Word count of abstract: 236; Word count of manuscript: 3254

References: 44; Figures: 3; Tables: 4

Title page

Title: Effect of sex, age and body measurements on heart weight, atrial, ventricular, valvular and subepicardial fat measurements of the normal heart

Running title: Effect of sex, age and body on the heart

JD WESTABYa, E ZULLOa, LM BICALHOa, RH ANDERSONa, MN SHEPPARDa

aCRY Cardiovascular Pathology Unit, Cardiovascular Clinical Academic Group, Molecular and Clinical Sciences Research Institute, St. George's, University of London.

We have no conflicts of interest to declare.

Corresponding author: Dr Joseph Westaby, CRY Cardiovascular Pathology Unit, Cardiovascular Clinical Academic Group, Molecular and Clinical Sciences Research Institute, St George’s University of London, London, SW17 0RE.

Tel: 0208725 5112; Fax: 020 8725 5139; Email: jwestaby@sgul.ac.uk

Original article.

**Abstract and key words**

Aims

Descriptive morphological studies of the normal heart are lacking. Previous autopsy studies have focussed mainly on heart weight. We characterise the normal heart by providing normal dimensions of the atria, ventricles, valves and subepicardial fat, comparing the findings in terms of sex, age and body measurements.

Methods

From 3602 referrals to our cardiovascular pathology unit, pathological criteria used for the classification of a morphologically normal heart were a weight of below 500 grams in males, and below 400 grams in females. Diseased hearts were excluded on anatomical and histological evaluation.

Results

We diagnosed 1062 morphologically normal hearts. Mean age at death was 34±12, with a male predominance (701, 66%). Age was similar in females and males (35±13 vs 34±12). Females had a significantly lower heart weight (285±55 vs 374±64). Sex was an independent predictor of most measurements.

The atrial and ventricular cavities were significantly larger in males. All ventricular measurements of muscle thickness were larger in males. All valvular circumferences were larger in males. In contrast, subepicardial fat was significantly thicker in females in 6 of 7 regions. This is the first study to provide a calculator to give expected values according to sex, age, height and weight.

Conclusions

Major differences between the sexes exist in the morphologically normal heart. These variations should be considered when assessing cardiac structure in imaging for risk stratification and diagnosis in the cardiomyopathies, as well as in treatment outcomes.

Key words

Morphologically normal heart, sex, atria, ventricles, valves, subepicardial fat.

**Text**

1. **Introduction**

Sudden cardiac death (SCD) is a leading cause of mortality, accounting for around half of all deaths from cardiovascular disease and the majority are due to coronary artery disease (Wong et al., 2019). Sudden adult death syndrome, where the heart is morphologically normal, is an important finding particularly in young individuals dying suddenly (Fabre and Sheppard, 2006). The presence of a normal heart at autopsy, with negative toxicology, points to an underlying electrical abnormality in myocyte channels referred to as channelopathy (Lahrouchi et al., 2017).

Whilst there is rich literature regarding the pathology of structural heart disease (Finocchiaro et al., 2019; Miles et al., 2019; Westaby et al., 2021), little has been written on the morphologically normal heart. Previous autopsy studies have focused on organ weights (Kitzman et al., 1988; de La Grandmaison et al., 2001; Wingren and Ottosson, 2015; Skurdal and Nordrum, 2016). This study is the first to characterise the normal heart in a cohort of individuals dying suddenly in terms of the heart weight, fossa size and patency, coronary artery dominance, dimensions of the atria, ventricular chambers and muscle thickness, valve circumference and subepicardial fat and correlate with sex, age, body weight, body height, body mass index (BMI) and body surface area (BSA). This will act as a baseline for future studies as well as correlation with imaging.

1. **Methods**

The study is undertaken at the Cardiac Risk in the Young (CRY) Cardiovascular Pathology Laboratory based at St George’s University of London. The centre receives cases of SCD from throughout the United Kingdom. Cases with morphologically normal hearts were identified from a cohort of 3602 referrals between 2013 and 2020.

BMI is calculated as weight (kilograms)/height2 (metres2). BSA is calculated by square root of (height (centimetres) x weight (kilograms)/3600).

Diagnostic criteria used for the classification of a morphologically normal heart are a heart weight of below 500grams in males, and below 400grams in females. Cases with significant coronary artery disease, valvular disease, congenital disease or hypertension are excluded. Cardiomyopathies, myocarditis and infiltrating diseases, such as amyloid, are excluded on histology.

All hearts were examined macroscopically and microscopically in a formalin fixed state. Coronary artery dominance is judged on the basis of the artery extending to the cardiac crux. The left atrium is measured between the ostia of the left and right superior pulmonary veins, and from the atrioventricular junction to the superior surface (Figure 1). The right atrium is measured from the mouth of the inferior caval vein to the tip of the appendage and between the mouths of the inferior and superior caval veins (Figure 1). The fossa ovalis is measured from inferior to superior and from anterior to posterior and a 2millimeters2 probe is introduced to assess for patency (figure 2). Macroscopic measurements of the thickness of the ventricular wall and subepicardial fat, along with the cavitary diameters, are taken at a midventricular level (figure 3). The right ventricular outflow tract wall is measured anteriorly 10millimeters below the pulmonary valve. Muscular wall measurements exclude the trabeculae and papillary muscles. The ascending aorta circumference is measured 20 mm above the aortic valve.

All the variables are explored and summarized according to their statistical type; categorical data as frequencies and percentages, and continuous data as means and standard deviations. Two-sided T-test is used to compare normally distributed continuous variables. The Chi-square test is used to assess independency between two categorical variables with Fisher’s exact test used when cross tabulations exhibit numbers smaller than 5. The Kruskal-Wallis test is used to assess non-normally distributed or ordinal variables. Multiple linear regression is used to determine the most important predictors of outcome variables. The statistical software package SPSS package 27 is utilised to perform these tests.

Ethical and research governance was prospectively reviewed and approved for this study (10/H0724/38). All examination conforms to the standards set out by the United Kingdom Human Tissue Authority. In coronial autopsies, examination was undertaken under the jurisdiction of her majesty’s coroner. In hospital autopsies, informed consent for examination was gained from the highest qualifying relative as set out by the United Kingdom Human Tissue Authority. The investigation conformed to the principles outlined in the Declaration of Helsinki.

1. **Results**

There are complete measurements taken from 1062 morphologically normal hearts, referred between 2013 and 2020. The mean age at death is 34±12years with a male predominance (n=701, 66%, ratio 1.9:1). Females died at a similar age to males (35±13years vs 34±11years). The mean BMI is 27±6kilograms/metre2, which is similar between females and males (27±6kilograms/metre2 vs 27±6kilograms/metre2). As expected, BSA is lower in females compared to males (1.83±0.27metres2 vs 2.04±0.24 metres2, p<0.001) (Table 1).

3.1 Heart weight

The mean heart weight is 344±74grams. Females have a significantly lower heart weight than males (285±55grams vs 374±64grams, p<0.001) (Table 2). Sex is the most important predictor of heart weight. We found that heart weight increases with BSA and age (Table 3).

3.2 Coronary artery dominance

The pattern of coronary arterial arrangement is right dominant in 999 (94%), left dominant in 52 (5%), and co-dominant in 11 (1%). These proportions are similar for females and males (Table 2).

3.3 Atrial measurements

In the right atrium, the mean transverse length between the mouth of the inferior caval vein and the tip of the right atrial appendage is 57±12millimeters. The mean longitudinal length between the orifices of the inferior and superior caval veins is 41±10millimeters.

In the left atrium, the mean transverse length between the left and right superior pulmonary veins is 42±9millimeters. The mean longitudinal length between the atrioventricular junction and the superior surface of the left atrium is 37±8millimeters.

The measurements of both right and left atria are all significantly smaller in females compared to males (all p<0.011) (Table 2 and Figure 1). The atrial measurements all increase with age (Table 3).

3.4 Oval fossa measurements and patency

The oval fossa has a mean width of 15±5millimeters with a mean height of 14±4, and did not show differences between sexes. The fossa is patent in 111 of 917 cases (12%) (Figure 2). Probe patency is present in a slightly higher proportion of females than males (45, 14% vs 66, 11%) but this did not reach significance (Table 2). The fossa measurements increase with age (table 3).

3.5 Right ventricular chamber and muscle wall measurements

The mean diameter of the right ventricular chamber is 29±6millimeters. The mean right ventricular muscle wall thickness is 2.7±1.0millimeters for the anterior wall, 2.8±1.0millimeters for the lateral wall, 3.6±1.0millimeters for the inferior wall, and 3.2±1.0millimeters for the right ventricular outflow tract.

The right cavitary diameter is significantly smaller in females when compared to males (p<0.001). The diameter increases with BSA (Table 3). The right ventricular muscle wall thickness measurements are all significantly smaller in females when compared to males (all p<0.001) (Table 2 and Figure 3). Sex is the most important predictor of all muscle wall thickness measurements (Table 3).

3.6 Left ventricular chamber and muscle wall measurements

The mean diameter of the left ventricular chamber is 31±7millimeters. The septal wall thickness is 13±3millimeters. The mean left ventricular muscle wall thickness is 12±2millimeters for the anterior wall, 12±2millimeters for the lateral wall, and 12±2millimeters for the inferior wall.

The left cavity diameter is significantly smaller in females when compared to males (p=0.007). The diameter increases with BSA (Table 3). The left ventricular muscle wall thicknesses are all significantly smaller in females when compared to males (all p<0.007) (Table 2 and Figure 3). Sex is the most important predictor of all muscle wall thickness measurements (Table 3).

3.7 Subepicardial fat measurements

The mean thickness of the right ventricular subepicardial fat is 1.2±1.3millimeters for the anterior wall, 2.8±2.2millimeters for the lateral wall, 0.2±0.6millimeters for the inferior wall, and 0.9±1.2millimeters for the right ventricular outflow tract.

The mean thickness of the left ventricular subepicardial fat is 0.7±1.3millimeters for the anterior wall, 0.3±0.8millimeters for the lateral wall, and 0.1±0.6millimeters for the inferior wall.

All fat measurements are all significantly greater in females when compared to males (all p<0.045) (Table 4 and Figure 3). Fat measurements increase with age (Table 3).

3.8 Valvular measurements

The mean tricuspid valvular circumference is 97±14millimeters. The mean pulmonary valvular circumference is 54±10millimeters. The mean mitral valvular circumference is 77±13millimeters. The mean aortic valvular circumference is 52±8millimeters and the mean ascending aortic circumference is 53±8millimeters. All valvular circumferences, and the ascending aortic circumference, are significantly smaller in females when compared to males (all p<0.001) (Table 2). All valvular and ascending aorta circumferences increase with age (Table 3).

We attach an excel file which will give expected values and limits for the examined cardiac measurements based upon age, sex, body height and body weight (supplementary file).

1. **Discussion**

Here, for the first time, we provide comprehensive morphological analysis of a large cohort of normal hearts over a range of ages. The are striking differences between the male and female hearts. Whilst it is well recognised that the male heart is heavier than the female heart (de La Grandmaison et al., 2001; Wingren and Ottosson, 2015; Skurdal and Nordrum, 2016), this is the first study regarding the detailed constitution of the heart chambers.

4.1 Heart weight

Several previous studies have demonstrated that heart weight varies by sex, age and BSA but not correlated with body weight, body height or BMI. Kitzman et al reported on 765 hearts found an average heart weight of 280g for a 70kg female and 349g for an 80kg male similar to us (female: 285g; male: 374g). In contrast to our study, they found body weight was a better predictor of normal heart weight than BSA (Kitzman et al., 1988). De La Grandmaison et al, found an average heart weight of 365g in males and 312g in females which correlated best with age and BMI (de La Grandmaison et al., 2001). Wingren and Ottoson analysed 27,645 adult heart weights confirming that males had heavier hearts and was related to BMI, height and sex (Wingren and Ottosson, 2015). Skurdal and Nordrum, found average heart weight to be 316g in females compared to 395g in males relating it to sex and body weight(Skurdal and Nordrum, 2016). Gaitskell et al, found average heart weight was 388g in males and 338g in females and related it to sex and BSA(Gaitskell et al., 2011).

The average heart weights in these studies are comparable but higher than in our study of 285g in females and 374g in males. This is most likely due to the inclusion of hearts with weights above 400g in females and 500g in males which we would consider abnormal.

4.2 Atrial dimensions

This is the first study of atrial dimensions at autopsy. This is a novel method of measurement and these results will provide a reference for future studies as there is increasing emphasis on atrial disease including atrial fibrillation and atrial cardiomyopathy (Goette et al., 2016). All four atrial measurements are smaller in females and increase with age. A previous imaging study has demonstrated that atrial measurements are smaller in females confirming our findings (Kou et al., 2014a). Previous imaging studies of healthy living individuals have shown varied results on the effect of age on the atria (Aurigemma et al., 2009; D’Ascenzi et al., 2019). One previous study using echocardiography has reported that age, height and weight are independent predictors of left atrial width (Aurigemma et al., 2009) whereas another study has reported that age did not have an effect on atrial size (D’Ascenzi et al., 2019). The European Association of Echocardiography recommends a normal left atrial transverse diameter should be 27-40millimetres which is similar to our measurement (Evangelista et al., 2008).

4.3 Oval fossa

This is the first study on the size of the oval fossa. There was no difference between sexes. Three anatomical studies of 500-1000 hearts have identified probe patency of the oval fossa in 17-35% of individuals which is very variable (Thompson and Evans, 1930; Seib, 1934; Hagen et al., 1984). Patent oval fossa is a risk factor for paradoxical embolization and ischaemic stroke and is therefore important to document in the normal population (Homma and Sacco, 2005). An echocardiographic study of 1000 consecutive living patients found a patent fossa in 9.2% similar to our study (Fisher et al., 1995).

4.4 Ventricular chamber and wall measurements

Post mortem ventricular chamber diameter is unique to our study and has not been previously reported. Our findings confirm that males have large cavities with increase with body surface area.

Three large studies of living patients using echocardiography and magnetic resonance imaging have reported greater sizes of both ventricular chambers in males compared to females (Pfaffenberger et al., 2013; Kou et al., 2014b). The European Association of Cardiovascular Imaging provides normal values of cardiac chamber dimensions assessed by resonance imaging which are indexed by body surface area (Petersen et al.). This supports our finding that body surface area is the most important predictor of both ventricular chamber diameters as well as sex and age.

In contrast to our study, Kitzman et al, found no difference between one ventricular muscle wall measurement of unknown location in males and females and found no correlation with age and body surface area (Kitzman et al., 1988). Their reported means are 3.4-4.0mm for the RV and 10.8-12.6mm for the LV. Our measurements are similar to those reported for the younger age group in Kitzman’s report.

Echocardiography and resonance imaging demonstrates that both septal and inferior LV wall measurements are larger in males than females (Kou et al., 2014b; Pelà et al., 2016). A resonance imaging study reported RV mass was greater in males (Kawut et al., 2011). These reports confirm that males have greater myocardial muscle wall measurements.

4.5 Valve circumference

Kitzman et al, found larger circumferences in males (Kitzman et al., 1988). The tricuspid circumference was largest followed by the mitral circumference. The aortic and pulmonary valve circumferences were the smallest and of similar size. All valves showed increases with age which we confirm.

4.6 Coronary arterial dominance

Coronary artery dominance shows disparity between anatomical and living patient studies and between methods used for assessment. Historical pathological estimates range from 34-71% right dominance, 20-48% left dominance and 9-20% co-dominant in studies of 230-350 hearts (Paulsen and Vetner, 1973; von Lüdinghausen, 2003). There is no explanation for this wide variation. A more precise study of 1453 cases using post mortem coronary angiogram reports 81% right dominance, 9.1% left dominance and 10% codominance similar to us (Knaapen et al., 2013).

Our study observed a lower proportion of co-dominance but is within the lower end of the estimates for left dominance examined by angiography. Left dominant circulation has been associated with poorer prognostic outcomes in individuals with and without coronary disease assessed by computed tomography angiography. The presence of a left dominant circulation is an independent risk factor for extensive myocardial infarction and death (Veltman et al., 2012).

4.7 Subepicardial fat thickness

Our previous study of 148 normal hearts has demonstrated that RV subepicardial fat increased with age and was thicker in females than males. Subepicardial fat was greatest in the lateral right ventricle wall, followed by the anterior wall with little or no fat present posteriorly. The lateral wall subepicardial thickness ranged from 0-12mm (Tansey et al., 2005). A post mortem study of 56 cadavers has used photography to quantify the extent of subepicardial fat of diseased hearts (Silaghi et al., 2008). They also found that age independently correlated with subepicardial fat extent and the presence and stage of coronary artery disease. They do not comment on differences by sex.

4.8 Subepicardial fat associations and role in disease

Increased subepicardial fat has been related to myocardial mass (Corradi et al., 2004). Corradi et al, studied 117 hearts comparing normal hearts to those with hypertrophy and/or ischaemia and found that there was a preserved ratio of fat to muscle in myocardial hypertrophy suggesting that fat increases with muscle thickness.

Increased subepicardial fat has also been related to smoking and elevated heart rate (Miyazawa et al., 2018). Miyazawa et al, used computed tomography to examine 623 men aged 40-79 years without a history of cardiovascular disease following them up for an average of 4.7 years finding that current smoker status and increased heart rate were independently associated with increases in subepicardial fat volume.

The most established and promising relations, however, are to acute coronary events (Nakanishi et al., 2014) and atrial fibrillation (Hatem and Sanders, 2014). Nakanishi et al, examined 517 non-obese patients with coronary disease using computed tomography following them up for 4 years and found that increases in subepicardial fat volume, despite risk management, were associated with increased high risk or obstructive plaques. The largest report demonstrating the relationship between atrial fibrillation and subepicardial fat derives from the Framingham Heart study. Computed tomography was used to examine 3217 participants, of which 54 developed atrial fibrillation. Pericardial fat volume was found to be associated with prevalent atrial fibrillation following adjustment for risk factors including body mass index (Thanassoulis et al., 2010). It is therefore surprising that females have greater subepicardial fat despite having a lower prevalence of both ischaemic heart disease and atrial fibrillation (Chugh et al., 2014; Khan et al., 2020).

The extent of fat may also play a role in reducing the effects of catheter ablation (Nascimento Matos et al., 2020; Zipse et al., 2020). Nascimento Matos et al, followed up 575 patients undergoing pulmonary vein isolation for atrial fibrillation (Nascimento Matos et al., 2020). Subepicardial fat was quantified using computed tomography. They found that subepicardial fat was a strong independent predictor of atrial fibrillation relapse outperforming other recognised clinical risk factors. Zipse et al, used bovine myocardium with subepicardial fat to assess different radiofrequency ablation strategies (Zipse et al., 2020). They found that increasingly thick subepicardial fat attenuated lesion size regardless of strategy.

There are various reports on the role of subepicardial fat in disease. It has been proposed to regulate granulopoiesis, and to serve as a source for inflammatory mediators leading to cardiovascular disease (Horckmans et al., 2018). It has also been linked to fibrosis and function following myocardial infarction as well as myocyte dysfunction in diabetes (Greulich et al., 2012; Horckmans et al., 2018). The differences in subepicardial fat between the sexes should be adjusted for when assessing for disease.

4.9 Limitations

Our study will include cases where SCD is due to channelopathies. Future comparison to hearts with a non-cardiac cause of death will be valuable. Our study is based on fixed hearts and future comparison with fresh hearts will be important. Due to the relatively lower numbers of old aged individuals in our sample population, it is uncertain how the calculator will perform in these individuals. Interval between death and post mortem was not available and hearts with death in systole were included. These variables have not been accounted for and may influence cavity diameters.

1. **Concluding remarks**

When evaluating the abnormal, it is of central importance to understand the normal. Sex is the most important predictor of cardiac anatomy. We provide a calculator to give expected values according to sex, age, height and weight. The fact that subepicardial fat is higher in females should be accounted for when examining this population. The fact the female heart is smaller with regard to the measurements of muscle wall thickness, chamber diameters and valvular circumferences should be accounted for so as not to under call pathologic alterations. This is further emphasised by the fact that sex is a modifier of cardiovascular health, disease, and medicine where discrepant outcomes occur for a variety of reasons (Mauvais-Jarvis et al., 2020).

1. **Funding and Acknowledgements**

Cardiac Risk in the Young fund the Cardiac Risk in the Young Centre for Cardiovascular Pathology.

1. **Conflicts of interest**

We have no conflicts of interest to declare.

1. **Notes on Contributors**

Dr Joseph Westaby BSc, MSc, DIC, BM BS, PhD, CHAT, FRCPath, RCPathME

NIHR Academic Clinical Lecturer in Cardiovascular Pathology, Histopathologist and Autopsy Practitioner

Cardiac Risk in the Young Cardiovascular Pathology Laboratories, St George’s, University of London

Miss Emelia Zullo BSc

Laboratory Technician and MSc Student

Cardiac Risk in the Young Cardiovascular Pathology Laboratories, St George’s, University of London

Miss Luciana Bicalho BSc MRes

Laboratory Technician

Cardiac Risk in the Young Cardiovascular Pathology Laboratories, St George’s, University of London

Professor Robert H Anderson BSc, MBChB, MD, PhD, FRCPath

Professor of Paediatric Cardiac Morphology

Cardiac Risk in the Young Cardiovascular Pathology Laboratories, St George’s, University of London

Professor Mary N Sheppard BSc MB BCh BAO MD FRCPath

Professor of Cardiovascular Pathology and Head of Department of the Cardiac Risk in the Young Cardiovascular Pathology Laboratories

Cardiac Risk in the Young Cardiovascular Pathology Laboratories, St George’s, University of London

1. **References**

Aurigemma GP, Gottdiener JS, Arnold AM, Chinali M, Hill JC, Kitzman D. 2009. Left Atrial Volume and Geometry in Healthy Aging: The Cardiovascular Health Study. Circ Cardiovasc Imaging 2:282.

Chugh SS, Havmoeller R, Narayanan K, Singh D, Rienstra M, Benjamin EJ, Gillum RF, Kim YH, McAnulty JH, Zheng ZJ, Forouzanfar MH, Naghavi M, et al. 2014. Worldwide epidemiology of atrial fibrillation: A global burden of disease 2010 study. Circulation 129:837–847.

Corradi D, Maestri R, Callegari S, Pastori P, Goldoni M, Luong TV, Bordi C. 2004. The ventricular epicardial fat is related to the myocardial mass in normal, ischemic and hypertrophic hearts. Cardiovascular Pathology 13:313–316.

D’Ascenzi F, Piu P, Capone V, Sciaccaluga C, Solari M, Mondillo S, Henein M. 2019. Reference values of left atrial size and function according to age: should we redefine the normal upper limits? International Journal of Cardiovascular Imaging 35:41–48.

Evangelista A, Flachskampf F, Lancellotti P, Badano L, Aguilar R, Monaghan M, Zamorano J, Nihoyannopoulos P. 2008. European Association of Echocardiography recommendations for standardization of performance, digital storage and reporting of echocardiographic studies. European Journal of Echocardiography 9:438–448.

Fabre A, Sheppard MN. 2006. Sudden adult death syndrome and other non-ischaemic causes of sudden cardiac death. Heart 92:316–320.

Finocchiaro G, Papadakis M, Tanzarella G, Dhutia H, Miles C, Tome M, Behr ER, Sharma S, Sheppard MN. 2019. Sudden Death Can Be the First Manifestation of Hypertrophic Cardiomyopathy: Data From a United Kingdom Pathology Registry. JACC: Clinical Electrophysiology.

Fisher DC, Fisher EA, Budd JH, Rosen SE, Goldman ME. 1995. The incidence of patent foramen ovale in 1,000 consecutive patients. A contrast transesophageal echocardiography study. Chest 107:1504–1509.

Gaitskell K, Perera R, Soilleux EJ. 2011. Derivation of new reference tables for human heart weights in light of increasing body mass index. Journal of Clinical Pathology 64:358–362.

Goette A, Kalman JM, Aguinaga L, Akar J, Cabrera JA, Chen SA, Chugh SS, Corradi D, D’Avila A, Dobrev D, Fenelon G, Gonzalez M, et al. 2016. EHRA/HRS/APHRS/SOLAECE expert consensus on atrial cardiomyopathies: definition, characterization, and clinical implication. Europace 18:1455–1490.

Greulich S, Maxhera B, Vandenplas G, Wiza DH de, Smiris K, Mueller H, Heinrichs J, Blumensatt M, Cuvelier C, Akhyari P, Ruige JB, Ouwens DM, et al. 2012. Secretory products from epicardial adipose tissue of patients with type 2 diabetes mellitus induce cardiomyocyte dysfunction. Circulation 126:2324–2334.

Hagen P, Scholz D, Edwards W. 1984. Incidence and size of patent foramen ovale during the first 10 decades of life: an autopsy study of 965 normal hearts. Mayo Clin Proc 59:17–20.

Hatem SN, Sanders P. 2014. Epicardial adipose tissue and atrial fibrillation. Cardiovascular Research 102:205–213.

Homma S, Sacco RL. 2005. Patent foramen ovale and stroke. Circulation 112:1063–1072.

Horckmans M, Bianchini M, Santovito D, Megens RTA, Springael JY, Negri I, Vacca M, Eusanio M di, Moschetta A, Weber C, Duchene J, Steffens S. 2018. Pericardial adipose tissue regulates granulopoiesis, fibrosis, and cardiac function after myocardial infarction. Circulation 137:948–960.

Kawut SM, Lima JAC, Barr RG, Chahal H, Jain A, Tandri H, Praestgaard A, Bagiella E, Kizer JR, Johnson WC, Kronmal RA, Bluemke DA. 2011. Sex and race differences in right ventricular structure and function: The multi-ethnic study of atherosclerosis-right ventricle study. Circulation 123:2542–2551.

Khan MA, Hashim MJ, Mustafa H, Baniyas MY, Suwaidi SKBM al, AlKatheeri R, Alblooshi FMK, Almatrooshi MEAH, Alzaabi MEH, Darmaki RS al, Lootah SNAH. 2020. Global Epidemiology of Ischemic Heart Disease: Results from the Global Burden of Disease Study. Cureus 12.

Kitzman D w., Scholz D g., Hagen P t., Ilstrup D m., Edwards W d. 1988. Age-Related Changes in Normal Human Hearts During the First 10 Decades of Life. Part II (Maturity): A Quantitative Anatomic Study of 765 Specimens From Subjects 20 to 99 Years Old. Mayo Clinic Proceedings 63:137–146.

Knaapen M, Koch AH, Koch C, Koch KT, Li X, Rooij PC van, Tijssen JGP, Peters RJ, Wal AC van der, Damman P, Winter RJ de. 2013. Prevalence of left and balanced coronary arterial dominance decreases with increasing age of patients at autopsy. A postmortem coronary angiograms study. Cardiovascular Pathology 22:49–53.

Kou S, Caballero L, Dulgheru R, Voilliot D, Sousa C de, Kacharava G, Athanassopoulos GD, Barone D, Baroni M, Cardim N, Gomez De Diego JJ, Hagendorff A, et al. 2014a. Echocardiographic reference ranges for normal cardiac chamber size: results from the NORRE study. European Heart Journal - Cardiovascular Imaging 15:680–690.

Kou S, Caballero L, Dulgheru R, Voilliot D, Sousa C de, Kacharava G, Athanassopoulos GD, Barone D, Baroni M, Cardim N, Gomez De Diego JJ, Hagendorff A, et al. 2014b. Echocardiographic reference ranges for normal cardiac chamber size: Results from the NORRE study. European Heart Journal Cardiovascular Imaging 15:680–690.

La Grandmaison GL de, Clairand I, Durigon M. 2001. Organ weight in 684 adult autopsies: New tables for a Caucasoid population. Forensic Science International 119:149–154.

Lahrouchi N, Raju H, Lodder EM, Papatheodorou E, Ware JS, Papadakis M, Tadros R, Cole D, Skinner JR, Crawford J, Love DR, Pua CJ, et al. 2017. Utility of Post-Mortem Genetic Testing in Cases of Sudden Arrhythmic Death Syndrome. J Am Coll Cardiol 69:2134–2145.

Lüdinghausen M von. 2003. The Clinical Anatomy of the Coronary Arteries. Dvances in Anatomy Embryology and Cell Biology.

Mauvais-Jarvis F, Bairey Merz N, Barnes PJ, Brinton RD, Carrero JJ, DeMeo DL, Vries GJ de, Epperson CN, Govindan R, Klein SL, Lonardo A, Maki PM, et al. 2020. Sex and gender: modifiers of health, disease, and medicine. The Lancet 396:565–582.

Miles C, Finocchiaro G, Papadakis M, Gray B, Westaby J, Ensam B, Basu J, Parry-Williams G, Papatheodorou E, Paterson C, Malhotra A, Robertus JL, et al. 2019. Sudden Death and Left Ventricular Involvement in Arrhythmogenic Cardiomyopathy. Circulation 139:1786–1797.

Miyazawa I, Ohkubo T, Kadowaki S, Fujiyoshi A, Hisamatsu T, Kadota A, Arima H, Budoff M, Murata K, Miura K, Maegawa H, Ueshima H. 2018. Change in pericardial fat volume and cardiovascular risk factors in a general population of Japanese men. Circulation Journal 82:2542–2548.

Nakanishi K, Fukuda S, Tanaka A, Otsuka K, Jissho S, Taguchi H, Yoshikawa J, Shimada K. 2014. Persistent epicardial adipose tissue accumulation is associated with coronary plaque vulnerability and future acute coronary syndrome in non-obese subjects with coronary artery disease. Atherosclerosis 237:353–360.

Nascimento Matos D, Ferreira AM, Cavaco D, Sousa A, Freitas P, Rodrigues G, Carmo J, Abecasis J, Costa F, Santos AC, Carmo P, Saraiva C, et al. 2020. Epicardial fat volume outperforms classic clinical scores for predicting atrial fibrillation relapse after pulmonary vein isolation. European Heart Journal 41.

Paulsen S, Vetner M. 1973. Anatomical variations of the coronary arteries and origin of blood supply to sinoauricular and atrioventricular nodes determined on the basis of postmortem coronary angiography. Acta Pathologica Microbiologica Scandinavica Section A Pathology 81 A:784–790.

Pelà G, Crocamo A, Li Calzi M, Gianfreda M, Gioia MI, Visioli F, Pattoneri P, Corradi D, Goldoni M, Montanari A. 2016. Sex-related differences in left ventricular structure in early adolescent non-professional athletes. European Journal of Preventive Cardiology 23:777–784.

Petersen S, Khanji M, … SP-EH, 2019 undefined. European Association of Cardiovascular Imaging expert consensus paper: a comprehensive review of cardiovascular magnetic resonance normal values of cardiac. academic.oup.com.

Pfaffenberger S, Bartko P, Graf A, Pernicka E, Babayev J, Lolic E, Bonderman D, Baumgartner H, Maurer G, Mascherbauer J. 2013. Size matters! Impact of age, sex, height, and weight on the normal heart size. Circulation: Cardiovascular Imaging 6:1073–1079.

Seib GA. 1934. Incidence of the patent foramen ovale cordis in adult american whites and american negroes. American Journal of Anatomy 55:511–525.

Silaghi A, Piercecchi-Marti M-D, Grino M, Leonetti G, Alessi MC, Clement K, Dadoun F, Dutour A. 2008. Epicardial adipose tissue extent: relationship with age, body fat distribution, and coronaropathy. Wiley Online Library 16:2424–2430.

Skurdal AC, Nordrum IS. 2016. A retrospective study of postmortem heart weight in an adult Norwegian population. Cardiovascular Pathology 25:461–467.

Tansey DK, Aly Z, Sheppard MN. 2005. Fat in the right ventricle of the normal heart. Histopathology 46:98–104.

Thanassoulis G, Massaro JM, O’Donnell CJ, Hoffmann U, Levy D, Ellinor PT, Wang TJ, Schnabel RB, Vasan RS, Fox CS, Benjamin EJ. 2010. Pericardial fat is associated with prevalent atrial fibrillation: The framingham heart study. Circulation: Arrhythmia and Electrophysiology 3:345–350.

Thompson T, Evans W. 1930. Paradoxical Embolism. QJM: An International Journal of Medicine os-23:135–150.

Veltman CE, Graaf FR de, Schuijf JD, Werkhoven JM van, Jukema JW, Kaufmann PA, Pazhenkottil AP, Kroft LJ, Boersma E, Bax JJ, Schalij MJ, Wall EE van der. 2012. Prognostic value of coronary vessel dominance in relation to significant coronary artery disease determined with non-invasive computed tomography coronary angiography. European Heart Journal 33:1367–1377.

Westaby JD, Miles C, Chis Ster I, Cooper STE, Antonios TF, Meijles D, Behr ER, Sheppard MN. 2021. Characterisation of hypertensive heart disease: pathological insights from a sudden cardiac death cohort to inform clinical practice. J Hum Hypertens (in press; doi: 10.1038/s41371-021-00507-6).

Wingren CJ, Ottosson A. 2015. Postmortem heart weight modelled using piecewise linear regression in 27,645 medicolegal autopsy cases. Forensic Science International 252:157–162.

Wong CX, Brown A, Lau DH, Chugh SS, Albert CM, Kalman JM, Sanders P. 2019. Epidemiology of Sudden Cardiac Death: Global and Regional Perspectives. Heart Lung and Circulation.

Zipse MM, Edward JA, Zheng L, Tzou WS, Borne RT, Sauer WH, Nguyen DT. 2020. Impact of epicardial adipose tissue and catheter ablation strategy on biophysical parameters and ablation lesion characteristics. Journal of Cardiovascular Electrophysiology 31:1114–1124.

**Tables**

Table 1. Demographics for females and males.

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
| Variable | Unit | Summary statistic | Normal hearts (n=1062) | Female (n=361) | Male (n=701) | p value |
| Age | Years | Mean±SDMedian, Q1-Q3Range | 34±1233, 25-4118-100 | 35±1333, 25-4318-100 | 34±1132, 25-4018-82 | 0.119 |
| Sex | Male:Female | NumbersRatio | 701:361 1.9:1 | 361- | 701- |  |
| Height | cm | Mean±SDMedian, Q1-Q3Range | 174±10175, 167-181140-208 | 166±9166, 161-172140-193 | 179±8178, 173-184150-208 | **<0.001** |
| Weight | kg | Mean±SDMedian, Q1-Q3Range | 81±2080, 67-9129-176 | 74±2170, 60-8535-160 | 85±1883, 73-9329-176 | **<0.001** |
| BMI | kg/m2 | Mean±SDMedian, Q1-Q3Range | 27±626, 22-3010-58 | 27±725, 21-3113-53 | 27±626, 23-2910-58 | 0.898 |
| BSA | m2 | Mean±SDMedian, Q1-Q3Range | 1.96±0.271.97, 1.78-2.131.16-3.02 | 1.83±0.271.80, 1.65-1.991.17-2.80 | 2.04±0.242.03, 1.90-2.161.16-3.02 | **<0.001** |

The table gives the age, sex, height, weight, body mass index (BMI) and body surface area (BSA) for the overall cohort and broken down into males and females.

Table 2. The cardiac findings.

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
| Variable | Unit | Summary statistic | Normal heart (n=1062) | Female (n=361) | Male (n=701) | p value |
| Heart weight |  g | Mean±SDMedian, Q1-Q3Range | 344±74347, 291-396134-498 | 285±55282, 243-328134-399 | 374±64375, 334-419191-498 | **<0.001** |
| **Atria** |  |  |  |  |  |  |
| Right atrium 1 | mm | Mean±SDMedian, Q1-Q3Range | 57±1260, 50-6524-100 | 54±1255, 45-6025-80 | 58±1360, 50-6524-100 | **<0.001** |
| Right atrium 2 | mm | Mean±SDMedian, Q1-Q3Range | 41±1040, 35-5020-80 | 40±1040, 35-4520-80 | 42±1040, 35-5020-80 | **0.006** |
| Left atrium 1 | mm | Mean±SDMedian, Q1-Q3Range | 42±940, 35-5016-85 | 41±940, 35-4520-80 | 43±940, 35-5016-85 | **0.002** |
| Left atrium 2 | mm | Mean±SDMedian, Q1-Q3Range | 37±835, 30-4014-70 | 36±835, 30-4015-70 | 37±835, 30-4014-70 | **0.010** |
| **Oval fossa** |  |  |  |  |  |  |
| Width | mm | Mean±SDMedian, Q1-Q3Range | 15±515, 12-184-40 | 15±515, 12-1844-33 | 16±415, 12-185-40 | 0.337 |
| Height | mm | Mean±SDMedian, Q1-Q3Range | 14±414, 10-163-35 | 14±512, 10-163-33 | 14±414, 10-163-35 | 0.310 |
| Probe patent | Count | Number/Total(percentage) | 111/917(12%) | 45/312(14%) | 66/605(11%) | 0.122 |
| **Right ventricle** |  |  |  |  |  |  |
| Diameter |  mm | Mean±SDMedian, Q1-Q3Range | 29±630, 25-3510-48 | 28±627, 25-3010-45 | 30±630, 25-3510-48 | **<0.001** |
| Anterior wall | mm | Mean±SDMedian, Q1-Q3Range | 2.7±1.03.0, 2.0-3.00.5-8.0 | 2.4±1.02.0, 2.0-3.00.5-6.0 | 2.8±1.13.0, 2.0-3.01.0-8.0 | **<0.001** |
| Lateral wall | mm | Mean±SDMedian, Q1-Q3Range | 2.8±1.03.0, 2.0-3.01.0-10.0 | 2.5±1.02.0, 2.0-3.01.0-7.0 | 2.9±1.13.0, 2.0-3.01.0-10.0 | **<0.001** |
| Inferior wall | mm | Mean±SDMedian, Q1-Q3Range | 3.6±1.04.0, 3.0-4.01.0-10.0 | 3.3±0.83.0, 3.0-4.01.0-6.0 | 3.7±1.04.0, 3.0-4.01.0-10.0 | **<0.001** |
| RVOT wall | mm | Mean±SDMedian, Q1-Q3Range | 3.2±1.03.0, 3.0-4.01.0-7.0 | 2.9±1.03.0, 2.0-3.01.0-7.0 | 3.4±1.03.0, 3.0-4.01.0-6.0 | **<0.001** |
| **Left ventricle** |  |  |  |  |  |  |
| Diameter | mm | Mean±SDMedian, Q1-Q3Range | 31±730, 25-358-49 | 30±730, 25-358-49 | 31±730, 27-3510-48 | **0.007** |
| Septal wall | mm | Mean±SDMedian, Q1-Q3Range | 13±312, 11-157-21 | 12±211, 10-137-20 | 13±313, 12-158-21 | **<0.001** |
| Anterior wall | mm | Mean±SDMedian, Q1-Q3Range | 12±212, 10-147-20 | 11±210, 10-127-20 | 12±212, 11-147-20 | **<0.001** |
| Lateral wall | mm | Mean±SDMedian, Q1-Q3Range | 12±212, 11-147-22 | 11±211, 10-127-20 | 13±212, 11-158-22 | **<0.001** |
| Inferior wall | mm | Mean±SDMedian, Q1-Q3Range | 12±211, 10-137-20 | 11±210, 10-127-20 | 12±212, 11-147-20 | **<0.001** |
| **Valves and aorta** |  |  |  |  |  |  |
| Tricuspid valve circumference | mm | Mean±SDMedian, Q1-Q3Range | 97±1495, 90-10550-145 | 92±1390, 81-10055-130 | 99±14100, 90-11050-145 | **<0.001** |
| Pulmonary valve circumference | mm | Mean±SDMedian, Q1-Q3Range | 54±1050, 50-6025-106 | 51±950, 45-5525-80 | 56±1055, 50-6030-106 | **<0.001** |
| Mitral valve circumference | mm | Mean±SDMedian, Q1-Q3Range | 77±1375, 70-8545-125 | 74±1170, 65-8045-115 | 79±1480, 70-9045-125 | **<0.001** |
| Aortic valve circumference | mm | Mean±SDMedian, Q1-Q3Range | 52±850, 45-5533-90 | 50±750, 45-5535-75 | 53±850, 50-6033-90 | **<0.001** |
| Ascending aortic circumference | mm | Mean±SDMedian, Q1-Q3Range | 53±850, 50-6035-85 | 51±850, 45-5535-80 | 54±855, 50-6035-85 | **<0.001** |
| **Coronary artery dominance** |  |  |  |  |  |  |
| Right | Count | Number/Total(percentage) | 999/1062(94%) | 336/361(93%) | 663/701(95%) | 0.559 |
| Left | Count | Number/Total(percentage) | 52/1062(5%) | 20/361(6%) | 32/701(5%) |
| Codominance | Count | Number/Total(percentage) | 11/1062(1%) | 5/361(1%) | 6/701(1%) |

The heart weight, dimensions of the atria, dimensions of the oval fossa and probe patency, cavity size and muscle wall thickness for right ventricle and left ventricle, valve and ascending aorta circumferences and coronary artery dominance are given for the entire cohort and broken down by sex (RVOT: right ventricular outflow tract).

Table 3. Multiple linear regression analysis results.

|  |  |  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- | --- | --- |
| Variable | AdjustedR2 | Summary statistic | Intercept | Sex= Female | Body surface area | Age | Body weight | Height | Body mass index |
| Heart weight | 0.525p<.000 | CoefficientImportance | 56.682 | 68.276**0.45** | 133.0530.42 | 1.4420.13 | **-** | **-** | **-** |
| **Atria** |  |  |  |  |  |  |  |  |  |
| Right atrium 1 | 0.045p<.000 | CoefficientImportance | 30.873 | -3.078**0.43** | 21.0510.26 | 0.0680.16 | -0.2100.15 | **-** | **-** |
| Right atrium 2 | 0.031p<.000 | CoefficientImportance | 12.305 | - | - | 0.1080.46 | **-** | 14.601**0.54** | - |
| Left atrium 1 | 0.032p<.000 | CoefficientImportance | 15.897 | - | 21.087**0.43** | 0.0860.32 | -0.2230.25 | - | - |
| Left atrium 2 | 0.036p<.000 | CoefficientImportance | 12.458 | - | - | 0.115**0.60** | - | 11.6770.40 | - |
| FossaWidth | 0.041p<.000 | CoefficientImportance | 2.307 | - | - | 0.072**0.68** | - | 6.0980.32 | - |
| Fossa Height | 0.041p<.000 | CoefficientImportance | 2.089 | - | - | 0.072**0.74** | **-** | 5.3840.26 | **-** |
| **Right ventricle** |  |  |  |  |  |  |  |  |  |
| Diameter | 0.035p<.000 | CoefficientImportance | 22.832 | -1.4830.44 | 3.387**0.56** | - | - | - | - |
| Anterior muscle | 0.040p<.000 | CoefficientImportance | 2.377 | -0.348**0.76** | - | - | 0.0050.24 | - | - |
| Lateral muscle | 0.035p<.000 | CoefficientImportance | 2.221 | -0.359**0.69** | - | 0.0060.10 | - | - | 0.0180.22 |
| Inferior muscle | 0.047p<.000 | CoefficientImportance | 3.234 | -0.401**0.76** | - | - | - | - | 0.0190.20 |
| RVOT muscle | 0.071p<.000 | CoefficientImportance | 3.007 | -0.554**0.91** | - | - | - | - | 0.0150.09 |
| Anterior fat | 0.056p<.000 | CoefficientImportance | -0.128 | 0.2440.14 | - | 0.025**0.79** | - | **-** | 0.0160.07 |
| Lateral fat | 0.082p<.000 | CoefficientImportance | 0.006 | 0.3990.09 | - | 0.052**0.83** | **-** | - | 0.0340.08 |
| Inferior fat | 0.003p<.000 | CoefficientImportance | 0.126 | 0.075**1.00** | **-** | - | - | - | - |
| RVOT fat | 0.081p<.000 | CoefficientImportance | -0.658 | - | - | 0.029**0.91** | - | - | 0.0200.09 |
| **Left ventricle** |  |  |  |  |  |  |  |  |  |
| Diameter | 0.047p<.000 | CoefficientImportance | 17.610 | - | 5.613**0.74** | 0.0700.26 | - | - | - |
| Septal muscle | 0.111p<.000 | CoefficientImportance | 11.780 | -1.650**0.85** | - | - | - | - | 0.0620.15 |
| Anterior muscle | 0.110p<.000 | CoefficientImportance | 10.491 | -1.332**0.89** | 0.9530.11 | - | - | - | - |
| Lateral muscle | 0.086p<.000 | CoefficientImportance | 11.232 | -1.246**0.92** | 0.7510.08 | - | - | - | - |
| Inferior muscle | 0.084p<.000 | CoefficientImportance | 10.561 | -1.105**0.89** | 0.7980.11 | - | - | - | - |
| Anterior fat | 0.011p<.000 | CoefficientImportance | 0.289 | 0.2000.44 | **-** | 0.010**0.56** | - | - | - |
| Lateral fat | 0.041p<.000 | CoefficientImportance | -0.873 | 0.1360.12 | 0.3460.19 | 0.013**0.69** | - | - | - |
| Inferior fat | 0.013p<.000 | CoefficientImportance | -0.384 | 0.108**0.38** | 0.1750.25 | 0.0040.37 | - | - | - |
| **Valves & aortic circumference** |  |  |  |  |  |  |  |  |  |
| Tricuspid valve | 0.082p<.000 | CoefficientImportance | 46.154 | -3.9400.26 | - | 0.1620.33 | **-** | 26.452**0.42** | **-** |
| Pulmonary valve | 0.089p<.000 | CoefficientImportance | 16.167 | -2.4470.16 | - | 0.160**0.50** | - | 19.1050.34 | **-** |
| Mitral valve | 0.059p<.000 | CoefficientImportance | 46.705 | -3.587**0.34** | 24.4630.21 | 0.1270.32 | -0.2540.13 | - | - |
| Aortic valve  | 0.210p<.000 | CoefficientImportance | 28.392 | -2.9820.13 | - | 0.279**0.83** | - | 8.6730.04 | - |
| Ascending aorta | 0.281p<.000 | CoefficientImportance | 22.805 | -2.7020.07 | - | 0.343**0.88** | - | 11.0590.04 | - |

Multiple linear regression analysis to predict heart weight, atrial measurements, ventricular diameter, muscle thickness, epicardial fat thickness, using age, sex, weight, height, body mass index and body surface area. The most important predictors of heart weight and muscle wall thickness is sex.

Table 4. Epicardial fat thickness.

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
| Variable | Unit | Summary statistic | Normal heart (n=1062) | Female (n=361) | Male (n=701) | p value |
| **Right ventricle** |  |  |  |  |  |  |
| Anterior fat | mm | Mean±SDMedian, Q1-Q3Range | 1.2±1.31.0, 0-2.00-8.0 | 1.4±1.51.0, 0-2.00-8.0 | 1.1±1.21.0, 0-2.00-7.0 | **0.002** |
| Lateral fat | mm | Mean±SDMedian, Q1-Q3Range | 2.8±2.22.0, 1.0-4.00-15.0 | 3.1±2.52.0, 1.0-5.00-15.0 | 2.6±2.12.0, 1.0-3.00-15.0 | **0.002** |
| Inferior fat | mm | Mean±SDMedian, Q1-Q3Range | 0.2±0.60, 0-00-6.0 | 0.2±0.70, 0-00-6.0 | 0.1±0.50, 0-00-5.0 | **0.045** |
| RVOT fat | mm | Mean±SDMedian, Q1-Q3Range | 0.9±1.21.0, 0-1.00-9.0 | 1.0±1.31.0, 0-1.00-8.0 | 0.8±1.10, 0-1.00-9.0 | **0.029** |
| **Left ventricle** |  |  |  |  |  |  |
| Anterior fat | mm | Mean±SDMedian, Q1-Q3Range | 0.7±1.30, 0-1.00-10.0 | 0.8±1.40, 0-1.00-10.0 | 0.6±1.20, 0-1.00-7.0 | **0.011** |
| Lateral fat | mm | Mean±SDMedian, Q1-Q3Range | 0.3±0.80, 0-00-9.0 | 0.4±0.90, 0-00-9.0 | 0.3±0.80, 0-00-6.0 | 0.082 |
| Inferior fat | mm | Mean±SDMedian, Q1-Q3Range | 0.1±0.60, 0-00-9.0 | 0.2±0.80, 0-00-9.0 | 0.1±0.40, 0-00-4.0 | **0.027** |

The table shows the epicardial fat thicknesses for the anterior, lateral and inferior walls of the right and left ventricular as well as the right ventricular outflow tract (RVOT).

**Figure legends**

Figure 1. The atrial measurements for female and male hearts. The left atrium (LA) is measured between the ostia of the left and right superior pulmonary veins (LSPV and RSPV), and from the atrioventricular junction to the superior surface. The right atrium (RA) is measured from the mouth of the inferior caval vein (ICV) to the tip of the appendage (RAA) and between the mouths of the inferior and superior caval veins (SCV). Note the larger measurements in males.

Figure 2. The normal oval fossa which may be probe patent. The left panels illustrate the oval fossa from the right atrium and the right panels illustrate the oval fossa from the left atrium. The top panels show a case which is not probe patent and the bottom panels show a case where there is probe patency (circled in red). LAA: left atrial appendage; MV: mitral valve; OF: oval fossa; PP: probe patent; RAA: right atrial appendage; SCV: superior caval vein; TV: tricuspid valve.

Figure 3. The ventricular measurements for female and male hearts. Both the subepicardial fat (yellow) and muscle wall thicknesses (pink for females and blue for males) are measured in the anterior, lateral and inferior positions on the right ventricle (RV) and left ventricle (LV). The interventricular septum (IVS) is measured. Muscle wall thickness measurements exclude the trabeculae. Chamber diameters are measured between the mid septum to the lateral wall including the trabeculae. All these measurements are taken at a midventricular level. Note the larger muscle wall thickness and chamber diameters in males and the larger subepicardial fat thickness in females.