Could Post-mortem Computed Tomography Angiography Inform Cardiopulmonary Resuscitation Research?

Guy N. Rutty^a, Claire Robinson^b, Jasmin Amoroso^a, Tim Coats^c, Bruno Morgan^b

^a University of Leicester, East Midlands Forensic Pathology Unit, Robert Kilpatrick Building, Level, Leicester Royal Infirmary, Leicester LE2 7LX, UK

^b University of Leicester, Radiology Department, Windsor Building, Leicester Royal Infirmary, Leicester, LE2 7LX, UK

^c University of Leicester, Cardiovascular Sciences, Jarvis Building, Leicester Royal Infirmary, Leicester, LE2 7LX, UK

*Corresponding author. Fax: 0044 116 252 3274.

E-mail address: gnr3@le.ac.uk (G.N.Rutty)

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Abstract

Aim: Firstly, to develop an optimised chest compression post mortem computed tomography angiography protocol in the adult human during closed chest compression to investigate cardiopulmonary resuscitation blood flow, and secondly to provide preliminary observations of post-mortem anatomical cardiac chamber movement using a novel radiolucent static chest compression device.

Methods: Variable volumes of radiological contrast agent were injected intravenously into a series of consented human cadavers. Each cadaver had chest compressions delivered with a LUCASTM2 mechanical chest compressor. Following each cycle of chest compressions, each cadaver was imaged with a Toshiba Aquilion CXL 128 slice computed tomography (CT) scanner to investigate the extent of contrast distribution. A chest compression simulator was then designed and built to allow static CT imaging of 1cm incremental cadaver chest compressions to a depth of 5cm.

Results: Mechanical compressions: Ten cases were recruited for the CT angiography component of the study. Two were subsequently excluded from the study at the time of the initial, non-contrast PMCT scan. A further case was recruited in Emergency Department (ED). CT demonstrable antegrade arterial contrast distribution was achieved in 2 cases. The other 7 cases, including that undertaken in ED shortly after death, showed venous retrograde flow. Incremental compressions: Five new cases underwent incremental chest compression imaging. All cases demonstrated compression of the sternum, ribs, atria and great vessels. The right and left ventricles were not compressed, but moved laterally and inferiorly, further into the left chest cavity. The left hemi-diaphragm, stomach and liver moved inferiorly. The sternum, ventricles, hemi-diaphragm, stomach and liver all moved back to their original position on incremental release.

Conclusion: The study suggests that with further protocol modification and access to human cadavers as near to death as possible, chest compression post mortem computed angiography (CCPMCTA) could be used as a model for the study of human vascular flow and heart movement during CPR.

Introduction

There is good evidence that cardiopulmonary resuscitation (CPR) protocols can increase return to spontaneous circulation (ROSC) if started early enough and particularly if the patient is in a shockable rhythm. The 2015 International Consensus on Cardiopulmonary Resuscitation (CPR) and Emergency Cardiovascular Care (ECC) Science with Treatment Recommendations (CoSTR) provide protocols and a summary of the published evidence for the delivery of basic and advanced life support.^{1,2} However, the experimental evidence-base behind these protocols is weak, due to the difficulties in undertaking human research.

Closed (external) chest compressions (CCC) as a form of CPR have been described in the literature for over 100 hundred years³, but were introduced into modern medical practice by the work of Kouwenhoven *et al.*⁴ in 1960. The principle was that CCC caused forward blood flow, but this was almost immediately questioned.⁵ Researchers have subsequently developed two mechanistic theories for CCC; the ‘cardiac pump’ and the ‘thoracic pump’ theories. Most research for these theories derives from animal models, and limited human research exists.⁶ Echocardiography has been used to show dynamic human heart anatomy during chest compressions.⁷⁻¹⁴ Computed tomography (CT) has been used to define the optimum hand position to deliver chest compressions in adults and children.¹⁵⁻¹⁸

In recent years, post-mortem computed tomography (PMCT) has become established throughout the world as an adjunct and more recently a replacement to the invasive autopsy.¹⁹ Techniques have been developed to deliver and circulate computed tomography (CT) contrast agents during PMCT, to simulate contrast enhanced CT used in clinical imaging. Three methods have emerged: multiphase PMCT angiography, targeted coronary artery PMCT angiography and chest compression PMCT angiography (CCPMCTA).²⁰ CCPMCTA was developed in Japan, where surgical management of the newly deceased patient, which is required for other PMCTA techniques, is traditionally shunned.²¹⁻²³ In CCPMCTA within 3 hours of death up to 150ml of clinical contrast is delivered via a peripheral vein, usually an antecubital fossa vein, by pump injector, at a rate of 1-2mls/s. Whilst injecting, chest compressions are delivered for 2 mins at a rate of 100 compressions/min. A pump injector is used, as initial studies found that hand injection followed by chest compressions did not cause diagnostically useful arterial angiograms, with most contrast showing retrograde flow into the venous system.^{20,24,25}

We propose that PMCTA may provide a novel “reverse-translation” human method to study the effect of cardiac compression on blood flow during CCC by dynamically imaging anatomical changes of the cardiac chambers during chest compression, and demonstrating any subsequent circulation of injected CT contrast agents. This could provide a measurement of the efficacy of CCC, allowing development of improved protocols.

The aim of this study was firstly to develop an optimised chest compression post mortem computed tomography angiography protocol to investigate cardiopulmonary resuscitation blood flow in the adult human during closed chest compression, and secondly to provide preliminary observations of post-mortem anatomical cardiac chamber movement using a novel radiolucent static chest compression device.

Methods

Ethics

The study has full ethical approval from the National Research Ethics Service (NRES) (04_Q2501_64, Amendment 7 and 10), and University Hospitals of Leicester Research and Development Office (UHL09523).

Recruitment

We prospectively selected non-consecutive natural death study participants referred and authorised by Her Majesty's (HM) Coroners for invasive autopsy examination. The cases were selected based on the first suitable referral for post-mortem investigation received by secure fax machine on a chosen study day. Exclusion criteria were: age less than 18, known transmittable disease (e.g. tuberculosis, HIV or hepatitis C), a shoulder width of greater than 70cm and weight over 125kg. Participants were recruited when consent was gained for study participation from the relatives of the deceased.²⁶ In one case contrast agent injection and chest compressions were performed in ED shortly after the cessation of advanced life support and pronouncement of life extinct, to provide a physical state as close as possible to that during standard CPR. This was necessarily without relatives' consent, as approved by the ethics committee, but informed consent was acquired prior to any further research procedures and subsequent PMCT imaging.

Post Mortem Computed tomography

A whole body PMCT scan was performed using a Toshiba Aquilion CXL 128 slice scanner (120 kVp, 300 mA and 128 x 0.5 mm slice thickness, matrix 512 x 512) reconstructed to 1mm (head and neck, chest abdomen and pelvis) or 2mm (legs) slices. All PMCT was undertaken on a hospital scanner outside standard clinical hours so as not to disrupt patient care. The anonymized DICOM images were transferred to an Apple Mac Pro workstation with OsiriX v4.0 64-bit software (Pixmeo, Switzerland) for review.

Chest Compression PMCTA

To standardise the delivery rate and depth of the chest compressions a LUCASTM2 mechanical chest compressor was used for all cases. The device was used in both continuous compressions and 30:2 modes for period of up to 4 mins, with breaks at 2 mins for CT imaging. During imaging the device was removed from the patient as the device size would not permit it to pass through the CT scanner bore. The back plate remained in place. Following scanning further cycles of chest compressions were applied followed by repeat scanning. Modifications were made to the chest compression protocol for each subject from lessons learnt from previous attempts, in order to generate arterial – venous directional flow during CCPMCTA. The injection protocols are summarised in Table 1. The contrast was delivered through bolus venous injection, both with, and without the aid of a pump injector (Supplementary Files; Table 1). We introduced up to 3000mls of Volplex 4% w/v Solution for Infusion (Beacon Pharmaceutical Limited, England) to pre-load the venous and arterial system in cases 5 to 8, to investigate whether post-mortem vascular collapse could influence the contrast distribution. Heparin or similar pharmaceutical agents were not used in this study with any case involving contrast agent injection.

A further case (Case 9, Table 1) was recruited within the Emergency Department (ED) to provide a physical state as close as possible to that during standard CPR. Contrast was injected into a left arm peripheral vein. Two minutes of chest compression using a LUCASTM2 device were delivered immediately after the contrast and flush injections. The volume of contrast (20mls of contrast and flush, no heparin) was selected to simulate a drug (such as adrenalin) injection during CPR in order to see how far the injected liquid moved from the peripheral vein during a round of CPR.

PMCT incremental cardiac compression

To directly visualise the changes of the cardiac chambers during chest compressions a novel radiolucent device was designed and built locally to depress the chest at static 1cm increments, and, following each incremental compression, would allow scanning whilst in place (Figure 1). The novel device utilised the LUCASTM2 backboard and suction cup.

Each cadaver was positioned on the CT scan table and the plunger was advanced so it was resting on the chest, over the lower third of the sternum. The first 4 cadavers were then CT scanned without contrast. One 1cm spacer was then removed from each side of the arch, and the screws tightened to advance the plunger and depress the chest by 1cm. The cadaver was then rescanned. This process was repeated to a depth of 5cms. The 1cm spacers were then reintroduced incrementally, with repeat scanning. In the first case, due to the shape and small size of the cadaver, padding was required to support the body. Subsequently, the device was re-designed to allow for variations in chest size.

The 5th case had contrast medium introduced directly first into the left, and then the right atrium. A suitable rib space to the right of the sternum at the level of the atria was first identified on PMCT. A 15cm long needle (Rocketmedical, Rocket Introducer Needle, Rocket Medical PLC, Washington UK) was then advanced angled towards the atria based on the PMCT images. The passage of the needle tip through the right and then left atria walls could be felt by a change in resistance. Position was confirmed by repeat CT scanning. 30mls of 40% Niopam 340 diluted with water was then injected into the left atrium. The needle was slowly withdrawn to position the tip the right atrium. 30mls of 40% Niopam 340 diluted with water was then injected into the right atrium. The needle was then withdrawn, and the chest compression simulator was attached as above.

Results

Recruitment

A total of 10 cases were recruited for the CCPMCTA component of the study. Two were excluded from the study at the time of the initial, non-contrast PMCT scan. The first had a tension pneumothorax and the second had extensive rib fractures. One further case was recruited in ED, giving a total of 9 cases studied. The demographics, post mortem interval (PMI) and the circumstances of the deaths for the 9 cases are shown in Supplementary Files; Table 2.

A further 5 cases were recruited for the incremental chest compression imaging. The demographics, post mortem interval (PMI) and the circumstances of the deaths for these cases are shown in Supplementary Files; Table 3.

Vascular contrast distribution

The first cadaver recruited had an extended post mortem interval and extensive post mortem intravascular air formation and was used for procedural development only. This cadaver was excluded from assessment of circulation, leaving a study group of 8 cases.

In all 8 cases, retrograde venous flow occurred first. Contrast passed inferiorly into the inferior vena cava reaching the iliac veins and abdominal organs, and superiorly into the superior vena cava, jugular veins and the brain (Figure 2).

In 2 of 9 cases ante grade flow through to the great vessels was achieved following intravenous contrast bolus and prolonged chest compressions. Contrast passed superiorly into the head and inferiorly as far as the renal arteries in both cases (Figure 3). Preloading the venous, or the venous & arterial vasculature with Volplex (cases 5 to 8) did not help generate forward flow.

Emergency Department case

The single bolus of 20mls contrast agent was seen to reach the right side of the heart, then with retrograde flow into the IVC and hepatic veins, but no forward movement into the pulmonary arteries, left heart, aorta or coronary arteries was achieved (Figure 4).

Cardiac movement during incremental chest compression

The chest compression simulator caused a consistent pattern of thoracic and abdominal organ movement in all cases. Sternal depression caused compression of the right and left atria, and the great vessels against the thoracic spine. The ribs took on an exaggerated angulation at the point where rib fractures are often caused by chest compressions. The right and left ventricles were not compressed, but moved laterally and inferiorly, further into the left chest cavity. The left hemi-diaphragm, stomach and liver moved inferiorly. The sternum, ventricles, hemi-diaphragm, stomach and liver all moved back to their original position on incremental release (Figure 5).

In the case where contrast was injected directly into the right and left atria, some contrast entered the right and left ventricles as well as moving forwards into the ascending aorta and flowing backwards into the venous system of the liver. During the incremental sternal depressions, the contrast present within the ascending aorta moved away from the heart and did not return during release. Contrast passed through the mitral valve through to the aortic valve without expanding the left ventricle (Figure 5).

Discussion

A key to survival following cardiac arrest is the ability of CPR to bridge the gap between cardiac arrest and effective intervention (such as defibrillation) by delivering oxygenated blood to the brain to maintain brain function integrity. This is dependent on CPR creating a circulation through the lungs into the cranial arteries. The intention of our study was to develop a model

using chest compressions and PMCT that could be used to study blood flow and internal organ movement in a human cadaver to study CPR.

In agreement with previously published papers using CT to consider the optimal position for chest compressions¹⁵⁻¹⁸, the incremental chest compression part of our study shows that the principal cardiac structures compressed by the sternum are the atria (and thoracic great vessels), not the ventricles. This explains why, for example, the right atrium, the right ventricular outflow track and to a lesser extent the left atrium, can be bruised or ruptured during CPR as these structures are compressed between the sternum and the thoracic spine²⁷. The pattern of rib compression seen in our model also illustrates, using PMCT, the location where rib fractures may occur in association with CPR. Slight positioning above or below the anatomical area overlying the atria will lead to compression mediastinal or diaphragmatic / sub-diaphragmatic structures.

Although the atrial chambers contained blood, air or, in one case, contrast, the ventricles were not shown to expand with displaced fluid from the atria, but rather moved away from the point of chest compression. The model also suggested in a single case that contrast moved from the left atria to the ascending aorta via the open valves in a 'U' shaped movement without significant expansion the ventricle. Trans-oesophageal echocardiography (TOE) has reported blood flow through the mitral and aortic valves and demonstrated valve movement and directional blood flow during CPR⁸⁻¹³. As our current model images the static, not the dynamic heart, we currently have not been able to image such movement or flow using PMCT. The static incremental nature of the movement may also effect the observed movement of contrast through the heart. However, dynamic cadaver imaging of compressions could investigate heart and valve movement as well as patterns of contrast flow both within the chambers and across the valves. This could be combined with TOE and intravascular pressure wire monitoring.

An advantage of contrast-enhanced PMCT over TOE, when studying the effect of chest compressions, is that PMCT can show contrast (and therefore blood) flow resulting from CPR into the wider vasculature. We believe that this technique will allow researchers to gain a better understanding of the processes occurring during CCC and in doing so, develop better strategies to try and increase post-ROSC survival.

We only examined 5 cm of sternal compression, although the device can provide at least 10cm of incremental compression. As the device provides incremental imaging, the effect on thoracic and sub-diaphragmatic structures with compression less than or beyond the recommended current guidelines could in future be examined in the full range of adulthood, in both sexes, with a range of body build. Although the device used provides static, not dynamic compression the location of fractures commonly encountered to the sternum, and rib cage and the reason they form at this location can be demonstrated using the device. Additionally, the actual internal compression distance, as opposed to the external compression distance can be measured. This might inform compression guidelines particularly for those of smaller or larger thoracic frames.

Our observations with regards to the vascular distribution of contrast are comparable to our understanding of those observed and reported within Japan (Personal communication 2016,²⁰⁻²³). The experience from Japan, where they recommend that CCPMCTA is used within 3 hours of death, shows predominantly retrograde venous flow, although they demonstrate antegrade circulation if a pump is used to inject contrast. The post mortem interval (PMI) of our cases, except for the case undertaken in ED shortly after death, was beyond this three-hour cut-off. The prolonged post mortem interval (PMI) will cause changes in the physical state, including rigor mortis which, if present to the heart at the time of our experiments, could reduce the compliance of the ventricles. Cadaver vascular collapse, which may affect subsequent cadaver vascular flow²⁸ may be another explanation for the failure of the model to generate an antegrade flow although time dependent cadaver aortic collapse occurs below the level of the diaphragm and cadaver histological examination of the pulmonary vasculature usually shows non-collapsed, blood filled vessels within the time frame of our study subjects. For both our ED case and the Japanese experience, rigor would not be expected to affect the heart so soon after death, and yet both groups have found retrograde flow. In other studies, at a similar post-mortem interval we have found that the left ventricle can be dilated using angiography catheter delivered contrast, which suggests cardiac rigor is not present and so this may not be the reason behind the observation of retrograde flow during CPR.

We did not use heparin or a similar pharmaceutical product as such products are not used, to our knowledge, during PMCTA or CPR (unless pulmonary thromboembolism is suspected). Although post mortem clots are encountered at autopsy throughout the arterial and venous system, these are not reported to effect contrast distribution during targeted coronary artery or multiphase PMCTA. Thus, we do not believe that post mortem clots are the reason for our observation with regards to retrograde flow although we intend to investigate this further with our ongoing research.

As CPR is known to generate forward flow then one explanation for our observations relates to the volume of flow generated. CPR is considered to create bulk oscillation of blood, which results in a considerably lower volume of net blood flow per compression. Thus, we may have achieved antegrade contrast flow, but at a level undetectable by our contrast enhanced CT method. However, CT is highly sensitive to the concentrations of iodinated contrast used in this study, and we imaged before any significant extra-vascular contrast leak would be expected. Also, we also failed to achieve antegrade flow after direct injection of high density contrast into the left atrium. Therefore, any undetected antegrade flow would have to be very low. The use of alternative contrast media at different concentrations can be considered to study this consideration further.

Weale and Rothwell-Jackson reported in 1962 that there was an associated rise in venous pressure, almost equal to arterial pressure, during chest compressions.²⁹ They suggested that changes in thoracic pressure, rather than cardiac compressions, were acting as the pump during chest compressions. According to Sanders *et al.*, this observation was forgotten in light of the clinical usefulness and efficacy of chest compressions.⁵ Our observation of retrograde venous flow within the superior vena cava and jugular venous system using CCPMCTA is consistent with this 1962 observation.

Our experience suggests that the concept of the heart being squeezed by CPR leading to a normal (anterograde) arterial flow pattern is over-simplistic. Retrograde venous flow may be more common. The Japanese realised as far back as 2004 that CCPMCTA did not provide adequate post mortem angiography from intravenous contrast injection followed by chest compressions alone, and that the use of a pump injector was required. They achieved ‘forward-flow’ angiography using pump injection of 150mls of contrast delivered at 1-2mls/s with 2 minute cycles of chest compressions. If the Japanese experience translates to CPR this would suggest that chest compressions may work better at achieving circulation, and delivering intravenously injected drugs to the heart, if accompanied by a steady delivery of crystalloids during chest compression. We suggest that CCPMCTA performed immediately after conformation of death, for example within ED recruited subjects, would provide a human model to explore this proposal in a human model as well as aspects of CPR such as rate, depth, hand position, the effect of chest girth and physiological build as well as drug administration and the effect of fluid delivery.

Summary

Our study suggests that CCPMCTA could be used as a model for the study of human vascular flow and heart movement during CPR. The use of our incremental chest compression device demonstrate the structures that are compressed and the way in which the thoracic and abdominal organs move during incremental static chest compressions using CT imaging. By adding contrast to the chambers of the heart, and undertaking dynamic CT imaging, possibly in combination with TOE and aortic root pressure wire monitoring, vascular flow during CPR could be studied.

We set out to establish a human CT contrast cadaver model showing antegrade flow during CPR and have failed to achieve this in most cases. Despite the limitations of the model due to the effects of a prolonged post-mortem interval, our findings and those previously from Japan undertaken closer to death, has illustrated a previously little acknowledged or considered observation that it is possible for CPR to generate a net retrograde not antegrade flow. This work has demonstrated the feasibility of a method that can be undertaken within a very short time after death and thus provides a new tool for researchers in this field.

Conflict of interest statement

The LUCASTM2 device used in the ED case was provided by Physio-Control, Solihull, UK. The authors have no financial or research conflict with Physio-Control or any other conflict to declare.

Disclosures

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Tables and Figure legends

Table 1.

Details of the contrast medium, number of injections, method of injection, chest compression time and sessions used per case.

Table 2

The demographics, post mortem interval (PMI) and the circumstances of the deaths for the 9 cases included in the CCPMCTA study.

Table 3

The demographics, post mortem interval (PMI) and the circumstances of the deaths for the 5 cases included in the incremental chest compression study.

Figure 1

Fig 1. (a). A rigid arch with adjustable plunger was constructed from radiolucent material. Dimensions were calculated to fit within the CT bore and cope with adult male and female anterior – posterior chest diameter (based on measurements from the consented PMCT archive at Leicester). This could be attached to the LUCAS™2 backboard. A series of 1cm Perspex spacers were used to allow for up to 5cm of incremental compression and return to normal position of the chest. (b) AP and (c) lateral PMCT images of the device correctly sited.

Figure 2

Fig 2. Retrograde contrast passed inferiorly into the inferior vena cava reaching the iliac veins and abdominal organs, and superiorly into the superior vena cava, jugular veins and the brain. J=left internal jugular vein, A= right atrium, V=right ventricle, P=pulmonary arterial vasculature, L=liver, K=kidney.

Figure 3

Fig 3. Antegrade and retrograde contrast distribution following prolonged chest compressions. A=aorta, IVC=inferior vena cava, P=pulmonary arterial tree, PV=pulmonary veins, RV=right ventricle, LV=left ventricle, C=carotid artery

Figure 4

Fig 4. 20mls of contrast injected in ED is observed to show retrograde distribution. The contrast is seen to be distributed into the hepatic veins (arrows). SVC=superior vena cava, LA=left atrium, Pa=pulmonary artery, L=liver, IVC=inferior vena cava.

Figure 5

Fig 5. Sequential 1cm static compression of a case. The plunger is seen to start over the atria (RA = right atrium) and great vessels, not the ventricles (LV = left ventricle). With each centimetre of depression the sternum moves inwards towards the thoracic spine, the ribs bend at the anterior lateral aspect, and the atria and great vessels are compressed against the thoracic spine. The left ventricle moves lateral and downwards, without chamber compression as the plunger moves inwards such that it all but disappears from the image window.