EFFECTS OF THE

AORTA TO CORONARY BYPASS OPERATION

ON THE

RESTING SYSTOLIC TIME INTERVALS

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THESIS

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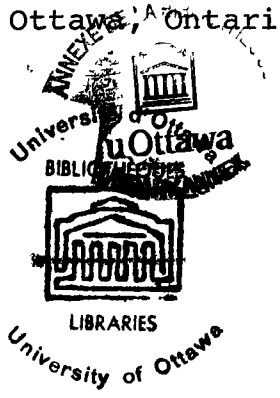

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CHAPTER I

THE PROBLEM

Introduction

New medical advances in the technological aspects of coronary care have provided physicians with an increasing spectrum of management techniques to arrest or at least decrease the serious effects of coronary artery disease on the human population. One technique now being employed to minimize the mortality and morbidity rates related to the disease is the aorta-to-coronary bypass operation (1,4,41,60). The purpose of this operation is to reestablish adequate coronary blood flow, hence oxygenation and contractility in the ischaemic myocardium. Secondary to the above mentioned effect is the relief of ischaemia and the resulting pain (angina pectoris) associated with the ischaemia.

However, it is recognized by many that if the bypass surgery is to remain effective, it should be complemented with a comprehensive rehabilitation program. Progressive physical activity is an important aspect of such a program and is considered to be one factor which may enhance the patient's psychological status and his ability to return to an optimal, productive life as early as possible.

Techniques available to physicians for clinically assessing the success of the operation for the purpose of initiating adequate physical therapy appear to be limited. The most practical assessment technique is the subjective feeling of the patient, i.e., whether or not his angina persists. If the patient's angina is not relieved, or worsens, the attending cardiologist may elect to recatheterize the patient for an haemodynamic reevaluation. Reevaluations prove somewhat problematic in terms of the limited catheterization facilities compared to the number of patients scheduled and the question of whether or not the results outweigh the inherent risks of the technique. An exercise tolerance test may be employed to determine both the functional capacity of patients treated by a coronary bypass operation and whether the ischaemia appears under the stress of exercise. The results of a tolerance test are also valuable to rehabilitation teams as they provide information necessary to prescribe exercises for successful therapy programs. However, the tolerance test cannot provide a precise view of myocardial function on a beat-to-beat basis, i.e., the ability of the heart to develop pressure and eject the blood into the systemic circulation. Such precise measures would provide additional information relating to the success of the coronary bypass

operation as well as part of the basis for the prescription of adequate exercise intensities for successful rehabilitation of the post-operative cardiac patient. A technique which could provide a measure of contractility and ability of the heart to eject blood from the ventricles and which has recently gained prominence is the systolic time intervals (24, 25, 42, 57, 63, 68, 79, 80, 81, 86). It is non-invasive as it does not require any puncturing of the skin, and may be used outside the hospital setting. With this technique, rehabilitators may obtain information about the intrinsic qualities of the myocardium and present at the same time, tests of overall functional capabilities. Together these give a more complete scope of the effects of the coronary bypass operation on the cardiac patient.

Statement Of The Problem

This study was designed to determine the effects of the aorta-to-coronary bypass operation on selected components of the resting systolic time intervals. The intervals to be studied are: the total systole ($Q\text{-}A_2$), ejection time (LVET), pre-ejection time (PEP), isovolumic contraction period (ICP) and the PEP/LVET ratio.

Scope

This study was designed to assess the effects of the

aorta-to-coronary bypass operation on selected components of the resting systolic time intervals (STI). The subjects used in this study were divided into three groups:

1. A control group of 12 normal subjects. None of these people demonstrated high risks in terms of heart disease.

2. A group of 12 cardiac patients, all presenting clinical evidence of at least 2-vessel coronary artery disease (CAD) determined by catheterization. All members of this group were scheduled to undergo a coronary bypass operation within a short time period after pre-operative evaluation. This group was subdivided into two groups of 6 members each, depending upon which cardiacs did or did not return for post-operative reevaluation.

2A. A group of those cardiac patients who did not return for a post-operative reevaluation. This group was referred to as the non-returning cardiac group.

2B. A group of those cardiac patients who did return for post-operative reevaluation. This group was referred to as the returning cardiac group.

Testing of all subjects was held in the Kinanthropology biokinetics laboratory at the University of Ottawa. The

cardiacs reported early in the morning of their respective evaluation days whereas the normals reported at selected times during the day. The non-returning cardiacs(group 2A) reported only once as did the normals whereas the returning cardiacs (group 2B) reported twice, once pre-operatively and once approximately two months post-operatively. The components of the STI were recorded by the simultaneous recording of the electrocardiogram (EKG), heart valve sounds, or phonocardiogram (PCG) and carotid arterial pulse wave (CPW).

Groups 1, 2A and 2B were statistically analyzed to determine if any differences existed among the groups in the STI components and PEP/LVET ratios at the initial testing session. Group 2B was statistically analyzed on a pre- to post-operative basis to determine if any changes in the STI components and PEP/LVET ratios occurred as a result of the treatment.

Limitations

1. The number of cardiac patients involved in the pre-operative evaluation was limited to 12 and only 6 of these persons returned for post-operative reevaluation.

2. Mitral closure within the first heart sound (S_1) was difficult to determine with reliability and thus presented a possible variance in the determination of the

isovolumic contraction period component of the STI.

3. The study was limited to the extent that the STI are a valid measure of myocardial contractility.

4. The study was also limited (for ethical reasons) by the absence of a cardiac control group. This group would not have undergone the coronary bypass operation. Therefore, it was not possible to establish a well balanced experimental design.

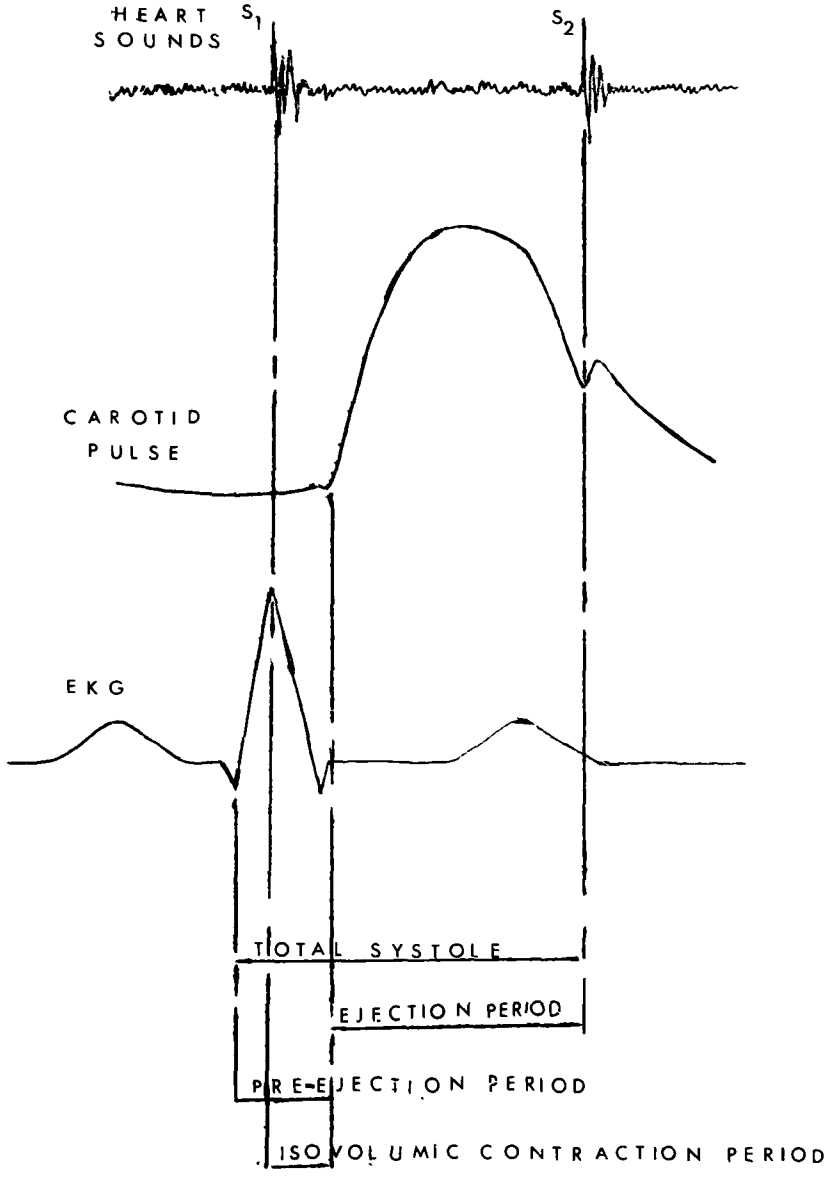
Definitions

The Systolic Time Intervals (STI) are distinct intervals of the systolic definitions phase of the cardiac cycle separable by electrical and mechanical events within that phase.

Total Systole ($Q-A_2$) is the total electrical and mechanical duration of ventricular systole (Fig. 1). It begins at the initiation of ventricular depolarization (Q-wave of EKG) and lasts until the beginning of ventricular relaxation as indicated by aortic valve closure (A_2). A_2 is the first high frequency vibrations of the second heart sound (S_2) on the PCG recording. The interval is directly related to heart rate and may be corrected in this relationship according to the regression formula proposed by Weissler (79).

Left Ventricular Ejection Time (LVET) is the period during which the blood is actively being ejected from the left ventricle into the systemic circulation (Fig. 1). It commences at the beginning of the rise of the carotid arterial pulse wave (CPW) and ends at the nadir of the dicrotic notch of the same wave. The interval is directly related to heart rate and may be corrected in this relationship according to the regression formula

Fig. 1, Systolic Time Intervals



proposed by Weissler (79).

Pre-ejection Period (PEP) is that time between the beginning of the spread of electrical activity in the ventricles (Q-wave of EKG) until the beginning of ejection (Fig. 1). This period is comprised of ventricular electrical activity leading to myocardial cellular contraction without any changes in the ventricular volume. The time interval is calculated by subtracting the LVET from the $Q\text{-}A_2$.

Mechanical Systole (MS) is that time between mitral closure (S_1) and aortic closure (S_2).

Isovolumic Contraction Period (ICP) is that time during which the ventricles are actively compressing the blood volume to increase intraventricular pressures from that of diastole to that of the systemic circulation without any volume changes (Fig. 1). This period is determined by subtracting LVET from MS. It may also be determined by subtracting the time of Q to S_1 from that of the PEP.

Heart Rate (HR) is the rate per minute and is calculated by measuring the R to R^1 interval and dividing by 60.

The subscript 'c' when appearing after the abbreviations $Q\text{-}A_2$ and LVET indicates that the associated component of the STI has been corrected for heart rate.

CHAPTER II

REVIEW OF LITERATURE

Introduction

The purpose of this study was to determine the effects of the coronary bypass operation on selected components of the resting systolic time intervals (STI). A review of the literature focused attention on the history, physiology and pathophysiology of each component interval. A brief review of the coronary bypass operation is then presented along with its possible implications relating to the resting STI components.

The Systolic Time Intervals

Basic studies of cardiac dynamics were documented in the early research of Wiggers (87). The cardiac cycle was divided into the commonly known phases of systole and diastole. These phases were then subdivided based upon their inherent electrical and mechanical properties. It was observed that unlike diastole, systolic events were significantly changed by such haemodynamic alterations as heart rate and blood pressure. Thus, the principle was established and confirmed that the events of systole were

influenced by blood volume and pressure changes within the ventricular chamber and contractility changes within the ventricular myocardium (29, 30, 58, 78, 84).

Shortly after Wigger's (87) observations, Katz (35) noted that all of the significant phases of systole could be identified by studying the combined recordings of ventricular valve sounds, arterial pulse wave curves and the electrocardiogram. This was the initial step in the formulation of the STI.

Blumberger (9) reported that while ventricular volumes on a beat-to-beat basis were quite variable, the mechanical events of systole were normally quite consistent. Wallace (76) observed the independent effects of heart rate, stroke volume and catecholamines on the LVET and ICP components of the STI. The results of this study were confirmed by Raab (57).

Total Systole ($Q-A_2$)

Total electro-mechanical systole extends from the Q-wave of the EKG to the aortic component of the second heart sound (A_2) (44, 78). The period begins at the initiation of electrical depolarization of the ventricles from the atrio-ventricular node. The ventricular myocardium contracts in response to the stimulus, compresses and ejects its blood volume. At the beginning of diastole

and ventricular relaxation the ventricular-systemic pressure differential during ejection reverses causing aortic valve closure. Closure of this valve is reflected in the first high frequency vibrations of the second heart sound on the PCG recording.

Wayne (78) suggested that the $Q\text{-}A_2$ is not a direct measure of myocardial performance but indicates fluctuations in the absolute time from ventricular stimulation to aortic valve closure. The interval is made up of two sub-components PEP and LVET which may fluctuate within the time range independent of the overall time of the $Q\text{-}A_2$. This measure demonstrates a direct and linear relationship to heart rate whether the heart rate changed spontaneously, by atrial pacing or by pharmacological interventions (9, 21, 29, 38, 39, 66, 77, 80, 81). The interval was shown to be correctable for heart rate fluctuations by the regression formula ($Q\text{-}A_{2c}$ = 2.1 x HR + measured value) proposed by Weissler (78). Positive preloading (increased end-diastolic volume or pressure) was not found to cause significant changes in this interval (9, 21, 29, 32, 77, 85). The constancy was related to positive and negative changes in the LVET and PEP respectively, of identical magnitude. The mechanism involved in these fluctuations was based upon an increased preload which in turn exerted a strain on the elastic component at the myofibrillar level in the ventricular

wall. This created a decreased difference of pressures between the ventricle and systemic circulation at the end of diastole. The overall effect was a lesser time taken to meet systemic pressures (shorter PEP) and a longer time to eject the increased blood volume (longer LVET). Chronic systemic pressure (afterload) increases resulted in a longer PEP and a shorter LVET with no apparent alterations in the total interval. The alterations in the sub-intervals were caused by a longer time taken to meet increased systemic pressures, and a shorter time of ejection perhaps because of an inability of the ventricle to sustain high enough pressures to eject its entire blood volume (88). On the other hand, transient afterload increases caused prolongation of the $Q\text{-}A_2$ because both subcomponents were lengthened. The factors causing this alteration were the longer time taken by the ventricles to attain elevated systemic pressures and the longer time taken to eject the entire ventricular volume (9, 30). When the preload and afterload were maintained, pharmacological interventions providing positive inotropic or force related effects on the myocardium were found to cause possible heart rate increases but definite reductions in the $Q\text{-}A_2$ through PEP and LVET shortening (9, 30, 57, 82, 84).

Few researchers have offered the $Q\text{-}A_2$ as a valuable diagnostic or prognostic indicator of pathophysiological conditions of the heart. Harley (29) noted in his pressure-flow studies that it was lengthened when the heart was slowed after bundle branch blockades were induced. Most of this influence was the result of a prolonged PEP while the LVET remained constant or increased as the heart rate declined. Bennett (8) found that a direct relationship existed between the degree of myocardial infarction and shortening of the $Q\text{-}A_2$ interval. His findings were supported by his observations that those persons demonstrating highest pulmonary diastolic pressures tended to show the shortest interval times in spite of comparable heart rates. It was not indicated, however, whether or not his intervals were corrected for heart rate. The intervals' reaction to myocardial failure led him to believe that it could be offered as a prognostic indicator of ventricular failure in AMI. Martin (42) noted that the $Q\text{-}A_2$, when related to its components PEP and LVET, could reflect small beat-to-beat changes in preload and afterload, thus supporting the hypothesis of Bennett (8). Other researchers questioned the clinical value of the interval as in many cases of AMI, the components of the

interval would alter significantly but the ($Q-A_2$) would not (18, 80, 81, 85). In many cases the effect of high levels of circulating catecholamines immediately following an AMI appeared to obscure the $Q-A_2$'s response to ventricular failure. These situations predisposed inaccurate conclusions when diagnoses were based on predicted characteristics of this interval following an AMI (8, 13, 18, 28, 32, 79). In addition, tests of patients with chronic ischaemia or a three-week old AMI demonstrated almost complete normality of this interval providing not over a certain amount of the myocardium was affected (22, 32).

Left Ventricular Ejection Period (LVET)

The LVET is that time of the $Q-A_2$ when the blood is actively being expelled from the ventricles. The absolute time of this period is determined from the beginning of the upstroke of the CPW and extends to the nadir of the dicrotic notch of the same wave. Normally, it is approximately 292 ± 19 msec (64). Martin (42) established a range for the value of 175 to 385 msec . However, Blumberger's (9) range was found to be 200 to 310 msec. Martin (42), using catheterization recordings to compare his non-invasive findings, concluded that a delay anywhere

from 10 to 35 msec could be expected on the CPW in any given patient in differing physiological conditions. Notwithstanding these deviations, the ejection time was found to be identical whether measured internally or externally. The LVET, like the $Q\text{-}A_2$ was found to be directly and linearly related to heart rate whether the latter was again changed spontaneously, by inotropic intervention or by atrial pacing (9, 21, 30, 38, 56, 57, 66, 79, 80, 81, 86). This relationship was assessed by Weissler (79) for the correction of LVET according to heart rate and indicated by the formula ($LVET_c = 1.7 \times HR$ + measured value) where $LVET_c$ indicates the corrected value.

Increases in preload were found to cause lengthening of the $LVET_c$ but the deviations were not beyond the range ascribed to the interval for the normal ventricles (29). In the failing left ventricle, however, the increase in time taken for ejection was extended beyond the range limit, indicating an inability to eject its entire volume within a prescribed time frame determined by the heart rate (22, 32, 38, 77, 85). Transient afterload increases and extreme decreases were found to prolong the ejection period (9, 30). The former alteration was noted to increase the flow

resistance against which the ventricle had to empty its entire volume while the latter permitted an earlier commencement of ejection of blood. This then required more time to allow the necessary pressure gradient to form, causing aortic valve closure at the termination of systole.

Clinically, the $LVET_c$ may be shortened as a result of a severe AMI or chronic failure in spite of no apparent changes in the haemodynamic determinants, preload and afterload (18, 28, 44, 64, 66, 80, 81, 85). The above authors were found to be in agreement over the clinical value of the $LVET_c$ as it was reported to accurately reflect declining ventricular performance in terms of cardiac output and stroke volume. It was also noted that the $LVET_c$, like other intervals of the STI, did not deviate greatly from normal in persons with functionally mild heart disease. Conversely, as mentioned above, those who showed marked ventricular dysfunction by any cause also demonstrated significant interval deviations from normal. Its clinical importance is reported to be quite conclusive as "... for a given period of ejection, the volume of blood delivered by the ventricle will be determined by the rate of fibre shortening and the initial chamber volume from which the fibre shortening

takes place" (39).

Pre-ejection Period (PEP)

The PEP is the time from the beginning of electrical depolarization, to the time of ejection of blood from the ventricles (24, 57). It commences at the Q-wave of the EKG and ends at the beginning of the upstroke of the CPW. Extensive studies have been carried out on this interval because one of its components, ICP, was found to relate directly to the ability of the ventricles to compress the blood to systemic pressure levels. Barring depolarization deviations, the PEP through the sub-component appears to measure one factor of cardiac performance, contractility (3, 9, 24, 30, 32, 42, 45, 57, 58, 73). As briefly mentioned above, the determinants of the PEP are chiefly the QRS duration or electrical conductance by the ventricular myocardium (32), and dp/dt which indicates the rate of change of pressure per unit time produced by the contracting ventricular myocardium as it compresses an unchanging blood volume (3, 32, 45, 58, 62, 73).

The time range for this period as determined by Blumberger (9) was 50 to 105 msec, although 'normals' seldom deviated beyond 70 to 100 msec. Frank (24), on the other hand, determined that the means for this period

was 105 ± 1.7 msec, indicating some discrepancy between Blumberger's (9) range and his own.

The PEP was noted to shorten with increasing heart rate induced pharmacologically, but not by atrial pacing or vagal blockade (30, 45, 57, 58, 72). A linear relationship to heart rate was established by Weissler (77) in the equation PEP_c = -0.4 x HR + measured value, whereby PEP_c indicates the corrected PEP for a certain heart rate. Other authors found, however, that the PEP did not correlate highly at all with heart rate or haemodynamic fluctuations (22, 24, 64). Increases in preload were found to decrease the PEP (32, 58, 62) while increases in afterload were noted to increase the interval (9, 11). In the former condition the increased diastolic volume and pressure in the normal ventricle would decrease the pressure difference between the ventricle and systemic circulation, thereby taking less time, at the same dp/dt, to reach the latter's pressure level. The latter condition would conversely predispose the ventricle to taking a longer time to build up to a higher pressure before ejection would take place. Inverse pressure differences to those above would cause the PEP interval to change in the opposite direction for each situation.

Chronically impaired ventricular function in the face

of no obvious alterations in preload or afterload revealed a prolonged PEP (8, 18, 80, 81, 85). In the recording periods soon after an AMI, however, apparent increased catecholamines seemed to normalize the PEP, whereas lengthening would otherwise be noted as above because of the failing left ventricle being unable to generate a substantial compression rate. The resultant effect of this offset would be to assess a normally functioning left ventricle in the presence of an AMI (13, 18, 22, 28, 32, 77). Other authors also found that the PEP exhibited little or no relationship between interval responses and types of myocardial impairement (24, 25, 44, 64). In addition, it was proposed that the low correlation to heart rate and high dependance on haemodynamic determinants tended to obscure any significance in its value as a clinical measure of contractility (24, 44, 64). The apparent normality of the PEP in spite of a failing left ventricle was thought to be caused by post-infarction circulating catecholamines resulting in a normal compression factor, or, circulatory deterioration which would decrease afterload and, therefore, the time required for the ventricle to meet systemic pressures (28, 32).

Isovolumic Contraction Period (ICP)

The ICP is that time period of the PEP during which

the ventricles are actively compressing the blood to increase intraventricular pressures from that of diastole to systemic pressures without any blood volume change. Its absolute time is calculated by subtracting the LVET from the mechanical systole (S_1-A_2). Reeves (58) studied the PEP and ICP and demonstrated that dp/dt is the prime determinant of the duration of the latter interval. Harris (30) used adrenergic stimulators and blockers to demonstrate how the ICP and PEP could be altered directly by changes in the inotropic state of the myocardium. Martin (42) related the ICP to internally retrieved measures of the same interval and found that the externally measured PEP reflected more accurately the internal ICP with correlations of $r = 0.90$ and 0.77 respectively than did the externally measured ICP. He and Harris (30) concluded that the ICP changes are identical to the PEP in that only the mechanical aspect dp/dt in both intervals could be altered by pharmacological interventions. Other authors indicated that the ICP was inversely proportional to contractility changes only and did respond in an identical fashion to the PEP under the influences of haemodynamic alterations (9, 11, 24, 45, 58, 73).

The time range for the interval was estimated by Blumberger (9) to be 15 to 45 msec which appeared to be

quite short with respect to the mean established by Frank (24) of 49 ± 1.9 msec. The absolute time of the ICP and its calculation is the subject of much controversy in terms of determining the exact time of mitral valve closure in S_1 and the beginning of pressure rise in the ventricle (10, 14, 33, 42). Frank (24) found that these factors alone accounted for approximately 15 percent of deviations within his statistical analysis.

The clinical value of the ICP is still being questioned and studied as it is subject to difficulties similar to the PEP. Weissler (77) reported that the failing left ventricle, when demonstrating how pressure increase responses to electrical stimulation would reveal a prolonged ICP. Ahmed (3), on the other hand, found no relationship between ICP and ventricular impairment the latter was already angiographically proven. Frank (24) and Spodick (64) also found that the ICP did not relate in any way to cardiac clinical diagnosis, heart rate and haemodynamic variances, thereby inferring that its clinical value was doubtful.

Haemodynamic Ratio (PEP/LVET)

The PEP/LVET was originated by Weissler (80, 81) to reflect more accurately the directional fluctuations of the normal to impaired ventricle. He noted that the

resting normal value was 0.35 ± 0.04 and was minimally influenced by heart rate, hence the raw figures of PEP and LVET could be used for calculation purposes. It was, however, considerably influenced by myocardial deficiencies and its value increased in proportion to ventricular dysfunction. Deviations from normal assume that the LVET decreases and the PEP increases in order that failure trends may be indicated. Therefore, although the ratio is sensitive to failure, the latter must be detectable by the LVET and PEP measures before it will be shown mathematically. Weissler (80, 81) found that the ratio correlated well with stroke volume ($r = -0.72$) and stroke index ($r = -0.82$), the latter which relates stroke volume to body surface area. Garrard (25) did not find as good correlations with these indices of cardiac function but did agree with Weissler that it was excellent for determining changes in ejection fraction.

Aorta-to-Coronary Bypass Operation

Myocardial revascularization is a surgical technique for treating coronary artery disease. The ideal anatomical conditions for revascularization are the presence of critical obstructive lesions (75 percent of original lumen restriction) in the proximal portions of the coronary arteries, particularly in the left coronary system, with minimally involved distal vessels. Two clinical conditions that indicate a serious imbalance between coronary blood supply, as a result of coronary restriction, and myocardial oxygen requirement are chronic and disabling angina pectoris which does not respond to ordinary medical treatment and unstable angina pectoris or preinfarction angina (71).

Coronary blood flow may be surgically restored by bypassing the areas of vascular disease in the proximal coronary arteries. The procedures involve direct anastomoses of reversed segments of saphenous vein between the ascending aorta and the patent segments of one or more of the three major arteries (36). Revascularization improves blood flow to the cardiac muscle thus relieving cardiac pain (angina) and improves cardiac function. Revascularization can thus improve the quality of a person's life and it may also decrease the likelihood of further myocardial embarrassment,

thereby increasing the possibility of long-term survival (71).

This form of medical treatment, now widely used, has not been proven to be 100 percent effective in its objectives (27, 65).

As previously indicated, the effects of limiting ischaemia resulting from severe coronary artery disease on the resting STI components may be a shortening of the $LVET_c$ and lengthening of the PEP with no change in the $Q\text{-}A_{2c}$. The PEP/LVET would show, as a result, an increased value. The ICP component may show alterations similar to the PEP. Successful revascularization would appear to have a normalizing effect on the components of the resting STI. The $LVET_c$ would lengthen, the PEP and ICP would shorten and the PEP/LVET ratio would be reduced towards the 'normal' range.

A study of the effects of myocardial revascularization in patients with left ventricular dysfunction by Johnson (34) noted that the $LVET_c$ was increased and the PEP was reduced as was the PEP/LVET ratio. A similar study by Matlof (43) on the other hand, demonstrated no lasting significant effects on the STI components of one group which had a transiently decreased LVET in the post-operative period and a sustained PEP reduction in the other group, either from enhanced ventricular function or the effects of adrenergic stimulation.

Summary

The studies reviewed presented various views concerning the physiology and clinical value of the STI variable. The $Q\text{-}A_{2c}$ was found to be influenced primarily by its sub-components PEP and $LVET_c$, thus, was generally considered to be of little clinical value other than determining changes in the time of A_2. The $Q\text{-}A_{2c}$'s value was enhanced when it was used in comparison with components PEP and $LVET_c$. The $LVET_c$ was accepted by the authors reviewed as being one interval which could, alone, appear to most accurately reflect changes in ventricular function. The PEP and ICP were noted to be closely related in their ability to reflect contractility measures. Their clinical value in this respect was the subject of much controversy as they did not appear to respond to varying physiological and pathological influences. The haemodynamic ratio, PEP/LVET, was accepted by the authors reviewed as being a variable which could accurately reflect directional fluctuations in ventricular performance.

The aorta-to-coronary bypass operation for the revascularization of the ischaemic myocardium has not been found to be 100 percent effective in its objectives.

Studies concerning the effects of this operation on the resting STI components have provided inconclusive results.

CHAPTER III

METHODOLOGY

This study was designed to assess the effects of the coronary bypass operation on selected components of the resting STI. The following is a presentation of the subjects, protocol, measuring equipment and statistical analysis used in this study.

Subjects

Twenty-four subjects participated in this study. They were divided initially into two basic groups, cardiac patients and normal control subjects, each group consisting of 12 members. The cardiac patient group was further subdivided into two classifications, those who did return for a post-operative re-evaluation and those who did not. The subjects were classified on the following basis:

1. A control group of 12 subjects considered to be hospital normals. These subjects had no demonstrable high risks in terms of heart disease and reported no

symptoms of cardiac abnormalities, when asked.

2. A group of 12 cardiac patients (Table 1), all of whom presented clinical evidence of at least two-vessel coronary artery disease. Every member of this group was scheduled to undergo a coronary bypass operation in a short time following pre-operative evaluation. This group was subdivided into the following categories:

2A. a pre-operative cardiac patient group of six subjects, all of whom did not return for post operative reevaluation, and

2B. a pre-operative cardiac patient group, all of whom did return for post-operative reevaluation.

As indicated by the above group divisions, only six cardiac patient subjects were tested both pre- and post-operatively (group 2B), while six cardiacs were tested only pre-operatively (group 2A). The normal subjects were tested only once to provide STI control values. All of the cardiac patients were under the care of a cardiologist and a cardiovascular surgeon, both of whom were directly involved in the assessment and rehabilitation programme sponsored by the Department of Kinanthropology of the University of Ottawa.

TABLE 1

AGE, HEIGHT, and WEIGHT of CARDIAC PATIENTS

Group	Subject	Age	Height (ins)	Weight
2A Non-returning	JB	43	68.0	173
	SC	57	64.0	138
	LG	41	67.5	170
	MK	66	67.5	175
	JL	39	67.3	167
	BT	45	68.3	167
2B Returning	EB	61	66.5	155
	LC	58	67.0	166
	RG	36	66.0	147
	WL	41	70.3	145
	WN	45	67.5	184
	JR	44	67.0	173

Protocol

Testing was conducted in the Kinanthropology biokinetics laboratory where the cardiacs reported early in the morning of their respective evaluation day. The normal subjects reported to the same location under similar conditions with the exception that they were tested at selected times during the day. All subjects experienced identical laboratory conditions during each evaluation.

The cardiac patients were assessed pre-operatively, then underwent coronary bypass surgery within a few days of their evaluation at the laboratory. The post-operative patients (group 2B) returned for reevaluation approximately 60 days after surgery. During this 60-day recovery period, they were given post-operative therapy by the Department of Physiotherapy of the Ottawa Civic Hospital. The patients were then admitted to an exercise-rehabilitation programme sponsored by the Department of Kinanthropology at the University of Ottawa.

The STI were recorded for each subject immediately preceeding a modified Balke exercise tolerance test (ETT). All subjects were familiarized with the immediate environment and the fundamental aspects of the testing procedure. They were then required to sit in a comfortable chair

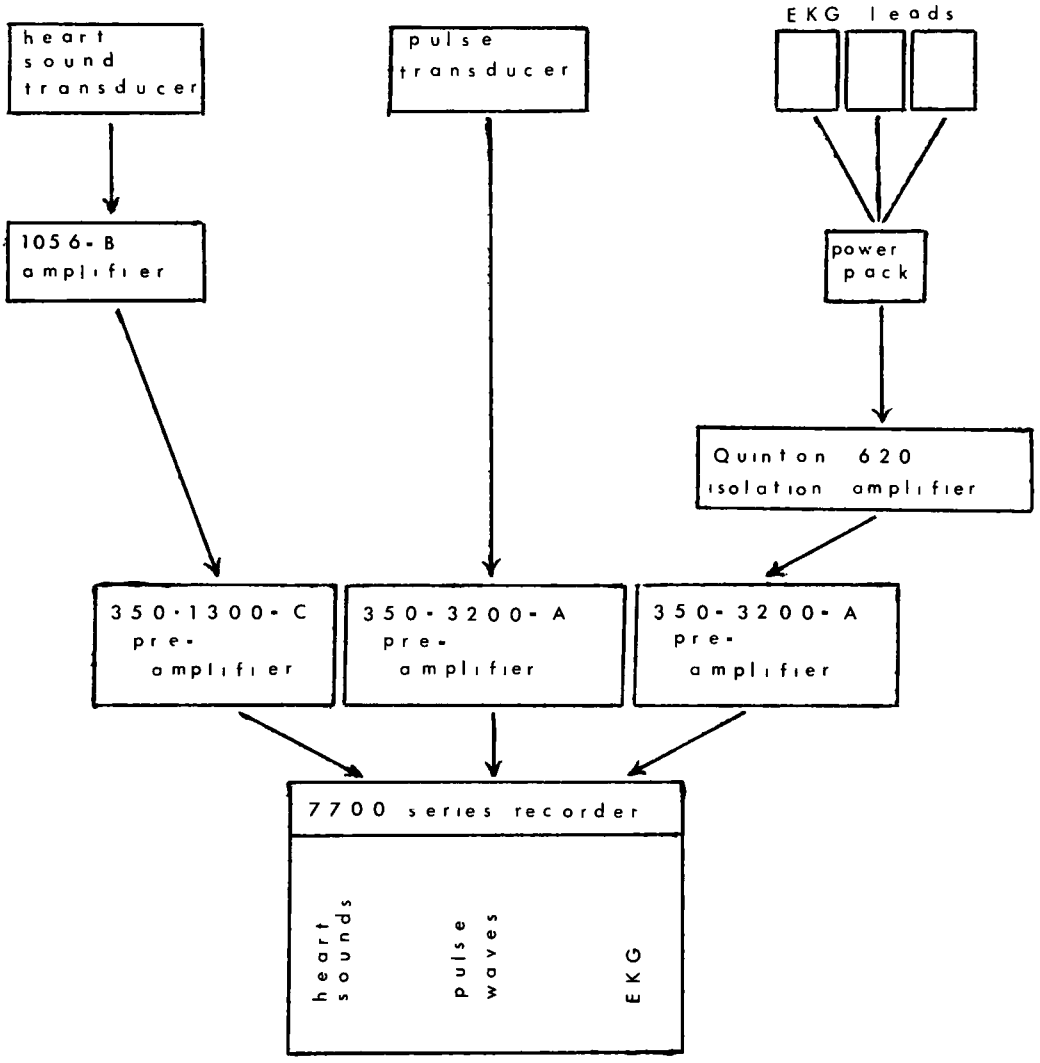
beside a treadmill and the STI recording devices. The STI were then recorded. Immediately following this, the patients were given an ETT on the treadmill. The ETT consisted of walking at 2 mph, beginning at 0% grade and proceeding through increasing treadmill elevations of 3.5%. Each level was maintained for 3 minutes (App. C). The ETT was terminated when the patient either attained his maximum functional working capacity, or complained of angina, dyspnea or fatigue.

The C-M-5 electrode arrangement for recording the EKG was used, whereby the leads were placed on the manubrium sterni, xyphoid process and V-5 position. A cardiophone was then positioned at the apex region of the chest where the left ventricular valve vibrations could be monitored. The carotid pulse wave (CPW) pickup, a pneumatically driven hollow disc, was placed on the carotid artery and was held in position by a suction ring and an elastic collar. The recording paper was then run at 100 mm/sec at which speed the most accurate recordings were made. The recordings were run at this speed until a minimum of fifteen consecutive pulse beats were recorded.

Equipment (Fig. 2)

The EKG was recorded on the V-lead by a Quinton #620 isolation amplifier and the impulses were transmitted to

Fig. 2, Measuring Equipment Schematic



a Sanborn 350-3200-A pre-amplifier to be recorded at a calibration of 1 mv = 1 cm deflection.

The quartz-filament cardiophone was connected to a Hewlett-Packard #1506-B heart-sounds ampliflier. The signals from this ampliflier were transmitted to a Sanborn 350-1300-C DC-coupling pre-amplifier.

The carotid pulse transducer, which was pneumatically activated, relayed the pulsations to an electrical impulse generator which transmitted the signals to a Sanborn 350-3200-A pre-amplifier.

All Sanborn 350 pre-amplifiers were connected to a Sanborn Series 7700 recorder which recorded the heart sounds, CPW and EKG simultaneously.

Statistical Analysis

The components Q-A_2 and LVET were corrected for heart rate according to the regression formulae proposed by Weissler (79). The returning and non-returning cardiac groups (2A and 2B) and the normal control group were statistically compared using a one-way analysis of variance (63) to determine if there were any significant differences among the groups for each of the components of the STI and for the PEP/LVET ratios. The returning cardiac group (2B) was statistically analyzed pre- and post-operatively using the Student's 't'-test

to determine if there were any significant changes in the group STI components and PEP/LVET ratios as a result of the coronary bypass surgery.

CHAPTER IV

RESULTS AND DISCUSSION

This study was designed to assess the effects of the coronary bypass operation on selected resting STI components. The results of this study will be discussed in terms of their basic interpretation, in their relationship to other similar studies and their comparisons with exercise tests conducted at each evaluation time.

Statistical analysis of the STI components and PEP/LVET ratios of the two cardiac groups (2A and 2B) and the normal group indicated that, at the onset of this study, there were no significant differences among the groups (table 2). Statistical analysis of the STI components and PEP/LVET ratios of the returning cardiac group (2A) on a pre- to post-operative basis indicated that there were also no significant changes in any of these variables as a result of corrective surgery (Table 3). This second result suggested that there were no apparent alterations in resting ventricular function resulting from the coronary bypass surgery. The lack of statistically significant differences between the groups at the

TABLE 2

STI COMPONENT AND PEP/LVET RATIO MEANS; STANDARD DEVIATIONS AND F RATIOS OF THE PRE-OPERATIVE CARDIAC AND NORMAL CONTROL GROUPS

Component	Normal Control Group (n=12)	Cardiac Groups: Pre-operative (non-returning) (n=6)	Cardiac Groups: Pre-operative (returning) (n=6)	F Ratio
$Q\text{-}A_{2c}$	.377 ± .02	.384 ± .02	.387 ± .02	0.513
$LVET_c$	.271 ± .02	.286 ± .01	.269 ± .01	2.545
PEP	.107 ± .01	.098 ± .02	.106 ± .03	0.514
ICP	.039 ± .01	.038 ± .01	.040 ± .02	0.043
PEP/LVET	.39 ± .08	.35 ± .13	.42 ± .11	1.448

Critical Value for $F_{95}(2, 21) = 3.47$

TABLE 3

STI COMPONENT AND PEP/LVET RATIO MEANS, STANDARD DEVIATIONS AND 't' VALUES OF THE SURGICAL CARDIAC GROUP (n=6)

Component	Pre-operative Value	Post-operative Value	t Value
$Q\text{-}A_{2c}$	.387 ± .02	.389 ± .02	0.004
$LVET_c$	.269 ± .01	.280 ± .06	0.026
PEP	.106 ± .03	.101 ± .03	0.028
ICP	.040 ± .03	.040 ± .01	0.002
PEP/LVET	.42 ± .11	.41 ± .01	0.000

Critical Value for t_{95} = 2.228

initial and final testing sessions may have been caused by the small number of subjects, the high variability of individual values, or both. This could have created a situation whereby any significant mean STI component alterations were obscured by large standard deviations. On the other hand, the STI components may have demonstrated that at rest there is really no detectable ventricular dysfunction as a result of no limiting ischaemia being present (56). Some authors have suggested, however, that the value of the STI in the clinical setting is doubtful in attempting to assess the effects of coronary artery disease on ventricular performance (28, 32, 44, 51).

A normalizing trend in the PEP/LVET ratio due to the coronary bypass operation was not demonstrated in the comparison between the pre- and post-operative cardiac groups (Table 3). In effect, the maintenance of such a high value appeared to indicate that the abnormally short LVET and long PEP relationship was sustained in this group of patients in spite of corrective surgery.

In comparison with the studies by Johnson (34) and Matlof (43) reviewed above, the results of this study were found to agree only with one group in Matlof's study. The 'normalizing' trend demonstrated by Johnson was not apparent in this study, nor were there any changes in any of the components such as the PEP shortening in Matlof's

second group. The results of this study were on the other hand, in agreement with the second month post-operative period evaluation of Matlof's first group whereby the STI components showed no overall changes in relation to the pre-operative values.

As previously mentioned in the experimental protocol, the STI were recorded immediately preceding an exercise tolerance test (ETT). The results of this test (App. B) indicated a general increase in the ability of the post-operative cardiacs to tolerate work stress. This 'general' enhancement in their functional capacities was not associated with comparable 'normalizing' trends in the STI components or PEP/LVET ratios. Increases in haemodynamic function are known to be directly related to increases in stroke volume, ejection fraction, contractility and heart rate. In light of this, definite increases in work load tolerances were demonstrated by four of the six returning cardiacs. Increases in heart rate were noticed in three of those that endured the higher stresses while the fourth increased his work tolerance response without a great heart rate increase. The increases in functional capacities of these subjects, of which stroke volume is a factor, was not reflected in the STI components. As a result of the comparison of these two forms of haemodynamic assessment, the general functional im-

-provement, supposedly as a result of the coronary bypass operation, as was shown by the post-operative cardiacs in the ETT, was not reflected in the resting STI components nor in the PEP/LVET ratios. Since the resting STI components and PEP/LVET ratios did not reflect the changes in functional improvement, the lack of agreement suggests two possibilities. Either the resting STI is a good measure of myocardial function and improvements in the cardiacs' functional capacity are not related to improvements in myocardial function, or, the improvement in the cardiacs' functional capacity is related to improvements in myocardial function and the resting STI is not a good objective measure of myocardial function.

Based on present knowledge of the cardiovascular response to exercise, the general improvements of four of the six returning cardiacs demonstrated in the ETT appears to support the latter possibility.

CHAPTER V

SUMMARY , CONCLUSIONS , RECOMMENTATIONS

Summary

This study was designed to assess the effects of the bypass operation on the resting STI. The subjects used in this study were divided into three groups:

1. A control group which consisted of 12 normal subjects. None were considered high risk subjects in terms of demonstrating symptoms of heart disease.

2A. A pre-operative cardiac subject group which consisted of six cardiac patients, all presenting clinical evidence of at least two-vessel CAD. All members of this group were scheduled to undergo a coronary bypass operation in a short time after pre-operative evaluation. The members of this group did not return for a post-operative evaluation and were termed the non-returning cardiacs group.

2B. A group of six cardiac patients, all presenting clinical evidence of at least two-vessel CAD. All members of this group were scheduled to undergo a coronary bypass operation in a short time after pre-oper-

-ative evaluation. The members of this group did return for a post-operative reevaluation and were termed the returning cardiacs group.

Testing was conducted in the Kinanthropology bio-kinetics laboratory. The subjects reported early on their respective evaluation day. The normal subjects reported under similar conditions but at random times during the day. This group and the non-returning cardiacs group (2A) reported for testing only once while six of the cardiacs group (2B) reported twice, once pre-operatively and once post-operatively under identical laboratory conditions. The STI were recorded by the simultaneous recording of the EKG, PCG, and CPW.

Groups 1, 2A and 2B were statistically analyzed to determine if any differences existed among the groups in the STI components and PEP/LVET ratios at the initial testing session. Group 2B was statistically analyzed on a pre- to post-operative basis to determine if any changes in the STI components and PEP/LVET ratios occurred as a result of the coronary bypass surgery.

Statistical analysis of the STI components and the PEP/LVET ratios of the two cardiac groups (2A and 2B) and the normal group indicated that, at the onset of this study there were no significant differences among the groups. Statistical analysis of the same variables of the returning cardiacs group (2A) on a pre- to post-operative basis

indicated that there were also no significant changes in any of these variables as a result of corrective surgery.

It was found that the STI components did not agree well with the findings of Johnson (34) and the second of the two groups in Matlof's study (43). The transient effects of the operation in the first group of Matlof's study were in agreement with the findings of this study. The absense of change in the STI components of the post-operative returning cardiacs and the maintenance of an abnormally high PEP/LVET ratio appeared to contradict the increases in functional capacities demonstrated by these subjects at the second exercise testing session.

Conclusions

The results of this study indicated that there were no statistically significant effects of the aorta-to-coronary bypass operation on the selected components of the resting systolic time intervals.

Recommendations

It is felt that the protocol used in the cardiac assessment part of the rehabilitation programme could be altered to include serial measurements of the STI. By this method, a more accurate assessment of overall haemodynamic performance may be noticed if one, two, or a series of measures were made over a period of a few months.

Serial measurements may be able to detect extremes and midpoints of the spectrum of myocardial performance described by cardiacs as they go into and come out of acute episodes of circulatory distress.

REFERENCES

1 Adam, M., Mitchell, B.F., Long term Results with Aorta-to-Coronary Bypass Vein Grafts, Annals of Thoracic Surgery. 14:1, 1972.

2 Agress, C., Wegner, S., Forrester, J.S., An Indirect Method for Evaluation of Left Ventricular Function in Acute Myocardial Infarction. Circ. 46: 291, 1972.

3 Ahmed, S.S., Levinson, G.E., Schartz, C.J., Ettinger, P.O., Systolic Time Intervals as Measures of the Contractile State of the Left Ventricular Myocardium in Man, Circ. 46: 555, 1972.

4 Amsterdam, E.A., et al., Saphenous Vein Bypass Graft for Refractory Angina Pectoris: Physiologic Evidence for Enhanced Blood Flow to the Ischemic Myocardium, Amer. J. Card. 26:623, 1970.

5 Aronow, W.S., Isovolumic Contraction and Left Ventricular Ejection Times, Amer. J. Card. 26: 238, 1970.

6 Aronow, W.S., Cassidy, J., Uyeyama, R., Effect of Position on the Resting and Post Exercise Phonocardiogram, Chest. 61: 439, 1972.

7 Benestad, A.M., The Deteriorative Effect of Myocardial Infarction upon Physiological Indices of Work Capacity, Acta Med. Scand. 191: 67, 1972.

8 Bennett, E.D., Smitten, C.S., Sowtan, E., An Assessment of the Haemodynamic Alterations in Acute Myocardial Infarction Using the Systolic Time Intervals, Thorax. 26: 632, 1971.

9 Blumberger, K.J., Meiners, S., Studies of Cardiac Dynamics. Cardiology and Encyclopedia of the Cardiovascular System. Part 4, Vol. II, (New York: McGraw-Hill and Co., 1959) p. 372.

10 Boerth, R.C., Covell, J.W., Mechanical Performance and Efficiency of the Left Ventricle During Ventricular Stimulation, Amer. J. Physiol. 221: 6, 1971.

11 Braunwald, E., Moscovitz, H.L., et al., Timing of Electrical and Mechanical Events of the Left Side of the Human Heart, J. Appl. Physiol. 8: 309, 1955.

12 Bristow, J.D., Van Zee, B.E., Judkins, M.P., Systolic and Diastolic Abnormalities of the Left Ventricle in Coronary Artery Disease, Circ. 42: 219, 1970.

13 Buyukozturk, L., Kimbris, D., Segal, B., Systolic Time Intervals, Amer. J. Card. 28: 183, 1971.

14 Bush, C.A., Lewis, R.P., Leighton, R.F., Fontana, M.E., Weissler, A.M., Verification of Systolic Time Intervals and the True Isovolumic Contraction Time From the Apex Cardiogram by Micromanometer Catheterization of the Left Ventricle and Aorta, Circ. 42, Sup. III: 121, 1970.

15 Cureton, T.K., Cundiff, E.E., Sloniger, E.L., du Toit, S.F., "Further Studies of the Pre-Ejection Intervals as Related to Physical Stress and Endurance Performances". Experiment Report, University of Illinois.

16 Dadario, R.C., Freis, E.D., Kinetocardiogram, Phonocardiogram and Arterial Pulse Waves During acute Haemodynamic Changes, Circ. 34: 423, 1966.

17 De Geest, H., Levy, M.N., Bieske, H., Lipman, R.I., Depression of Ventricular contractility by Stimulation of the Vagus Nerves, Circ. Res. 17: 222, 1965.

18 Diamant, B., Killip, T., Indirect Assessment of Left Ventricular Performance in Acute Myocardial Infarction, Circ. 42: 579, 1970.

19 Diamond, G., Forrester, J.S., et al., Mean Electromechanical $\Delta P/\Delta t$, Amer. J. Card. 30: 335, 1972.

20 Dock, W., Further Evidence for the Purely Valvular Origin of the First and Third Heart Sounds, Amer. Heart, J. 30: 332, 1945.

21 Fabian, J., Epstein, E.J., Coulshed, N., Duration of Phases of Left Ventricular Systole Using Indirect Methods, I: Normal Subjects, Br. Heart J. 34: 874, 1972 (A).

22 Ibid., II Acute Myocardial Infarction, p. 882, 1972 (B).

23 Flessas, A.P., Kumar, S., Spodick, D.H., Effects of the Valsalva Maneuver on the Cardiac Systolic Intervals: Beat-to-Beat vs. Timed Analysis. Amer. Heart J. 80: 522, 1970.

24 Frank, M.N., Kinlaw, W.B., Indirect Measurement of Isovolumetric Contraction Time and Tension Period in Normal Subjects, Amer. J. Card. 10: 800, 1962.

25 Garrard, E.L., Weissler, A.M., Dodge, H.T., The relationship of Alterations in Systolic Time Intervals to Ejection Fraction in Patients with Cardiac Disease, Circ. 42: 455, 1970.

26 Gibson, D.G., Chamberlain, D.A., Coltart, D.J., Mercer, J., Effects of Changes in Ventricular Activation in Cardiac Haemodynamics in Man, Br. Heart J. 33: 397, 1971.

27 Glenn, W.W.L., Some Relections in the Coronary Bypass Operation, Circ. 45 (1): 375, 1972.

28 Hamosh, P., Cohn, I.M., et al., Systolic Time Intervals and Left Ventricular Function in Acute Myocardial Infarction, Circ. 45: 375, 1972.

29 Harley, A., Starmer, C.F., Greenfield, J.C. Jr., Pressure flow Studies in Man. An Evaluation of the Duration of the Phases of Systole, J. Clin, Invest. 48: 895, 1969.

30 Harris, W.S., Schoenfeld, C.D., Weissler, A.M., Effects of Adrenergic Receptor Activation and Blockade on the Systolic Pre-ejection Period, Heart Rate, and Arterial Pressure in Man, J. Clin, Invest. 11: 1704, 1967.

31 Harrison, T.R., Dixon, K. et al., The Relation of Age to the Duration of Contraction, Ejection, and Relaxation of the Normal Human Heart, Amer. Heart J. 67: 189, 1964.

32 Hodges, M., Halpern, B.L., Friesenger, G.C., Dagenais, G.R., Left Ventricular Pre-ejection Period and Ejection time in Patients with Acute Myocardial Infarction, Circ. 45: 933, 1972.

33 Inoue, K., Young, G.M. et al., Isometric Contraction Period of the Left Ventricle in Acute Myocardial Infarction, Circ. 42: 79, 1970.

34 Johnson, A.D., O'Rourke, R.A., Karliner, I.S., Burian, C., Effect of Myocardial Revascularization on Systolic Time Intervals in Patients with Left Ventricular Dysfunction, Circ. 45-46 (sup. 1): 91, 1972.

35 Katz, L.M., Feil, H.S., Clinical Observations on the Dynamics of Ventricular Systole: I Auricular Fibrillation, Arch. Intern. Med. 32: 672, 1923.

36 Kouchoukos, N.T., Kirkun, J.W., Coronary Operations for Ischemic Heart Disease, Modern Concepts of Cardiovascular Disease, 61: 47, 1972.

37 Kumar, S., Spodick, D.J., Study of the Mechanical Events of the Left Ventricle by Atraumatic Techniques: Comparison of Methods of Measurement and their Significance, Amer. Heart J. 80: 401, 1970.

38 Leighton, R.F., Weissler, A.M., Weinstein, P.B., Wooley, C.F., Right and Left Ventricular Systolic Time Intervals, Amer. J. Card. 27: 66, 1971.

39 Levine, H.J., Neill, W.A., Wagman, R.J., Krasnow, N., Gorlin, R., The effects of Exercise on Mean Left Ventricular Ejection Rate in Man. J. Clin. Invest. 41: 1050, 1962.

40 Luisada, A.A., The Second Heart Sound in Normal and Abnormal Conditions, Amer. J. Card. 28: 150, 1971.

41 Manley, J.C., Johnson, W.D., Flamma, R.J., Lepley, D. Jr., Objective Evaluation of the Effects of Direct Myocardial Revascularization in Ventricular Function Utilizing Ergometer Exercise Testing, Amer. J. Card. 26: 648, 1970.

42 Martin, C.E., Shaver, T.A., Thompson, M.E., Reddy, J.R., Leonard, J.J., Direct Correlation of External Systolic Time Intervals with Internal Indices of Left Ventricular Function in Man, Circ. 44: 419, 1971.

43 Matlof, H. et al., Cardiac Function Assessed by Systolic Time Intervals After Aortocoronary Saphenous Vein Bypass Surgery, Br. H. J. 35: 714, 1973.

44 McConahay, D.R., Martin, C.M., Cheitlin, M.D., Resting and Exercise Systolic Time Intervals, Circ. 45: 592, 1972.

45 Metzger, C.C., Chough, C.B., Kroetz, F.W., Leonard, J.J., True Isovolumic Contraction Time. Amer. J. Card. 25: 434, 1970.

46 Milesis, C.A., Cureton, T.K., "Indirect Measurement of Selected Intervals of the Cardiac Dynamic Cycle in Adolescents and Middle Aged Men", Study Presented to the Midwest Meeting of AAHPER, Milwaukee, Wis., March 1968.

47 Molnar, S., Franks, D.B., Jette, M., Cureton, T.K., Cardiac Time Components of Boys 7-14 Years of Age., Med. & Sci. In Sports. 3, (1): 12, 1971.

48 Montoye, H.J., Willis, P.W., Howard, G.E., Keller, J.B., Cardiac Pre-ejection Period: Age and Sex Comparisons. J. Gerontology. 26, (2): 208, 1971.

49 Nandi, P.S., Pigott, V.M., Spodick, D.H., Sequential Cardiac Responses During the Respiratory Cycle: Patterns of Change in Systolic Intervals, Chest. 63 (3): 380. 1973.

50 Nazarov, I.L., Musiichuck, Yu. I., Influence of Body Position on Phases of Cardiac Cycle in Healthy Subjects. Tera. Arkhiv. 37, (10): 106, 1965.

51 Parker, M.E., Just, H.G., Systolic Time Intervals in Coronary Artery Disease as Indices of Left Ventricular Function: Fact or Falicy? Br. H. J. 36: 368, 1974.

52 Perloff, J.K., Value and Limitations of Systolic Time Intervals in Patients with Acute Myocardial Infarction, Circ. 45: 929, 1972.

53 Perry, J.M., Garrard, C.L. Jr., (abstract) Systolic Time Intervals - Relation to Severity of Coronary Artery Disease and Left Ventricular Dysfunction Circ. 41-42 (supp. III): 121, 1970.

54 Pigott, V.M., Spodick, D.H., Effects of Normal Breathing and Expiratory Apnea on Duration of the Phases of Cardiac Systole, Amer. Heart J. 82: 786, 1971.

55 Pigott, V.M., Spodick, D.H., Effects of Normal Breathing and Experatory Apnea on Duration of the Phases of Cardiac Systole, Amer. Heart J. 82: 786, 1971.

56 Pouget, J.M., Harris, W.S., Mayron, B.R., Naughton, J.P., Abnormal Responses of the Systolic Time Intervals to Exercise in Patients with Angina Pectoris, Circ. 43: 289, 1971.

57 Raab, W., Training, Physical Inactivity, and the Cardiac Dynamic Cycle, J. Sport, Med., and Physical Fitness. 6: 38, 1966.

58 Reeves, J.T., Hefner, L.L., Jones, W.B., Coghlan, C., Prieto, G., Carroll, J., The Haemodynamic Determinants of the Rate of Change of Pressure in the Left Ventricle During Isometric Contraction, Amer. Heart J. 60: 745, 1960.

59 Robinson, B., The Carotid Pulse. II Relation of External Recordings to Carotid, Aortic, and Brachial Pulses, British Heart J. 25: 61, 1962.

60 Ross, D.M., Gonzalez-Lavin, L., Frazier, T., Saphenous Vein Bypass for Direct Coronary Revascularization, Thorax. 27: 1, 1972.

61 Selzer, A. Cohn, K., Functional Classification of Cardiac Disease: A Critique, Amer. L. Card. 30: 306, 1972.

62 Siegel, J.H., Sonneblick, E.H., Isometric Time-Tension Relationships as an Index of Myocardial Contractility, Circ. Res. 12: 597, 1963.

63 Sokal, R.R., Rohlf, F.J., Biometry W.H. Freeman and Co., 1969.

64 Spodick, D.H., Door, C.A., Calabrese, B.F., Detection of Cardiac Abnormality by Clinical Measurement of Left Ventricular Ejection Time, J.A.M.A. 209: 239, 1969 (A).

65 Spodick, D.H., Coronary Revascularization, (editorial) Circ. 44: 302, 1971 (A).

66 Spodick, D.H., Kumar, S., Left Ventricular Ejection Period, Amer. Heart J. 76: 70, 1968.

67 Spodick, D.H., Humar, S., Flessas, A.P., Spiratanaban, A.D., Sosler, G., Klints, R.B., Muench, H., Relationship of Paper Speed to Precision of Pulse Wave Measurements: A Multiple Observer Study of Left Ventricular Ejection Time, Aerospace Medicine. 40: 707, 1969 (B).

68 Spodick, D.H., Meyer, M., St. Pierre, J.R., Effect of Upright Tilt on the Phases of the Cardiac Cycle in Normal Subjects, Card. Res. 5: 210, 1971 (B).

69 Spodick, D.H., Muench, H., Ejection Time and Electrocardiogram in Screening for Heart Disease, Geriatrics. 26: 93, 1971.

70 Stafford, R.W., Harris, W.S., Weissler, A.M., Left Ventricular Systolic Time Intervals as Indices of Postural Circulatory Stress in Man, Circ. 41: 485, 1970.

71 Swan, Dr. H.J.C., U.C.L.A. Conference: Myocardial Revascularization for Acute and Cardiac Coronary Heart Disease. Ann. Int. Med. 79: 851, 1973.

72 Taggart, P., Parkinson, P., Carruthers, M., Cardiac Responses to Thermal, Physical and Emotional Stress, British Med. J. 3: 71, 1972.

73 Talley, R.C., Meyer, J.F., McMay, J.L., Evaluation of the Pre-ejection Period as an Estimate of Myocardial Contractility in Dogs, Amer. J. Card. 27: 384, 1971.

74 Tharp, G.D., The Validity of Using Cardiac Function Tests for Measuring Fitness. (Thesis Abstract, University of Nebraska).

75 Vaisrub, S., editor, Nontraumatic Approaches to Assessing Cardiac Performance J.A.M.A. 216: 2007, 1971.

76 Van Bogaert, A., New Concept on the Mechanism of the First Heart Sound, Amer. J. Card. 18: 253, 1966.

77 Wallace, A.G., Mitchell, J.H., Skinner, N.S., Duration of the Phases of Left Ventricular Systole, Circ. Res. 12: 611, 1963.

78 Wayne, H.H. Noninvasive Technics in Cardiology, Year Book Medical Publishers Inc., 1973

79 Weissler, A.M., Garrard, C.L., Systolic Time Intervals in Cardiac Disease, Modern Concepts of Cardiovascular Disease, 1, 2, 1971.

80 Weissler, A.M., Harris, W.S., Schoenfeld, C.D., Systolic Time Intervals in Heart Failure in Man, Circ. 38: 149, 1968.

81 Weissler, A.M., Harris, W.S., Schoenfeld, C.D., Bedside Techniques for Evaluation of Ventricular Function in Man, Amer. J. Card. 23: 377, 1969.

82 Weissler, A.M., Gamel, W.G., Grode, H.E., Effect of digitalis on Ventricular Ejection in Normal Human Subjects, Circ. 29: 721, 1964.

83 Weissler, A.M., Harris, L.C., White, G.D., Left Ventricular Ejection Time Index in Man, J. Appl. Physiol. 18: 919, 1963.

84 Weissler, A.M., Kamen, A.R., Bornstein, R.S., Schoenfeld, C.D., Cohen, S., The effect of Deslanoside on the Duration of the Phases of Ventricular Systole in Man, Amer. J. Card. 15: 152, 1965.

85 Weissler, A.M. Peeler, R.G., Roehill, W.H., Relationships between Left Ventricular Ejection Time, Stroke Volume, and Heart Rate, in Normal Individuals and Patients with Cardiovascular Disease, Amer. Heart J. 62: 367, 1961.

86 Whitsett, T.L., Naughton, J., The effects of Exercise on Systolic Time Intervals in Sedentary and Active Individuals and Rehabilitated Patients with Heart Disease, Amer. J. Card. 27: 352, 1971.

87 Whittaker, A.V., Shaver, J.A., Gray, S., Leonard, J.J., Sound-Pressure Correlates of the Aortic Ejection Sound, Circ. 39: 275, 1969.

88 Wiggers, C.J., "Studies on the Consecutive Phases of the Cardiac Cycle. II. The Laws Governing the Relative durations of Ventricular Systole and Diastole," Amer. J. Physiol. 56: 439, 1921.

89 Wiley, J.F., Effects of Body Position and Exercise on Left Ventricular Intervals, Med. in Sci. and Sport. 2, (3): 132, 1970.

90 Willems, J.L., Roelandt, J., de Geest, H., Kesteloot, H., Joossens, J.V., The Left Ventricular Ejection Time in Elderly Subjects, Circ. 42: 37, 1970.

91 Zaret, B.L., Strauss, H.W., Hurley, P.J., Pitt, B., Left Ventricular Ejection Fraction and Regional Myocardial Performance in Man Without Cardiac Catheterization, Circ. 41-42, supp III: 120, 1970.

APPENDIX A

RAW DATA

Table A-1

Systolic Time Intervals For Pre-Operative Cardiacs

(Non Returning Group)

Units = Seconds

Subject	$Q\text{-}A_{2c}$	$LVET_c$	PEP	ICP
JP	.365	.284	.077	.031
SC	.375	.276	.099	.037
LG	.398	.286	.117	.053
MK	.386	.277	.101	.037
BT	.367	.303	.123	.057
JL	.410	.289	.068	.015

Table A-2

Systolic Time Intervals For Pre-Operative Cardiacs

(Returning Group)

Units = Seconds

Subject	$Q\text{-}A_{2c}$	$LVET_c$	PEP	ICP
EB	.389	.282	.062	.020
LC	.419	.282	.132	.051
RG	.402	.274	.120	.056
WL	.388	.255	.126	.055
WN	.347	.256	.095	.030
JR	.374	.264	.102	.029

Table A-3

Systolic Time Intervals For Returning Post-Operative Cardiacs

Units = Seconds

Subject	$Q\text{-}A_{2c}$	$LVET_c$	PEP	ICP
EB	.402	.329	.063	.029
LC	.413	.277	.119	.058
RG	.385	.251	.123	.051
WL	.385	.264	.117	.036
WN	.354	.280	.076	.026
JR	.395	.284	.109	.042

Table A-4

Systolic Time Intervals For Normal Subjects

Units = Seconds

Subject	$Q\text{-}A_{2c}$	$LVET_c$	PEP	ICP
RC	.372	.256	.112	.039
WC	.415	.309	.107	.041
HD	.363	.269	.089	.026
KD	.403	.289	.118	.046
MJ	.357	.272	.084	.036
FK	.357	.248	.107	.047
JK	.369	.271	.113	.039
EL	.372	.260	.117	.050
EM	.360	.262	.127	.041
DM	.389	.261	.095	.039
PT	.401	.292	.106	.037
JW	.362	.263	.104	.036

APPENDIX B

EXERCISE TOLERANCE TEST RESULTS

Pre and Post-Operative Exercise Tolerance Test Performances of the Returning Cardiac Subjects

Initials	Pre-Operative Mph/% Grade		Hr	Post-Operative Mph/% Grade		Hr
EB	2/	10.5	142	2/	7.0	136
LC	2/	3.5	126	2/	7.0	143
RG	3/	12.5	141	2/	3.5	108
WL	2/	17.5	130	3/	17.5	128
WN	2/	7.0	106	2/	17.5	167
JR	2/	7.0	141	3/	12.5	148

APPENDIX C

EXERCISE TOLERANCE TEST PROTOCOL

UNIVERSITY OF OTTAWA - CARDIAC UNIT

EXERCISE TESTING SESSION

TREADMILL TEST PROTOCOL*

Ensure that the patient has complied with all the preliminary instructions issued to him, ie. sleep, food, drugs, etc.

Patient will read and sign the consent form if he has not already done so. The test should have been previously authorized by the patient's physician and the form duly signed.

Ensure that all recordings forms are ready. Enter all required data, including age and target heart rate.

Pre-exercise 12 lead EKG. Patient should be made to relax for at least 5 min., prior to the recording.

Draw 10 ml of venous blood (if so indicated by the test supervisor).

Attach the electrodes for the exercise test - CM5 position: top of sternum, bottom of sternum (xiphoid process), V_5 position (on rib prominence),

Direct patient to treadmill.

Sit patient on semi-reclining chair on treadmill. Strap phonocardiogram to the patient so that good quality heart sounds are obtained. Place the transducer on the carotid artery. Ensure that the written record indicates a good upstroke and diacrotic notch. Take a simultaneous recording for 10 seconds.

Connect patient to EKG control panel .

Attach blood pressure cuff to the left arm and take a sitting and resting blood pressure measurement. Record standing EEKG.

Initiate subject to treadmill walking. Set treadmill at 1.5 mph, 0% grade. Once subject is comfortable, have him walk for a three minute period.

Rest for 3 minutes. Set clock.

Test will begin on the command of the test supervisor.

Serial	MPH	% GRADE	MIN
1	2	0	0
2	2	3.5	2
3	2	7.0	4
4	2	10.5	
5	2	14.0	6
6	2	17.5	8
If required			
7	3	12.5	0
8	3	15.0	3
9	3	17.5	6
10	3	20.0	9
11	3	22.5	12

During exercise, take EECG each minute of test for 5 secs: ie. between the 55th and 60th secs. If arrhythmias occur, run ECG continuously and mark on paper the speed, grade and time on the EKG write-out.

At every minute, record heart rate (b/ min) from digital read out. Record blood pressures at every 2nd minute.
At end of test, slow-down the treadmill to 1.5 mph and decrease elevation to 0% grade. Have patient walk for 2 minutes. Take recordings as for actual test
When exercise test ends, have patient sit on semi-reclining chair on treadmill. Record pre-ejection intervals. These should be taken at exactly 15 seconds after the patient stops exercising.
Draw 10 ml of venous (if so indicated in para. 6 above)
During recovery, record EKG and heart rate every minute for six minutes. Take blood pressure readings at the 2nd, 4th and 6th minute of the recovery.
At 6th minute of recovery, record pre-ejection intervals.
Disconnect patient, remove electrodes and blood pressure cuff.

* Modified from Naughton, J.,Rehabilitation for Patients with Coronary Heart Disease. Paper presented at the Post Graduate Course on Physiology and Psychology of Exercise Testing and Training of Coronary Disease Patients and Coronary Prone Subjects, Airlie, Virginia, April 1972.

ABSTRACT

The purpose of this study was to determine the effects of the aorta-to-coronary bypass operation on selected components of the resting systolic time intervals (STI). A group of 6 cardiac patients were evaluated pre-operatively and 60 days post-operatively using the STI and an exercise tolerance test (ETT) as objective measures of myocardial performance. The STI were recorded by the simultaneous recording of the electrocardiogram (EKG), the phonocardiogram (PCG), and the carotid pulse wave (CPW). The selected intervals were the $Q\text{-}A_{2c}$, $LVET_c$, PEP, ICP, and PEP/LVET ratio. The STI values and the PEP/LVET ratios of the two cardiac pre-operative groups and of the normal control group were analysed by a one-way analysis of variance to determine if any significant differences existed among them at the onset of this study. The pre- and post-operative STI values and the PEP/LVET ratios of the cardiacs receiving corrective surgery were compared to determine if any significant changes occurred as a result of the treatment.

The study indicated that, at the initial testing session, there were no statistically significant dif-

ferences in the STI values and the PEP/LVET ratios among the cardiac and normal control groups. There were also no statistically significant changes in the STI values and the PEP/LVET ratios of the cardiacs who received corrective surgery.

It was concluded that the patients who underwent the coronary bypass operation in this study demonstrated no statistically significant changes in the selected components of the resting STI values and the PEP/LVET ratios.