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Changes in heart dimensions in decomposition using porcine and ovine animal models: a method for estimating decomposed human heart weight

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ABSTRACT

Heart weight is a routine measurement at post-mortem examination. An increased heart weight is associated with pre-existing heart disease and sudden cardiac death. However, during decomposition, the heart weight measured at post-mortem examination may not reflect the weight at the time of death. A previous study showed that heart dimensions can be used to estimate heart weight. This study documents the changes in heart dimensions during decomposition using porcine and ovine animal models. It shows that, in contrast to heart weight, which decreases in the post-mortem interval, the heart dimensions increase. Heart width increases and subsequently plateaus at the end of the 18 days to a mean of 6 and 8 mm in ovine and porcine hearts, whereas the length continues to increase. Using the results of this study, in conjunction with previous published literature, the heart width may be used as a parameter to estimate heart weight and determine cardiac hypertrophy at the time of death in decomposed hearts.

Introduction

Hypertrophy of the heart (or cardiac hypertrophy) is commonly encountered in forensic pathology and is associated with sudden cardiac death [1–4]. Gross examination for cardiac hypertrophy includes assessing the heart size (by heart dimensions) and heart weight [4]. Heart weight is well studied compared with heart dimensions (width and length), where the literature on heart dimensions is scant, and the relationship between heart weight and cardiac hypertrophy has only recently been established [5,6]. These studies showed that the dimensions of the heart, especially the width (measured along the base of the heart), can be used as an estimator for heart weight and can be used to identify cardiac hypertrophy. The justification for this is that the width of the heart measures the fibrous annulus ring of the heart which is more robust to the effects of rigour mortis [5]. Thus, it is

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possible that the width of the heart may also be robust during decomposition in the late post-mortem interval. Furthermore, in our practical international experiences, the heart appears to enlarge during decomposition. Understanding the extent to which decomposition affects thedimensions of the heart is therefore of use as a potential specific parameter to identify cardiac hypertrophy.

In bodies showing signs of decomposition with loss of parenchymal tissue, weighing the heart may underestimate the actual weight at the time of death. As such, most often, the post-mortem report would merely record the 'residual weight'. Given the relationship between heart dimensions and heart weight, and the proposed underlying reason and characteristics, we hypothesize that the heart dimensions (specifically the width of the heart) may be of utility in estimating heart weight and determining cardiac hypertrophy in cases showing decomposition.

This study aims to document the serial changes in heart dimensions during decomposition using porcine and ovine animal models to explore the potential utility in using heart dimensions to estimate heart weight and determine cardiac hypertrophy.

Materials and methods

Animal model selection

Normal human hearts weigh approximately between 150 and 300 g in females and 200 and 400 g in males[4,7,8]. The porcine (pig) heart weighs ~400 g and an ovine (sheep) heart weighs ~200 g. This study subsequently used ex-vivo commercially sourced porcine and ovine hearts to model normal adult male and female hearts. Furthermore, porcine and ovine models are established animal models for human cardiology studies[9,10].

Sample collection

This study was conducted under the animal ethics approval of The University of Queensland, Production and Companion Animal Ethics Committee (ANRFA Certificate Number: 2024/AE000061). A total of 59 hearts from porcine ($n = 29$) and ovine ($n = 30$) sources were obtained from a local abattoir. Each heart, enclosed within its pericardial sac, was excised from the thoracic viscera at the level of the major vessels using a standardized approach shortly after slaughter by abattoir staff. The hearts were segregated by species and transported in sealed plastic bags in batches of six to the anatomic pathology laboratory at the School of Veterinary Science, The University of Queensland (Gatton, Queensland). Upon arrival, these bags were placed on plastic trays and stored in a cold room maintained between 1 and 3℃ until processing commenced.

Sample processing

Heart processing occurred within 12 hours of arrival at the laboratory, which included obtaining weight and dimensions. Initial preparation at the laboratory included retrimming the tissues in a standardized dissection by a trained prosector to ensure a consistent sample was assessed. Briefly, the pericardial sac was elevated and removed along with the great vessels at the level of the pericardial attachment to create a standardized specimen,

mirroring heart removal in human post-mortem examination. The hearts were not dissected as to maintain the structural integrity for measurements. As such, the residual blood clots in the heart chambers were not thoroughly removed, however the animals were exsanguinated during the slaughter process minimizing residual cardiac chamber blood.

Data collection

The study was conducted over a duration of 18 consecutive days. Initial weights were measured on day 1 using a calibrated scale (Philips HR2385/00) accurate to the nearest gram. The heart was positioned with its caudal (posterior) aspect down, allowing visualization of its cranial (anterior) surface (Figure 1).

Hearts were then measured using a calibrated forensic ruler to obtain the width and length of the heart to the nearest millimetre (mm) (Figure 2). Similar to post-mortem examination in human, the width was measured along the base of the heart (i.e. distance from the obtuse to the acute margin at the posterior atrio-ventricular sulcus in humans). The length was measured from the apex to crux cordis (i.e the point at which the atrio-ventricular sulcus meets the posterior interventricular sulcus). Different from human, the width and length of the animal hearts are not perpendicular to each other. Between measurement intervals, each heart was individually sealed in a zip-lock bag to minimize dessication and placed in a portable cooler at ambient temperature in a secured, sheltered area of the laboratory. This aims to mimic in-vivo decomposition and prevent scavenger predation and mummification.
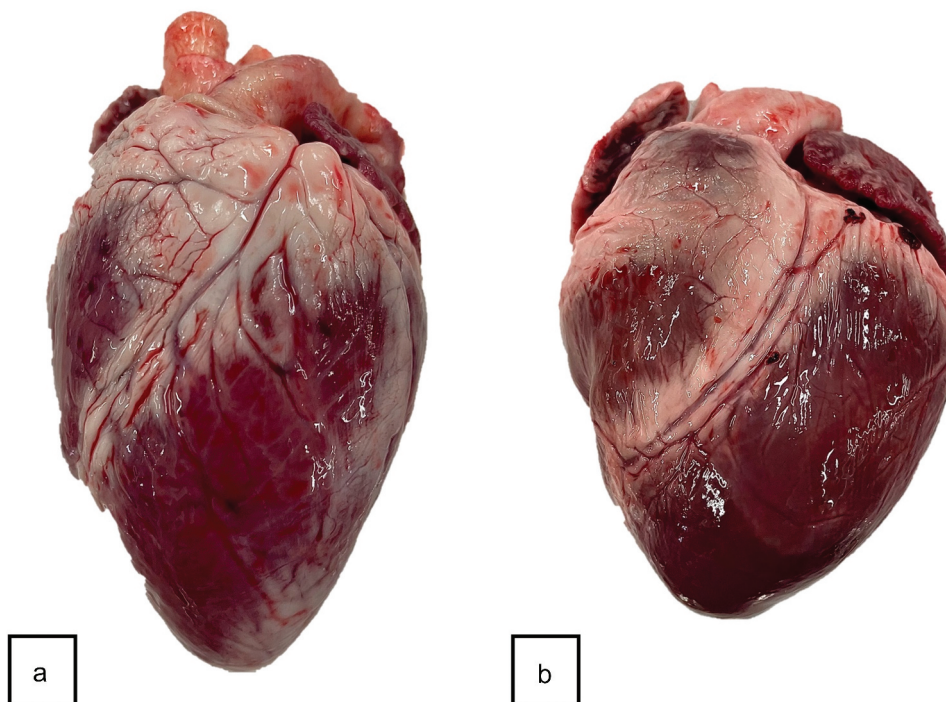

Figure 1. Cranial (anterior) view of the ovine (a) and porcine (b) heart after standardized preparation on the day of slaughter.
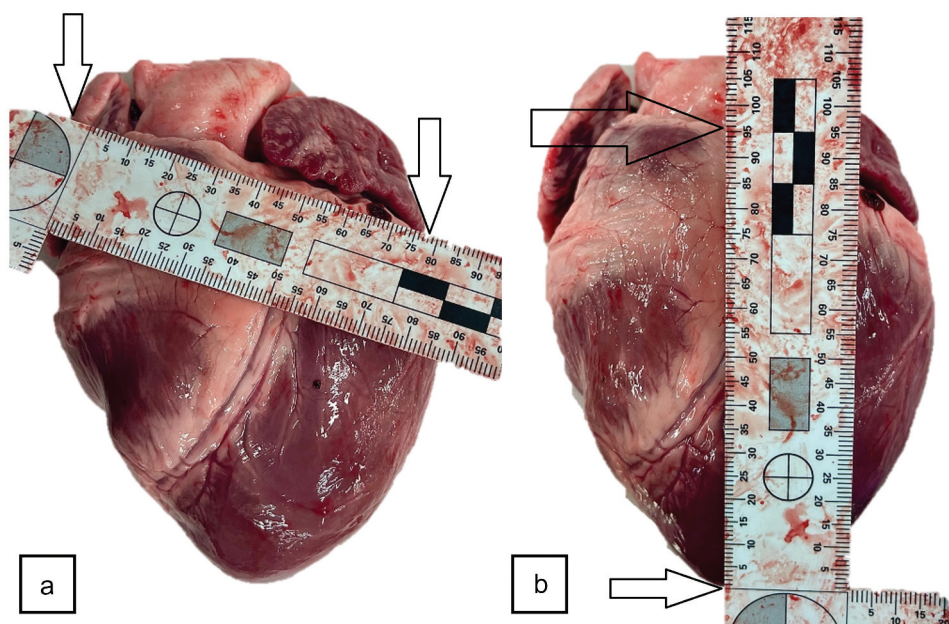
Figure 2. Illustration using the porcine heart showing measurement of the heart width (a) and length (b) with arrows showing where the measurements were taken.

Additional measurements of weight, width, and length along with a general description of the shape, texture and colour of the hearts were recorded at the initial timepoint and on days 2, 3, 5, 6, 7, 8, 10, 13, 14, and 18 of the study period. At the end of the study period the hearts were completely flattened when placed on a dissection table and some showed obvious fragmentation, so the experiment was discontinued on day 18. Once all the measurements were taken, the hearts were disposed of in accordance with the veterinary mortuary protocol.

Statistical analysis

Statistical analysis was performed using R (open source, R studio 2022.07.01, Build 554). Continuous variables were presented as mean, median, minimum, maximum and standard deviation (s.d). Discrete variables were represented as counts.

Pearson's correlation with subsequent univariate analysis were used to establish the relationship between heart weight and heart dimensions. The Kruskal Wallis (due to the presence of outliers and non-parametric distribution) and post hoc Dunn's test was used to establish the day when the end point had been reached (i.e. no significant change in at least three consecutive measurements) for either width or length.

Results

General morphological changes

Throughout the study duration, the cardiac tissues exhibited progressive softening and flaccidity resulting in a flatter profile and more rounded cardiac silhouette. By day 5, evidence of sulphoxy-haemoglobin accumulation became apparent, causing a greenish hue to the purge fluids. The fluid content within the sealed bags showed a gradual increase over time, with a marked escalation observed around day 8, coinciding with visible breakdown of residual post-mortem blood clots within the cardiac chambers. By the 13th day, the tissues had significantly deteriorated, displaying signs of ramollissement/softening and fragmentation on handling. Concurrently, there was a noticeable darkening and greying of the tissue (Figure 3).

Gross examination revealed that the porcine hearts tended to soften and lose turgidity more rapidly compared with the ovine hearts. This species difference was also reflected in the greater accumulation of purge fluid observed in the zip-lock bags containing porcine hearts as opposed to those containing ovine hearts.

Porcine heart

On day 1, the mean heart weight was 401 g (s.d: 116.7, median: 380, min: 221, max: 733). The mean width and length of the hearts were 76.6 mm (s.d: 8.4, median: 79, min: 58, max: 90) and 90.6 mm (s.d: 15.0, median: 88., min: 62, max: 120), respectively. Person's correlation coefficient between heart weight with width and length were 0.77 ($p < 0.05$) and 0.87 ($p < 0.05$) respectively. Univariate linear regression coefficients to estimate heart weight using width and length were 12.5 ($p < 0.05$, $r^2 = 0.61$) and 12.6 ($p < 0.05$, $r^2 = 0.77$)
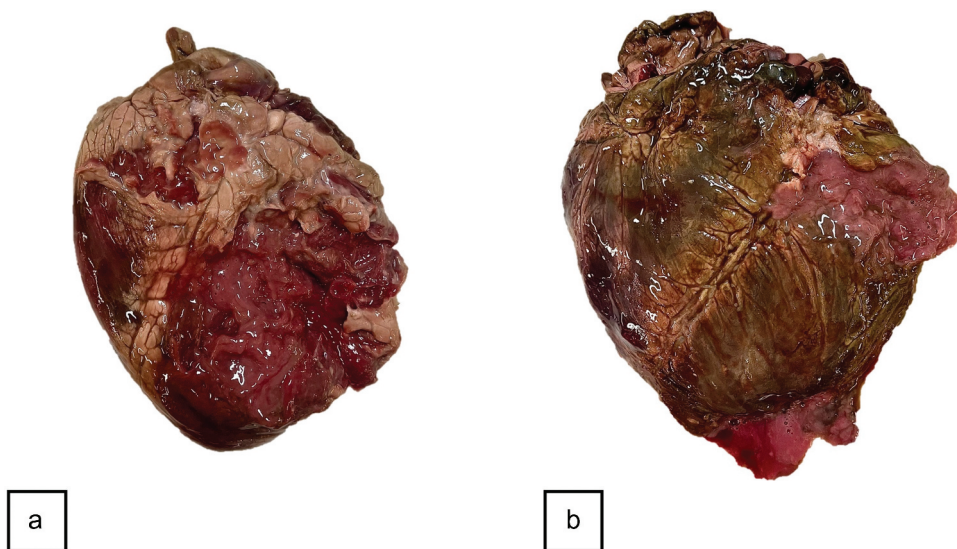

a

b

Figure 3. Ovine (a) and porcine (b) heart on day 18 of the study, showing significant disintegration and discolouration of cardiac muscle tissue.

respectively. At the conclusion of the study the mean heart weight was 323.8 g (s.d: 49.6, median: 323.8, min: 221, max: 402) with an average loss of 15.8% of initial heart weight.

The change of width and length are shown in Figure 4. The mean width fluctuated in the first week and subsequently decreased. Kruskal-Wallis analysis for the second week (days 8, 10, 12, 13, and 17) showed no significant changes in the width of the heart, indicating end point ($p > 0.90$). The mean change (days 8, 10, 13, 14, and 18) was 8 mm (s. d: 8.31, median: 9, min: −16, max: 25). The length gradually increased and continued to show significant differences with a mean change on day 18 of 31.1 mm (s.d: 8.7, median: 32, min: 14, max: 46).

Ovine heart

At day 1, the mean heart weight was 165 g (s.d: 29.9, median: 166, min: 125, max: 275). The mean width and length of the hearts were 56 mm (s.d: 5.7, median: 56, min: 47, max: 74) and 62.9 mm (s.d: 4.5, median: 63., min: 52, max: 73), respectively. Person's correlation coefficients between heart weight and width and length were 0.74 ($p < 0.05$) and 0.43 ($p < 0.05$) respectively. Univariate linear regression coefficients to estimate heart weight using width and length were 3.85 ($p < 0.05$, $r^2 = 0.54$) and 2.9 ($p < 0.05$, $r^2 = 0.19$), respectively. At the conclusion of the study the mean heart weight was 146 g (s.d: 29.3, median: 147, min: 107, max: 256) with an average loss of 12% of initial heart weight.

The change of width and length are shown in Figure 5. Similar to the porcine heart, mean width fluctuated in the first 10 days and Kruskal-Wallis analysis between days 13, 14, and 18 showed no significant changes in the width of the heart indicating end point ($p > 0.76$). The mean change (days 13, 14, and 18) was 6.3 mm (s.d: 6.6, median: 6, min: −5, max: 33). The length gradually increased and continued to show significant differences with a mean change on day 18 of 25.7 mm (s.d: 4.3, median: −26, min: 15, max: 36).

Discussion

This study used animal heart models to document the changes in heart dimensions during decomposition. It showed that the dimensions ultimately increased with decomposition. The mean width increased by 8 and 6 mm in porcine and ovine heart,
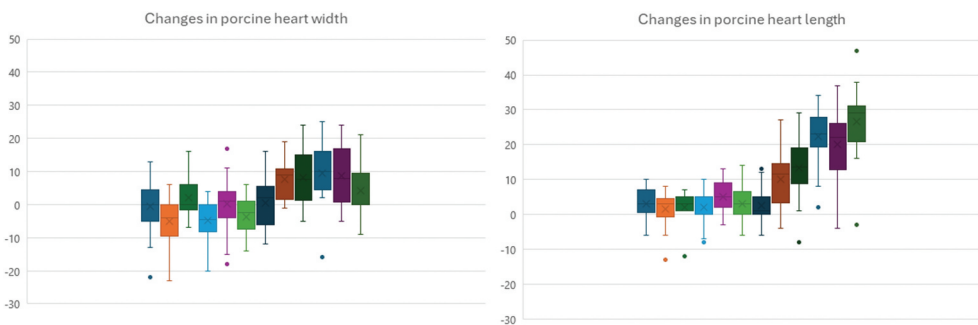

Figure 4. Changes (y-axis) in porcine heart width ($W_n - W_0$, n = day(s) lapsed in mm) and length ($L_n - L_0$, n = day(s) lapsed in mm) through days 2, 3, 5, 6, 7, 8, 10, 13, 14, and 18 (x-axis).

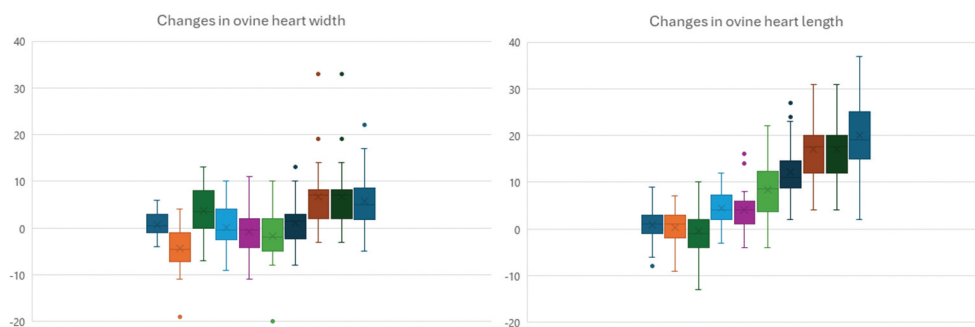**Figure 5.** Changes (*y*-axis) in ovine heart width (W_n-W_0, *n*=day(s) lapsed in mm) and length (L_n-L_0, *n*=day(s) lapsed in mm) through days 2, 3, 5, 6, 7, 8, 10, 13, 14, and 18 (*x*-axis).

respectively when the hearts had reached their endpoint (i.e. no further statistical changes and the heart being 'pancaked' and/or showing tissue fragmentation). The length of the heart continued to increase by the end of the study and the mean increase was 31 and 26 mm for porcine and ovine heart respectively.

Weighing the heart is routine in human post-mortem examination. The heart weight is compared with established referenced tables, charts and/or online calculators [4,7,8,11–16]. If the heart weight is increased, the heart can be defined macroscopically as having cardiac hypertrophy [1,2,4]. Cardiac hypertrophy is associated with sudden cardiac death, and it is critical to identify it at post-mortem examination [1,2,4]. In the post-mortem examination the heart weight is assumed to be equivalent to that during life. However, during the post-mortem interval, decomposition can occur resulting in loss of fluid and subsequent parenchymal tissue. As such, heart weights measured in decomposed bodies may be lower than during life. This would result in underappreciation of cardiac hypertrophy in decomposed bodies.

Currently, there is no established method in estimating/back calculating the heart weight at the time of death in decomposed bodies. Two recent studies documented that heart dimensions, especially heart width, can be used to estimate heart weight and to identify cardiac hypertrophy, as it is intuitive that a visually larger heart should weigh more [5,6]. In our experience, different from heart weight, heart dimensions tend to increase, rather than decrease, in a decomposing heart. However, there is scant literature investigating this observation or proof of this.

This study showed that, in porcine and ovine hearts, the width and length of the heart ultimately increases, which was expected. However, in the initial 7–10 days, the width appears to fluctuate and gradually increase, whereas the length increased consistently throughout the study period. This may be explained by structural heterogenicity between the fibrous annulus and musculature. The relatively ridged fibrous annulus ring may have been partially folded, rendering a decreased width when placed on the dissection board whilst the heart musculature started to soften and lose structural integrity during decomposition., The length of the heart, which comprises musculature, followed a more gradual increasing trend without much fluctuation. The initial fluctuation of the heart width was considered minimal and the mean changes for both porcine and ovine were less than 1.5 mm.

In this study the 'end point' was initially determined when the heart was completely flattened and/or showing obvious fragmentation. This was then confirmed by demonstrating three consecutive days with no significant changes in either heart width or length. At the end points for both porcine and ovine hearts, mean width increased by 8 and 6 mm respectively, whereas the length continued to increase. During decomposition when the musculature loses its integrity, the heart gradually become flattened/'pancaked'. It is probable that the fibrous ring was more robust in maintaining the width of the heart. This characteristic can be potentially used to calculate/estimate heart weight at the time of death in humans.

Applications of results to human subjects

Heart weight can be estimated using the equation: heart weight (g) $= -298 + 5.29\,8*$ heart width (mm) ($R^2 = 0.71$) [6], and the thresholds for heart width in estimating heart weight of >400 g and >500 g for cardiac hypertrophy in female and male are 118.5 mm and 129.5 mm respectively [5]. With the result from this study, heart width may potentially be used as a guide in determining cardiac hypertrophy in decomposed human subjects (Figure 6).

During decomposition, the heart weight decreases and dimensions increase. As heart weight decreases in decomposition, an increased heart weight in a decomposed heart would be suggestive of cardiac hypertrophy. When a heart exhibits decomposition and the heart weight is not increased, either the heart weight was normal at the time of death or there was a substantial loss of parenchymal tissue in a hypertrophied heart, leading to an artifactual normal heart weight. In this situation, the dimension of the heart can be useful in determining cardiac hypertrophy. If the heart weight estimated by the heart width is normal or below the threshold, then there is no cardiac hypertrophy. If the heart weight estimation using heart
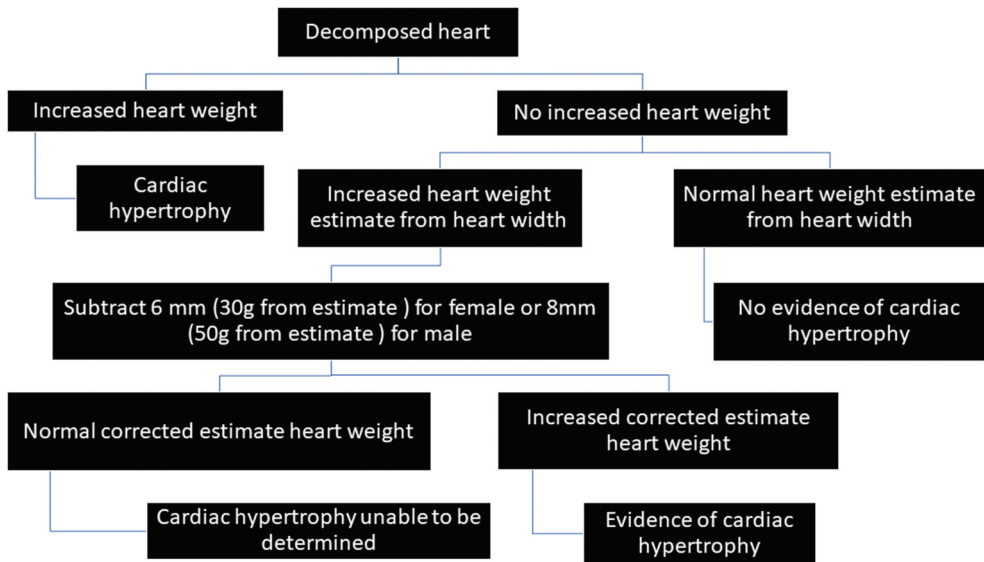

Figure 6. Proposed flow chart in assessing heart weight/cardiac hypertrophy from combining the findings from the literature and the presented study. The heart weight estimation from heart width used here is: heart weight (g) $= -298 + 5.29\,8*$ heart width (mm) ($R^2 = 0.71$) [6]. Thresholds for heart width in estimating heart weight of >400g and >500g are 118.5 mm and 129.5 mm [5].

width is increased or above the threshold, using the results in this study, the mean change in heart width would be approximately 6 mm and 8 mm overestimation for female and male, respectively. This would translate to 30–50 g overestimation in heart weight. If this was considered, and the heart weight estimate remains increased, the heart weight would be suggestive of cardiac hypertrophy.

Limitation

Using heart enlargement as a marker for cardiac hypertrophy

Heart enlargement is assessed by the physical dimension of the heart, and when it is enlarged it is termed as cardiomegaly. Whereas heart weight is used to determine cardiac hypertrophy macroscopically. Intuitively, there is an association between heart dimension and heart weight, as in a larger heart should weight heavier. This study uses heart dimensions in estimating heart weight in decomposed cases. However, it needs be highlighted that cardiomegaly and cardiac hypertrophy are not equivalent and some pathology can cause cardiomegaly and not hypertrophy and vice versa.

Use of ex-vivo animal heart models

This study used ex-vivo porcine and ovine heart models to study the changes in heart dimensions in decomposition, aiming to translate the results to human subjects. There are clear limitations with this method, which include non-human subjects, healthy animal hearts, and ex-vivo nature of the study. Using human hearts (healthy and/or pathological) and serially measuring heart dimensions during decomposition in-vivo is practically not feasible in routine post-mortem examination, and non-intrusive methods (imaging techniques) are not validated in the post-mortem population. Owing to the serial observations needed in this study, the animal hearts were not dissected, and blood clots were not completely removed from the heart chambers when the hearts were weighed. This is contrary to the standard/recommended method in human post-mortem examination [17–19] and additional error is expected if this method of assessment is translated to human post-mortem examination. Although having its limitations and not completely translatable to human subjects, the model chosen (ex-vivo porcine and ovine hearts to model healthy male and female hearts) is a practical and feasible method to serially measure heart dimensions during decomposition. Translating the results from the study to humans needs to be done with great caution and, when feasible, validation studies on humans would be ideal in the future. Furthermore, this study only used normal animal hearts, further studies on pathological animal hearts, particularly with an element of fibrosis, is recommended.

End point establishment

The end point in this study was determined when the heart is flattened, showing tissue disintegration, and/or having no significant change in dimension, which was at 18 days. Decomposition is a very variable process with multiple factors contributing to the rate, intensity and characteristics [1–3], This study was not designed to model the wide range of factors affecting decomposition. It only attempted to examine the change in heart dimension during putrefaction, the most common type of decomposition. The results of this study cannot be directly translated to different types of decomposition or hearts that have gone beyond the 'end point'.

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Disclosure statement

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