LUND UNIVERSITY

Infarct transmurality and adjacent segmental function as determinants of wall thickening in revascularized chronic ischemic heart disease.

Ugander, Martin; Cain, Peter; Perron, Annick; Hedström, Erik; Arheden, Håkan

Link to publication

Infarct Transmurality and Adjacent Segmental Function as Determinants of Wall

Thickening in Revascularized Chronic Ischemic Heart Disease

Martin Ugander MD, Peter Cain MBBS PhD, Annick Perron MSc, Erik Hedström BSc,

Håkan Arheden MD PhD

Cardiac MR Group, Department of Clinical Physiology

Lund University Hospital, Lund, Sweden

Short title: Determinants of wall thickening in chronic IHD

Corresponding author:

Associate Professor Håkan Arheden, MD PhD

Department of Clinical Physiology

Lund University Hospital

SE-22185 Lund, Sweden.

Telephone: +4646173328

Fax: +4646151769

E-mail: hakan.arheden@med.lu.se

Summary

BACKGROUND: There are many factors which influence regional left ventricular wall thickening (WT) in ischemic heart disease (IHD). We used magnetic resonance imaging (MRI) to explore, in patients with chronic IHD, how regional WT is affected by both infarct transmurality (IT) and the function of adjacent segments. We also compared these findings with a group of healthy volunteers (controls). METHODS: Twenty patients (20 men, mean age 63, range 45-80 years) were imaged with cine MRI for function and delayed enhancement MRI for infarction six months after revascularization. Twenty age and sex matched controls underwent cine MRI. Short axis images were analyzed using a 12-segment per slice model in four midventricular slices per subject. RESULTS: WT and IT were inversely related (R^2=0.11, p<0.001). WT of non-infarcted segments in patients was lower than corresponding segments in controls (5.1 vs. 4.6 mm, p<0.001). WT in patients decreased with an increasing number of dysfunctional adjacent segments (p<0.001) and increasing IT (p<0.001). WT was more strongly influenced by the number of dysfunctional adjacent segments (t=-22.93, p<0.001) than by IT (t=-4.50, p<0.001). CONCLUSIONS: The number of dysfunctional adjacent segments is a greater determinant than infarct transmurality on regional wall thickening.

Key words: magnetic resonance imaging, regional left ventricular function, infarction

Introduction

Regional left ventricular myocardial function in patients with ischemic heart disease (IHD) may be affected by infarct transmurality (Weiss *et al.*, 1981; Lieberman *et al.*, 1981; Ellis *et al.*, 1985; Mahrholdt *et al.*, 2003; Ingkanisorn *et al.*, 2004), stunning (Braunwald & Kloner, 1982), hibernation (Rahimtoola, 1989) and the function of adjacent segments (Sakai *et al.*, 1985; Force *et al.*, 1986; Mahrholdt *et al.*, 2003). A number of regional quantitative echocardiographic measures of regional function have been developed over the past two decades. These measures of regional myocardial function, however, are often taken in isolation without regard to the function of surrounding segments (Hatle & Sutherland, 2000). The presence of three dysfunctional adjacent segments has been shown to influence the relationship between wall thickening and infarct transmurality in chronic IHD (Mahrholdt *et al.*, 2003). Yet, the effect of the number of dysfunctional adjacent segments has not to date been elucidated. Therefore, we used MRI to further explore the extent to which infarct transmurality and the function of adjacent segments affects regional left ventricular wall thickening in patients with IHD. We also compared the function of non-infarcted segments in patients with that of healthy volunteers (controls). Furthermore, patients with IHD were imaged six months after revascularization in an attempt to minimize the influence of stunning and hibernation on wall thickening.

Methods

Study population

The study was approved by the local institutional committee on human research and all subjects provided written informed consent. Inclusion criteria for patients included either elective first time coronary artery bypass grafting or first time ST-elevation acute myocardial infarction treated with acute percutaneous coronary intervention. All patients were imaged six months after revascularization of all stenosed vessels. Inclusion criteria for controls included the absence of a history of cardiovascular or systemic disease, blood pressure less than 140/90 mmHg and a normal electrocardiogram. Exclusion criteria for all subjects were absence of sinus rhythm, claustrophobia or contraindications for MRI.

MR Imaging

All imaging was performed in the short axis plane during end-expiratory apnea using a 1.5T system (Magnetom Vision, Siemens, Erlangen, Germany). An ECG-triggered cine gradient echo sequence (resolution 1.6 x 1.6 x 8 mm, gap 2 mm) was used for imaging regional LV function in all subjects (Engblom *et al.*, 2004). Infarct imaging was only performed in patients and was performed using a delayed contrast enhancement (DE) sequence consisting of a segmented inversion recovery turbo fast low-angle shot sequence (Simonetti *et al.*, 2001) (resolution 1.6 x 1.6 x 8 mm, gap 2 mm, inversion time set to null normal myocardium). DE MRI was commenced 20 minutes after intravenous injection of 0.2 mmol/kg body weight of an extracellular contrast agent (Magnevist®, gadopentetate dimeglumine, Gd-DTPA, Schering). This approach has been shown to enhance infarcted myocardium (Kim *et al.*, 1999) due to an increased tissue distribution volume of Gd-DTPA in infarcted regions (Tong *et al.*, 1993; Arheden *et al.*, 1999; Flacke *et al.*, 2001; Klein *et al.*, 2004).

Slice and segmental model

All studies were quantitatively analyzed using a 48-segment model with four midventricular short axis slices and 12 segments per slice. All images were analyzed using software developed in-house (Cain *et al.*, 2005). Quantification of wall thickening and scar, was performed along radial profiles from the geometrical centroid of the left ventricular lumen at every other degree, yielding 180 measurements per short axis slice. For any given parameter, the mean value of the 15 measurements (corresponding to 30 degrees) per segment was used as the value for a given segment. The four slices analyzed for all subjects were three, four, five and six cm from the apex, respectively. Slices closest to the base and apex were excluded to minimize errors in assessment of wall thickness introduced by the partial volume effect and atrioventricular plane movement, respectively.

Wall thickening

Wall thickening was defined as the change in radial wall thickness between end diastole and end systole. End diastole and end systole were defined globally as the time frame in which the volume of the LV was largest and smallest, respectively. Using cine images, endocardial and epicardial borders of the left ventricle were manually delineated in diastole and systole, excluding papillary muscles. Delineation was performed blinded to patient identity and delayed enhancement images. Wall thickness in diastole and systole were then quantified along radial spokes emanating from the centroid of the endocardial delineation. Wall thickening was defined as the difference between end systolic wall thickness and end diastolic wall thickness.

Dysfunctional adjacent myocardial segments

Dysfunction in a given segment was defined as wall thickening less than 30% (Chan *et al.*, 2000). This corresponded to a cut off of less than 3.5 mm wall thickening

in our population. Figure 1 describes the adjacent segment model. An adjacent segment was defined as a myocardial segment that bordered a given segment in the same short axis slice or the same segment in a more basal or apical slice. This resulted in a maximum of four adjacent segments. Hence, the model did not consider segments that were diagonally adjacent in a more basal or apical slice.

Variation in wall thickening between adjacent segments

In order to better understand the inter-relationship of wall thickening of adjacent myocardial segments, we explored the difference in wall thickening between adjacent segments for both controls and patients. Twelve segments from the midventricular slice located four cm from the apex were analyzed in all 20 patients and all 20 controls. This yielded 240 differences in wall thickening for analysis in each subject.

Infarction

Using delayed enhancement images, the area of hyperenhancement and the endocardial and epicardial borders of the left ventricular myocardium were delineated manually. Wall thickness and infarct thickness were quantified along radial spokes at 2 degree intervals and averaged according to the 12 segment model. Infarct transmurality was defined as infarct thickness divided by total wall thickness from the same DE image.

Statistics

Wall thickening is presented graphically as mean ± SEM. Data is given as mean ± SD unless otherwise specified. A p-value of less than 0.05 was considered statistically significant. Variation between data was tested by an independent t-test or one-way ANOVA with post hoc Bonferroni correction as appropriate. Correlation between variables was calculated using linear regression. Differences between standard deviations was tested by the F-test. A linear mixed model assuming compound

symmetry (SPSS for Windows, Release 11.0.1) was used to explore the effect size of

parameters contributing to wall thickening.

Results

Twenty patients, all men, and 20 age and sex matched controls were prospectively identified. Table 1 summarizes the characterics of the study population. No patient sought medical attention for chest pain during the time between revascularization and CMR imaging.

Wall thickening and infarct transmurality

Overall, there was a negative correlation between wall thickening and infarct transmurality (R^2=0.11, p<0.001). Figure 2 shows wall thickening for controls, and the relationship between wall thickening and quartiles of infarct transmurality for patients with chronic IHD. There was a decrease in wall thickening with increasing infarct transmurality. Mean wall thickening ± SD (mm) was 5.1 ± 1.8 for controls and 4.6 ± 1.9, 3.7 ± 2.1, 3.1 ± 1.8, 2.9 ± 2.5, and 2.5 ± 2.1 for patients with IT of 0%, 1-25%, 26-50%, 51-75% and 76-100%, respectively (p<0.001 by ANOVA). Wall thickening in non-infarcted segments in patients was lower than in corresponding segments in controls (p<0.001). In patients, all quartiles of infarct transmurality differed in wall thickening compared to 0% infarction (p<0.001). Also, the latter two quartiles of infarct transmurality differed in thickening compared to the quartile with 1-25% infarction (p=0.02 for both 51-75% and 76-100% infarction).

Wall thickening and function in adjacent segments

Figure 3 shows the relationship between wall thickening and the number of dysfunctional adjacent myocardial segments according to infarct transmurality. Mean wall thickening differed significantly according to number of dysfunctional adjacent segments (p<0.01 by post hoc ANOVA for all). The difference in wall thickening between 0% and other quartiles of infarct transmurality with the same number of dysfunctional segments was significant in a minority of subgroups, see Figure 3 for

details.

The results of the linear mixed model confirmed that the number of dysfuntional adjacent segments had a greater influence on wall thickening than did infarct transmurality (Table 2).

Variation in wall thickening between adjacent segments

Figure 4 shows histograms of the difference in wall thickening between adjacent segments in the same slice. This measure followed a normal distribution in both controls and patients. Figure 5 shows the SD for the mean difference in wall thickening according to postion of the adjacent segment. The SD for patients was consistently larger than the SD for controls (Same slice: 1.6 mm vs. 1.2 mm, F=1.72, p<0.001, Basal slice: 2.7 mm vs. 1.9 mm, F=1.49, p=0.001, Apical slice: 2.5 mm vs. 2.1 mm, F=1.41, p=0.004). The SD for apically and basally located adjacent segments was significantly greater than for adjacent segments in the same slice. See Figure 5 for details.

Discussion

The main findings of this study are that quantitative regional myocardial function is influenced moreso by the function of adjacent segments and to a lesser extent by infarct transmurality. Also, the average segmental wall thickening was greater in controls than in segments without infarction in patients with IHD.

The effect of the number of dysfunctional adjacent segments

The current study illustrates that regional myocardial thickening decreases with increasing numbers of dysfunctional adjacent segments and that this has a more powerful effect on wall thickening than infarct transmurality. Previous studies have recognized the presence of both reduced function in viable myocardium in the peri-infarct zone (Sakai *et al.*, 1985; Force *et al.*, 1986), and, paradoxically, normal function in infarcted segments otherwise surrounded by normal myocardium (Mahrholdt *et al.*, 2003). Techniques such as myocardial strain assessed by echocardiography have been used to assess regional myocardial function in an attempt to overcome the tethering effect of adjacent myocardial function (Urheim *et al.*, 2000). Although such techniques probably reflect the local function of a given myocardial segment, our results suggest that the radial function of adjacent myocardial segments is an important factor in determining regional myocardial function *per se*.

The effect of infarct transmurality

Our study showed a trend of decreasing wall thickening with increasing infarct transmurality. Notably, wall thickening for controls and patients with varying severity of infarct transmurality showed considerable variation and overlap. Thus, it may not be possible to discriminate between different infarct transmuralities based on wall thickening alone. Earlier studies using experimental acute infarction models describe a threshold phenomenon encompassing a marked decrease in regional function when

infarct transmurality exceeds approximately 20-40% transmurality (Lieberman *et al.*, 1981; Ellis *et al.*, 1985). This discrepancy between acute and chronic infarction may likely be due to the presence of post-ischemic stunning (Braunwald & Kloner, 1982) in these acute models. The current study, however, imaged patients six months after revascularization in an attempt to overcome the effect of such stunning. Our data demonstrates that mean wall thickening decreases mostly between 0% and 50% infarct transmurality and changes little once infarct transmurality exceeds 50%, which is supported by findings of a recent study (Ingkanisorn *et al.*, 2004). In comparison, Mahrholdt *et al* found a more pronounced decrease in wall thickening with >75% infarct transmurality. The differences between the results of the current study and those of Mahrholdt *et al* may be attributable to the presence of infarction in multiple vessel territories in the current patient population. Also, we quantified radial wall thickening using a centroid while Marhholdt *et al* used a centerline method.

Variation between adjacent segments

The greater variation of the difference in wall thickening between adjacent segments in patients compared to controls likely reflects a greater regional heterogeneity in wall thickening in patients, possibly due to an influence of infarction. The greater variation in wall thickening between basally or apically adjacent segments compared to those within the same slice is likely inherent to measurement methodology. Since wall thickening is measured by manual delineation of the myocardium, the wall thickening of adjacent segments in the same slice are based on a contiguous manual delineation. The measurement of wall thickening for apically or basally adjacent segments, however, is based on a separate delineation in a different slice and therefore is prone to greater variations in measurement.

Study limitations

Residual stunning or hibernation as a cause of reduced wall thickening could not completely be excluded because no coronary angiography or myocardial perfusion imaging was performed at the time of CMR. These potentially uncontrolled effects, however, may have been of limited influence considering that no patient sought health care for chest pain-related symtoms in the time between revascularization and CMR imaging. Other factors that are known to influence regional contractility include wall stress and left ventricular geometry. These factors have been studied extensively in acute myocardial infarction using various techniques (Pfeffer & Braunwald, 1990; Bogaert *et al.*, 2000; Jackson *et al.*, 2003) but never with the simultaneous accurate knowledge of infarct extent and transmurality offered by DE CMR. The current study did not examine wall stress or left ventricular geometry and this is a limitation. Future studies designed to control for these factors may offer greater insight into the relationship between infarction and function.

Wall thickening was only assessed in four midventricular slices. Therefore, the most apical and the most basal slice only had at most three adjacent segments, whereas the two middle slices had at most four adjacent segments. Therefore, the number of dysfunctional adjacent segments may have been underestimated by at most one segment in approximately 50% of the segments. Also, radial wall thickening was analyzed using a common centroid instead of the centerline method. The centroid method may be less accurate in patients with deformed left ventricles after infarction.

Conclusions

The results of the present study have demonstrated that regional wall thickening is more affected by the number of dysfunctional adjacent segments than infarct transmurality. Furthermore, regional myocardial function was shown to vary greatly in both normal myocardium and myocardium with varying degrees of infarction. Taken

together, these results underscore the difficulty of using resting function alone to accurately assess the extent and severity of mycardial infarction in revascularized chronic ischemic heart disease. Direct infarct imaging with DE CMR is an important tool in this setting.

Acknowledgements

References

Arheden H, Saeed M, Higgins CB, Gao DW, Bremerich J, Wyttenbach R, Dae MW, Wendland MF. Measurement of the distribution volume of gadopentetate dimeglumine at echo-planar MR imaging to quantify myocardial infarction: comparison with 99mTc-DTPA autoradiography in rats. *Radiology* (1999); **211**: 698-708.

Bogaert J, Bosmans H, Maes A, Suetens P, Marchal G, Rademakers FE. Remote myocardial dysfunction after acute anterior myocardial infarction: impact of left ventricular shape on regional function: a magnetic resonance myocardial tagging study. *J Am Coll Cardiol* (2000); **35**: 1525-1534.

Braunwald E, Kloner RA. The stunned myocardium: prolonged, postischemic ventricular dysfunction. *Circulation* (1982); **66**: 1146-1149.

Cain P, Ugander M, Palmer J, Carlsson M, Heiberg E, Arheden H. Quantitative Polar Representation of Left Ventricular Myocardial Perfusion, Function and Viability using SPECT and Cardiac Magnetic Resonance: Initial Results. *Submitted* (2005).

Chan J, Wahi S, Cain P, Marwick TH. Anatomical M-mode: A novel technique for the quantitative evaluation of regional wall motion analysis during dobutamine echocardiography. *Int J Card Imaging* (2000); **16**: 247-255.

Ellis SG, Henschke CI, Sandor T, Wynne J, Kloner RA. Relation between the transmural extent of acute myocardial infarction and associated myocardial contractility two weeks after infarction. *Am J Cardiol* (1985); **55**: 1412-1416.

Engblom H, Hedstrom E, Palmer J, Wagner GS, Arheden H. Determination of the left ventricular long-axis orientation from a single short-axis MR image: relation to BMI and age. *Clin Physiol Funct Imaging* (2004); **24**: 310-315.

Flacke SJ, Fischer SE, Lorenz CH. Measurement of the gadopentetate dimeglumine partition coefficient in human myocardium in vivo: normal distribution and elevation in acute and chronic infarction. *Radiology* (2001); **218**: 703-710.

Force T, Kemper A, Perkins L, Gilfoil M, Cohen C, Parisi AF. Overestimation of infarct size by quantitative two-dimensional echocardiography: the role of tethering and of analytic procedures. *Circulation* (1986); **73**: 1360-1368.

Hatle L, Sutherland GR. Regional myocardial function--a new approach. *Eur Heart J* (2000); **21**: 1337-1357.

Ingkanisorn WP, Rhoads KL, Aletras AH, Kellman P, Arai AE. Gadolinium delayed enhancement cardiovascular magnetic resonance correlates with clinical measures of myocardial infarction. *J Am Coll Cardiol* (2004); **43**: 2253-2259.

Jackson BM, Gorman JH, 3rd, Salgo IS, Moainie SL, Plappert T, St John-Sutton M, Edmunds LH, Jr., Gorman RC. Border zone geometry increases wall stress after myocardial infarction: contrast echocardiographic assessment. *Am J Physiol Heart Circ Physiol* (2003); **284**: H475-479.

Kim RJ, Fieno DS, Parrish TB, Harris K, Chen EL, Simonetti O, Bundy J, Finn JP, Klocke FJ, Judd RM. Relationship of MRI delayed contrast enhancement to irreversible injury, infarct age, and contractile function. *Circulation* (1999); **100**: 1992-2002.

Klein C, Nekolla SG, Balbach T, Schnackenburg B, Nagel E, Fleck E, Schwaiger M. The influence of myocardial blood flow and volume of distribution on late Gd-DTPA kinetics in ischemic heart failure. *J Magn Reson Imaging* (2004); **20**: 588-593.

Lieberman AN, Weiss JL, Jugdutt BI, Becker LC, Bulkley BH, Garrison JG, Hutchins GM, Kallman CA, Weisfeldt ML. Two-dimensional echocardiography and

infarct size: relationship of regional wall motion and thickening to the extent of myocardial infarction in the dog. *Circulation* (1981); **63**: 739-746.

Mahrholdt H, Wagner A, Parker M, Regenfus M, Fieno DS, Bonow RO, Kim RJ, Judd RM. Relationship of contractile function to transmural extent of infarction in patients with chronic coronary artery disease. *J Am Coll Cardiol* (2003); **42**: 505-512.

Pfeffer MA, Braunwald E. Ventricular remodeling after myocardial infarction. Experimental observations and clinical implications. *Circulation* (1990); **81**: 1161-1172.

Rahimtoola SH. The hibernating myocardium. *Am Heart J* (1989); **117**: 211-221.

Sakai K, Watanabe K, Millard RW. Defining the mechanical border zone: a study in the pig heart. *Am J Physiol* (1985); **249**: H88-94.

Simonetti OP, Kim RJ, Fieno DS, Hillenbrand HB, Wu E, Bundy JM, Finn JP, Judd RM. An improved MR imaging technique for the visualization of myocardial infarction. *Radiology* (2001); **218**: 215-223.

Tong CY, Prato FS, Wisenberg G, Lee TY, Carroll E, Sandler D, Wills J, Drost D. Measurement of the extraction efficiency and distribution volume for Gd-DTPA in normal and diseased canine myocardium. *Magn Reson Med* (1993); **30**: 337-346.

Urheim S, Edvardsen T, Torp H, Angelsen B, Smiseth OA. Myocardial strain by Doppler echocardiography. Validation of a new method to quantify regional myocardial function. *Circulation* (2000); **102**: 1158-1164.

Weiss JL, Bulkley BH, Hutchins GM, Mason SJ. Two-dimensional echocardiographic recognition of myocardial injury in man: comparison with postmortem studies. *Circulation* (1981); **63**: 401-408.

Tables

Table 1. Patient characteristics

	PCI (n=8)	CABG (n=12)	All (n=20)	Controls (n=20)
Age (y)	58 ± 8	66 ± 9	63 ± 9	60 ± 11
Male gender	8/8 (100%)	12/12 (100%)	20/20 (100%)	20/20 (100%)
Ejection fraction (%)	53.0 ± 6.2	44.2 ± 9.7	47.7 ± 9.4	59.2 ± 7.5
Time of imaging after revascularization (d)	176 ± 13	199 ± 21	190 ± 21	
Single vessel disease	7/8 (88%)	1/12 (8%)	8/20 (40%)	
Two-vessel disease	1/8 (13%)	6/12 (50%)	7/20 (35%)	
Three-vessel disease	0/8 (0%)	5/12 (42%)	5/20 (25%)	
Previous myocardial infarction...	8/8 (100%)	12/12 (100%)	20/20 (100%)	
...in LAD territory	6/8 (75%)	9/12 (75%)	15/20 (75%)	
...in LCX territory	1/8 (13%)	6/12 (50%)	7/20 (35%)	
...in RCA territory	2/8 (25%)	2/12 (17%)	4/20 (20%)	
Active smoker	3/8 (38%)	1/12 (8%)	4/20 (20%)	
Former smoker	3/8 (38%)	8/12 (75%)	11/20 (55%)	
Beta blocker	5/8 (63%)	10/12 (83%)	15/20 (75%)	
Angiotensin Converting Enzyme inhibitor	6/8 (75%)	4/12 (33%)	10/20 (50%)	
Angiotensin II receptor blocker	1/8 (13%)	0/12 (0%)	1/20 (5%)	
Calcium channel blocker	1/8 (13%)	0/12 (0%)	1/20 (5%)	
Diuretic	0/8 (0%)	3/12 (25%)	3/20 (15%)	
Statin	6/8 (75%)	11/12 (92%)	17/20 (85%)	
Oral anti-diabetic	0/8 (0%)	3/12 (25%)	3/20 (15%)	
Insulin	1/8 (13%)	2/12 (17%)	3/20 (15%)	

PCI = percutaneous coronary intervention, CABG = coronary artery bypass grafting,

LAD = left anterior descending, LCX = left circumflex, RCA = right coronary artery.

Table 2. Multivariate analysis of parameters contributing to segmental wall thickening.

Parameter contributing to wall thickening	Estimate	SE	*t*	p
Infarct transmurality of a given segment	-0.96	0.21	-4.50	<0.001
Number of dysfunctional adjacent segments	-1.10	0.05	-22.93	<0.001

SE indicates standard error. *t* indicates the *t* statistic which reflects the effect size of a parameter.

Figure Legends

Figure 1. A schematic diagram of the left ventricle showing the 12 segment per slice model in the four midventricular slices used for analysis (light grey). Four possible adjacent myocardial segments are shown (dark grey) in relation to a given segment (black). An adjacent segment could either be in the same slice (Same), or in a more apical (Apical) or basal (Basal) slice.

Figure 2. The relationship between wall thickening and quartiles of infarct transmurality. CIHD denotes patients with chronic ischemic heart disease. Error bars denote SEM. *** denotes $p < 0.001$. n denotes the number of myocardial segments in each group.

Figure 3. The relationship between wall thickening and number of dysfunctional adjacent myocardial segments according to infarct transmurality. Error bars denote SEM. * denotes $p < 0.05$ vs. 76-100%, † denotes $p < 0.01$ vs. 51-75% and ‡ denotes $p < 0.01$ vs. 76-100%. Numbers at the bottom of each bar denote the number of myocardial segments in each group.

Figure 4. Histograms showing an example of the normal distribution of the difference in wall thickening in millimeters between adjacent myocardial segments in the same slice. Data is shown for controls (A) and patients with chronic ischemic heart disease (B).

Figure 5. Standard deviation (SD) of the mean difference in wall thickening between adjacent segments. Data are presented for different relative locations of the adjacent

segment for both controls and patients with chronic ischemic heart disease (CIHD). The SD for patients was consistently larger than the SD for controls indicating a greater regional heterogeneity in wall thickening in patients. Both controls and patients exhibit a greater SD in apically and basally adjacent segments compared to adjacent segments in the same slice ($p<0.001$). Only controls had a significant difference between basally and apically adjacent segments ($p=0.03$).
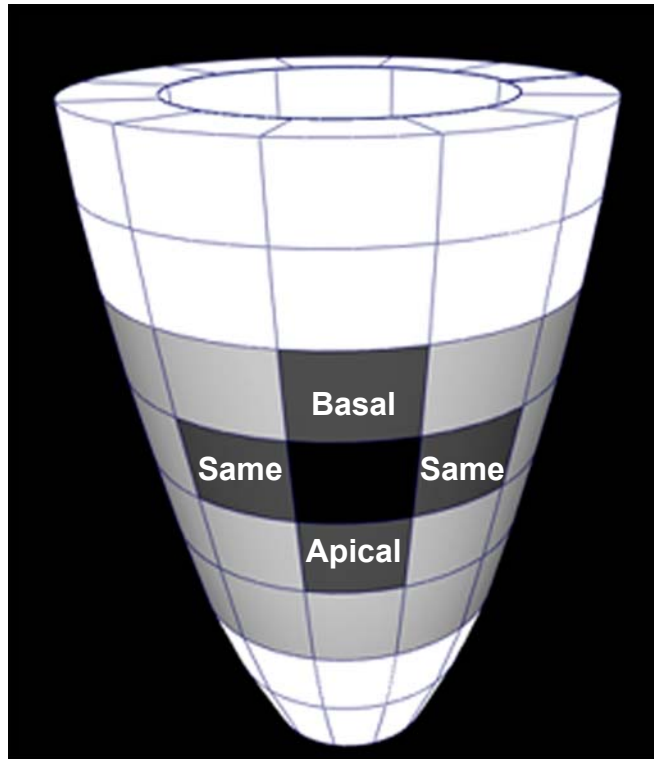
Figure 1

Figure 2
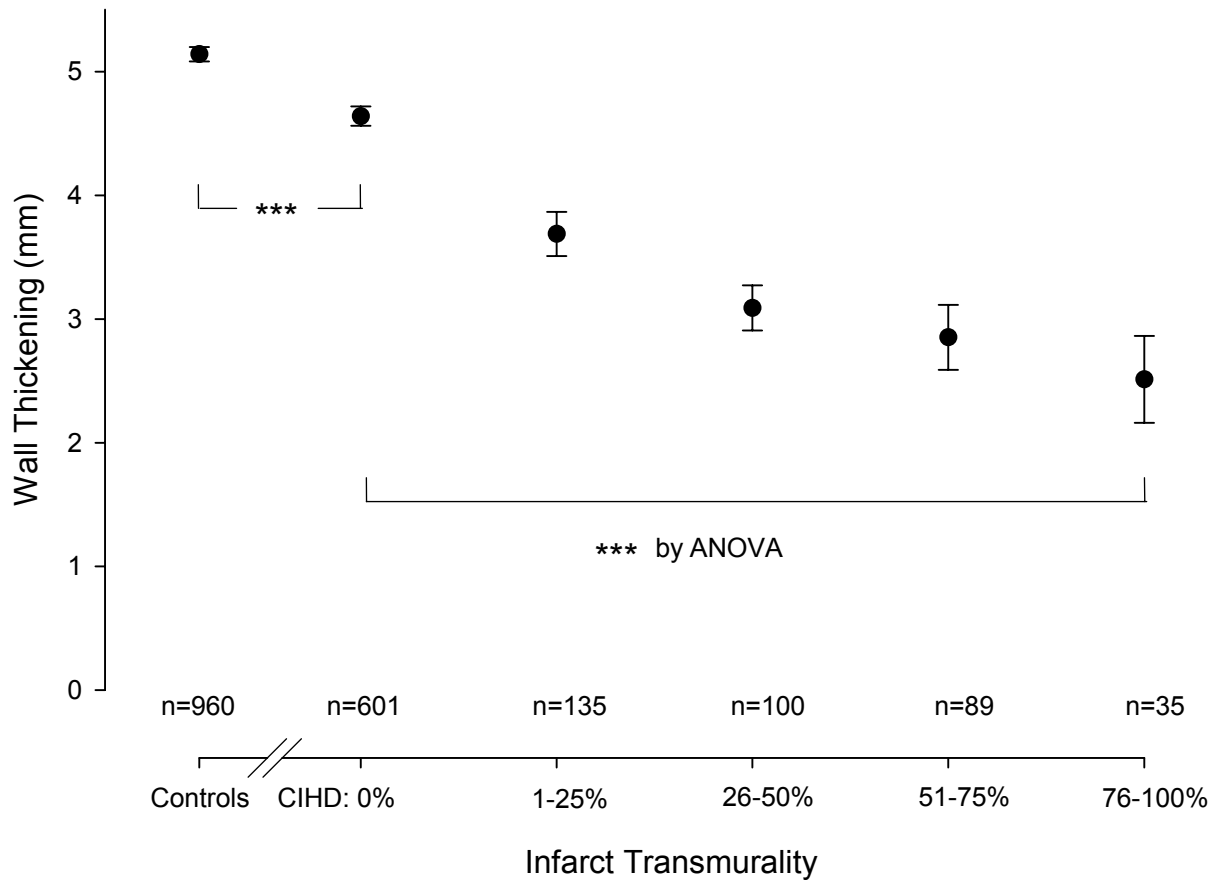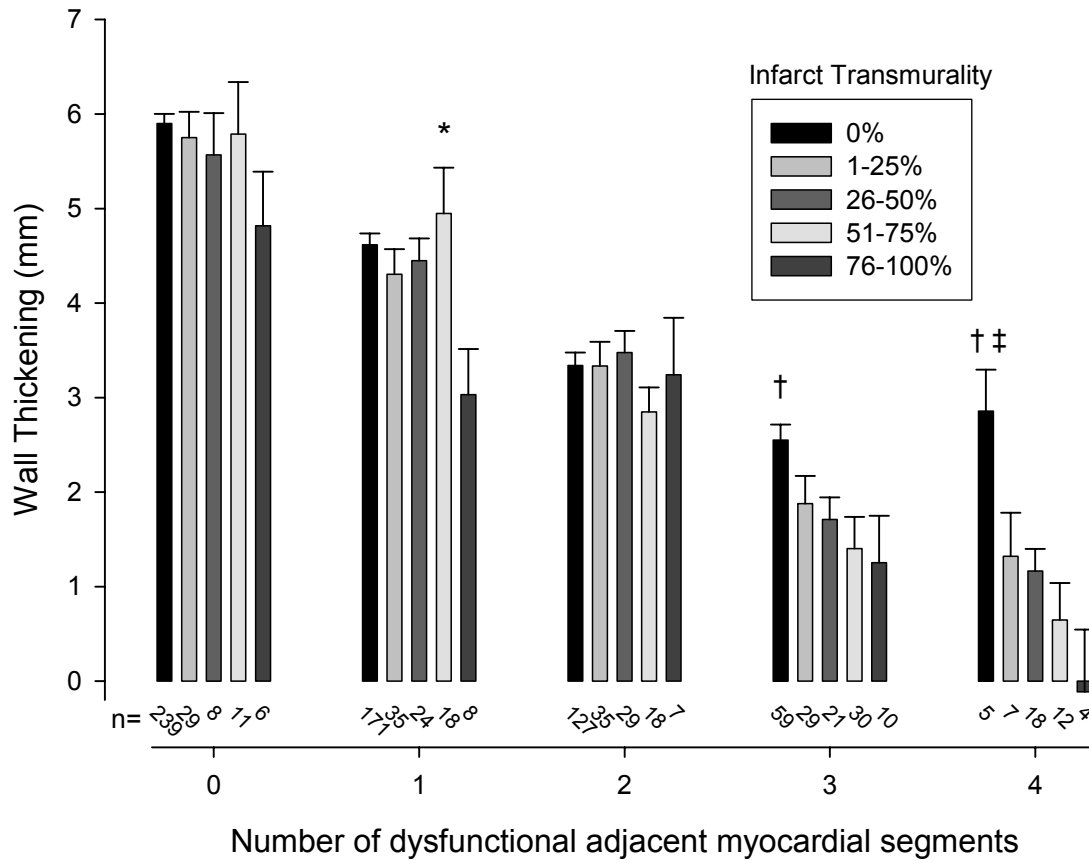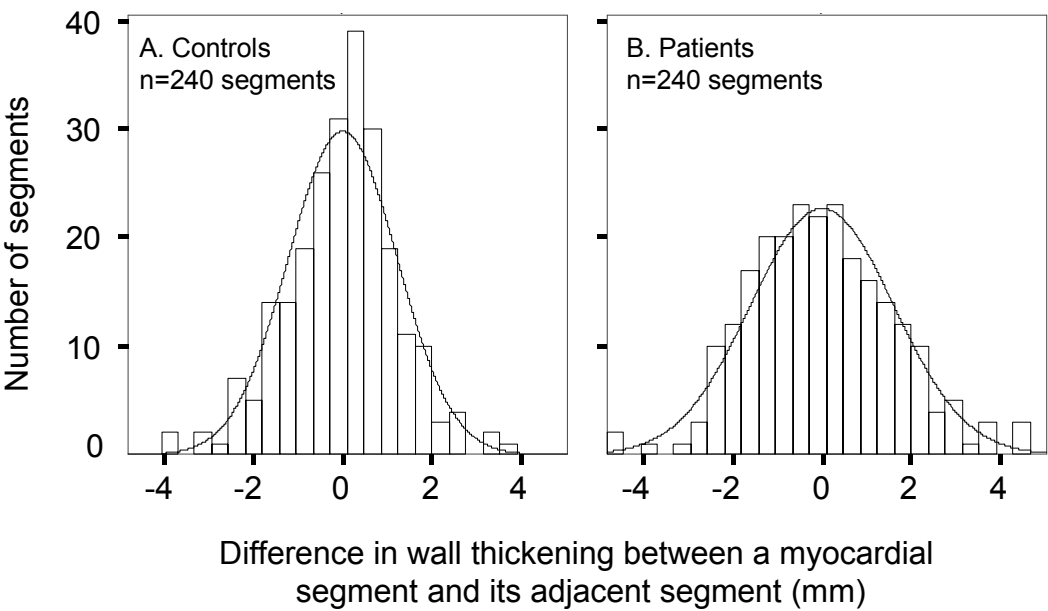
Figure 3

Figure 4

Difference in wall thickening between a myocardial
segment and its adjacent segment (mm)

Figure 5