clinical studies.[5–7] While some of these effects have been attributed to differences between "laboratory CPR" and "real life CPR on the street",[8] differences in chest configuration between humans and pigs are poorly understood, but may explain, in part, the observed problems to extrapolate interventions from bench to bedside.

The purpose of this study was to identify differences in mechanical characteristics of the human and porcine chest during CPR.

2. Materials and methods

2.1. Human data

The raw human data were obtained from an observational prospective study of out of hospital cardiac arrest patients between October 2004 and June 2005.[9] Approval for this study was obtained

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* Corresponding author. Tel.: +43 512 504 22400; fax: +43 512 504 22450.
E-mail address: hans.strohmenger@i-med.ac.at (H.-U. Strohmenger).

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from the Regional Committee for Research Ethics and the Norwegian Data Inspectorate. Patient data were collected prospectively by emergency medical services in London, England; Stockholm, Sweden; and Akershus, Norway.[9] Resuscitation attempts were performed according to the CPR 2000 guidelines.[10] In this study CPR recordings of 90 out of hospital cardiac arrest patients between 18 and 92 years were analysed.[11]

Chest compression and ECG data were obtained from custom built, CE marked Heartstart 4000SP defibrillators with extended measurement and data recording capabilities. This includes the forces that the sternum is exposed to, and the resulting accelerations of the sternum during chest compressions via pressure and acceleration sensors in a special chest pad. This pad was mounted on the lower part of the patient's sternum with double adhesive tape and the heel of the rescuer's hand was placed on top of the chest pad during CPR.[12] Chest compression depth was calculated continuously by the defibrillator by double integration of the effective acceleration signal.[12,13] Chest compression rate and depth were visualised on a screen. All channels, including the calculated chest compression depth, were recorded with a sampling rate of 500 Hz and digitalised with a resolution of 16 bit. Advanced life support (ALS) management information and demographic data of the patients were documented according to the Utstein guidelines,[14] and linked to the electronic data; details are described elsewhere.[12]

2.2. Porcine data

The animal dataset ($n = 14$) consisted of two parts: part one was seven pigs receiving CPR with manual chest compressions in a study of haemodynamic effects of adrenaline (epinephrine) during cardiopulmonary resuscitation (Oslo study).[8] The experiments were conducted in accordance with the "Regulations on Animal Experimentation" under The Norwegian Animal Welfare Authority Act and approved by the Norwegian Animal Research Authority. Domestic pigs of both sexes (weight, 23–30 kg) were anesthetised and instrumented for measurement of coronary perfusion pressure, cerebral blood flow, femoral blood flow, adrenaline plasma concentration, and slope ventricular fibrillation (VF) analysis. After 4 min of untreated VF, basic life support (BLS) was performed for 4 min, followed by ALS. Thirty seconds after initiation of ALS, adrenaline (0.02 mg/kg) was administered. Manual chest compressions were performed at a rate of 100 min^{-1} and interrupted every 15 chest compressions for 9 s to ventilate the animal twice. A custom built Heartstart 4000SP defibrillator, identical to the one used for collecting the human data above, was used for data recording. The chest pad was mounted on the lower part of the shaved sternum with double adhesive tape. Chest compression depth and rate were continuously displayed on the screen. Two lines on the screen at 30 and 38 mm depth served as target for a single investigator who performed the chest compressions.

The second part ($n = 7$) of the porcine data originated from a study evaluating effects of varying degrees of stomach inflation on haemodynamic and pulmonary function during CPR (Innsbruck study).[15] In the present study data from the group receiving 0 L stomach inflation ($n = 7$) were used for analysis (weight, 34–46 kg). This project was approved by the Austrian Federal Animal Investigation Committee, and animals were managed in accordance with the American Physiologic Society, institutional, and Utstein-style guidelines,[14] and the position of the American Heart Association on Research Animal Use, as adopted on November 11, 1984. Healthy domestic pigs of both sexes were anaesthetised and instrumented for evaluation of haemodynamic and pulmonary function. After 4 min of untreated VF, BLS was performed for 3 min. Subsequent ALS was started with a chest compression rate of 100 min^{-1} and target chest compression depth of 38–50 mm.[15] An identical

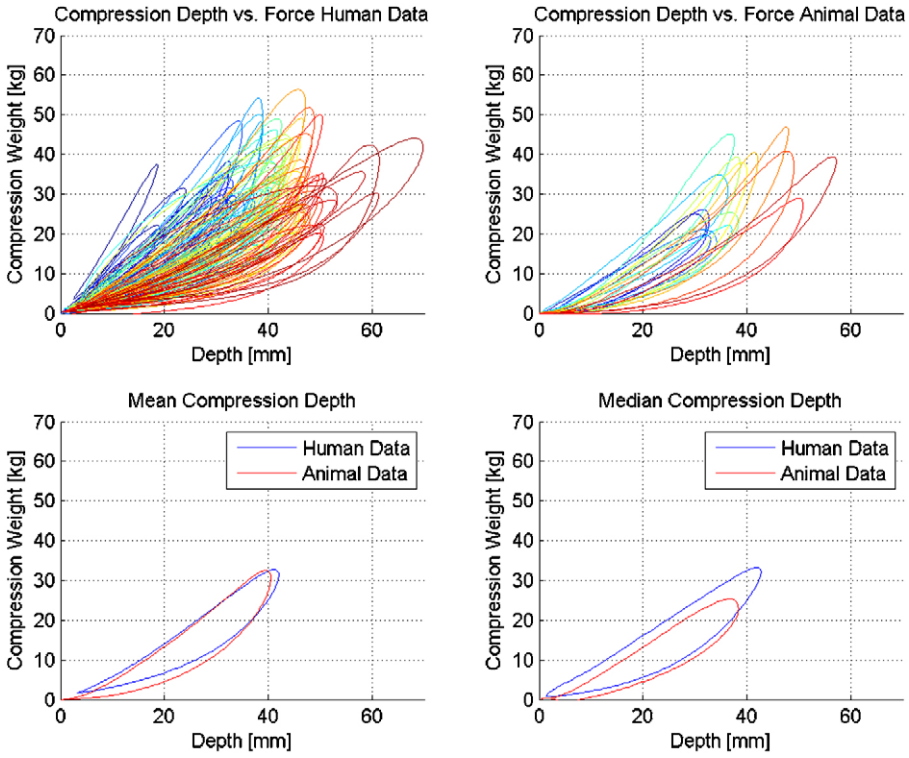

Fig. 1. Force–displacement curves describing human and porcine mechanical chest characteristics during CPR. The top left plot gives one force–displacement hysteresis for each observed patient, where line colours correspond to maximal reached compression depth. Top right shows the same kind of plots for the investigated pigs. The two plots bellow give the resulting mean and median curves for human and animal dataset.

custom built Heartstart 4000SP defibrillator was used to record data as in the human and the other porcine study. Chest compression force-displacement curves of the human and porcine dataset are shown in Fig. 1.

Because of variations in interruption to the chest compressions in the different study protocols, the actual number of chest compressions per minute was lower than the chest compression rate. Mean (±S.D.) number of chest compressions per minute for the first 240 chest compressions was 71.5 (±23.1) in the human dataset, 52.3 (±4.0) in pigs from Oslo and 90.6 (±4.5) in pigs from Innsbruck.

2.3. Analysis of chest characteristics

Programs written in Matlab version 7 (Natick, MA, USA) were used for signal processing, stiffness and viscosity calculations and for statistical analysis. The CPR traces of the human cardiac arrest patients and animals were analysed retrospectively.[16]

The recordings were used to calculate system parameters of a simple spring damper model in order to characterise the chest (Fig. 2) assuming that the elastic chest properties referring to the ribs and sternum can be described by the stiffness k. By analogy, the viscosity properties of the chest, assumed to refer to blood flow, tissue properties and movement of abdominal and thoracic viscera were included into this model as damping characteristic μ.[17] The physical properties of this model define the following differential equation:[18]

$$F_{\text{ext}} + mg = -kd - \mu\dot{d} - m\ddot{d} = F_{\text{el}} + F_{\mu} + F_{\text{m}} \quad (1)$$

with force components:

- F_{ext}: external force applied by the rescuer
- F_{el}: elastic force, caused by chest stiffness responsible for relaxation

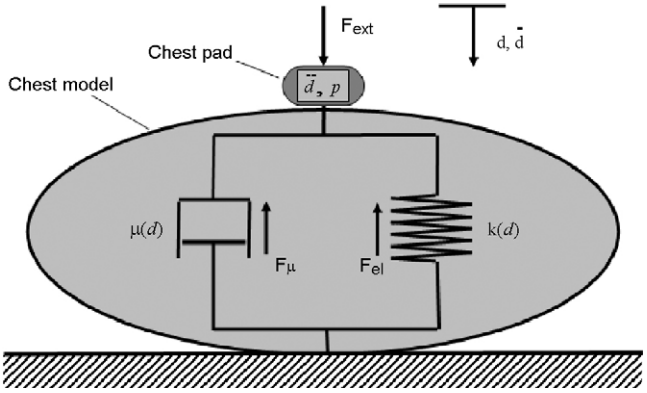

Fig. 2. Schematic of the second order spring dashpot model used to describe the human and porcine chest. The model consists of a parallel structure of a spring with stiffness $k(d)$ and a dashpot of viscosity $\mu(d)$. The chest pad, which includes acceleration and pressure sensors, is placed on top of the chest. The external force F_{ext} that is applied by a rescuer induces two system forces F_{el} and F_{μ} that correspond to the elastic and viscous behaviour of the chest.

- F_{μ}: damping force, caused by viscosity properties of the chest; responsible for delaying compression as well as relaxation process
- F_{m}: inertia force referring to the moving mass

and model parameters:

- d: chest displacement; its first and second derivates $\dot{d}$, $\ddot{d}$ describe vertical sternum velocity and acceleration
- m: moving mass
- k: chest stiffness
- μ: viscosity of the chest

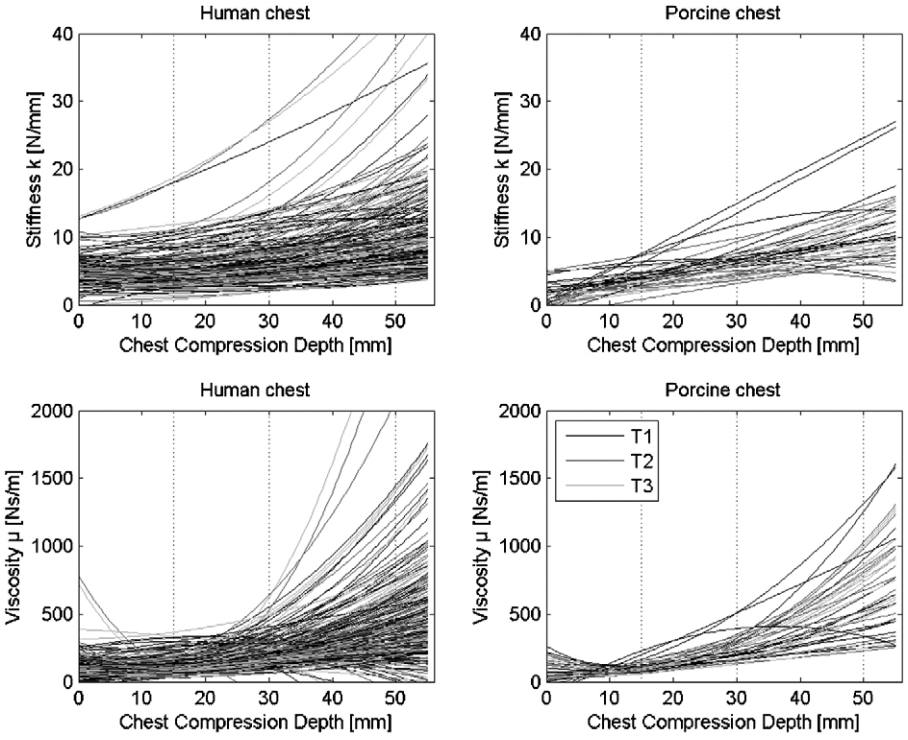

Fig. 3. Depth depended second order polynomial fits of chest stiffness k and chest viscosity μ for the human and porcine dataset. The line shade represents the different time periods of measurement; T1: at CPR onset (dark grey); T2: after 100 chest compressions (grey); T3: after 200 chest compressions (light grey). In most cases, a depth-dependent increase of k and μ is obvious for humans as well as for pigs. Note that these figure just give a general overview of all stiffness and viscosity curves, mean values and range are more emphasised in Fig. 4.

Inertia force F_m is rather small compared to elastic and damping force and was therefore not considered further.[16] From the depth and the force channel stiffness k and viscosity μ, were calculated the following way:[18]

Every single chest compression was extracted by finding all local maxima and minima in the force and chest compression depth traces. To increase the precision level single depth and force curves were tested for artefacts and smoothness and then averaged in groups of ten. For all N averaged chest compressions of one patient, the depth-dependent chest stiffness $k_n(d)$ and depth-dependent viscosity $\mu_n(d)$ were estimated by solving Eq. (1).

To enable comparisons, chest stiffness $k_n(d)$ and viscosity $\mu_n(d)$ were analysed for three displacement steps d at 15, 30 and 50 mm. If the depth of 50 mm was not reached during a chest compression, the 50 mm value of $k_n(50)$ and $\mu_n(50)$ was extrapolated from $k_n(d)$ and $\mu_n(d)$ using a second order polynomial curve fitting as second order polynomial fitting was found to be more adequate than simple linearisation (Fig. 3). However, in some cases (<5%) using second order polynomials resulted in a decrease of viscosity and negative values for $\mu_n(50)$. The calculated values were used for time-dependent investigations of individual changes of chest characteristics during ongoing CPR.

To assess possible changes in chest characteristics with the number of chest compressions applied, we extracted stiffness and viscosity values for three different time periods in each episode. To achieve better comparability of the three groups and to cover the influence of CPR, we used the number of chest compressions being applied to the patient as a measure for time. The first period of observation started after the first chest compression at the beginning of each episode, the second after 100 and the third after 200 chest compressions. For all periods, each lasting 40 chest compressions, mean stiffness $k(15)$, $k(30)$ and $k(50)$ and mean viscosity $\mu(15)$, $\mu(30)$ and $\mu(50)$ were computed.

2.4. Statistical analysis

Data were tested with the Jarque-Bera-test for normal distribution. As all stiffness and viscosity parameters of the human data fit to normal distribution, while none of the animal data do, all values are given as median and range. For analysing changes of chest stiffness k and viscosity μ over time, we compared the mean values at the onset of CPR to those values after 200 chest compressions using the Wilcoxon signed rank test. The Kolmogorov–Smirnov test was performed to compare the characteristics of the porcine chest to those of the human chest. Statistical difference was considered significant at $p < 0.05$.

3. Results

Human and animal chest stiffness k and viscosity μ values at all three periods of observation are as shown in Fig. 4 and Table 1.

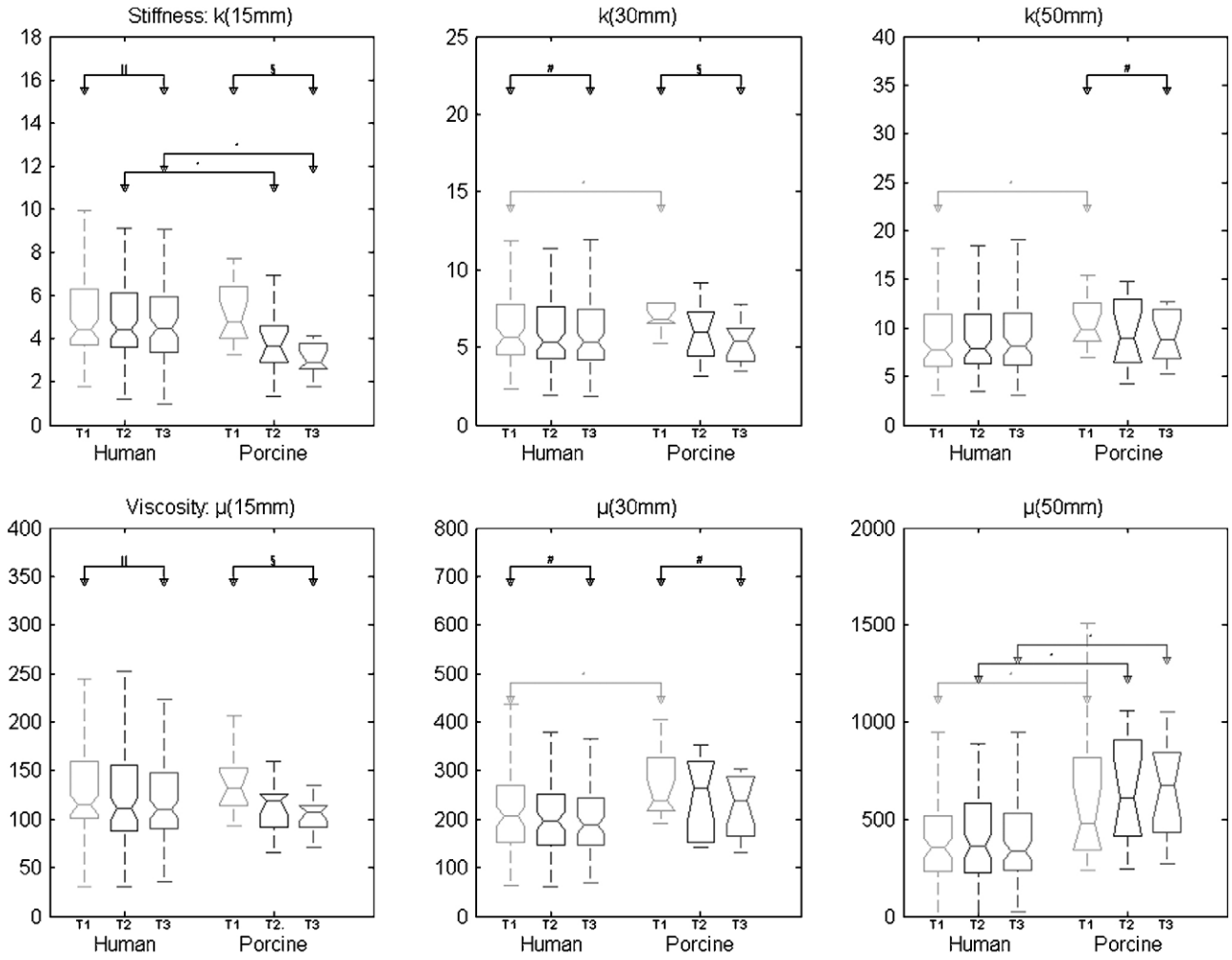

Fig. 4. The box plots show distributions of human and porcine stiffness parameter k and viscosity parameter μ for 15, 30 and 50 mm chest compression depths. Each parameter is given for three different time periods at CPR onset (T1), after 100 (T2), and after 200 chest compressions (T3). Significance levels of the Wilcoxon signed rank test for differences between starting and end point are symbolised above the box plots. '#', '||' and '§' correspond to $p < 0.05$, $p < 0.01$ and $p < 0.001$, respectively. Different distributions according to Kolmogorov–Smirnov test at $p < 0.05$ between human and porcine parameters are marked with a '*'.

Table 1
The median and range of porcine and human chest stiffness k and viscosity μ at 15, 30 and 50 mm depth corresponding to Fig. 4. The values are given for the three different time periods starting at CPR onset, after 100 and after 200 chest compressions.

Stiffness k		k(15 mm)	k(30 mm)	k(50 mm)
Human	T1	4.5 [1.8–18.5]	5.7 [2.3–25.3]*	7.7 [3.1–36.7]*
	T2	4.4 [1.2–18.3]*	5.4 [1.9–27.4]	7.9 [3.4–45.2]
	T3	4.5 [1.0–18.7]***	5.4 [1.8–26.8]	8.1 [3.0–41.6]
Porcine	T1	4.8 [3.3–7.7]	6.8 [5.3–14.7]	9.8 [6.9–24.7]
	T2	3.7 [1.3–6.9]	6.0 [3.2–9.2]	8.9 [4.2–14.8]
	T3	2.9 [1.8–6.0]	5.4 [3.4–7.8]	8.8 [5.2–12.7]
Viscosity μ		μ(15 mm)	μ(30 mm)	μ(50 mm)
Human	T1	115.2 [31.0–339.4]	206.0 [65.1–640.1]*	353.9 [−478.7–1640.1]*
	T2	110.8 [30.7–316.3]	196.8 [−30.0–632.3]	362.1 [−874.4–2513.7]**
	T3	110.1 [35.3–360.1]	187.7 [68.5–583.1]	338.7 [−536.4–3076.2]**
Porcine	T1	132.6 [93.4–219.6]	237.9 [191.6–572.3]	478.8 [236.3–1510.5]
	T2	119.8 [65.6–160.0]	265.7 [140.7–353.6]	610.7 [247.2–1059.0]
	T3	107.7 [70.6–134.6]	239.4 [131.1–303.5]	672.3 [272.7–1054.3]

Significance levels of the Kolmogorov–Smirnov test for differences between human and porcine chest characteristics are givens as * for $p < 0.05$, as ** for $p < 0.01$ and as *** for $p < 0.001$.

Within the animal dataset, a significant time-dependent decrease of chest stiffness k was found for all three chest compression depths. For 15 mm and 30 mm chest compression depth viscosity μ also decreased significantly, but a non-significant increase of $\mu(50)$ was found. The decrease of $\mu(15)$ and $\mu(30)$ was found in 12 out of 14 animals and can therefore be regarded as a consistent individual and not just an average effect. In the human dataset, significant differences were found between first and third period of observation for $k(15)$, $k(30)$, $\mu(15)$ and $\mu(30)$. Despite the fact that these changes were significant, the differences of absolute values (as represented by median and range) are quite small.

Comparison of human and animal data revealed similar chest stiffness and viscosity values in the beginning of the CPR episodes at 15 mm chest compression depth ($k(15)$ and $\mu(15)$). With increasing depth of chest compression, chest stiffness k and viscosity μ values in animals were significantly higher than the human values. However, after 200 chest compressions the only significant differences between pigs and humans were lower stiffness at 15 mm and higher viscosity at 50 mm chest compression depth for pigs.

4. Discussion

Both chest stiffness k and viscosity μ were initially higher in pigs than in humans at clinically relevant chest compression depths of 30 and 50 mm. At these chest compression depths there was a significant decrease in the pigs' chest stiffness over the first 200 chest compressions and as there was less change in humans, differences in stiffness between humans and pigs were no longer significant after 200 chest compressions. However, viscosity increased further and remained higher in pigs than humans at 50 mm chest compression depth.

In the present viscoelastic chest model described by Gruben et al.,[16] a dashpot is reported to reflect the damping characteristics of the chest. Damping is caused by mechanical chest compression of muscles and connective tissue as well as by movements of thoracic and abdominal viscera, and blood.[17] A spring connected in parallel to the dashpot reflects the elastic behaviour of the chest caused by the ribcage cartilage and bones and is described by the stiffness k.[19] With ongoing CPR the thoracic stiffness k for medium and deep chest compressions were comparable for humans and pigs which indicates that a similar static load will result in a similar displacement of the sternum in both species. Depending on chest compression depth, however, interspecies differences in viscosity will cause differences in the forces needed for chest compression.

Importantly, the viscose force component consumes to a certain degree energy applied by the rescuer. Thus, the rescuer needs to release the chest completely which reduces time until rescuer fatigue starts. The following simplified example illustrates what proportion of the force applied is consumed by damping. With triangular shaped chest compressions at a rate of 100 min^{-1}, one chest compression cycle lasts 0.6 s, and a chest compression depth of 50 mm results in a mean velocity of 0.166 ms^{-1}. Correspondingly, a medium viscosity $\mu(30)$ of 200 Ns/m would result in a viscous force component F_μ of 33 N. When assuming the same chest compression velocity until a depth of 50 mm which is an overestimation, viscosity at 50 mm depth, $\mu(50)$, of 350 Ns/m results in a viscous force component F_μ of around 58 N. The maximum elastic Force F_{el} needed to compress a chest with stiffness k of 8 N/mm to a depth of 50 mm would then be 400 N. Accordingly, the viscous component F_μ would be more than 10% (58 N/458 N) of the overall force. However, typical chest compressions are not triangular shaped, chest compression duration is shorter than relaxation duration, and movement is interrupted for a short moment in both in the maximum chest compression and decompression position. Thus, the CPR pattern is steeper and chest compression velocity faster than estimated above. In our study, we could measure maximum chest compression velocities of up to 0.5 ms^{-1} at chest compression depths of around 30 mm. With corresponding $\mu(30)$ this results in peak values of F_μ of up to 100 N which is 25% of F_{el}. Especially in mechanical devices, the pattern might even be steeper and velocities higher.[20,21] Thus, depending on how CPR is performed F_μ can become quite large which indicates also larger differences between humans and animals.

At low chest compression depths, the decrease of stiffness and viscosity with time was higher in pigs than in humans. In 39 patients without bystander CPR in the another study,[11] the average force needed to compress the chest 25 mm gradually decreased throughout the study from 13.5 kg to 12.2 kg (132.4 N to 119.7 N) after 1000 chest compressions but different time courses existed. One explanation for this interspecies difference might be due to the fact that a significant number of the patients in the present study had bystander CPR before study initiation which of course, was not the case for animals. Thus, an initial decrease in stiffness in humans might already have occurred. In both groups, the decrease of chest stiffness k could be explained by chest wall injuries and rib fractures which are a well known side effect of CPR.[22]

One reason for differences in chest viscosity between humans and animals might be located in the shape of the porcine chest and the structure of the sternum. The porcine anterior–posterior

chest diameter in our dataset was median 230 mm with a range of 219–247 mm, which is comparable to 253 ± 27 mm for males and 235 ± 30 mm for females in a study of 100 human adults.[23] However, for the human chest the frontal to anterior-posterior chest diameter is larger than in pigs (1.5 times vs. 1.25 times), which results in a different thoracic shape. Another possible reason for the differences found in chest viscosity in this study might be that we compare human data of a higher aged patient population to data of quite young pigs.

There are several limitations in this study. Although it is a strength that the animal data are combined from two laboratories using the same measuring device as in the human study with data from emergency medical services in three countries, the overall number of animals is still limited. While all pigs were of approximately the same size and age, there were significant variations in patient size and age. On the other hand, this should make the data representative, as most experiments are done in young pigs, and the present human dataset reflects a standard cardiac arrest patient population. There are also limitations regarding the model parameter calculations. Stiffness and viscosity at 50 mm were extrapolated using a second order polynomial in cases where 50 mm chest compression depth was not reached. This method delivered useful results for most cases; however in some cases, these fits resulted in values that were obviously much smaller than expected. It has also to be mentioned that most reliable measurements of viscosity are performed during the movement phase when the chest has a significant velocity. However, $\mu(50)$ is located near the turning point where the velocity is low, thus making an accurate measurement of μ more difficult. Accordingly $\mu(50)$ values might be less accurate than $\mu(30)$ values.

The spring-dashpot model is also only a very rough simplification of the chest and its environment. Thus, the compliance of the surface on which cardiac arrest patients received CPR was not taken into account. All pigs received CPR on a rigid surface, and according to the patient report forms, this was the case also for most patients (floor or ground outside). A soft surface would overestimate the chest compressions and thereby influence calculated chest properties.

In conclusion, human and porcine chest behave relatively similar during CPR with respect to chest stiffness, but differences in chest viscosity at medium and deep chest compression depth should at least be kept in mind when extrapolating porcine results to humans.

Conflict of interest

The study was supported, in part, by Laerdal Medical AS, Stavanger, Norway, which offers products and system solutions for health care providers with particular emphasis on emergency medicine and training. At the time of the study, Helge Myklebust and Joar Eilevstjønn and Jon Nysæther were employees of Laerdal Medical. Petter A. Steen was a Board member of directors of Laerdal Medical. Andreas Neurauter received financial support from Laerdal Medical for this research.

Appendix

Eq. (1) writes

$$F_{\text{ext}} + mg = -kd - \mu\dot{d} - m\ddot{d} = F_{\text{el}} + F_{\mu} + F_{\text{m}} \quad (1)$$

Inertial force is expected to be small therefore the moving mass of the chest can be ignored[16] and we get the following simplification:

$$F_{\text{ext}} = -kd - \mu\dot{d} = -kd - \mu v \quad (2)$$

Using MATLAB the following algorithm is performed for all data recordings:

- Detecting every single compression.
- Interpolating all detected curves to a standardised time base.
- Averaging force curves and depth cure for $n = 10$ subsequent compressions.
- Calculating velocity curve and acceleration curve from depth curve by differentiation.
- Splitting all curves into two parts: the compression and release part.
- By subtracting releasing force from compressing force at a specific compression depth × Eq. (2) becomes:

$$F_{\text{comp}} - F_{\text{release}} = k(d_{\text{comp}} - d_{\text{release}}) - \mu(v_{\text{comp}} - v_{\text{release}}) \quad (3)$$

as x_{comp} is equal x_{release} we derive:

$$F_{\text{comp}}(d) - F_{\text{release}}(d) = \mu(d)(v_{\text{comp}} - v_{\text{release}}) \quad (4)$$

- For each depth d the viscosity $\mu(d)$ is calculated by

$$\mu(d) = \frac{v_{\text{comp}}(d) - v_{\text{release}}(d)}{F_{\text{comp}}(d) - F_{\text{release}}(d)} \quad (5)$$

- Chest stiffness $k(x)$ is derived by solving:

$$k(d) = \frac{F_{\text{comp}}(d) - \mu(d)v_{\text{comp}}(d)}{d} \quad (6)$$

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