Published in final edited form as:

Invest Radiol. 2015 December ; 50(6): 384–391. doi:10.1097/RLI.0000000000000138.

Contrast-Enhanced C-arm Computed Tomography Imaging of Myocardial Infarction in the Interventional Suite

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Abstract

Objectives—Cardiac C-arm CT uses a standard C-arm fluoroscopy system rotating around the patient to provide CT-like images during interventional procedures without moving the patient to a conventional CT scanner. We hypothesize that C-arm computed tomography (CT) can be used to visualize and quantify the size of perfusion defects and late enhancement resulting from a myocardial infarction (MI) using contrast enhanced techniques similar to previous CT and magnetic resonance imaging studies.

Materials and Methods—A balloon occlusion followed by reperfusion in a coronary artery was used to study acute and subacute MI in 12 swine. ECG-gated C-arm CT images were acquired the day of infarct creation (n=6) or 4 weeks after infarct creation (n = 6). Images were acquired immediately following contrast injection, then at 1 minute, and every 5 minutes up to 30 minutes with no additional contrast. The volume of the infarct as measured on C-arm CT was compared against pathology.

Results—The volume of acute MI, visualized as a combined region of hyperenhancement with a hypoenhanced core, correlated well with pathologic staining (concordance correlation = 0.89, $p < 0.0001$, mean difference = $0.67 \pm 2.98 \text{ cm}^3$). The volume of subacute MI, visualized as a region of hyperenhancement, correlated well with pathologic staining at imaging times 5–15 minutes following contrast injection (concordance correlation = 0.82, $p < 0.001$, mean difference = $-0.64 \pm 1.94 \text{ cm}^3$).

Conclusions—C-arm CT visualization of acute and subacute myocardial infarction is possible in a porcine model but improvement in the imaging technique is important before clinical use. Visualization of MI in the catheterization lab may be possible and could provide 3D images for guidance during interventional procedures.

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Conflicts of interest: For the remaining authors, none were declared.

Keywords

myocardial infarction; imaging; C-arm computed tomography

Introduction

Computed tomography (CT) and magnetic resonance imaging (MRI) are used to quantify the size and age of a myocardial infarction (MI), and to predict segment dysfunction, functional recovery, and potential viability of the infarcted tissue (1–3). An imaging assessment of the size and viability of infarcted tissue is clinically relevant in deciding on revascularization strategy and monitoring patients who may be at risk for arrhythmias or sudden cardiac death (4). Both imaging modalities rely on the pharmacokinetics of contrast material (iodine or gadolinium) through healthy versus unhealthy myocardial tissue to delineate perfusion defects in newly infarcted tissue or an accumulation of contrast material that appears as late enhancement in scar.

Advances in C-arm mounted x-ray fluoroscopy systems have resulted in the ability to generate fully-3D CT images with contrast resolution approaching that of conventional CT within the cardiac catheterization laboratory (5). This allows the physician to obtain a CT image at any point during the procedure, without moving the patient. Initially, cardiac C-arm CT applications focused on left atrium volume imaging for navigation of electrophysiology procedures (6). New studies aim to extend C-arm CT applications for monitoring a delivered therapy. For example, the ability to visualize radiofrequency ablation lesions using C-arm CT has previously been described (7).

In this work, we demonstrate that cardiac C-arm CT can be used for low-contrast, soft tissue imaging to assess perfusion defects and late enhancement resulting from MI. We hypothesize that we can directly visualize MI and quantify the size and location of a suspected MI using contrast enhanced techniques.

Materials and Methods

Animal Preparation

The institution's Administrative Panel on Laboratory Animal Care approved this study. Fourteen female Yorkshire swine were anesthetized intramuscularly with 5–7 mg/kg Telazol and 0.05 mg/kg atropine and were maintained on 80% oxygen and 2–3.5% isoflurane with a respirator. Two venous and two arterial femoral access points were established using percutaneous puncture. Aspirin (81 mg) was given once the day before and heparin (300 IU/kg) was administered throughout the procedure to maintain activated clotting time over 300 s. Of the fourteen swine, six were studied in the acute MI model and eight were studied in the subacute MI model.

Acute Myocardial Infarction Porcine Model—The reperfused myocardial infarctions were created minimally invasively using a balloon catheter placed in the artery of interest (8). An AL 0.75 or 1.0 SH guide catheter (Mach 1, Boston Scientific, Natick, MA) was used to gain access to the left anterior descending (LAD, n=3) or left circumflex (LCX, n=3)

coronary artery in 6 swine (mean weight 54 ± 8.9 kg). Baseline coronary angiography was performed to determine the occlusion site and angioplasty balloon size. The artery was occluded using a 3.0 or 3.5 mm angioplasty balloon (Maverick Over-The-Wire, Boston Scientific, Natick, MA) inflated to 10–14 atm for 60 minutes followed by deflation over 3 minutes. C-arm CT image acquisition began 90 min after removal of the angioplasty balloon to ensure hemodynamic stability of the animal before iodine contrast injection and multiple breath holds. Postmortem tissue examination occurred at completion of the imaging.

Subacute Myocardial Infarction Porcine Model—Eight swine (mean weight 46 ± 3.2 kg) were prepared as described above and a myocardial infarction was induced using the same methods (LAD: $n=4$, LCX: $n=4$). C-arm CT imaging was performed 90 minutes after initial reperfusion and then the animals were transported to the animal facility for intensive-care unit recovery and support. Buprenorphine (0.01 mg/kg) was administered for 24 hours after infarction for pain management and aspirin (162 mg) was administered daily until the follow-up imaging procedure.

Approximately 4 weeks (28 ± 3 days) after infarct creation, the animals (mean weight 58 ± 5.1 kg) were anesthetized, intubated, and placed on a respirator. The C-arm CT imaging protocol was repeated, followed by postmortem tissue examination. A sustained release verapamil tablet (120 mg) was given the night before imaging to control ventricular rate during the imaging study.

C-arm CT Image Acquisition

Cardiac C-arm CT images were acquired by rotating a C-arm angiography system in forward and backward sweeps, 4 sweeps of 5 s each (5) (syngo DynaCT Cardiac, Siemens AG, Healthcare, Erlangen, Germany) (Figure 1). A total of 992 projection images (249 projection images/sweep) were acquired with electrocardiography for retrospective gating.

The first-pass C-arm CT acquisition began following a 42 s peripheral injection of iodinated contrast (Omnipaque 350 mg/mL, GE Healthcare, Princeton, NJ). A 6 F pigtail catheter was placed in the inferior vena cava and contrast was delivered using a power injector (Medtronic, Saarbrücken, Germany) programmed for a two-phase protocol of 150 mL of iodine (0.97 ± 0.15 g Iodine/kg) followed by 16 mL saline flush at 4 mL/s. The contrast injection was timed to the myocardial phase for optimization of myocardial perfusion assessment, distinct from the arterial phase timing utilized for CT coronary angiography. After the first-pass image, a series of C-arm CT images were acquired with no additional contrast at 1 minute and then every 5 minutes up to 30 minutes following contrast injection.

All images were acquired with 1.2 μ Gy/frame entrance dose at the detector. The tube voltage was 90 kV for the first-pass and 1 minute image acquisitions and all subsequent images in the time series were acquired with 70 kV, as lower kV improves low-contrast detectability (9). Breathing was held at inspiration for the duration of each image acquisition, approximately 24–30 seconds.

C-arm CT images were reconstructed using a Feldkamp algorithm, which has been previously described (5, 10). Images were reconstructed using retrospective ECG-gating

[selecting 248 of the available 992 projection images] at ventricular end-diastole (centered at 80% of the R-R interval) to minimize motion artifact. The 3D volume size was $512 \times 512 \times 300$ with an isotropic voxel resolution of 0.50 mm.

Postmortem tissue examination

After completing the imaging protocol (day 0 for the acute study and ~4 weeks for the subacute study), thioflavin S (2mg/50mL saline) was injected by hand into the LV over 3 minutes using a 6F pigtail catheter. Animals were immediately euthanized with a Beuthanasia solution (0.22 ml/kg intravenous dose) (D Special, Animal Health Corp., Union, New Jersey) and the heart was extracted. 10 mm thick, short-axis slices of the heart were photographed on both sides under natural and ultraviolet light (365nm, Spectroline ENF-280-C, Spectronics). Thioflavin S is a fluorescent dye for endothelium and intact capillaries fluoresce under UV light (11), where regions of microvascular obstruction (MO) appear dark, allowing for demarcation of MO (12). The myocardial sections were then placed in a 1% solution of 2,3,5-triphenyltetrazolium chloride (TTC) at 37° C for 20 min and photographed. TTC remains white in areas of necrosis and is enzymatically reduced to red in living tissue. Thus, white regions after TTC staining demarcate the area of infarction. Areas of infarction and MO were measured using hand planimetry and the volume was calculated by multiplying by slice thickness. Tissues samples from both acute and subacute studies were subsequently processed for routine histological analysis using hematoxylin and eosin (H&E) staining.

C-arm CT Data Analysis

C-arm CT images were analyzed offline using OsiriX (version 3.9.1 64-bit, Pixmeo, Geneva, Switzerland). The region of infarction was assessed using continuous, 5 mm multiplanar reformatted (MPR) short-axis slices in the first-pass through 15-minute images. An experienced observer, blinded to the results of pathologic staining, independently determined the infarct region by hand-tracing the regions of infarction, which were visualized as hyperenhancement with a variable hypoenhanced core in acute MI studies and as an area of hyperenhancement in the subacute MI studies. The hypoenhanced core in acute MI studies represented areas of microvascular obstruction. The total infarct region was defined as the sum of the area of hypoenhanced and hyperenhanced myocardium (acute studies) or the area of hyperenhanced myocardium (subacute studies). The volume was calculated by multiplying the area by the slice thickness. Due to the experimental nature of the study, the reader knew that the C-arm CT images were the result of an occlusion in either the LAD or LCx, but was not told which.

Next, first-pass through 30-minute images were loaded into 4D viewing to measure the contrast dynamics. Regions of interest (ROIs) were placed in the first-pass volume and propagated through consecutive volumes. The image intensity in Hounsfield units (HU) was measured in 3 regions: LV cavity, normal myocardium, and infarct core (1cm^2 ROIs). Additionally, the intensity was measured at the periphery of the infarct region in acute images only (0.6cm^2 ROI). The ROI for healthy myocardium was chosen within 2 cm of the infarct region so that image artifact levels would be similar.

Statistical Analysis

The C-arm CT measure of infarct volume was compared to the volume from TTC. The hypoenhanced volume measured on C-arm CT was compared to the volume of MO determined by thioflavin S. Agreement between C-arm CT and pathological measurements was assessed by means of concordance correlations, 95% confidence intervals (CI), means and standard deviations of differences, and Bland-Altman 95% limits of agreement. Contrast dynamics are illustrated using signal-intensity curves showing the mean intensity and standard deviation for all acute and subacute studies for each region of interest. Trend over time in differences in CT and pathology measurements were assessed by two-sided Jonckheere-Terpstra nonparametric test for trend. P values < 0.05 were considered significant. All statistical analyses were done with Stata Release 9.2 (StataCorp LP, College Station, TX).

Results

Acute study

Ventricular fibrillation occurred during balloon occlusion in two animals and during reperfusion in four animals. All animals were successfully defibrillated and able to complete the full 60-minute balloon occlusion followed by C-arm CT imaging. The mean heart rate during the imaging study was 88 ± 7 beats per minute.

The extent of acute infarction, visualized on C-arm CT axial slices as the combined area of hyperenhanced and hypoenhanced myocardium, visually corresponded with photographs of TTC staining (Figure 2 A, B). Figure 2D shows a non-significant ($p=0.35$) trend for increasing bias over time and visually a C-arm CT acquisition time of 1 or 5 minutes may provide the best agreement with pathology. The infarct volume quantified on C-arm CT images correlated well with TTC images, with a concordance correlation of 0.89 ($p<0.0001$) over all time points (95% CI = 0.78 – 0.94, mean difference = 0.67 ± 2.98 , Bland-Altman 95% limits = $-5.17 - 6.52$, $N=30$), see Figure 2 F,G. The infarct volume, as measured by pathology (TTC), ranged from 4.0–25.0 cm³. The mean and standard deviation of infarct volume on TTC and C-arm CT (1 min.) was 13.1 ± 6.7 cm³ and 13.7 ± 6.1 cm³, respectively.

MO was seen on C-arm CT as an area of hypoenhanced myocardium in 4 of 6 animals, two animals showing 1.5 and 1.6 cm³ on thioflavin S-stained tissue with no corresponding hypoenhancement on C-arm CT. The region of hypoenhancement was visualized beginning on the first-pass image and decreased in size in subsequent images, showing a significant ($p=0.0006$) change in the C-arm CT and pathology volume difference over time (Figure 2E). The concordance correlation for all time points was poor, 0.224, $p=0.260$ (95% CI = $-0.17 - 0.55$, mean difference = 0.96 ± 5.89 , Bland-Altman 95% limits = $-10.85 - 12.49$, $N=20$), although graphically (Figure 2E) a C-arm CT acquisition time of 1 or 5 minutes may provide the best comparison to pathology.

The contrast dynamics are illustrated in Figure 3A and the plot in Figure 3B shows the average intensity in each ROI for all subjects. The hyperenhanced region had a peak intensity of 127 ± 18 HU (36 ± 28 HU above the myocardium) 1 minute after contrast injection and the intensity remained high, near 100 HU, until 20 minutes. The region of MO, found

primarily in the core of the infarct, had a lower intensity than the enhanced tissue at all imaging time points (1 minute post contrast injection: average intensity 55 ± 33 HU, 56 ± 39 HU lower than myocardium). Over time, diffusion of contrast into the region of MO likely resulted in a slight increase in signal intensity.

H&E staining was performed on tissue samples from the center and border of the infarct zone and the histologic changes in the samples are representative of a peracute to acute, myocardial infarct. The border zone of the infarct showed edema and characteristics of early degenerative and necrotic changes to the myofibers, including loss of sarcoplasmic cross striations, contraction banding in the sarcoplasm, and some loss of nuclei.

Subacute study

Two animals died within 48 hours after the infarction due to complications with pulmonary edema and respiratory distress. One animal experienced ventricular fibrillation during the balloon occlusion and was successfully defibrillated. The mean heart rate during the subacute imaging study was 91 ± 12 beats per minute for the 6 animals. Image quality was degraded in two animals due to a heart rate >100 beats per minute, but was still sufficient to evaluate the infarct region.

The hyperenhanced region on C-arm CT images visually correlated with TTC-negative stained tissue (Figure 4 A–C). The difference in the volume of infarct between pathology and C-arm CT showed no significant bias over time ($p=0.43$), Figure 4D. The infarct volume quantified on C-arm CT images correlated well with TTC images, with a concordance correlation of 0.77 ($p<0.0001$) over all time points (95% CI = 0.59 – 0.88, mean difference = -0.68 ± 2.18 , Bland-Altman 95% limits = $-4.95 - 3.59$, $N=30$), see Figure 4 E,F. Based on studies of CT late enhancement, we would expect the agreement between C-arm CT images and pathology to be better at later time points and if we consider time points between 5–15 minutes, the concordance correlation slightly increases to 0.828 ($p<0.001$, 95% CI = 0.63 – 0.925, mean difference = -0.64 ± 1.93 , Bland-Altman 95% limits = $-4.44 - 3.16$, $N=18$). The infarct volume, as measured by pathology (TTC), ranged from 6.2–13.5 cm³. The mean and standard deviation of infarct volume on TTC and C-arm CT (5 min.) was 9.7 ± 3.2 cm³ and 9.8 ± 4.0 cm³, respectively.

A perfusion defect was found in 4 of 6 animals on the first-pass images of the infarct at 4 weeks, with a mean signal intensity of 55 ± 32 HU below normal myocardium (average intensity 77 ± 47 HU). All animals exhibited hyperenhancement on later images, as illustrated in Figure 5A, with the maximum signal intensity above normal myocardium of 49 ± 15 HU occurring at 5 min (average intensity 118 ± 30 HU). The average intensities of the cavity, infarct region, and remote myocardium for all subjects are shown in Figure 5B. Enhancement was noted on some images as early as 1 minute after contrast injection and the intensity remained above the remote myocardium, approximately at the level of the cavity, for the remainder of the image acquisitions.

H&E staining revealed histologic changes that are representative of a chronic-active myocardial infarct. The infarct region contained large regions of mature fibrous scar interrupted by regions of active inflammation. The junction between the scar tissue and

healthy myocardium appeared as a collar of inflammation composed of neutrophils, macrophages, and multinucleated giant cells.

Discussion

This study demonstrates that cardiac C-arm CT can be used to visualize both acute and subacute myocardial infarction in a porcine model. In the acute setting, the total infarction was delineated on C-arm CT images as a hyperenhancement with a variable hypoenhanced core, with MO appearing as the region of hypoenhancement. Based on the contrast dynamics and the trend over time of the difference in pathology and C-arm CT volume, the total infarct volume and regions of MO may be best discriminated at 1–5 minutes post contrast injection, which is consistent with the findings in Lardo et al. (12). Further studies should investigate the optimal imaging time. In the subacute setting, fibrous myocardial scar tissue was characterized by late enhancement, with peak enhancement on C-arm CT images obtained 5 minutes after contrast injection and good correlation across imaging times of 5–15 minutes post contrast injection.

CT and MR myocardial viability imaging

Functional information is derived from CE CT and MR imaging using the time-dependent contrast enhancement patterns throughout the infarct region compared to healthy myocardium. Contrast-enhanced CT and MRI give analogous enhancement patterns, since iodine and gadolinium contrast agents have similar molecular weight and diffuse comparably (13). In this study, we have shown that C-arm CT images of MI give enhancement patterns similar to reported signal levels in CT images of MI: hyperenhancing signals above healthy myocardium of 20–50HU for CT compared to 35–50 HU for C-arm CT, and hypoenhancement below healthy myocardium of approximately 50HU for CT compared to 55–65 HU for C-arm CT (2, 13, 14).

Myocardial viability imaging with CT has seen renewed interest and is being investigated as an alternative to MRI, particularly for patients with contraindications for MRI. CT has a shorter acquisition time and has better spatial resolution in the z-axis (smaller slice thickness), which helps mitigate partial volume effects associated with MRI (15). Although C-arm CT imaging has poorer temporal resolution and low contrast detectability than CT, C-arm CT has the additional benefit that images of MI could be acquired in the catheterization lab and used for guidance, without having to register a pre-procedural CT or MRI.

Clinical Significance

C-arm CT evaluation of an acute MI could be useful for improved diagnosis and differentiation of treatment plans with respect to prognosis. Without interfering with the primary angioplasty procedure, the imaging could be done immediately following primary angioplasty in the catheterization lab. Prior investigations have shown that the size of regions of LE and MO are strong predictors of follow-up myocardial segment dysfunction and lack of myocardial functional recovery (2, 15, 16). Therefore, the ability to directly measure infarct size and MO immediately after the initial intervention in the catheterization lab could provide early prognostic information.

The combination of real-time fluoroscopy and 3D cardiac imaging with a delineation of scar morphology using the C-arm CT system could be a valuable tool for image guidance during other complex cardiac procedures. For example, in the treatment of ventricular tachycardia, there is typically a structural substrate of ventricular scar involved in the arrhythmia (17). Visualization of the scar morphology could facilitate electroanatomical mapping (18) and guide the delivery of ablation lesions using the same system to acquire 3D images and fluoroscopically guide the mapping and ablation catheters. Lastly, tracking and validating the delivery of a cellular or molecular therapy with respect to the infarction could be done using the combined x-ray fluoroscopy and C-arm CT system and an appropriate contrast agent, such as microcapsules containing a radiopaque agent (19, 20).

C-arm CT Image Quality

This is the first study visualizing MI using C-arm CT and therefore a high-dose e ($4 \text{ sweep} \times 5 \text{ s}$, $1.2 \mu\text{Gy/frame}$ at 70 or 90 kV), high-contrast ($0.97 \pm 0.15 \text{ g Iodine/kg}$) protocol was used to guarantee good image quality. Further investigations to optimize iodine contrast and radiation dose should be conducted. Absorbed dose was measured in our lab on a porcine subject using 5 dosimeters (Dxposure Radiology Dosimetry System, high-sensitivity MOSFET dosimeters, Best Medical Canada, Ottawa, Ontario, Canada), 4 placed circumferentially 1 cm under the skin and 1 placed in the esophagus, yielding an effective dose of 27.3 mSv for the 70 kV protocol and 27.1 mSv for the 90 kV protocol. This estimate for the $4 \text{ sweep} \times 5 \text{ s}$ is in agreement with the dose reported by Wielandts et al. (21) of 5.5 mSv and 6.6 mSv according to ICRP 60 and ICRP 103, respectively, for a single-sweep protocol (giving an estimated dose of 22 mSv [ICRP 60] and 26.4 mSv [ICRP 103] for 4 sweeps). The actual dose is patient dependent due to the automatic exposure control, which controls the kVp and m*As per projection, and amount of collimation used.

Significant dose reduction could be obtained using a single-sweep acquisition protocol with rapid pacing or adenosine injection, although both methods carry additional risk to the patient, and rapid pacing is generally only acceptable when a pacing electrode is already needed for the intervention. We felt that ECG gating would provide the best opportunity to freeze cardiac motion and visualize soft tissue defects in the myocardium. A clear limitation of the ECG-gated acquisition is residual motion artifact from the large gating window, as illustrated in Figure 6 by the superior image quality of a post-mortem C-arm CT scan (B) compared to the ECG-gated image (A) acquired under the same contrast injection condition (5 min post iodine contrast injection) and same ventilated lung pressure. A single, 5s sweep acquisition without ECG gating was used for the postmortem scan. Future investigations should be conducted to develop single-sweep imaging and injection protocols to visualize myocardial scar tissue.

In comparison to conventional CT, where a scan can be performed with as little as 1 mSv (22), even a single sweep protocol at 5.5 mSv requires significantly more dose. In the context of relevant interventional procedures though, with effective doses ranging between 2.1– 59.6 mSv (23) for electrophysiology procedures and 0.9 – 44.7 mSv (24) for coronary angiography with percutaneous transluminal coronary angioplasty, an additional single-sweep C-arm CT may be acceptable if there is a proven benefit to guide the procedure.

Cardiac C-arm CT is a newer imaging modality and lower-dose protocols will arise from hardware improvements, such as more efficient flat-panel detectors, prospective ECG triggering, or use of a wedge filter, and new software reconstruction and motion compensation algorithms.

Compared with conventional CT, C-arm CT suffers from additional artifacts due to beam hardening, scatter, truncation, motion, and cone beam (25). In addition to the motion artifact discussed above, there are additional artifacts noted on the anterior and posterior wall of the LV, due to beam hardening and scatter. Figure 6C,D are images from a healthy, non-contrast enhanced heart that illustrate a higher-intensity artifact attributed to beam hardening (C) and a shading artifact on the posterior wall attributed to scatter (D).

Nonetheless, all image sets were of sufficient quality to measure the infarct.

Study Limitations

Although the C-arm CT observer was blinded from the postmortem tissue examination, due to the experimental nature of the study, it was known that the C-arm CT images were from an acute or subacute study and that the infarct territory was the result of an occlusion in either the LAD or LCx. This study did not consider infarctions in the right coronary artery.

Conclusions

Cardiac C-arm CT can visualize a myocardial infarction in an acute and subacute animal model. C-arm CT has the potential to provide myocardial infarction imaging in the catheterization lab.

Acknowledgments

The authors thank Wendy Baumgardner, RVT, LATg for her assistance with the animal care. The authors also acknowledge support from NIH grant R01 HL087917, Siemens AG, Healthcare Sector, Medtronic AG, and the Lucas Foundation.

Source of funding: Drs. Fahrig and Al-Ahmad receive research support from Siemens AG, Healthcare Sector and by NIH grant R01 HL087917 (Bethesda MD). Dr. Fahrig additionally receives a research grant from Medtronic AG (Saarbrücken, Germany) and the Lucas Foundation (Stanford, CA). Teri Moore is an employee of Siemens Medical Solutions, Erin Girard is an employee of Siemens Corporate Technology, and Guenter Lauritsch is an employee of Siemens AG, Healthcare Sector.

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Figure 1.

(A) The C-arm rotates 200° around the patient table with ECG monitoring. (B) A diagram showing how multiple sweeps are used to fill the 200° space required for a 3-dimensional reconstruction with views from a given cardiac phase.

Figure 2.

Axial C-arm CT image (window width = 200 HU, contrast = 120 HU) demonstrating a large lateral infarct acquired 1 min (A) after contrast injection and the corresponding TTC (B) and thioflavin S (C) stained images. The difference in measured C-arm CT volume to pathology volume over time is shown in (D) for the total infarct and (E) for the hypoenhanced core. (F) The total infarct volume measured on TTC compared to C-arm CT volume measurements, at 1 and 5 min, shown around the line of identity. (G) Bland-Altman plot of the infarct volume

at all measured time points (black lines demark the 95% limits of agreement = $-5.17 - 6.52$).

Figure 3. (A) Short-axis images from an acute study with a large anterior infarct showing a perfusion defect in the first-pass image. Contrast around the periphery eventually diffuses into the region of microvascular obstruction, making the hypointense region appear smaller over time. Window width = 400 HU, contrast = 150 HU. (B) The signal intensity in different regions is shown at all imaging time points.

Figure 4. C-arm CT images (window width = 390 HU, contrast = 130 HU) of a 4-week-old infarct 5 minutes after contrast injection showing late enhancement (red arrows) in the coronal (A) and short-axis (B) views. The TTC-negative area (red arrows) (C) qualitatively correlates with the short-axis image in (B). The difference in measured C-arm CT volume to pathology volume over time is shown in (D) for the total infarct. (E) The total infarct volume at 4 weeks, measured on TTC compared to C-arm CT, at 5 and 10 min., shown around the line of

identity. (F) Bland-Altman analysis of the infarct volume at all measured time points with black lines demarking the 95% limits of agreement at 5 min.

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Figure 5. (A) C-arm CT images from a subacute study demonstrating late enhancement throughout the mid and apical regions (red arrows) clearly visualized in images 5 min and later (window width = 330 HU, contrast = 100 HU). (B) The average signal intensity in different regions is shown at all imaging time points.

Figure 6.

Pre- (A) and post- (B) mortem C-arm CT images from the same animal demonstrate the residual motion artifact after retrospective ECG gating for the reconstruction. Examples of scatter and beam-hardening artifacts in reconstructions of a healthy subject demonstrating a high-intensity banding throughout a non-contrast enhanced heart (C) and a shading artifact on the posterior wall (D).