A CLINICALLY APPLIED ANATOMICAL STUDY OF THE CORONARY ARTERIES IN THE SOUTH AFRICAN POPULATION

By

NIRUSHA LACHMAN

Submitted in fulfilment of the Degree of Doctor of Philosophy

In the

Department of Anatomy
School of Basic and Applied Medical Sciences
Faculty of Health Sciences
University of Durban Westville
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DECLARATION

This study represents original work by the author and has not been submitted in any form to another University. Where use was made of the work of others, it has been duly acknowledged in the text.

The research described in this dissertation was supervised by

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To my Greatest Love

"For having known Whom there remains nothing more to be known"

CONTENTS

5. CHAPTER V – DISCUSSION

ABSTRACT

Interest in the anatomy of the coronary arteries dates as far back as the early 1500's, at a time when anatomical inquiry was being cautiously aroused. Whilst the later 1700's encouraged academic domination of anatomical study, significant documentation of the coronary arteries was only been established by the late 1800's to early 1900's. There is no doubt that this topic continues to remain dynamic, favoured for its value in applied clinical research. Indeed, technological advancement in the 21st century has transformed modern day anatomy into more than just a simple descriptive exercise. Whether to update standard literature, create ethnically specific banks of anatomical data, abate technical difficulties associated with coronary artery surgery or provide exciting interventional possibilities for clinicians, revisiting the anatomy of the coronary arteries is clearly warranted.

The objective of this investigation was to review the anatomy of the coronary arteries using a clinical approach in order to investigate the morphologic presentation of these vessels within the South African population. On a more clinically universal level, this study aimed to elucidate two focal areas of anatomical interest: extra-cardiac collaterals and myocardial bridges.

The investigation was conducted by means of micro-dissection, angiography, histology and scientific evaluation. A total of 323 sets of coronary arterial patterns consisting of patient angiograms (n=212) and cadaveric dissections (n=95) were studied. Specimens were harvested at post-mortem and angiograms and surgical reports were obtained from clinical centers within KwaZulu-Natal.

Results of this study confirmed the standard anatomical description of the coronary arteries as documented.

Within the South African population, the ramus marginalis artery was found to be present in 13.3% (Females: 10.7%; Males: 5.6% and Blacks: 18.0%; Indians: 6.6%; Whites: 1.4%). The LAD and LCX arteries arose from independent aortic ostia in 14.5%, (Females: 7.5%; Males: 15% and Blacks: 6.5%; Indians 50%; Whites: 35%). Right dominance was observed most frequently in 85.9% (Blacks: 82.3%; Whites: 83.6% and Indians: 86.4% and Males: 82.6%; Females: 89.2%). A bifid LAD artery was noted in 52%, (Females: 6.2%; Males: 8.7% and Blacks: 17.6%; Indians: 6.3 %; Whites: 4.5 %). In 27.7%, (Females: 24.0%; Males: 28.8% and Blacks: 29.5%; Indians: 50%; Whites: 20%) the LCX artery failed to continue along the atrio-ventricular groove. The conus artery arose from a high position off the RCA in 19.2%, (Females: 16%; Males: 21% and Blacks: 19.7%; Indians: 100%; Whites: 10%); and from an independent ostium in 3.61%, (Females: 4.0%; Males: 3.8% and Blacks: 4.9% only). The LCA measured 0.82cm (0.27-2.4cm), (Females: 0.84cm, Males: 0.96cm and Blacks: 0.88cm; Indians: 0.53 cm; Whites: 0.78cm).

Myocardial bridges were recorded on the RCA in 2.5% and on the LAD in 50.6%. The bridge pattern depicted myocardial loops to complete arterial investment and ranged

in length from 3.0 to 20.02 mm. Scientific evaluation of the intramural LAD indicated positive correlation between a straight appearance of the LAD on angiogram and a deep myocardial position upon surgical observation (mean "tortuosity index" =

1.147 [1.373-1.045] where 1= baseline for straightness). Results were confirmed in the correlated cadaveric investigation.

Extra-coronary collaterals were observed in 100% (n=9). The arterial pattern consisted of 1 to 2 main stems with secondary anastomotic branches. The average external diameter was measured to be 0.6mm (0.4-0.7mm), length 52.5mm (18-83mm) with at least 5 secondary branches (3-9) of external diameter 0.3mm (0.2-0.5mm).

Results of the histopathological investigation (n=20) indicated the presence of atherosclerotic disease within the intramural LAD artery segment (15%). A 60% incidence was recorded in the pre-mural segment and 25% incidence in the post-mural arterial segments. When analysed in terms of severity, the intramural segment reflected only mild signs of intimal alteration.

Although not statistically significant, mean values for coronary artery size differed between sexes. The findings were similar when evaluated in terms of the coronary artery anomalies studied. There were significant differences between ethnic groups in terms of the length of the LCA. Mean values showed that Indians had the shortest LCA's when compared with Blacks and Whites. The highest incidence of the ramus marginalis branch was recorded amongst Blacks. Separate origin of the LCX and LAD was highest amongst Indians and high in comparison to reports documented in other countries. A high origin of the conus artery was found to be dominant amongst Blacks. A low incidence of separate origin of the conus from the aorta was recorded in the South African population. These findings are significantly lower than that

reported in the literature. A right dominant system has the highest occurrence within this population. Statistical evaluation confirmed that neither sex, ethnicity, age nor height influenced dominance in a coronary arterial pattern. The presence and description of the bifid LAD has been recorded. Its occurrence is highest amongst Blacks. The anomalous path of the LCX has been documented and described. The significantly high occurrence of this disposition of the LCX within the South African population appears to be the highest reported find in the literature.

In terms of the presence and patterns of myocardial bridges, there are no observable differences between ethnic groups or sex. Results of this study confirm a relationship between the straight appearance of the LAD on angiogram and its anatomical presence.

Extra coronary collaterals have been successfully investigated and observed. Measurements of vessel dimensions and patterns have been recorded.

Results of the histopathological investigation illustrate that the intra-mural LAD artery is relatively protected from vascular disease. It does not however support the theory that in such a sub-myocardial position, the LAD artery is never prone to the damaging effects of atherosclerosis. The "cardio-protective" effect of a muscular bridge, whilst prevalent, is dependant on the thickness and extent of the bridge itself.

The anatomy of the coronary arteries has been successfully documented and a bank of data, specific for a South African population has been presented. Significant aspects of coronary arterial patterns have been discussed and interpreted in terms of its

clinical relevance. This study presents an original method for the investigation of ECC's using technologically advanced materials and equipment. In addition, a scientific method for confirmation of a "straight" appearance of the LAD artery has been developed in this study. Findings contribute to the bank of diagnostic indicators that may be used to predict myocardial bridges pre-operatively. Through the dissection experience of more than 150 hearts and observation of more than 200 angiograms, this study has been able to contribute to the anatomical description of the coronary arteries. In some ways new perspectives were afforded and on the same note, already existing concepts have been verified. The value of this study is enhanced by the potential clinical impact that such data is envisaged to create.

ACKNOWLEDGEMENTS

"Thy infinite gifts come to me only on those very small hands of mine.
Ages pass, and still thou pourest....
And still there is room to fill."

Rabindranath Tagore

I wish to begin this formal expression of my gratitude by acknowledging the generosity of those whose names I have been unable to recall. Indeed, no success is ever possible through solitary effort. The recognition that this work may receive therefore, belongs not to the name that precedes it,
but to those who have made it possible.

Samraj and Asha Lachman, my parents, for Their inspiration, support, encouragement and faith in my ability to succeed – for Their most integral contribution to my life and for the Love which I continue to receive in such abundance

Anusha and Samesh, my sister and brother for their Love, support and companionship in my life

Dr Heidi Bas, without whose kindness, Love, support and generosity this work would not have reached completion. With eternal gratitude and admiration for her contribution to my life and to this achievement

Dr Robert Acland, for his most treasured friendship, for his inspiration and unfailing support, for allowing me the use of his laboratory facilities, materials and intelligence, for his effort in creating the images that appear in this dissertation and for his selfless contribution to my life

Professor Kapil Satyapal, for his friendship, valued contribution to my career and for inspiring my academic growth

Professor Ebrahim Vanker, for his support, encouragement and faith in my ability throughout this work, for sharing with me his incredible knowledge and inspiration

Nalini Pillay, my dearest friend and co-researcher. For her presence in my life – for her Love and infinite encouragement and for her integral and devoted contribution to the success of this work

Nalini Pather, for her support and friendship and for the selfless hours of contribution and inspiration to my work and life

Mr Ganas Mathura, for his valued contribution to this work. For his commitment, expertise, encouragement and immense support throughout my academic endeavours.

Satya Baboolall, for his contribution to this study, for sharing with me his knowledge, time and intelligence and for his continuous support and encouragement

Mr Leonard Majola, for the unfailing attempts and persistence in ensuring that there were specimens for this study – my most sincere gratitude for his contribution

Lelika Ramsaroop, for her support and encouragement

Ranjeni Govender and **Sohana Sooful**, for their unconditional Love and friendship

Deven Naidoo, for all that was and continues to be possible because of his valued contribution to my life

Sharita, Shireen, Ashmita, Selina, for being the most loving and inspirational sisters

Poowa, Shani Poowa and the rest of the **Lachman Family**, for providing a home that is one of my biggest blessings, for their Love and unconditional support

Bickrum Lachman, for everything that he inspired me to be and for that which I am

Friends at the **Department of Anatomy – UDW**, for their support and encouragement

Sohan Mansingh, for his respect, dedication and help when it was most needed

Indirani Naidoo, for her efforts in making the interpretation of this study possible, for her support and friendship

Friends at the **Department of Human Biology – DIT**, for their support and unlimited encouragement

University of Durban Westville, Durban Institute of Technology, Forensic Pathology at the Nelson Mandela School of Medicine, Kornhouser Library at the University of Louisville, Entabeni Hospital Cath Lab, for allowing me the use of their facilities and the time and support in order to accomplish this work

Fresh Tissue Lab – University of Louisville, for the integral role it has played in the success of this work. And to all the intelligent and inspirational individuals that have passed through the lab - For the opportunities I have had to learn from them

Department of Radiography – DIT, for their continuous support throughout this research

Rosh Sundar – for her technical support, generosity and friendship
Doug Greenshield and the **Oppenheimer Trustees**, for their valued support and encouragement

Dr Ashwin Bhamdhev and **Logan Govender** at the Lancet Pathology Laboratory, for their contribution and support

My Friends: **Mary, Dennis, Neelam, Chakshu, Sandil, Cecily, Joan, Cedric, Anusha, Joy, Brian, John, Mala, Veena, Kavitha, Mary Pellerito**, for their Love and support

Brandon Madanjit and **Albert Hirasen** – for their support, kindness and expert assistance

Bette Levy, for her support, encouragement and inspiration in my life – and for being such a great mom

Judd Griffin, for believing in me, for his Love, support and selfless contribution to the success of this work

Dr's Wojciech Pawlina and **Stephen Carmichael**, for their friendship, support and encouragement in my career

Dr Kunwar Bhatnagar, for his support and encouragement and contribution to my spiritual growth

Makoto Tamai, for sharing with me his brilliance, talent and friendship, for allowing me to experience the energy of working with him and for his expert and artistic contribution to the images that appear in this dissertation

Lydia Shina, for the warmth of her friendship, support and unconditional Love, through whose encouragement, I have grown, accepted, understood and achieved

And **Kevin Goudy**, in whose wisdom I have unfaltering faith. For his unparalleled ability in being able to bring such clarity to my thoughts and such definition to my purpose. For his cherished support and inspiration in my life.

LIST OF FIGURES

LIST OF TABLES

LIST OF GRAPHS

LIST OF PLATES

LIST OF ABBREVIATIONS

ABBREVIATION	INTERPRETATION
AA	Aortic Arch
ANG	Angiogram
AP	Antero-posterior
BCT	Brachiocephalic Trunk
BF	Black Female
BM	Black Male
CAD	Cadaveric
ECC	Extra Coronary Collaterals
EXD	External Diameter
IDX	Index
IF	Indian Female
IM	Indian Male
ITA	Internal Thoracic Artery
IVC	Inferior Vena Cava
LA	Left Atrium
LAD	Left Anterior Descending Artery
LAO	Left Anterior Oblique
LAT	Lateral
LCA	Left Coronary Artery
LCO	Left Coronary Ostium
LCX	Left Circumflex Artery
LPA	Left Pulmonary Artery
LPV	Left Pulmonary Vein
PDA	Posterior Descending Artery
PT	Pulmonary Trunk
RCA	Right Coronary Artery
RAO	Right Anterior Oblique
RCO	Right Coronary Ostium
RCX	Right Circumflex
RM	Ramus Marginalis
RPA	Right Pulmonary Artery
RPV	Right Pulmonary Vein
RSA	Right Subclavian Artery
STJ	Sino-Tubular Junction
SVC	Superior Vena Cava
TDX	Tortuosity Index
WF	White Female
WM	White Male

CHAPTER I

INTRODUCTION

"The only means of strengthening one's intellect is to make up one's mind about nothing –

To let the mind be a thoroughfare for all thoughts, not a select party."

John Keats (1795)

1.1. INTRODUCTION

It may appear presumptuous perhaps, that one should decide to embark on a study of the anatomy of the coronary arteries. Such anatomical enquiry indeed, seems hardly novel, hardly revolutionary. Yet beyond the limitation of a preconceived notion that guards the philosophy of Anatomy, lies a field of potential resource that has transcended the platform of standard discovery in order to integrate the fundamentals of clinical science.

This dissertation attempts to review the anatomy of the coronary arteries using a clinical approach to investigate the morphologic presentation of these vessels within the South African population. On a more clinically universal level, this study aims to elucidate two focal areas of anatomical interest: extra-cardiac collaterals and myocardial bridges.

Although it has been presumed that the modern day anatomy is a simple descriptive exercise, recent advancement of imaging techniques has made possible the visualization of the coronary arteries with remarkable accuracy. Whilst such technical advancement undoubtedly favors clinical outcome, the variability of angiographic images in any given patient population, to some degree, often casts doubt with its interpretation, (Muriago et al., 1997). Reports of the incidences of these patterns in the patient population vary greatly and the fallacy of assessing coronary artery disease without taking into consideration the individual pattern becomes apparent, (Ochsner and Mills, 1978).

Coronary vessel anomalies although rare, are a recognized cause of myocardial ischemia and sudden cardiac death, (McConnell et al., 1995). Even though some are often considered to be

simple anatomical variations, controversy still exists over the functional importance as well as clinical and pathological implications of these findings.

Although recognized as a distinct anatomical entity, the surgical significance of the intramural coronary artery and its branches has evoked much interest. With the trend toward minimal access cardiac surgery, pre-operative identification of such anatomical variants of coronary artery disposition becomes increasingly relevant, if technical challenges that may be associated with its surgical presentation are to be abated. Whilst studies have been attempted, little has been achieved in terms of the precise identification of these arteries angiographically. In addition, the possibility of the relative immunity of an intra-myocardial segment of coronary artery from atherosclerosis, presents encouraging possibilities. From a clinical standpoint, such information regarding the histology of a vessel subject to such anatomical disposition may be valuable in the understanding and direction of a clinical outcome.

There are several clinical controversies derived from a discrepancy on the description and relations of the coronary vessels in human hearts of either sex and in different ethnic groups, (Caetano et al., 1995). Such controversy is not unfounded and has over the years become an issue of critical importance in appreciating the concept of patient specificity. The scientific notion of ethnicity as an influencing factor in creating a predisposition for anatomic variation cannot be ignored. Standard anatomical texts, while substantively clear, invariably present descriptions that appear to be widely based on data gathered from a predominantly, if not exclusively, Caucasian population. Although in terms of its anatomic value, such information makes for interesting study, its clinical impact, when measured against a diversity of target groups, may be limited. It is no longer a trend, but rather a necessity to up-date where available and create where deficient, banks of anatomical morphometric data that is ethnically relevant.

In assessing the clinical characteristics of coronary surgery patients, the gender profile with regards to peri-operative mortality has received some attention. Reports appear to indicate that although men present more frequently with coronary artery disease and for subsequent surgical procedures, women in a similar set-up tend to be associated with higher failure and mortality rates. In as much as understanding the physiological disposition of this population continues to be achieved, little has been presented on the anatomy of the coronary arteries within each group.

When O'Connor et al., (1996) investigated the effect of coronary artery diameter in patients undergoing coronary artery bypass surgery, their results indicated that females had smaller coronary arteries than males. After controlling for differences in body size, they concluded that smaller coronary arteries in females and smaller individuals influenced peri-operative mortality during coronary artery bypass procedures. However, information regarding coronary artery size

and its correlation between sex, race and body size is rarely available and as such, warrants further investigation.

When Gross (1921) in his landmark contribution, proclaimed the abundance of anastomoses that existed between coronary arteries, the idea not only gained popularity, but also established a basis from which several focal investigations then developed.

The possibility of naturally occurring alternate arterial channels offers critical clinical direction in dealing with coronary artery obstruction. Although the presence of arterial collaterals in the atherosclerotic heart may vary from none to extensive in the patient population, these inter-coronary anastomoses are important in myocardial revascularization and may allow communication with surgically created collaterals, (Oschner and Mills, 1978). Even more encouraging, is the idea of the existence of collateral sources beyond the confines of the cardiac boundaries.

With continuing advancement in medical research, to date, most procedures to revascularize the myocardium have been directed toward establishing bulk flow in large vessels. The new trend however, is toward establishing bulk flow to ischemic areas by inducing the growth of collateral vessels by the topical application of growth factors. Due to this trend, more information is now needed regarding the "normal" anatomy of collateral vessels that may connect the territories of the principle coronary arteries with systemic vessels. Such information would be valuable for planning treatment strategies for the induction of collateral vessels, and also, for evaluating the effectiveness of such treatments.

Surgically, these extra cardiac collaterals are thought to be the source of unwanted bleeding from coronary arteries in situations where a dry field is needed. They are also thought to interfere with cardioplegia and create warming of the myocardium by displacement of the cardioplegic solution, (Brazier, 1974). In addition, although the degree of participation of these vessels in a collateral role may be hypothetical, it seems a possibility that they may perhaps be in part responsible for the survival of a patient in the event of occlusion of both coronary arteries at their ostia, (Gross, 1921; Moritz et al., 1932).

Since Hudson et al's presentation on the existence of extra-coronary collaterals in 1932, this topic of coronary research has waned. Although Hudson et al., (1932) presentation provided the basis for the localization of ECC's, its value is limited by the use of invasive exploration techniques and technological limitations.

With only one significant report on experiments using animal models and a few random publications of case studies, coronary to systemic connections have to date not been approached with the anatomical enthusiasm it deserves.

The objectives of this study are presented in line with three major focus areas: Coronary Artery Anatomy, Myocardial Bridges and Extra-Coronary collaterals. This investigation aims to:

1. Record and compare data from various ethnic and sex groups within the South African population

2. Review the anatomy of the coronary arteries by means of cadaveric dissection and angiographic analysis.

3. Document the incidence and arrangement of myocardial bridges

4. Scientifically validate the association between the anatomical presence and angiographic manifestation of the intra-mural left anterior descending artery

6. Investigate the anatomy of the extra coronary collaterals

7. Examine by means of histological assessment, the predilection of atherosclerosis within an intra-mural artery

CHAPTER II

LITERATURE REVIEW

"In our study of Anatomy, there is a mass of mysterious Philosophy; and such as reduced the very Heathens to Divinity: yet, amongst all those rare discoveries and curious pieces I find the Fabrik of Man, I do not so much content my self as in that I find not, there is no organ or Instrument for the rational Soul"

Thomas Browne, 1643

CORONARY ARTERIES: HISTORICAL BACKGROUND

"Yet when we content ourselves with their discoveries and calmly believe that there is now no more place for new inventions, the sprightly edge of our own wit languisheth,
and we extinguish the lamp which they lighted to our hands"
William Harvey, (1653)

If one were to go by the accounts that standard historical literature banks present, it would appear that significant interest in the anatomy of the coronary arteries began somewhere within the early 1500's. The first recorded account of this arterial pattern however, dates back to when Leonardo Da Vinci (1452-1519) captured his artistic impression of a few oxen hearts during an exploration of the viscera within the thoracic cavity, (Angelini et al, 1999). Although visually effective, his descriptive account of the coronary arteries appears to be brief. The contribution of his early sketches however, is valuable in its point of bringing into attention the anatomy of the aortic valve, the coronary ostia and the proximal course of the right and left main stems. In addition, the depictions highlight the progressive decrease in coronary artery diameter as the vessels continue to reach the apex of the heart. Although lacking in anatomical detail, Da Vinci's sketches formed an important foundation for the initiation of further investigation of human hearts, (Figure 9).

Anatomical accounts of the coronaries continued along two lines of interest. Whilst Andreas Vesalius (1514-1564), brought to the fore a set of fundamental records, the "Tabulae Anatomicae" others such as Scarpa, (1794) and Cloquet, (1828) demonstrated dissections

through their own investigations with detailed artistic depictions, (Figures 1, 2, 3, 4, 5, 6, 7 and 8). In the early 1700's, Vieussens and Thebesius were the forerunners in descriptive coronary anatomy and in 1761, Morgagni clearly defined the two main coronary arteries. Encouraged by the need for such information, more anatomists began documenting the patterns illustrated by the coronary arteries. Noteworthy contributions by Bell and Bell, Cheselden and Quain and Sharpey in the 1700- 1800's and Gross, Schlesinger, Edwards, Baroldi, James and Roberts and Goldberg in the early 1900's have not only presented modern day anatomists with descriptions that to date remain unchallenged, but have also provided a foundation upon which clinical questions may be clarified and understood.

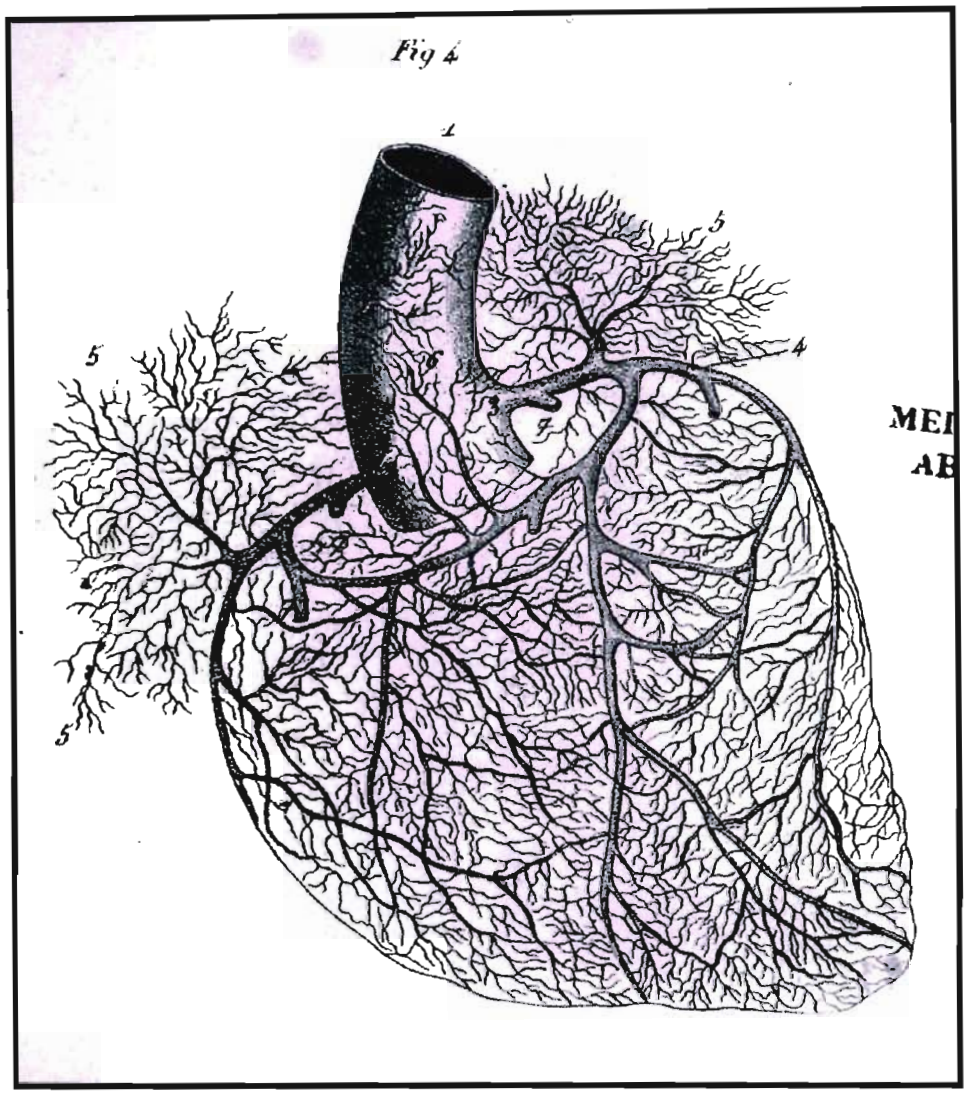


Figure 1: Coronary tree showing inter-coronary anastomoses

(Adapted from Cloquet, 1828)

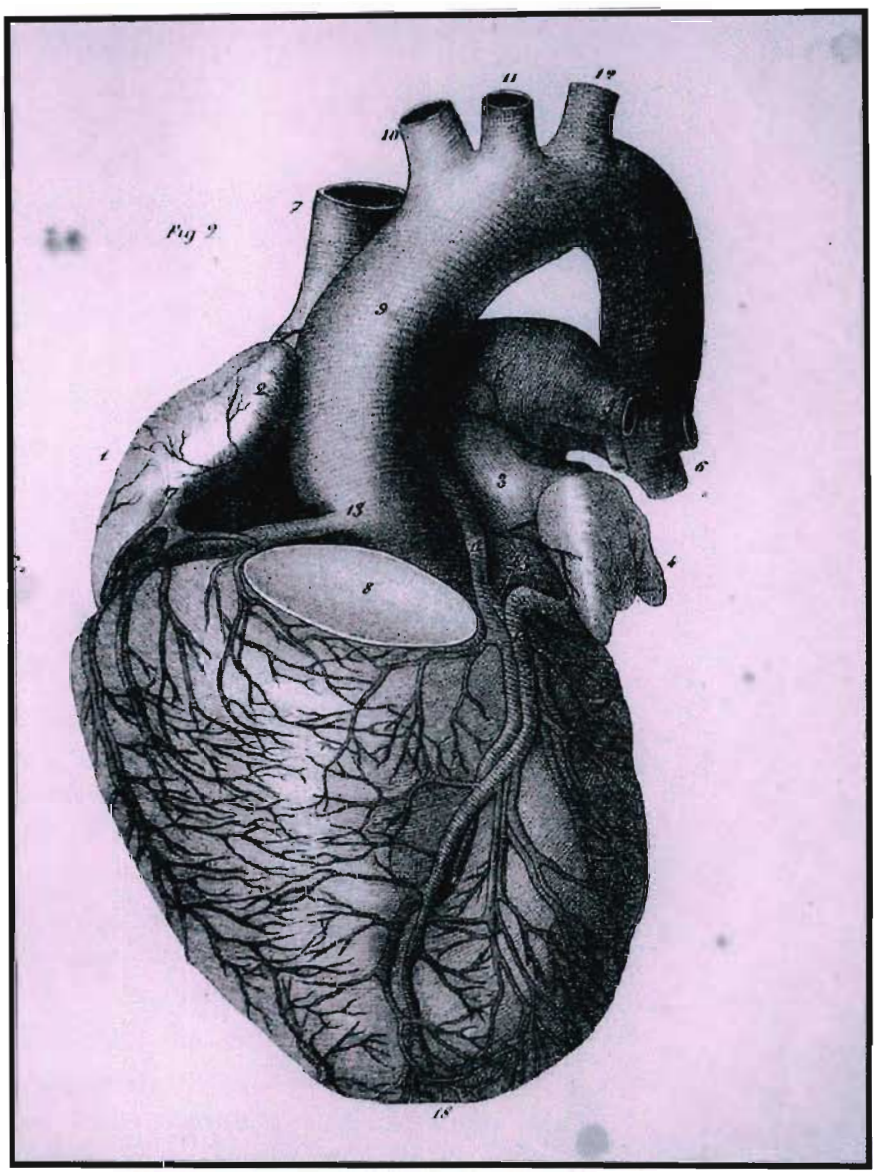


Figure 2: Lateral view of the heart showing the RCA and LCA and LAD artery

(Adapted from Cloquet, 1828)

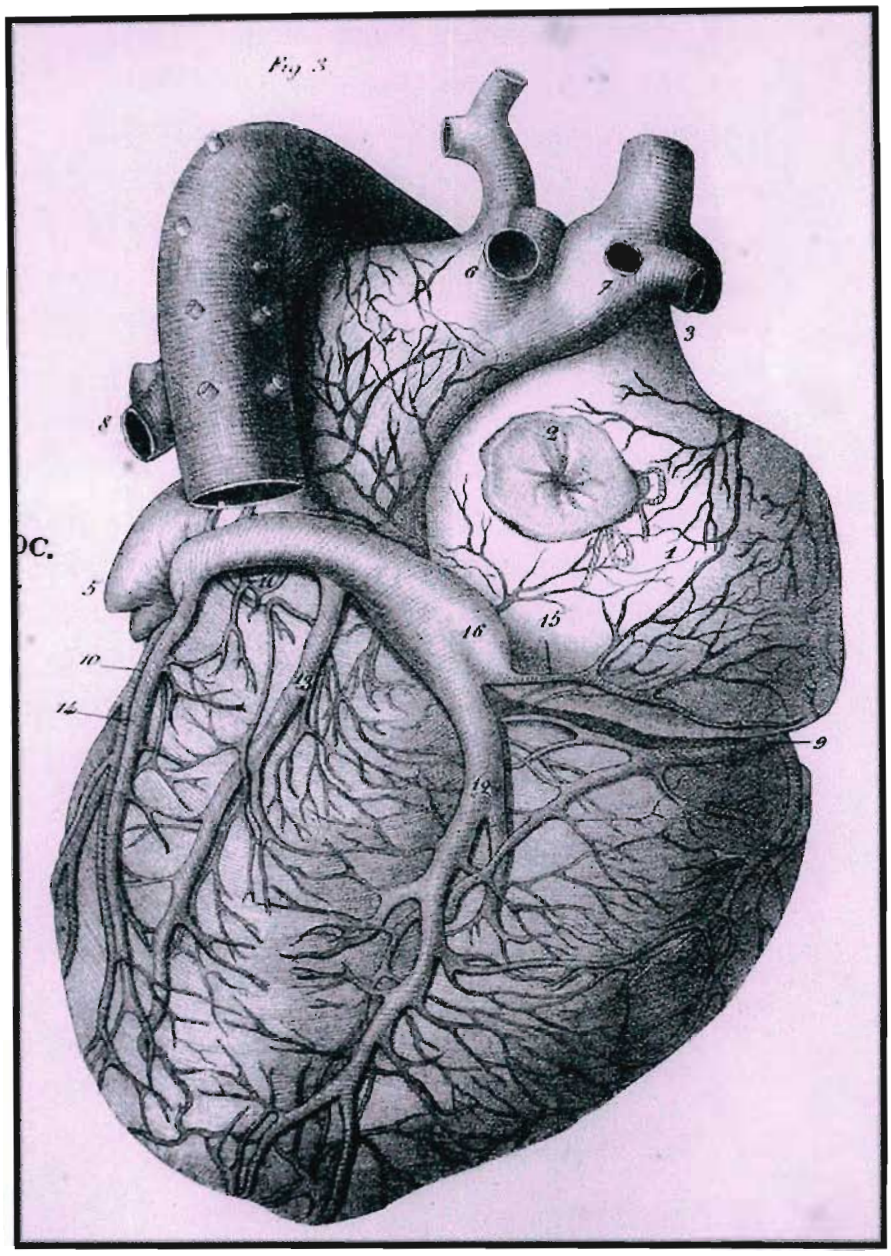


Figure 3: Posterior view showing the coronary sinus and branches to the posterior interventricular groove

(Adapted from Cloquet, 1828)

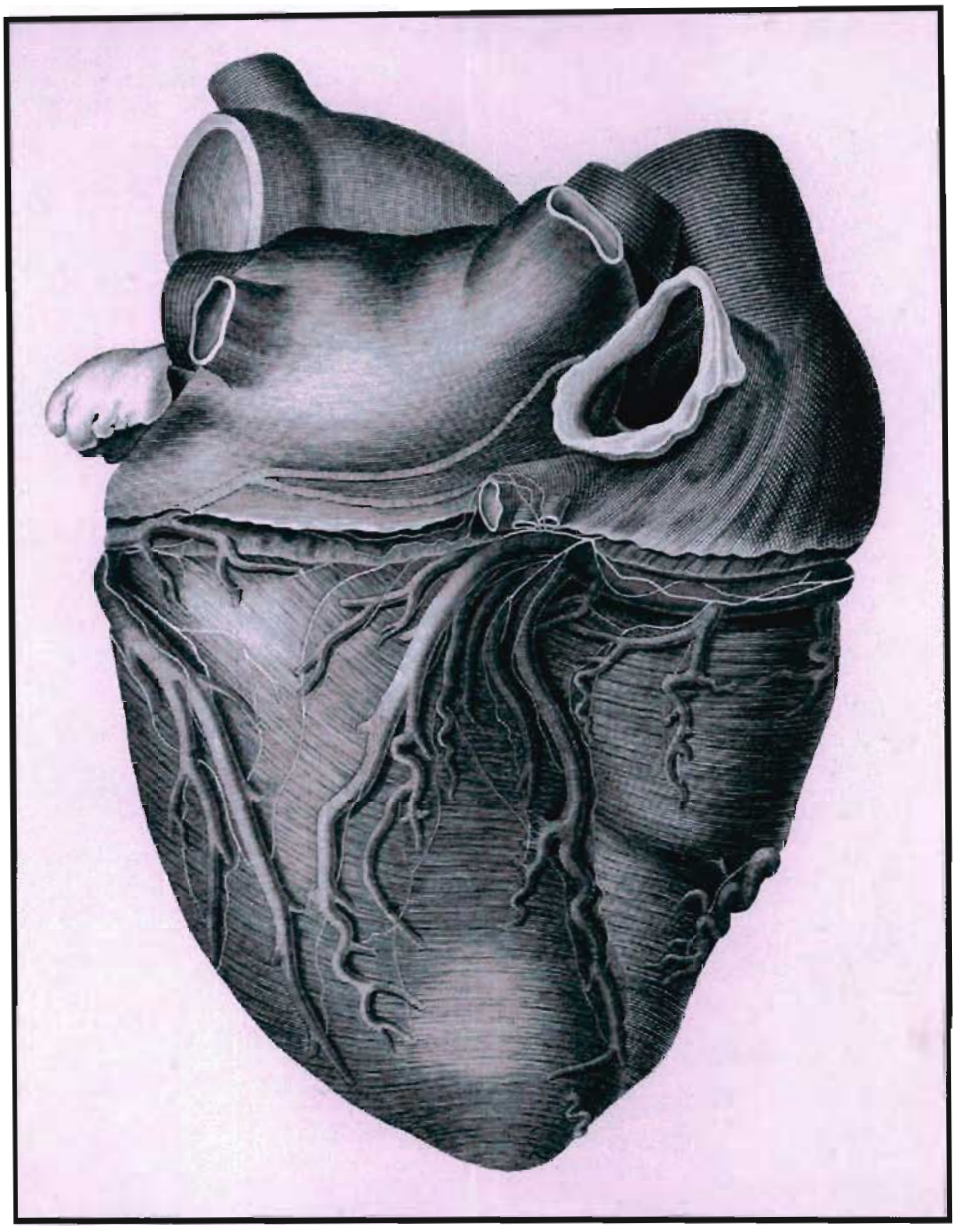


Figure 4: Posterior view of a "real right dominant" heart showing the RCA and LCX artery branches

(Adapted from Scarpa, 1794)

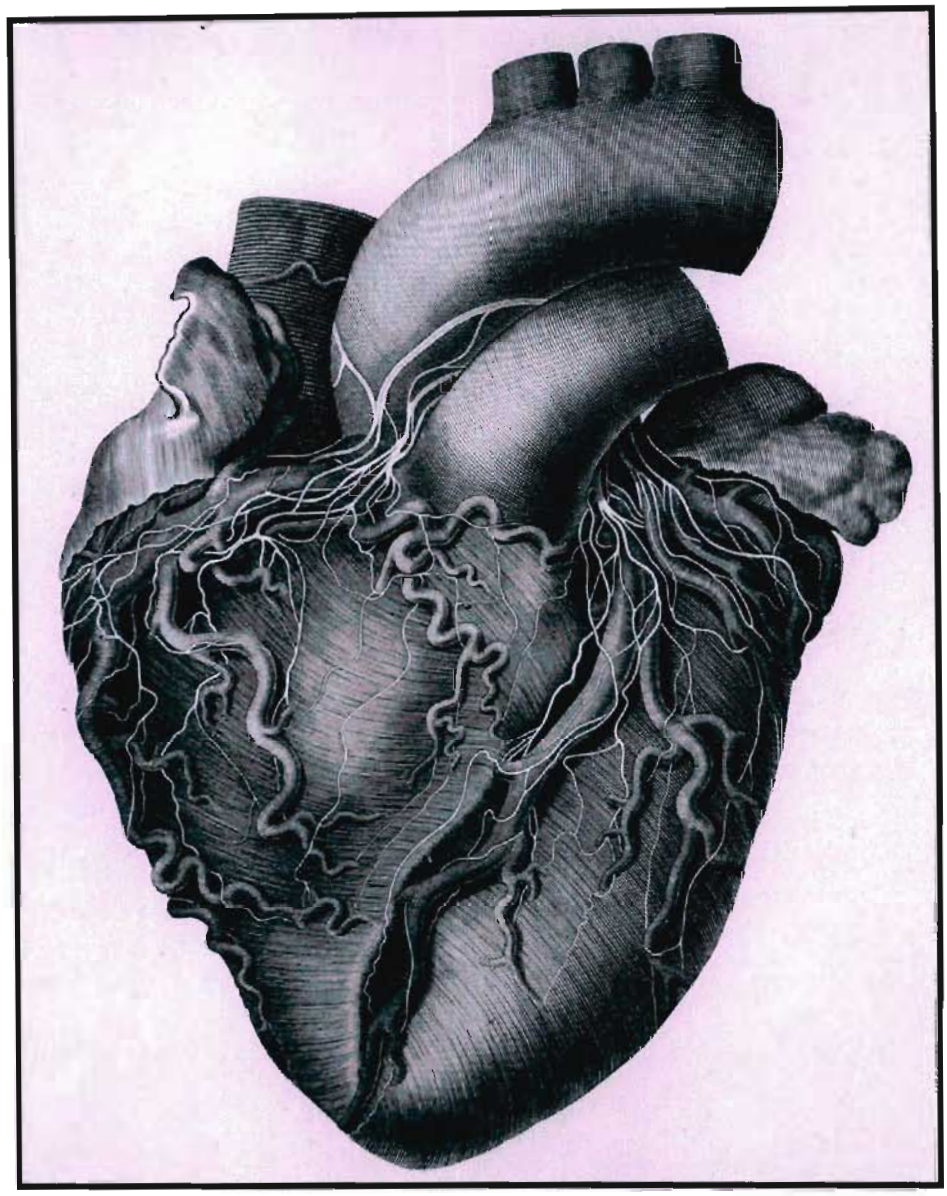


Figure 5: Left anterior view of the heart showing ventricular branches of the RCA and the LAD artery

(Adapted from Scarpa, 1794)

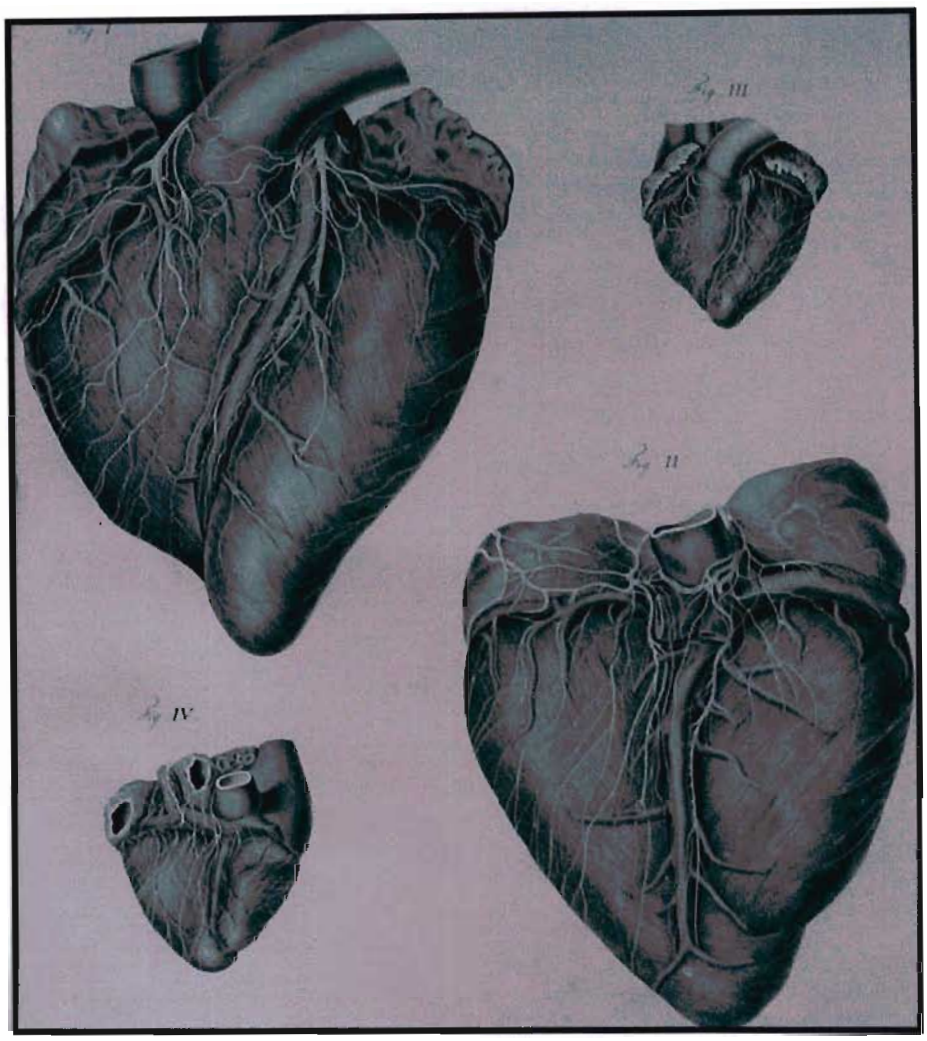


Figure 6: Anterior and posterior views of the coronary artery branches

(Adapted from Scarpa, 1794)

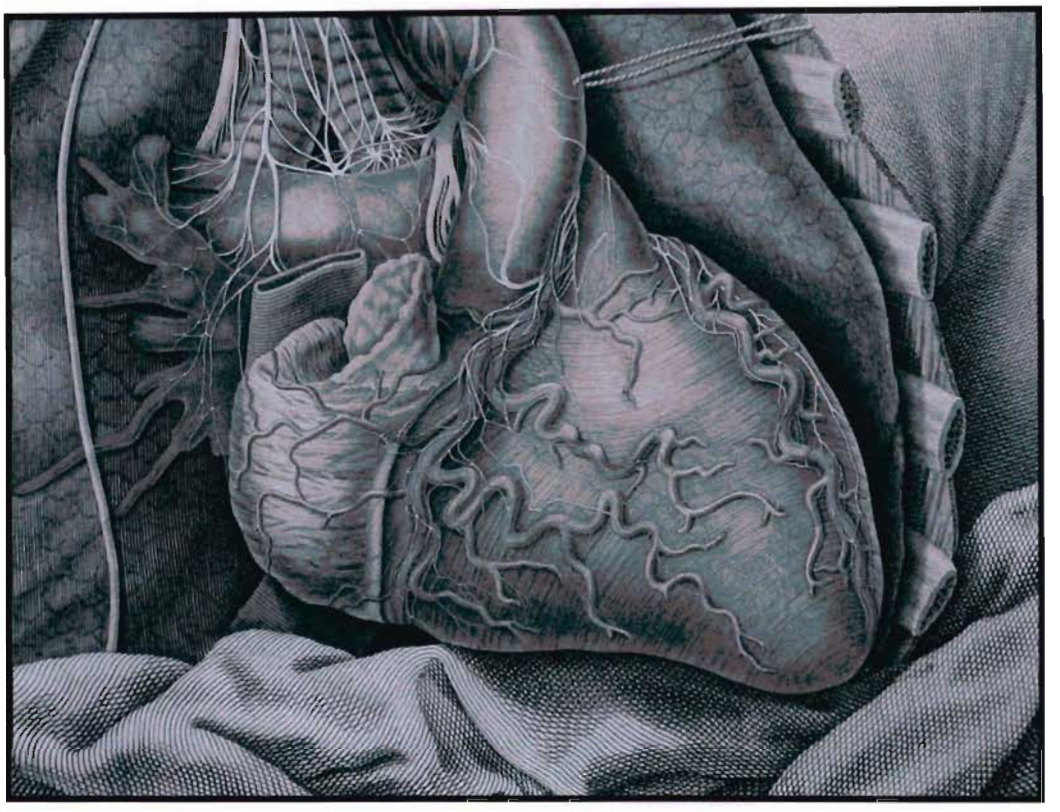


Figure7: The heart *in situ* demonstrating the RCA and branches

(Adapted from Scarpa, 1794)

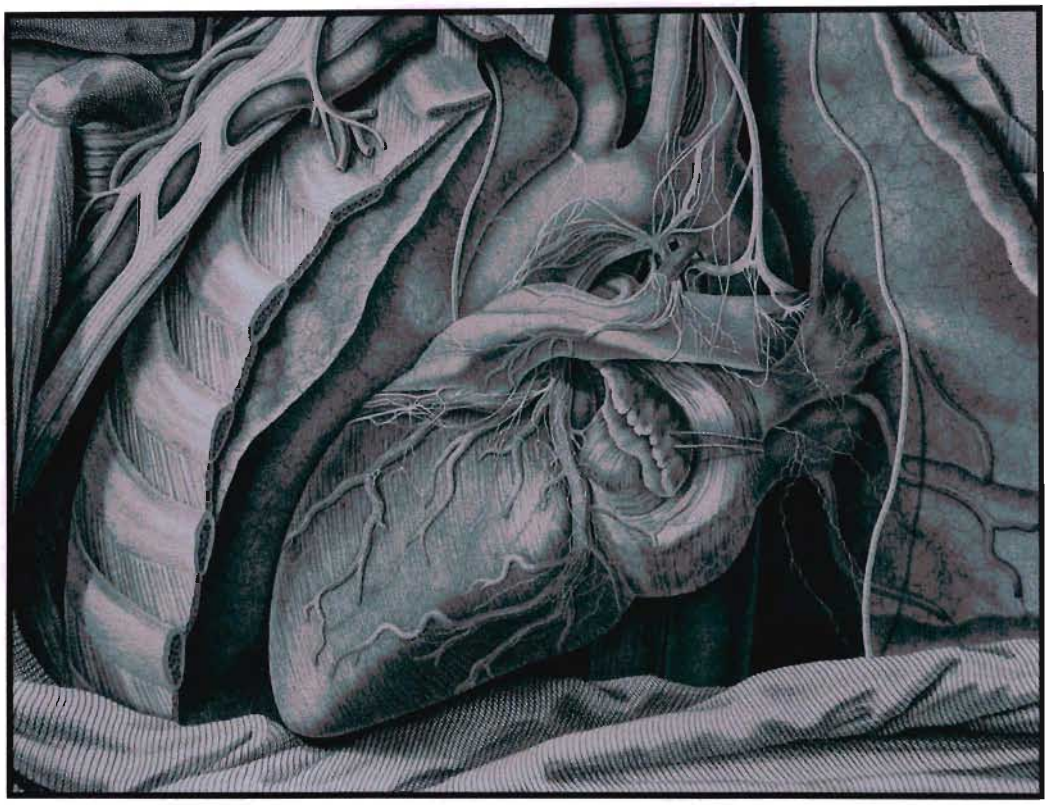


Figure 8:The heart *in situ* demonstrating the LCA branches

(Adapted from Scarpa, 1794)

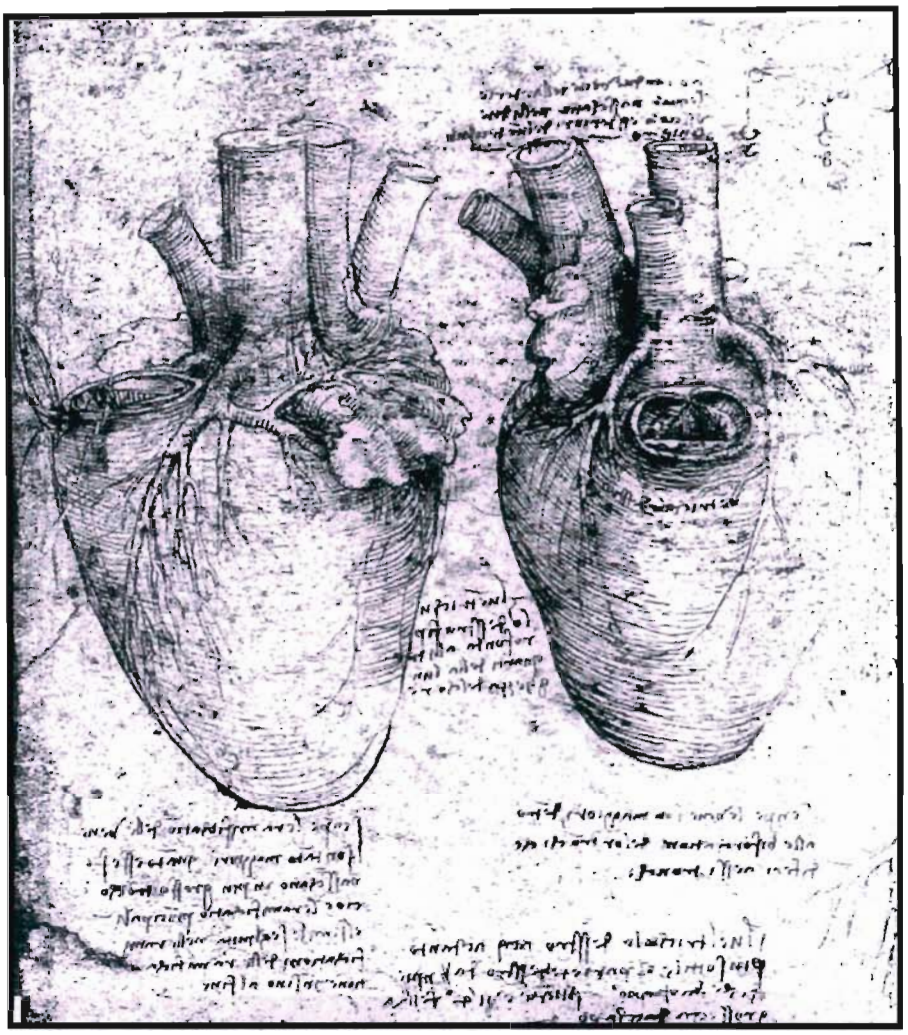


Figure 9:Early sketches of the coronary arteries

(Adapted from Leornardo Da Vinci,)

DEVELOPMENTAL ANATOMY OF THE CORONARY ARTERIES

"A man passes seed into a womb and goes his way, and anon another cause takes it in hand and works upon it and perfects a babe – what a consummation, from what a beginning"
Marcus Aurelius, (121-180)

The development of the coronary vessels is a dynamic process that involves a progressive, sequential, uninterrupted and irreversible series of events, (De la Cruz et al., 1999). The sequence of events begins with the appearance of stem cells that will form the coronary vascular system. The primitive coronary vessels are then formed and after a period of differentiation, connect with the aorta and establish the basic coronary distribution patterns around the heart.

Studies based on the development of chick embryos, (Hirakow, 1983) regarding the extra-cardiac origin of the epicardium from cells within the dorsal wall of the sinus venosus, formed the hypothesis that the vascular sub-epicardial network is also of extra-cardiac origin.

Viragh et al., (1993) found that the endothelial cells of the vessels were derived from the pro-epicardial organ, located in the region of the sinus venosus. From here, stem cells migrate inside the sub-epicardial space, in the regions corresponding to the atrioventricular and inter-ventricular grooves. In the adult heart, these grooves correspond to the location of the main coronary arteries. The authors suggested that the cardiac neural crest cells give rise to the smooth muscle and connective tissue of the tunica media of the great arteries, but not to the smooth muscle of the coronary artery media.

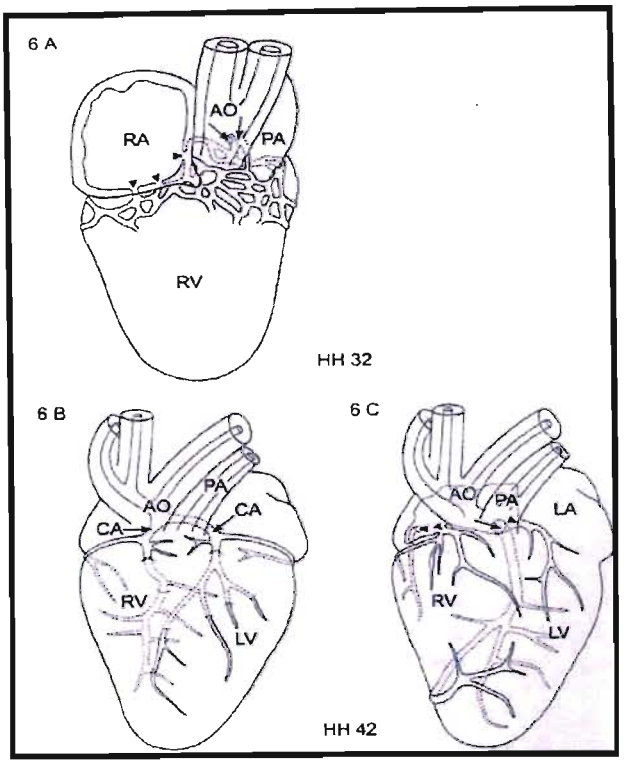


Figure 10: Early embryonic development of chick coronary arteries

(Adapted from Vrancken Peeters et al., 1997)

The sub-epicardial, primitive coronary vessels first appear as vascular lakes that eventually transform into arteries and veins. According to early studies (Gross, 1921), sub-epicardial vessels first appear in humans during the sixth week of development. More recently, (Hirakow, 1983) studies have shown that the coronary vessels first appear in situ, in the sub-epicardial space as endothelial tubes without a tunica media. These studies also describe essential features of the main coronary arteries as seen in the mature heart, already defined in the nine-week embryo. The author reported that early coronary vessels are composed of endothelium only (without tunica media) and that groups of sub-epicardial cells preceded the appearance of coronary vessels, (Figure 10).

Coronary vessels first appear when the truncal and ventricular septation is advanced. Essentially,

these initial coronary vessels consist of endothelial cells without a tunica media, yet they eventually give rise to both coronary arteries and veins. These vessels are therefore termed primitive coronary vessels, (De la Cruz et al., 1999).

The primitive coronary vessels connect initially with the sinus venosus and later, with the aortic root, (Hirakow, 1983). The study indicated that the first primitive coronary vessels to define themselves were the future coronary veins. Some reports hypothesize that the aortic endothelium may actively participate in connecting the primitive coronary network to the aorta by forming coronary buds at the level of the primordial semi-lunar cusp valves, (Angelini, 1989). Others have postulated that the aortic endothelium is passively entered by extensions of the sub-epicardial primitive network, (Bogers et al., 1988). It is important to remember however, that at the time of aorto-coronary connections, the walls of the coronary arteries are composed only of endothelium.

According to Waldo et al., (1994), multiple channels from the primitive coronary arteries are initially present near the so-called facing and non-facing aortic sinuses. However, only two channels usually persist - one in each facing sinus. The coronary arteries therefore, seem to connect consistently, only with the sinuses of Valsalva adjacent to the pulmonary artery.

Although the factors that determine the specific site of penetration of the coronary vessels on the aortic wall are still unknown, the findings that the two facing sinuses (aortic-pulmonary area) are formed by the truncal septum, while the non-facing sinuses are formed by the intercalated truncal swellings is of importance. Observations of an abundance of cardiac neural crest cells in the truncal septum (Waldo et al., 1994) may bear relevance to the understanding of the mechanisms involved in the appearance of the coronary artery connections with the aorta.

The establishment of the basic coronary artery patterns and their anatomic relationship to the heart, occur in two chronologic periods. First, the distribution of the primitive coronary vessels is defined in relation to the established architecture of the heart. Specific coronary-aortic connections are then established by a process of extensive vascular interaction, (De la Cruz et al., 1999).

At the time of development of the atrioventricular and inter-ventricular grooves, two vascular rings begin to develop during angiogenesis. The coronary atrioventricular ring, consisting of the right coronary and left circumflex arteries appears at the atrioventricular groove and the coronary inter-ventricular ring, consisting of anterior and posterior descending branches, appear at the inter-ventricular groove, (De la Cruz et al., 1999).

The final arterial pattern is further defined by interruption of these rings and development of the dependant myocardial masses. This occurs simultaneously, with the establishment of the coronary circulation with an aortic origin and drainage into the venous system. Although the caliber of the coronary arteries, is known to be influenced by arterial flow throughout embryogenesis, the specific mechanisms that determine coronary artery patterns and size are still under investigation.

THE ANATOMY OF THE CORONARY ARTERIES

"This name of coronary vessels is a very favorite one with anatomists, and is applied wherever vessels surround the parts which they belong to, however little this encircling may be like a crown, and it is thus that we have the coronary arteries of the stomach, coronary arteries of the lips, and coronary arteries of the heart... But these vessels of the heart are really very beautiful and have some things very peculiar in their circulation..... ."
Charles Bell, 1827

2.3.1. INTRODUCTION

Whether from actual dissection or citation, early anatomical accounts of the heart appear to agree in that the coronary arteries, usually two in number originate from the aortic base somewhere above the free margins of the semi-lunar valves. From here, they are said to continue on a course following the grooves that demarcate the boundaries between the atria and ventricles, (Bell and Bell, 1827; Gross, 1921; Fulton, 1965), (Figures 11, 12 and 13).

Although it may appear, after reviewing a number of key texts, (Williams et al., 1989; Angelini et al., 1999; Allwork, 1987, Moore et al., 1992) that this standard description would remain pretty much established, the concept of anatomical anomalies was by no means masked by the terseness with which the coronary arteries were alluded to. There are indeed reports of such deviation from the norm as early on as when Vesalius's famous "*Tabulae Anatomica*" showed the right coronary artery originating from the left coronary artery and coursing anterior to the pulmonary trunk. A single coronary ostium was reported by Fallopius (Venice, 1562), and in the centuries to follow, anatomists such as Cruvielheier (1834) and Hrytl (1879) presented findings of a similar nature.

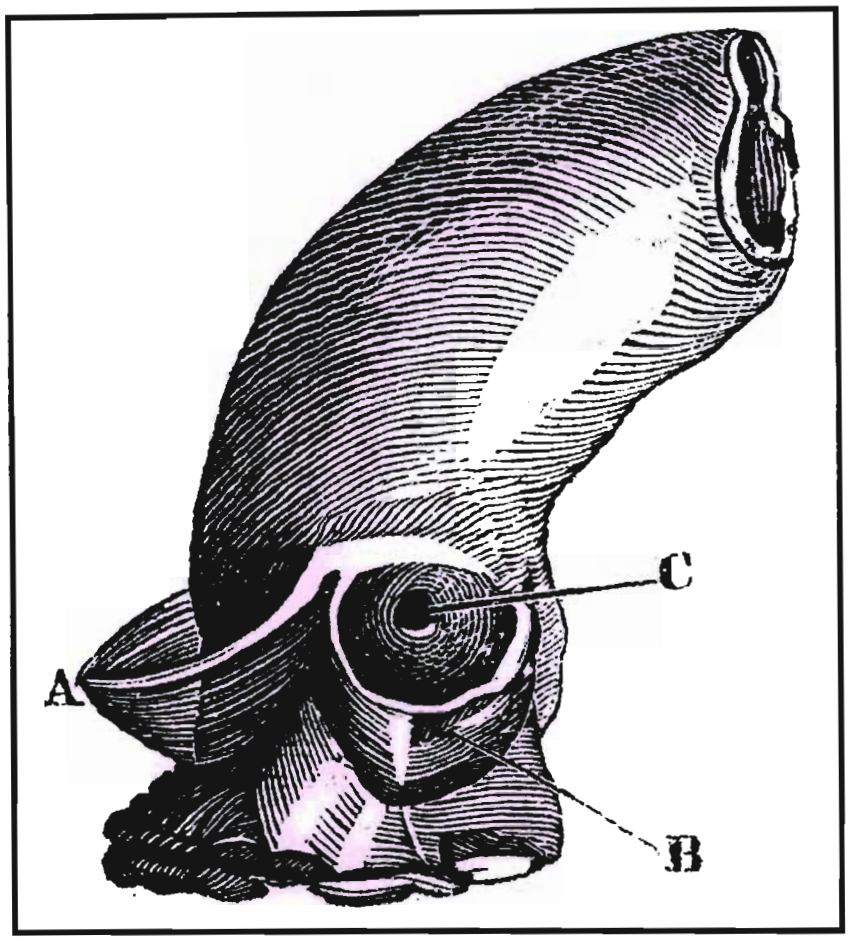


Figure 11: Coronary ostium (C) and aortic sinus (B)

(Adapted from Bell and Bell, 1827)

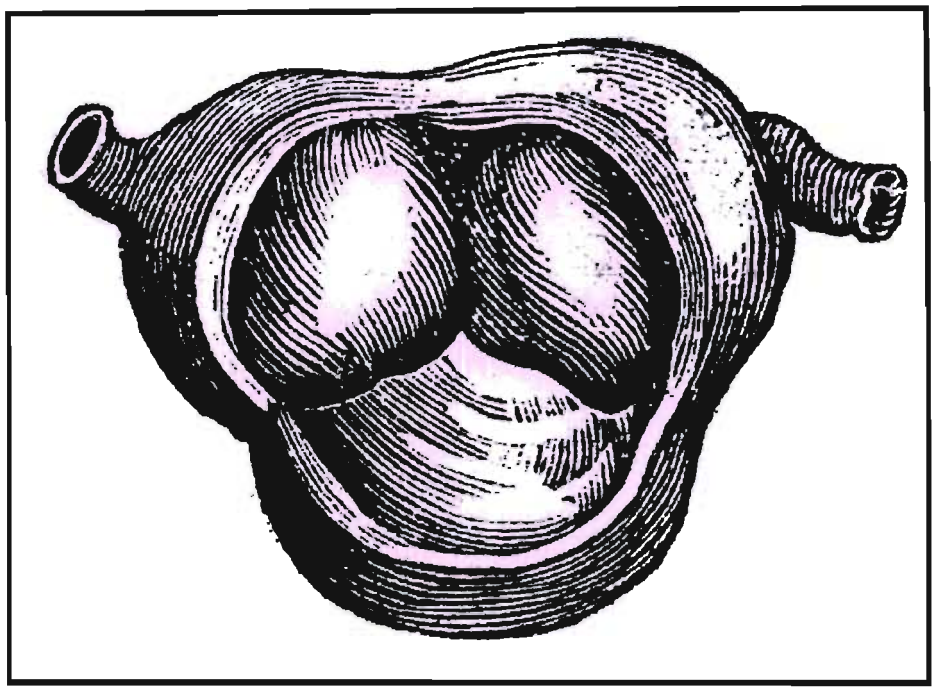


Figure 12: Dilated aortic sinuses and proximal stems of the RCA and LCA

(Adapted from Bell and Bell, 1827)

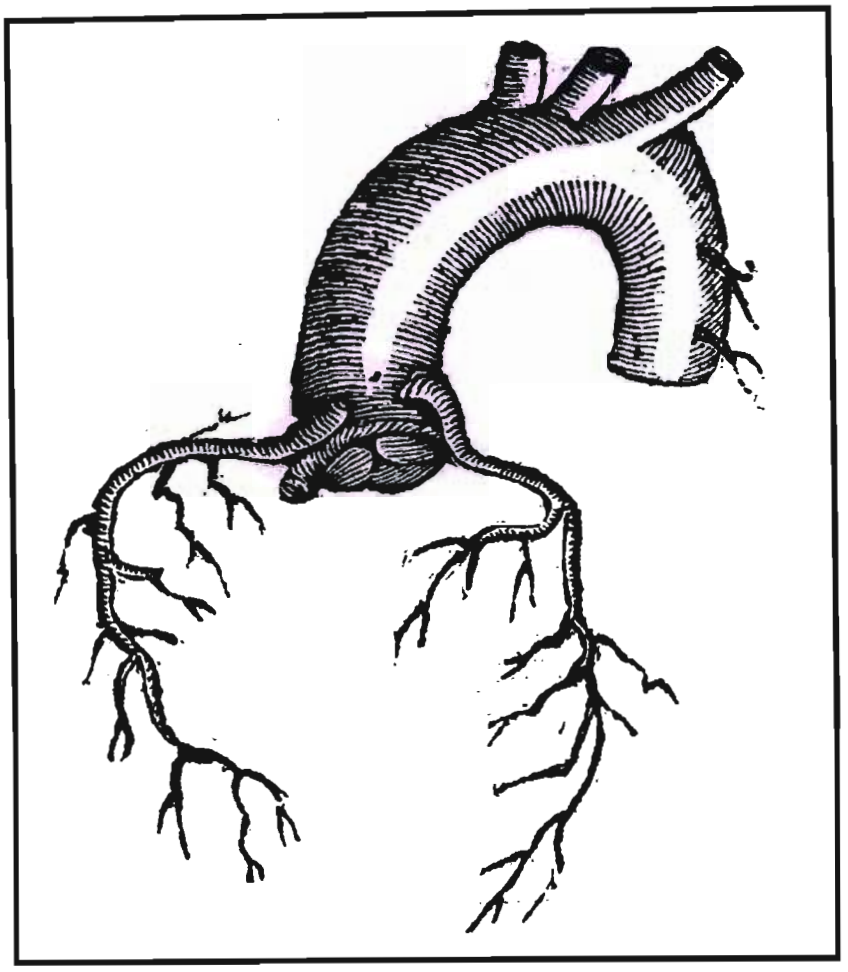


Figure 13: Right and left coronary tress

(Adapted from Bell and Bell, 1827)

At this point therefore, for any further description of the "normal" anatomy of the coronary arteries to be of anatomical value, it would need to be preceded by some understanding of what is meant by the term "normal".

There are three points of reference that may be considered in attempting to afford a basis from which one may begin to describe, classify or define the coronaries. In their dissertation on coronary artery anomalies, Angelini et al., (1999) proposed the following:

That a coronary artery may be defined as any artery or arterial branch that carries blood to the cardiac parenchyma, which may be any structure located within the pericardial cavity, (the cardiac parenchyma includes the myocardium, semilunar and atrioventricular valves, proximal part of the great vessels and the visceral pericardium);

That the name and distribution of a coronary artery or branch may be defined by the vessel's distal vascularization territory, and not by its origin;

And that the sinuses of Valsalva should be identified by their own topographic location and not by the artery that originates from it.

In the spectrum of coronary morphology, "normal" should therefore mean, "what is commonly observed" and "abnormal" or "anomalous", reserved for any form observed in less than 1% of the general population. Furthermore, this criterion is proposed by Angelini et al., (1999) to be the dividing line between "normality" which includes more frequent variants and "abnormality" which includes relatively infrequent variants (anomalies).

2.3.2. CORONARY ARTERY NOMENCLATURE

There are two schools of thought when considering the nomenclature of the coronary arteries. Anatomical texts, (Nomina Anatomica, 1989) employ the use of the following terminology to describe these vessels, (Figure 14):

The ***right coronary artery,*** (RCA) is used to indicate the main stem on the right side of the heart. Its main branches are the ***conus*** artery, right anterior and posterior ***ventricular rami***, the ***right marginal artery***, ***nodal*** arteries to the sinuatrial and atrioventricular nodes and the ***posterior interventricular artery***.

The left system originates from the ***left coronary artery,*** (LCA). Its main branches are the ***anterior interventricular artery***, left anterior and posterior ***ventricular rami***, ***anterior septal rami***, the ***circumflex artery***, (LCX) the ***left marginal artery*** and ***nodal arteries*** to the sinuatrial and atrioventricular nodes.

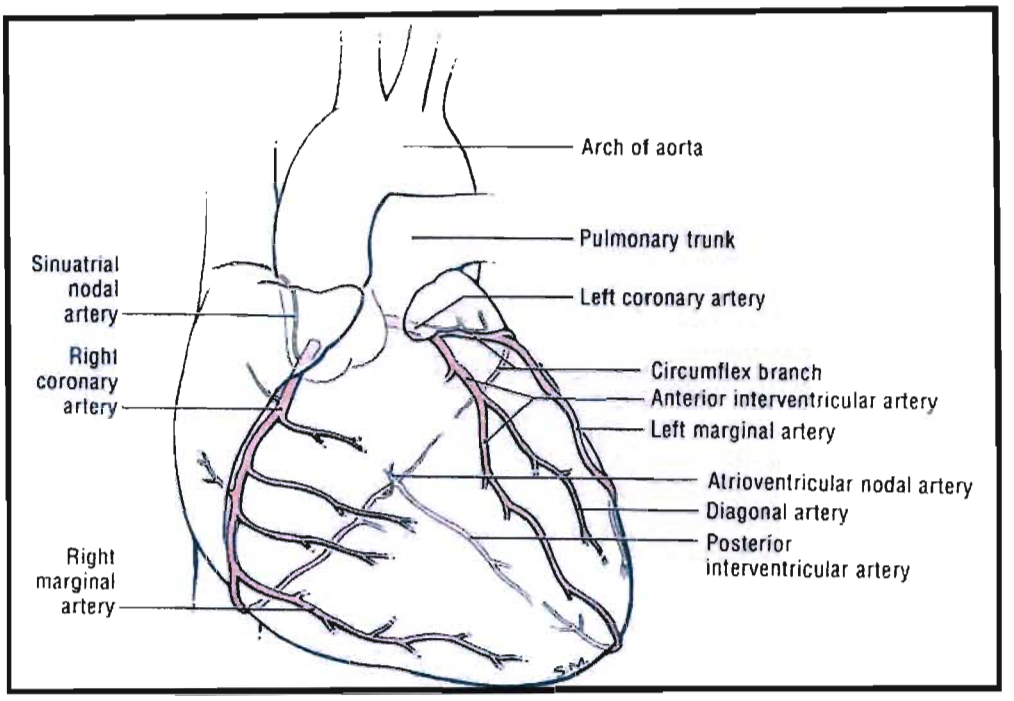


Figure 14: Anterior view of the coronary arteries

(Adapted from Moore, 1992)

Surgical texts, (Ochsner and Mills, 1978; Alexander et al., 1998) on the other hand employ the following terminology in their description of the arterial pattern: the main stem of the right system is called the right coronary artery, (Figure 15). Its main branches are the conus artery, nodal branches to the sinuatrial and atrioventricular nodes, the acute marginal artery, and the posterior descending artery.

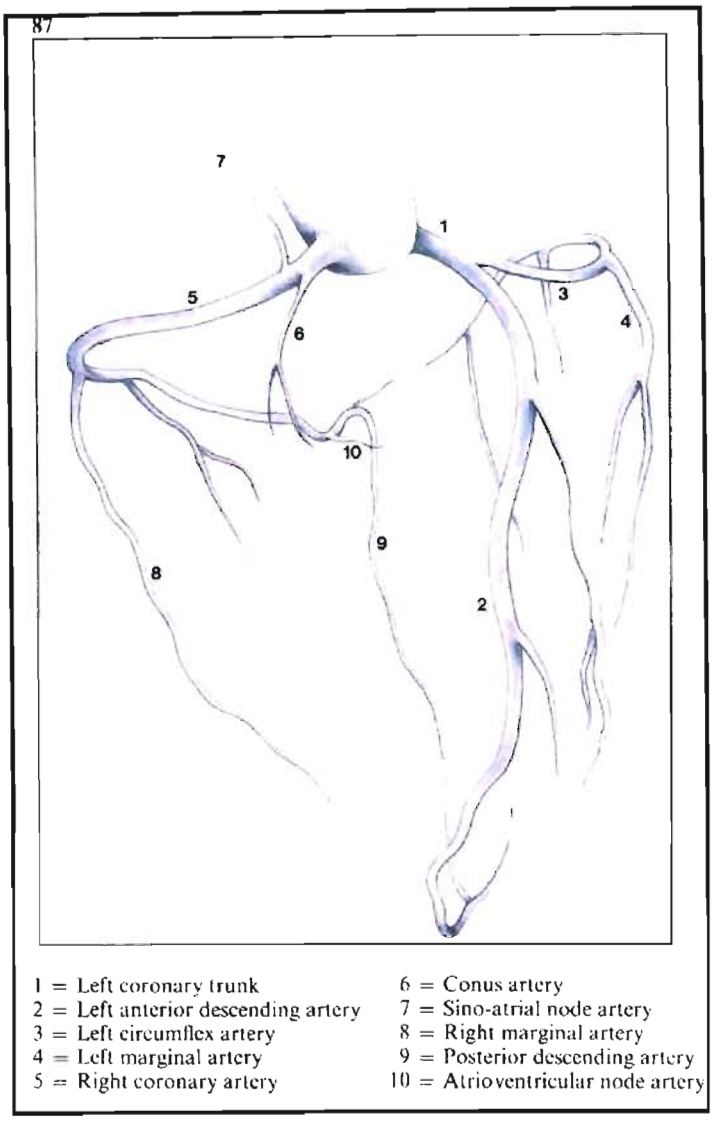


Figure 15: Surgical nomenclature of the coronary arteries

(Adapted from Farrer-Brown, 1977)

The LCA is referred to as the left main stem. Its branches are the left anterior descending, (LAD) with left diagonal branches and right septal perforators. The left circumflex, (LCX) gives off obtuse marginal branches and when dominant, the posterior descending artery, (PDA). When the left main artery terminates in a trifurcation, the branch arising between the anterior descending and circumflex is referred to as the ramus medianus or ramus marginalis, (Ochsner and Mills, 1978).

In view of making clear the distinction between anatomical and surgical nomenclature with regards to the coronaries, the following table has been formulated. For the purpose of facilitating this anatomical and clinical study, a list of the terminology employed in this dissertation is presented.

Table 1: Coronary artery nomenclature

ANATOMICAL	CLINICAL	CURRENT STUDY
Right coronary artery	Right coronary artery	Right coronary artery
Conus artery	Conus artery	Conus artery
Right marginal artery	Acute marginal artery	Right marginal artery
Posterior interventricular artery	Posterior descending artery	Posterior descending artery
SA and AV nodal arteries	SA and AV nodal arteries	SA and AV nodal arteries
Left coronary artery	Left main artery	Left coronary artery
Anterior interventricular artery	Left anterior descending artery	Left anterior descending artery
Left circumflex artery	Left circumflex artery	Left circumflex artery
Left marginal artery	Obtuse marginal artery	Obtuse marginal artery
	Diagonal artery	Diagonal artery
	Septal perforators	Septal perforators
	Ramus medianus / Ramus marginalis	Ramus marginalis

2.3.3. CORONARY ARTERY ORIGIN

The coronary arteries originate from the aortic root. Anatomically, the aortic root consists of three, usually equal sized semilunar leaflets, three inter-cuspal spaces and three aortic sinuses, and a sino-tubular junction, separating the aortic root from the ascending aorta, (Angelini et al., 1999). The aortic root lies behind, and slightly to the right of the pulmonary valve. The aortic and pulmonary valves have an adjacent contact point (the remnant of the aorto-pulmonary septum). The two aortic sinuses that adjoin the aorto-pulmonary contact point are in most cases, the points

of origin of the coronary arteries. They are termed "facing sinuses" as they face the pulmonary artery. Traditionally, the sinus opposite the aorto-pulmonary contact point is termed "non-facing" and therefore "non-coronary" sinus, (Figure 16).

Although two ostia are typically present, it is also normal to see a third, for a conal branch, or a third coronary artery, (*Reported incidence - 23-51%*). The left anterior descending and circumflex branches may sometimes originate separately, in the absence of a main trunk and as a result, produce a third or fourth coronary ostium (*Reported Incidence - 0.4-8.0%*), (Angelini et al., 1999).

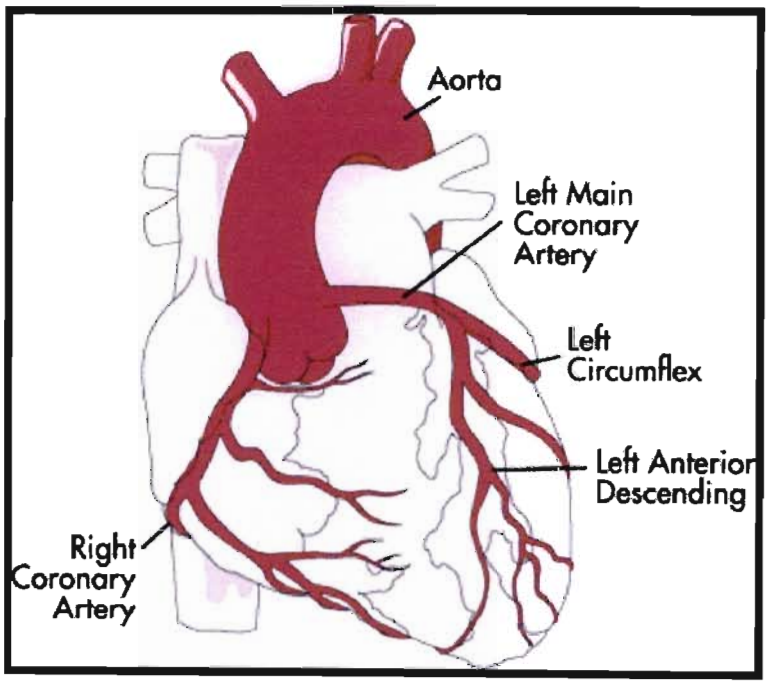


Figure 16: Coronary Artery Origin and Branches

The proximal segment of a coronary artery usually arises at a nearly orthogonal angle from the aortic wall. However, it is not uncommon to observe distinctly unusual angles of origination, (Roberts et al., 1982), (Figures 17, 18 a, b, c and d). Nerantzis and Marianou, (2000) have recently reported a "high" origin of both the coronary arteries from the left aortic wall.

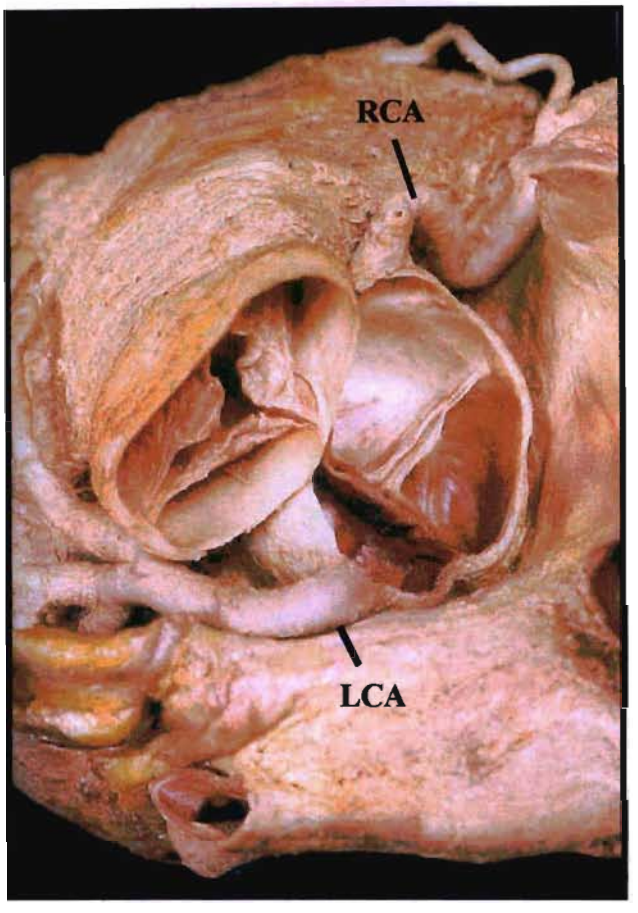


Figure 17: Origin of the coronary arteries from the aortic sinuses

(Adapted from Gosling et al., 2002)

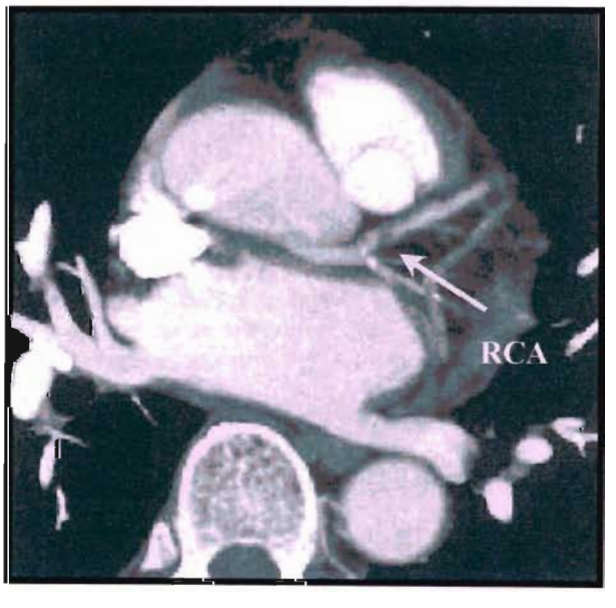


Figure 18 (a): CT image of the proximal coronary arteries

(Adapted from Electro Medica, 2000)

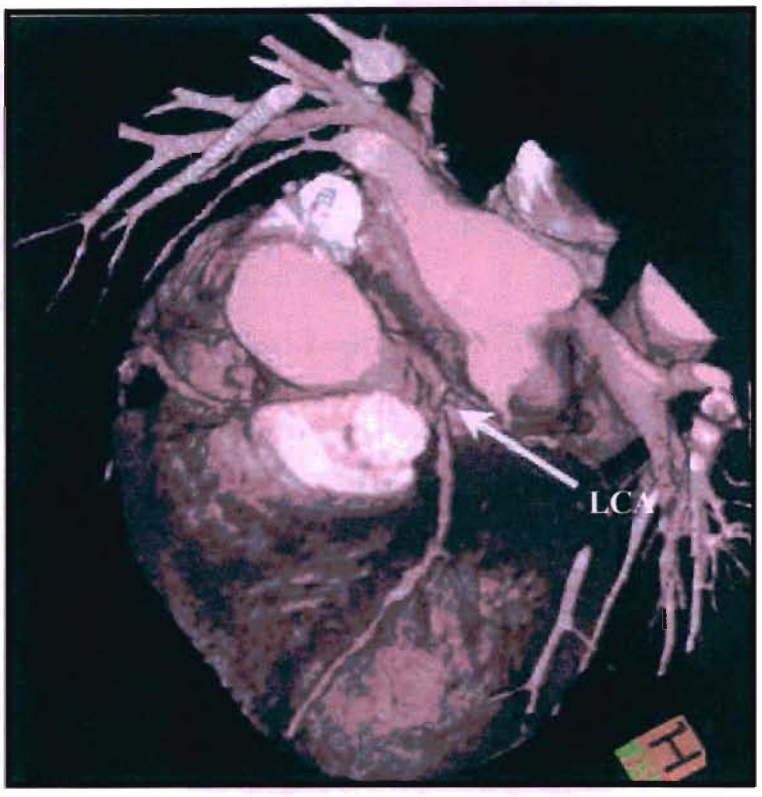


Figure 18 (b): CT image of the proximal coronary arteries

(Adapted from Electro Medica, 2000)

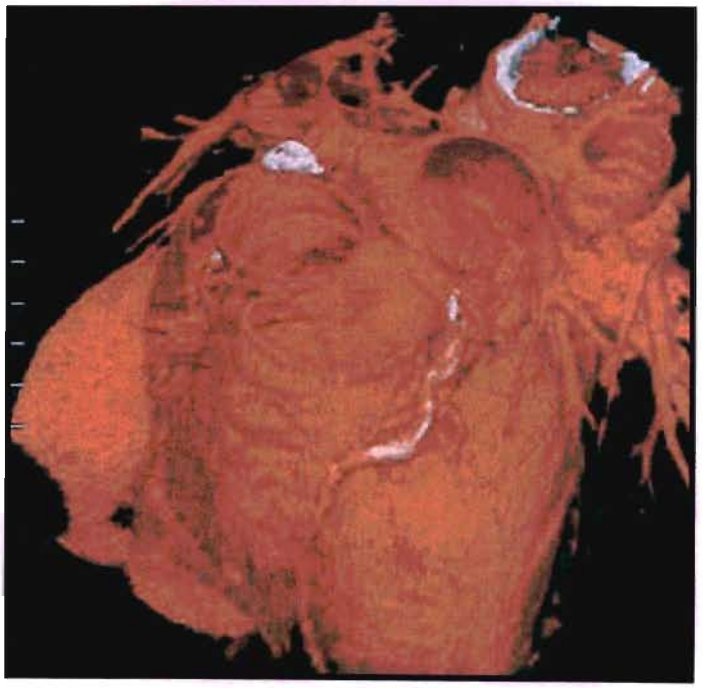


Figure 18 (c): CT image of the proximal coronary arteries

(Adapted from Multi Slice Clinical Data, 2000)

2.3.4. THE RIGHT CORONARY ARTERY

Origin and Course

The right coronary artery (RCA) usually arises from an ostium that is located below the sino-tubular junction in the middle of the right aortic sinus, (Williams et al., 1989 and Angelini et al., 1999). From here it is said to pass anteriorly and slightly to the right, between the right atrial appendage and pulmonary trunk. Upon reaching the atrioventricular sulcus, the artery then descends within the sulcus, almost vertically, to the inferior right margin of the heart and then curves around it to continue posteriorly to the region where the inter ventricular and inter-atrial grooves meet – a point referred to as the "crux" of the heart, (Moore, 1992 and Williams et al., 1989), (Figures 21 and 22).

Termination of the RCA usually ends a little to the left of the crux by anastomosing with the LCX in about 60% of individuals,, (Moore, 1992 and Williams et al., 1989), In 10%, it ends either near the right inferior border or in between this and the crux and in 20%, may reach the left border, infiltrating part of the LCX artery territory . The RCA may therefore stop at the acute margin, continue to the crux or left atrioventricular groove, or occasionally extend up to the territory of the LAD artery, (Figures 19 and 20).

Branches and Distribution

The branches of the RCA supply the right atrium and ventricle, parts of the left chambers and the atrioventricular septum.

The RCA gives off the ***right conus artery*** (as the 1st branch). The conus artery extends anteriorly to the lowest part of the pulmonary conus and uppermost part of the right ventricle. It commonly anastomoses with a corresponding branch from the LCA to form the "Annulus of Vieussens", an anastomotic circle around the pulmonary trunk.

The so-called "1st segment" of the RCA extending from its origin to the right inferior margin of the heart gives rise to anterior atrial and ventricular rami that diverge widely. The ***right anterior ventricular rami***, usually 2-3, extend toward the apex of the heart. The largest of these rami is called the ***right marginal artery***, (Figures 21 and 22) and is of great enough caliber to reach the apex in 93% of individuals, (Baroldi, 1966). When the right marginal artery is very large, the remaining ventricular rami may be reduced to 1 or may even be completely absent.

The "2nd segment" of the RCA, between the right border and the crux, gives off 1-3 small ***right posterior ventricular rami,*** (Figures 21 and 22) which supply the diaphragmatic surface of the right ventricle. Their size is dependant on the caliber of the right marginal artery and may sometimes be absent, (Williams et al., 1989).

As the RCA approaches its termination at the crux, it produces 1-3 posterior interventricular rami, only one of which travels in the inter ventricular sulcus. This prominent artery is known as the ***posterior interventricular artery*** (Figure 20). In most hearts, it remains as a single branch, but may be flanked by accompanying branches on either or both sides. The number of branches arising from the posterior interventricular artery depends on the existence of the flanking vessels. In approximately 10% of hearts, it appears to be is replaced by a branch of the left coronary system, (Angelini et al., 1999, Williams et al., 1989).

The ***atrial rami*** (Figure 19) are often described in anterior, lateral and posterior groups, frequently single small vessels. The anterior and lateral are usually double, very rarely triple and supply predominantly the right atrium. The posterior branch usually supplies the left and right atria, (Williams et al., 1989).

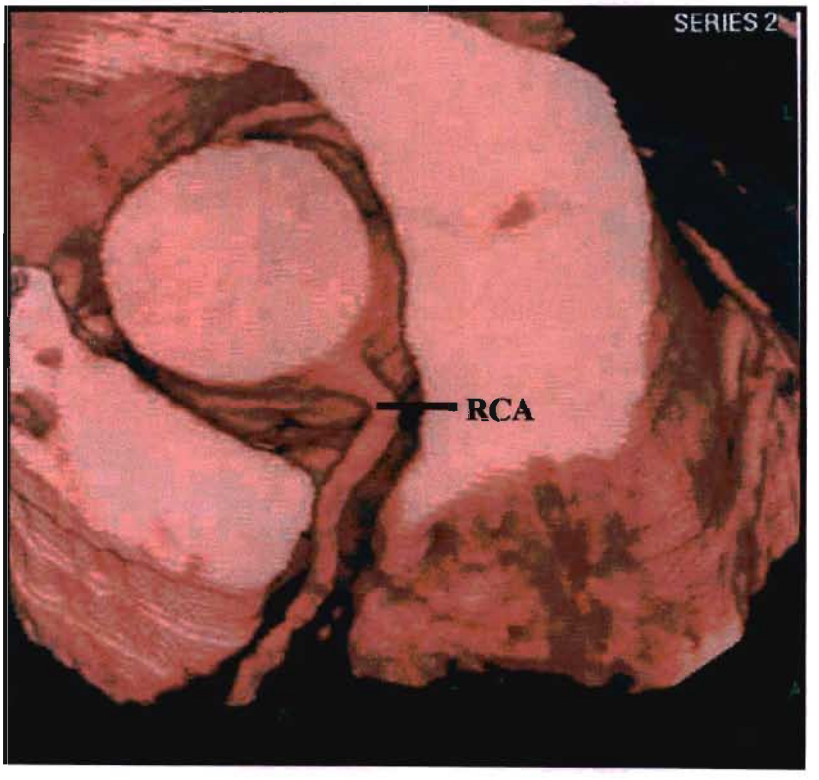


Figure 18 (d): CT image of the proximal coronary arteries

(Adapted from Multi Slice Clinical Data, 2000)

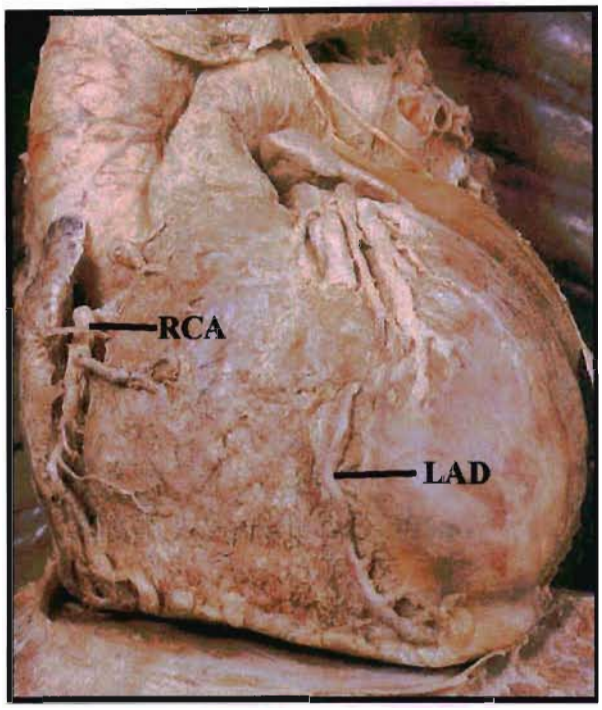


Figure 19: The RCA and LAD artery on anterior view in situ

(Adapted from Gosling et al., 2002)

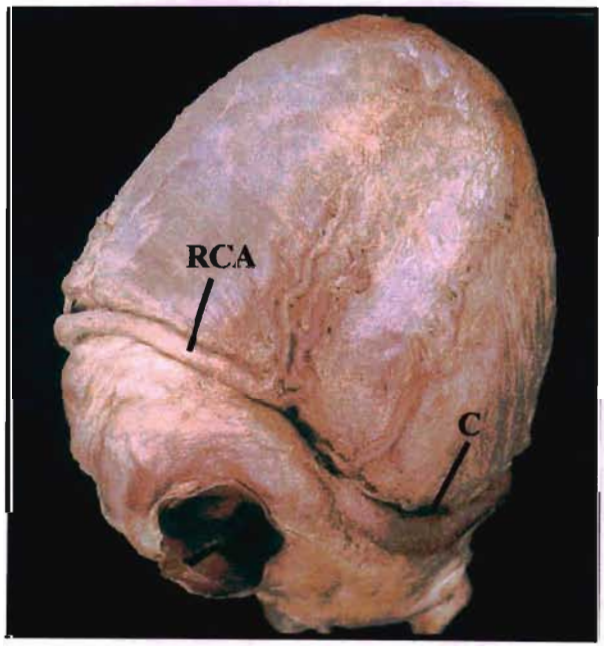


Figure 20: The posterior atrioventricular course of the RCA

(Adapted from Gosling et al., 2002)

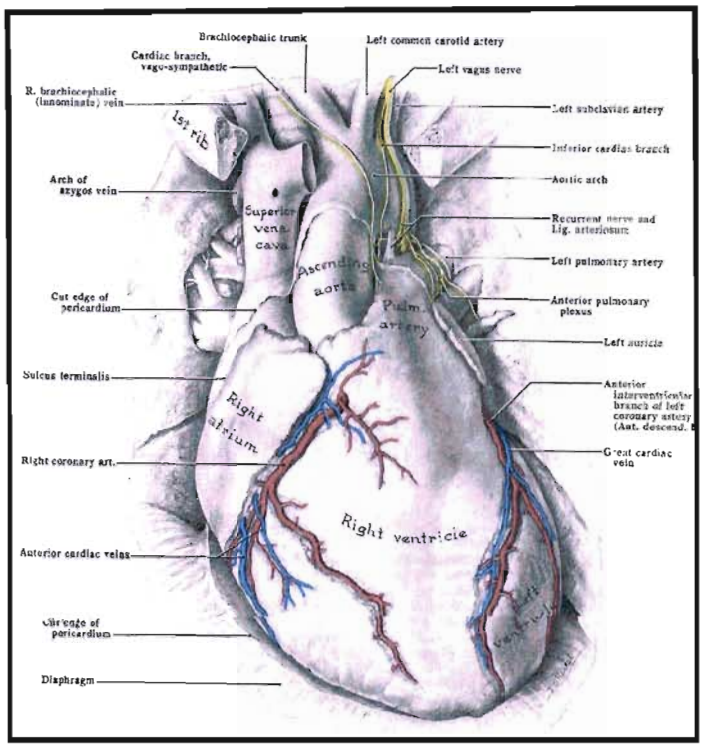


Figure 21: Right and left coronary artery branches

(Adapted from Anderson, 1978)

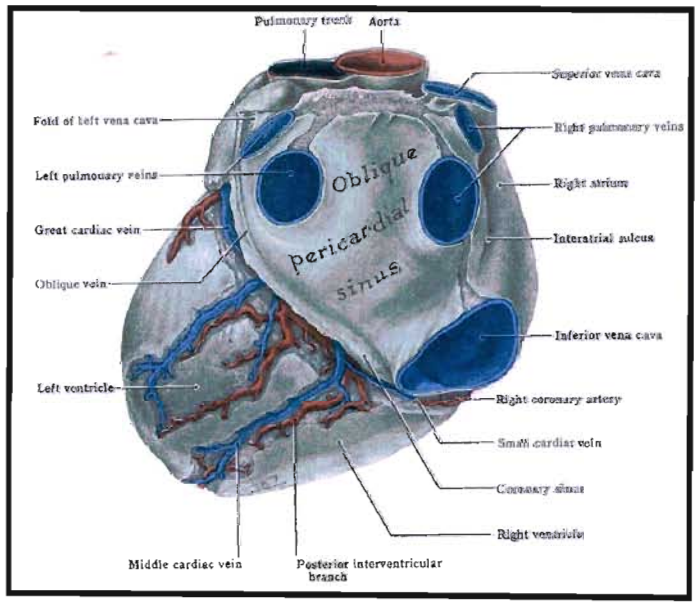


Figure 22: Coronary arterial supply to the posterior ventricular surfaces

(Adapted from Anderson, 1978)

The ***sino-atrial nodal artery,*** (Figures 23 and 24) is distributed largely to the myocardium of both atria but mainly the to the right. In 35% of cases, it may originate from the LCX artery, (Hutchinson, 1978). Irrespective of its origin, this artery branches around the base of the superior vena cava (SVC), forming an arterial loop, which gives rise to small atrial branches. A large branch traverses the sino-atrial node – termed by Spalteholz (1924) as the ***ramus crista terminalis.*** Since most of this branch supplies the atria, it is commonly termed "*main atrial branch*", (Baroldi, 1966). The ***septal rami*** are relatively short and numerous, but usually do not reach the apical septal parts. The largest posterior septal ramus usually supplies the atrioventricular node in up to 80% of hearts, (Hutchinson, 1978).

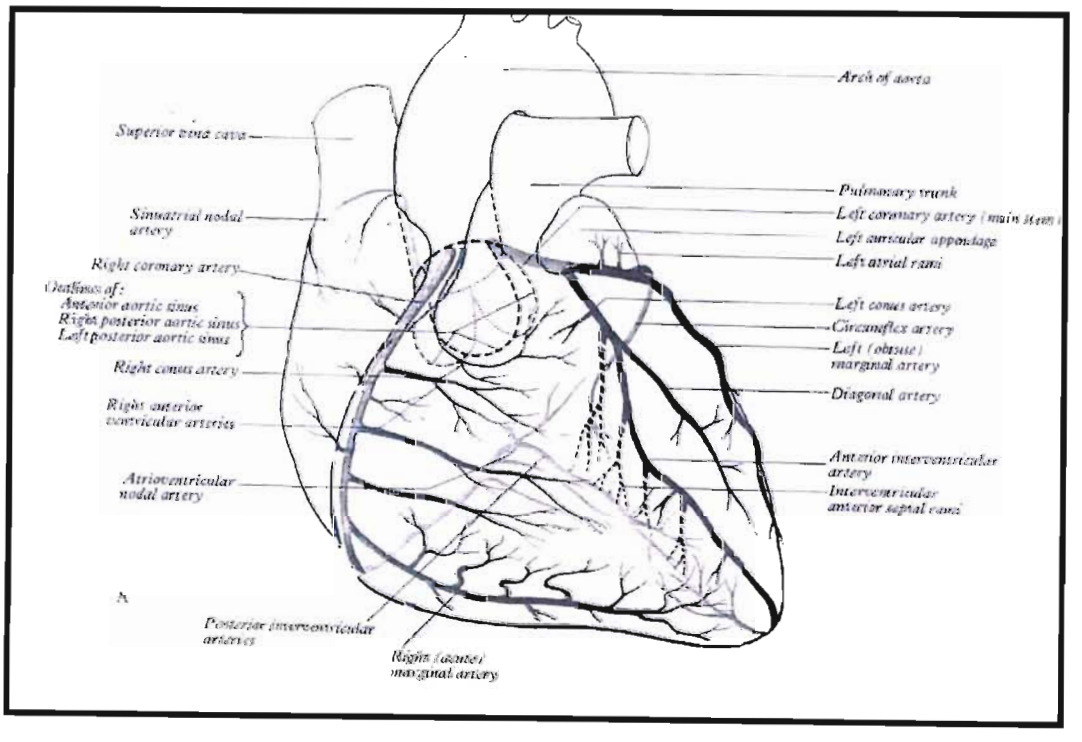


Figure 23: Terminating branches of the right and left systems

(Adapted from Williams et al., 1989)

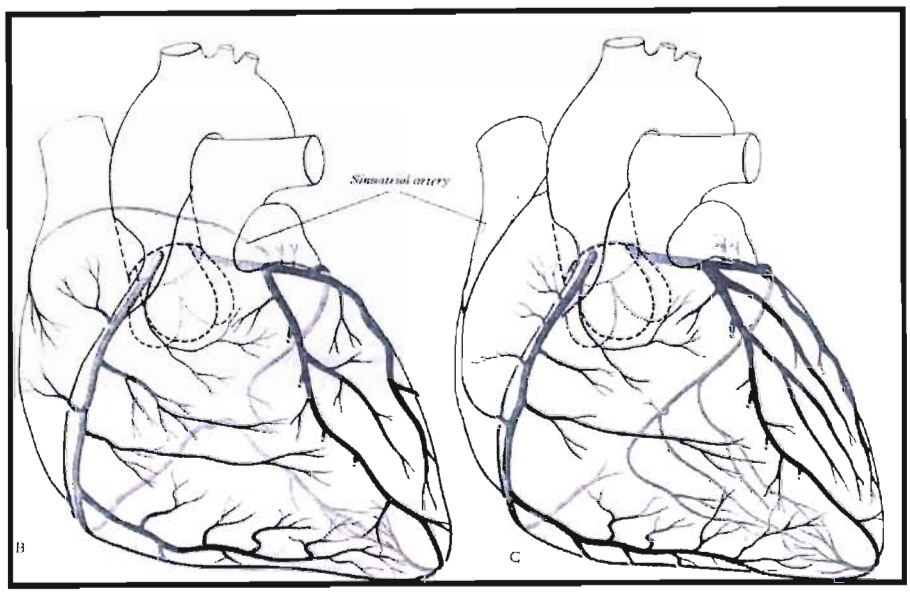


Figure 24: The contributions of the sino-atrial nodal artery

(Adapted from Williams et al., 1989)

2.3.4. THE LEFT CORONARY ARTERY

Origin and Course

The LCA usually arises from the middle portion of the left anterior sinus of Valsalva, just above the level of the free edge of the open aortic cusp and below the sino-tubular junction, Williams et al., 1989; Angelini et al., 1999). It lies between the pulmonary trunk and left atrial appendage in the atrioventricular sulcus. The artery is usually embedded in subepicardial fat and does not appear to produce any branches. Termination of the LCA is usually by bifurcation and sometimes, by trifurcation, (Figure 25).

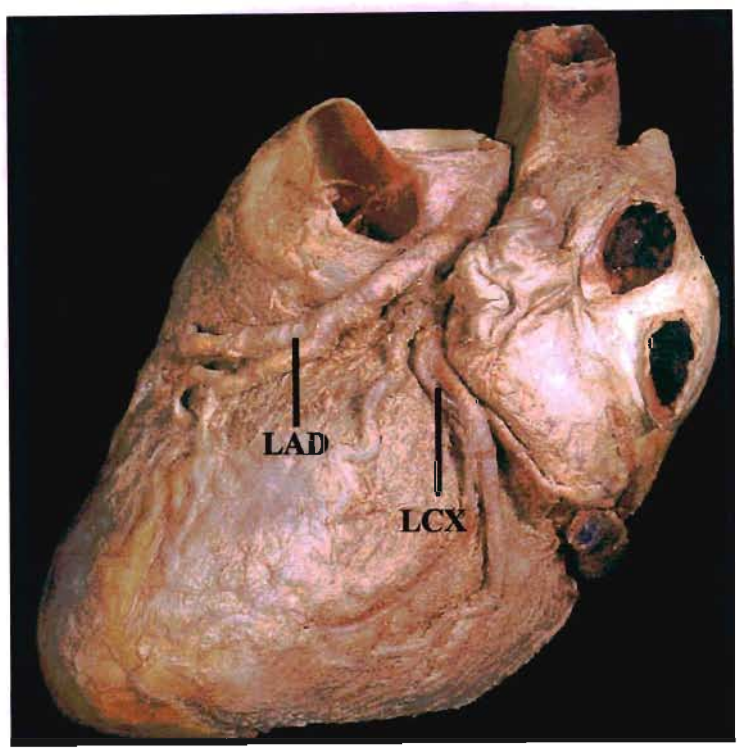


Figure25: The left coronary system showing LAD and LCX arteries

(Adapted from Gosling et al., 2002)

The ***LAD artery*** is commonly described as a continuation of the LCA. It descends obliquely forward and to the left in the anterior inter ventricular sulcus. It is sometimes deeply embedded, or crossed by bridges of myocardium and the great cardiac vein and its tributaries. The LAD artery passes toward the apex where it terminates in some hearts, but in most cases, it continues posteriorly by turning around the apex, where it terminates in an anastomosis with the twigs from the corresponding posterior inter ventricular artery. The LAD artery produces right and left anterior ventricular, septal and posterior rami. The right anterior ventricular rami are small and rarely number more than one or two.

As the LAD artery continues down the anterior surface of the left ventricle, 2 to 9 large left anterior ventricular arteries branch diagonally at acute angles from it. Larger branches reach the rounded left obtuse border. Often, a large branch may arise separately from the left coronary trunk, which then Two to nine large left anterior ventricular arteries branch at acute angles from the LAD to cross diagonally on the anterior surface of the left ventricle. Larger branches reach the rounded left obtuse border.

A small ***left conus artery*** often leaves the LAD near its origin and anastomoses with the corresponding vessels on the right, and with the vasa vasora of the proximal pulmonary trunk and the aorta. The ***anterior septal rami*** leave the LAD almost perpendicularly, passing back and down in the septum to supply the ventral aspect. Small posterior rami supply the posterior aspect from the apex.

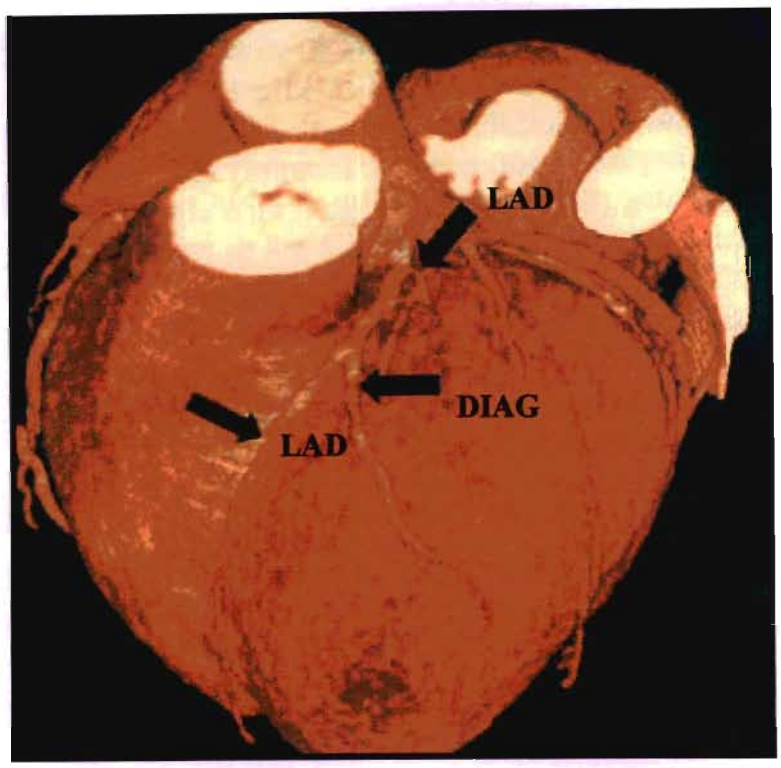


Figure 26: CT showing LAD artery and a diagonal branch

(Adapted from Multi Slice Clinical Data, 2000)

The ***LCX artery,*** (Figures 25 and 26) often in caliber comparable to that of the LAD artery, curves in the left atrioventricular sulcus, (usually overlapped by the left atrial appendage) round the left cardiac border, ending posteriorly at the crux in most hearts (Williams et al., 1989). Sometimes, it continues as a posterior inter ventricular artery. In 90%, a large branch, the ***left marginal artery (obtuse marginal)***, arises perpendicular to it, to ramify over the obtuse margin of the heart. Anterior ventricular branches run parallel to the diagonal branches of the LAD. The posterior inter ventricular branches are smaller and fewer and may be replaced by an inter ventricular continuation of the LCX artery. The rami from the LCX predominantly supply the left atrium.

The ***ramus intermedius,*** (Gorlin, 1976) or ***ramus marginalis,*** (Ochsner and Mills, 1978) is

defined as the intermediate vessel between the first diagonal and first obtuse marginal arteries. Its presence in the left coronary system depicts a trifurcation pattern in the termination of the LCA. It covers a variable extent of the free wall of the left ventricle, posterior to the territories of the vessels between which it emerges.

2.3.5. ANGIOGRAPHIC ANATOMY OF CORONARY ARTERIES

Visualization of the coronary arteries in the clinical set-up requires an appreciation of the anatomy of the coronary arteries as demonstrated on an angiogram. Bearing in mind that a radiographic image is a two dimensional presentation of a three-dimensional structure, proper understanding of the arterial pattern of the coronaries from this is angle is essential to accurate clinical interpretation.

Raphael et al., in 1980, demonstrated by means of corrosion casts, the anatomy of the coronaries as seen from standard arteriographic projections. These casts, when matched by superimposed angiograms, were easily able to define the arterial pattern from a correlative position, (Figures 27 and 28).

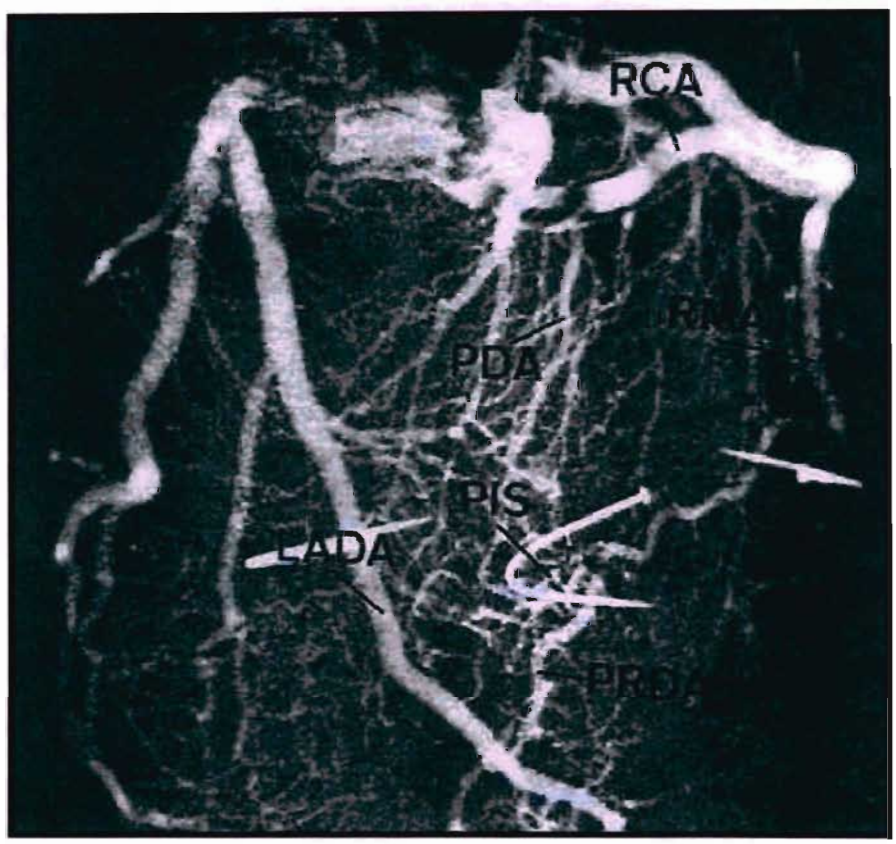


Figure 27: Angiogram of a post-mortem dissection

(Adapted from Nerantzis et al., 1994)

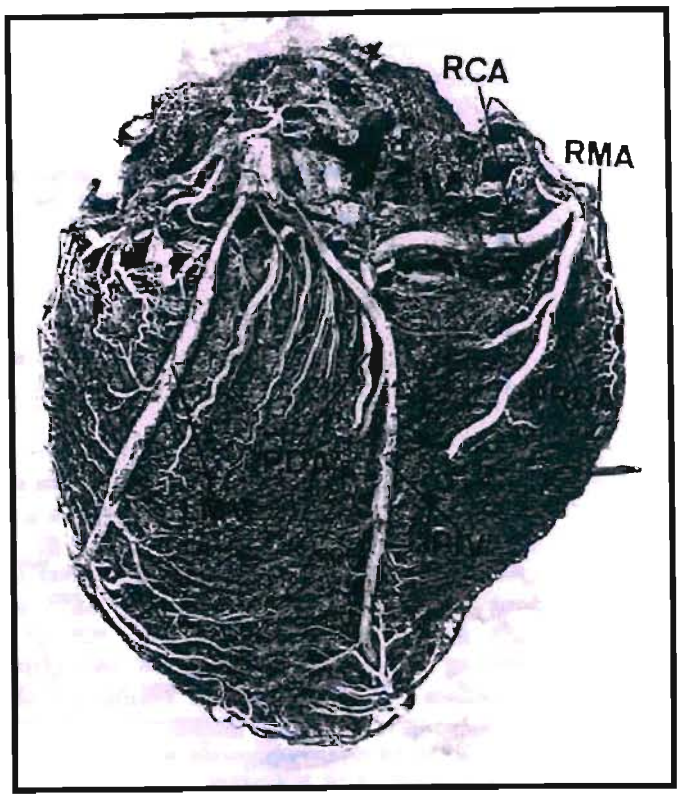


Figure 28: Arterial cast of a post-mortem dissection

(Adapted from Nerantzis et al., 1994)

Observation of the coronary arteries in the lateral projection

Due to the foreshortened appearance of the LCA in this view, any further interpretation of its anatomy may be of no value. The LAD and LCX however are clearly spread out in this view. The LAD courses anteriorly, giving off septal branches or septal perforators that appear to move away from the camera and diagonal branches that appear to move toward the camera, (Raphael et al., 1980). Although the proximal part of the vessel is not too clear, it is much more obvious after the first septal perforator. Characteristically, the LAD may be identified in this view by it being the most anterior vessel, with a large radius curve concave posteriorly, by its curve around the

apex of the heart and by the septal branches that arise from it. The septal branches may be distinguished from the diagonal branches in that they are straighter and run parallel. The diagonals have a characteristic tortuous appearance.

The LCX runs posteriorly in a curve that bulges anteriorly. Foreshortened at first, it becomes more visible as it continues posteriorly. Its termination is somewhat variable but usually gives off a small posterior descending artery as it approaches the crux of the heart. The branches that arise from it project laterally onto the free ventricular wall. Often, these obtuse marginal arteries may distort the view of the LCX. In this instance, the LCX may be recognized by its tighter curve and by its pattern of termination.

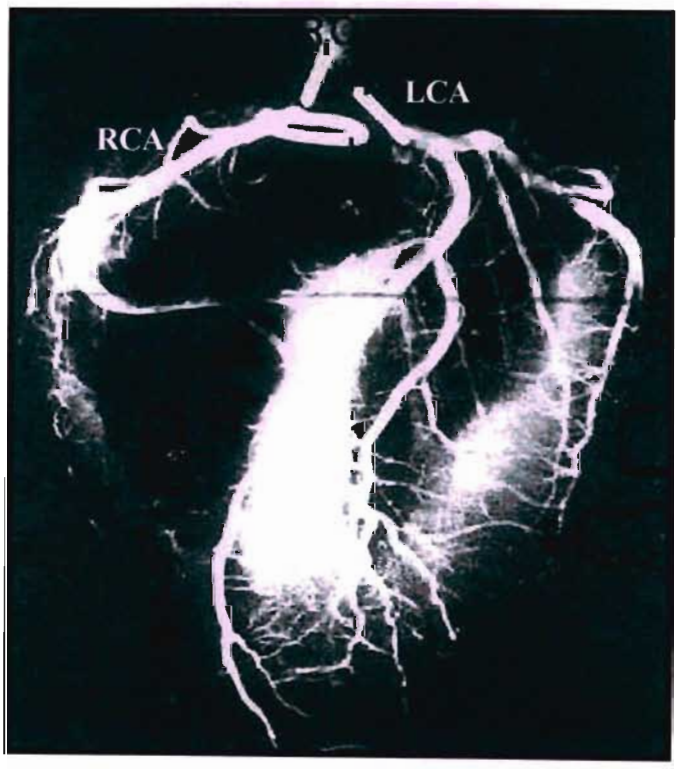


Figure 29: Post-mortem angiographic presentation of the coronaries

(Adapted from Nerantzis et al., 1994)

Observation of the coronary arteries in the left anterior oblique view

Picturing the LAD and LCX in this projection is not optimal. Vessels are often foreshortened and not diagnostically valuable. This view however is useful in distinguishing the diagonals from the septal perforators of the LAD, (Rodriquez and Robbins, 1965), (Figure 29).

In this view however, a more satisfactory picture of the RCA can be achieved. In a steep LAO position, the ostium and proximal RCA can be appreciated. The artery can be followed up until its point of termination beyond the crux as the large posterior descending artery in right dominant hearts. The ventricular branches, although foreshortened, may be recognised as they appear to pass towards the camera. The atrial branch may be seen to pass to the right and backward, with some visualization of the SA nodal branches. It is important to appreciate that in this view, the RCA in the atrio-ventricular groove is clearly demonstrable. In the event of left arterial dominance however, the RCA may appear much smaller, with a short trunk and often terminating in a spray of ventricular branches. In this case, no trunk is seen in the atrio-ventricular groove.

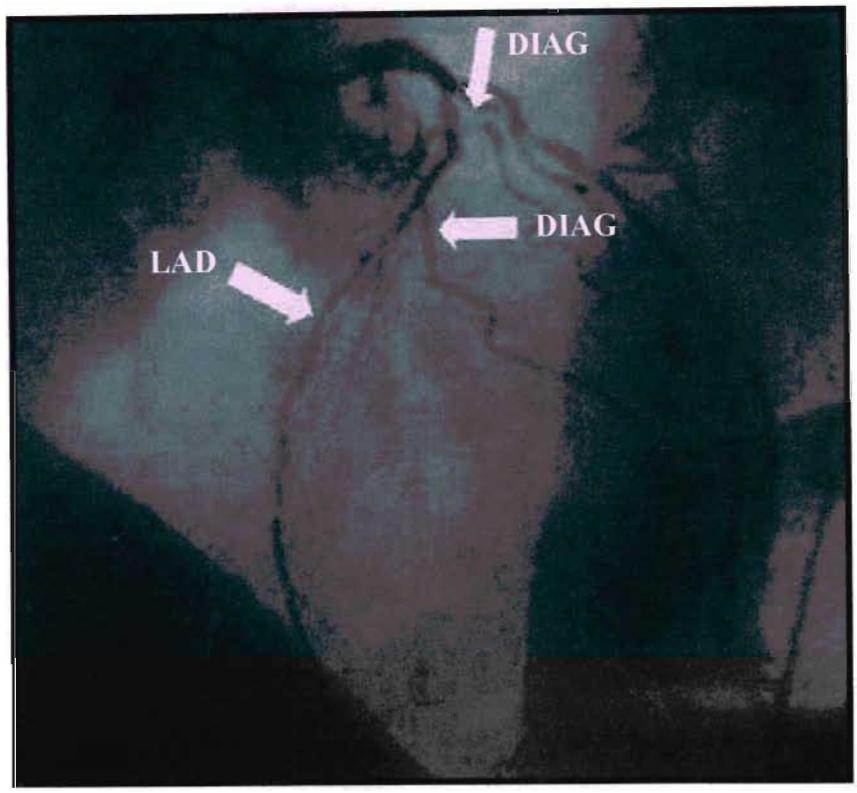


Figure 30: Branches of the left coronary system on a left anterior oblique view

(Adapted from Electro Medica Cardiology, 2000)

Observation of the coronary arteries in the right anterior oblique projection

The LCA main trunk may be identified via the points of origin of the LAD and LCX in the right anterior oblique projection. The LCX has a characteristic straight appearance in this view. A large obtuse marginal branch is often seen passing towards the camera. An intermediate free wall branch of a large obtuse marginal may often overlap the LAD and create a degree of distortion in visualization of the LAD and its branches. The LCX is seen clearly in this view as it continues around the atrio-ventricular groove.

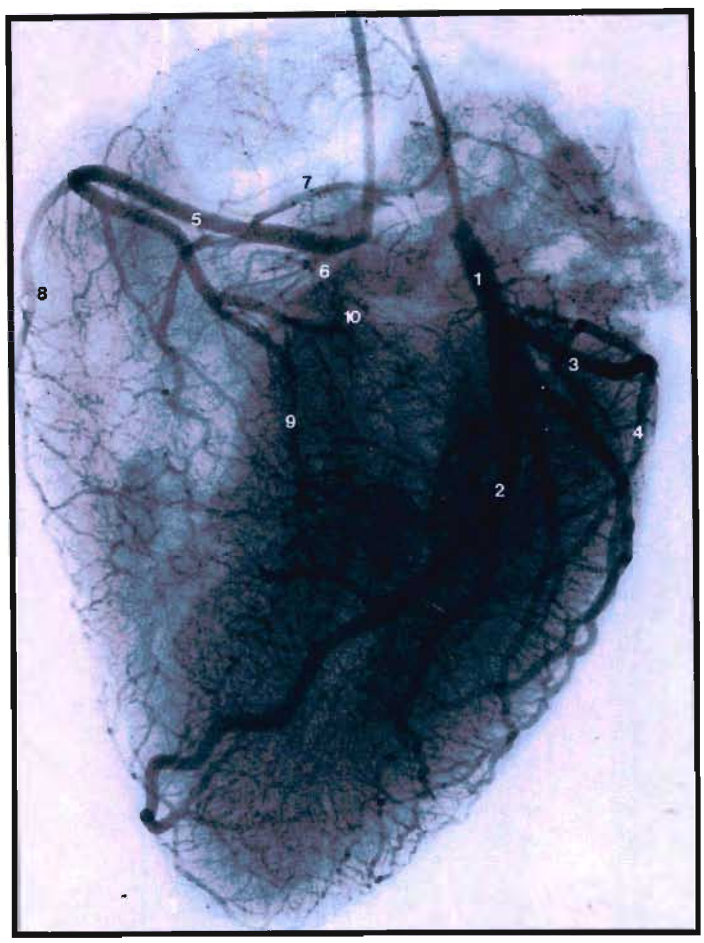


Figure 31: Post mortem angiographic demonstration of the LAD artery and branches

(Adapted from Fareer-Brown, 1977)

The LAD is most clearly observed in this projection. A certain degree of overlapping of the diagonals by the LAD may result when these branches lie in a similar position. This confusion may be limited by applying a cranio-caudal angulation in this view and the origin of the diagonals maybe identified. The LAD may be characterized in this position and further recognized by the straight septal perforators that arise from it and by the curved pathway it takes to reach the

inferior surface of the heart. The RCA is poorly visualized in the right anterior oblique view. Foreshortening of the vessel is extensive and thereby of little diagnostic value. RCA branches however are better seen as they run in the inter-ventricular groove. Visualization however is limited to the posterior inter-ventricular, posterior ventricular and acute marginal branches, provided that overlapping is minimal.

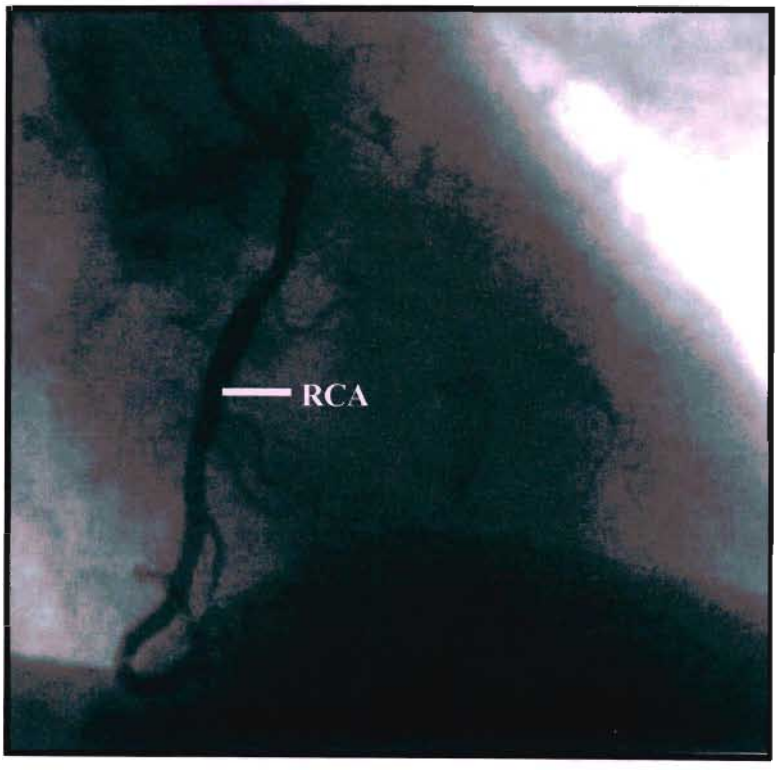


Figure 32: Angiographic view of the RCA and branches

(Adapted from Multi Slice Clinical Data, 2000)

CORONARY ARTERY ANOMALIES

"From under two of the semi-lunar valves of the aorta, which is ere it leaves the heart, arise two branches, sometimes but one, which are bestowed upon the heart. From the curved part of the aorta, arise the subclavian and carotid arteries... By some authors these vessels have been described in a different manner, But I believe their descriptions were for the want of human bodies taken from brutes, for I have never yet seen any variety in these vessels in human bodies, though I have, in the veins nearer the heart...."
William Cheselden, 1795

Whether for want of human bodies or not, the value of such principal observation lies unsurpassed. In as early as 1543, anatomical accounts of coronary artery anomalies were published when Andreas Vesalius demonstrated in one of his famous *"Tabulae Anatomica"*, a right coronary artery originating from the left coronary artery and coursing anterior to the pulmonary trunk. Similarly, Fallopius, in 1562 reported on his finding of a single coronary ostium. And well before any formal description of a coronary system was established, anatomists acknowledged the concept of anatomical variation.

When considering the spectrum of coronary artery anomalies, the difficulty in establishing a somewhat definitive classification is unavoidable. It is therefore best understood using a broad descriptive approach as applied by Angelini et al., (1999). His classification of coronary artery anomalies is based upon the origin and course of each major branch, its "intrinsic" anatomy and pattern of termination.

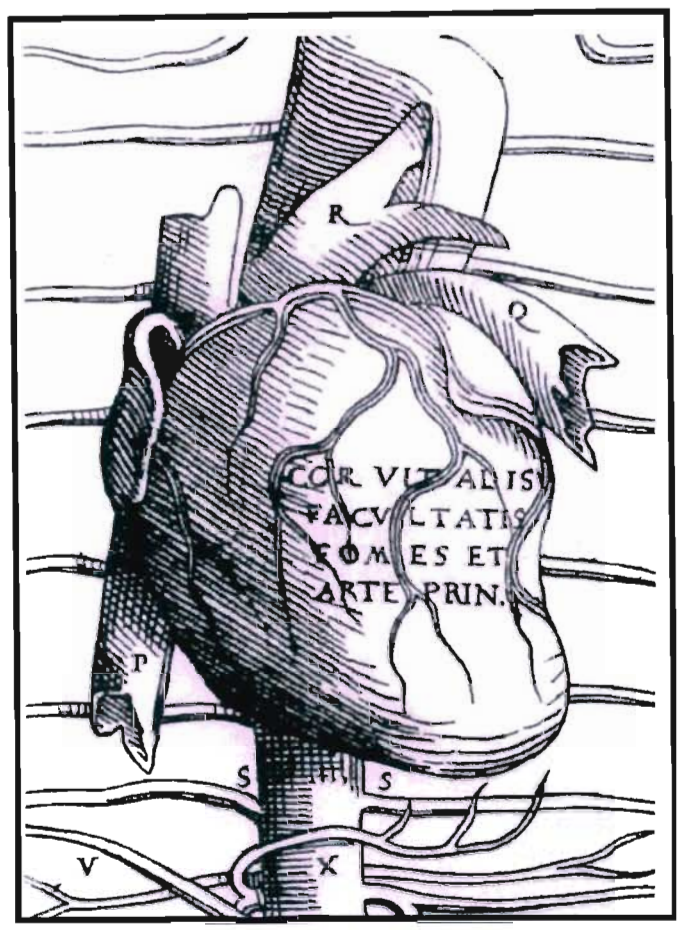


Figure 33: The coronary arteries from the "Tabulae Anatomica"

(From sketches of Vesalius, 1543)

2.4.1 ANOMALIES OF ORIGINATION AND COURSE

Absent Left Main Trunk

In the absence of the left main trunk, the LAD and LCX usually originate from separate ostia or as immediate branches from a common sinus opening.

The reported incidence ranges between 1.0% - 0.6%, (Vlodaver, 1972). The left main trunk is also considered to be absent when LAD or LCA originate ectopically, outside the left aortic sinus. In addition, when both LAD and LCA originate ectopically, the ectopic left main trunk may be present, if the LAD and LCA share a proximal conjoined stem of any length.

Coronary Ostial Location

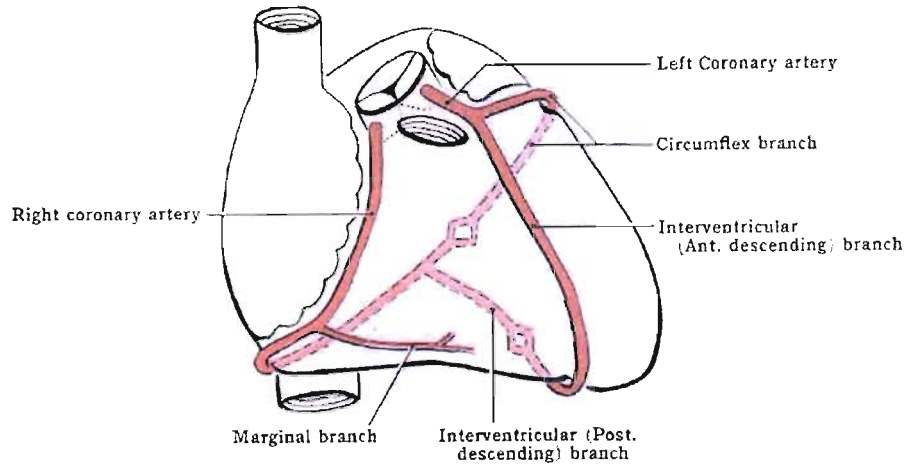
Anomalies may occur where the ostium lies within aortic root, near a proper aortic sinus or outside a normal aortic sinus. In the first occurrence, the ostial position may vary between a high, low or commissural position. A commissural ostium is defined as an ostium located less than 5mm away from an aortic valve commissure (or apex of the intercuspal triangle). In these cases, high origination tends to overlap with ectopic origination from the ascending aorta, therefore, ostium located less than 1cm above normal will be included in this group.

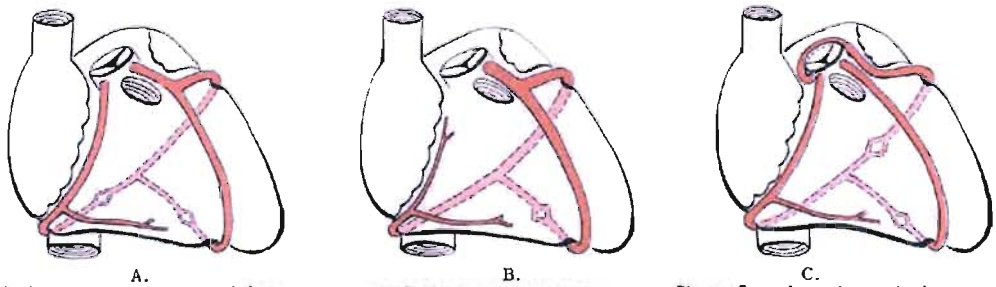
When the ostium is located outside the normal position, the ostium may be located in any of the following cardiac regions - right posterior aortic sinus, the ascending aorta, the left ventricle, the right ventricle or the pulmonary artery. The LCA, LAD and LCX may each in turn originate from the posterior facing sinus and the RCA may begin at the anterior right facing sinus. An ectopic location may occur outside facing sinuses of any coronary artery: anterior left sinus, pulmonary trunk, pulmonary branch, aortic arch, brachiocephalic artery, right carotid artery, internal mammary, bronchial artery, subclavian artery and even the descending thoracic aorta. This anomaly is usually considered insignificant and there has to date been only one reported case where the anomaly became clinically significant, (Angelini et al., 1999).

Location outside the aortic root in the ascending aorta usually involves the anterior, left surface of the aorta. The site ranges from origin just above the sino-tubular junction to the origin of the brachiocephalic artery (several centimeters above the aortic valve). The RCA is the most frequently ectopic artery, but the LCA (or separately LAD and LCX arteries) may also originate ectopically. In addition, reports of the presence of a single coronary artery have been documented, (Smith, 1950).

There are very few reports of the RCA originating from an otherwise normal left ventricle, just below a congenitally insufficient and stenotic aortic valve in adults. Angelini et al., (1999) propose that this should be described as "origination of a nutrient coronary vessel" providing flow to the myocardium from the left ventricle. This definition excludes unrelated anomalies such as aorto-left ventricular tunnel and sinusoidal coronary collaterals.

Origination from the right ventricle is usually a misnomer for a complex congenital heart condition in which pulmonary atresia and an intact ventricular septum co-exist with multiple right ventricular sinusoids. These functionally drain into neighboring coronary vessels during systole, but during diastole, they drain coronary blood into the right ventricle. This has never been observed in an otherwise normal heart.





A.
Left coronary artery supplying part of right coronary artery territory.

B.
A single coronary artery.

C.
Circumflex branch springing from right aortic sinus.

Figure 34: Anomalous patterns of origin of the main coronary arteries

(Adapted from Moore, 1992)

Anomalous origin of a coronary artery from the pulmonary artery

Cases of coronary arteries arising from the pulmonary artery are most frequently encountered in the pediatric group and very rarely seen in the adult. Although cases of the RCA, LCA, even both coronary arteries originating from the pulmonary artery, the anomalous origin of the LCA appears to be the most common, (Wald et al., 1971). Persistence of such an anomaly has its origin in the embryonic development of these vessels.

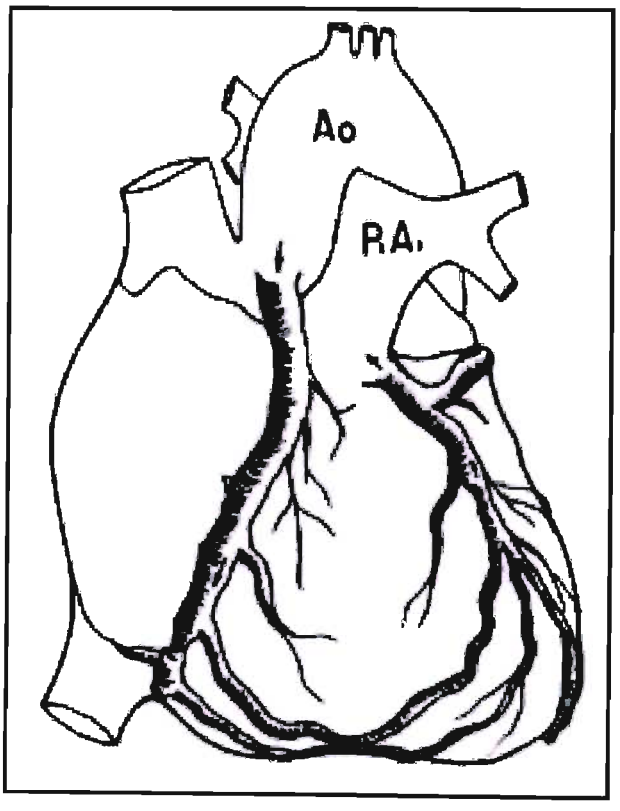


Figure 34: Anomalous origin of the LCA from the pulmonary artery

(Friedman and Child, 1994)

Narchi, (1999) proposes 2 possibilities. In the first, an abnormal septation of the cono-truncus during the development of the pulmonary artery and the aorta may occur. The second proposes an abnormal persistence of the pulmonary buds with an involution of the aortic buds.

Anatomically in this anomaly, the left coronary artery arises from the pulmonary artery, while the right normally arises from the anterior aortic sinus. In response to the physiological stresses that ensue, the anomalous coronary artery dilates, and collateral vessels from the normal right system develop and increase in relation to compensatory demands. However, since the pressure in the anomalous left coronary system is lower than the right, right to left shunting with retrograde flow into the pulmonary artery is likely to create a coronary steal syndrome. The degree of collateralization determines the degree of myocardial ischemia and if well developed, individuals may reach adulthood without signs of myocardial compromise.

Anomalous Pathways

As with any anomalous occurrence, it is safe to assume that the path hence taken by the artery is almost entirely re-routed. According, these commonly occurring pathways are described.

When a coronary artery arises from the opposite facing aortic sinus , the vessel's proximal course is abnormal, as the artery connects with the contra-lateral in situ vascular network. The artery may originate directly from the aorta or arise jointly by way of a mixed trunk (Vithoori et al., 1980) with the coronary artery that normally originates from that cusp. When the anomalous artery has an independent ostium, it is generally adjacent to the ostium normally arising from that sinus. The independent ostium usually has a slit like appearance. After arising from a contra lateral sinus, the

artery can take one of 5 or 6 pathways, each with a peculiar, but consistent topographic anatomy (Wang et al., 1997; Liberthson, 1989).

When the RCA arises from left anterior sinus the anomalous course may follow the posterior AV groove in which case it appears retro-cardiac. It may pass retro aortically, between the aorta and pulmonary artery. An intraseptal course may be taken or it may pass anterior to pulmonary outflow tract in a pre-cardiac route. When the retro-cardiac path is taken, the vessel proceeds behind the tricuspid and mitral valves at the posterior atrioventricular groove.

When the anomalous RCA takes this path, it constitutes the terminal branch of a dominant LCX artery, which reaches the right ventricular infundibulum. This condition may be classified as a single LCA, since no RCA originates from the right sinus. However, Angelini et al., (1999) comment on this as being an erroneous suggestion, implying that the individual has no RCA. They re-reiterate that in reality, the RCA is perfectly developed, although it has an anomalous pattern.

When the LCX artery originates from the right sinus with the RCA, and has a posterior course behind the atrioventricular valves, the anomalous trunk that extends to the crux of the heart is indeed a mixed trunk, not a simple RCA (Sagkan et al., 1994). Similarly, the coronary artery that arises from the left cusp is indeed the LAD, not a true LCA and therefore, the left main trunk is absent. In such cases, the condition should be known as "absent left main trunk with anomalous origination of the LCX artery from the distal RCA", (Angelini et al., 1999). Similarly, the entire LCA may arise from the right anterior sinus and follow a posterior course. In such a case, the proximal trunk is a mixed one, not a single RCA. The trunk distal to the crux of the heart is

considered an LCA that gives rise first to the circumflex - obtuse marginal and then to the LAD systems. Although this condition is known as single RCA, there is also indeed a well-developed LCA system, albeit of anomalous origin.

When a vessel assumes a retro-aortic path, it is most commonly associated with origination of coronary artery from the opposite sinus. However, it specifically involves the LCX artery originating from the right sinus. This occurs in 0.1% to 0.9% (Baltaxe and Wixon, 1977, Bjork, 1966) and includes cases where the LCX has separate origin adjacent to RCA and those in which it has a joint origin with the RCA. This course continues just next to the posterior wall of the aorta in the transverse sinus sulcus and eventually reaches its normal location in the atrioventricular groove.

The same retro-aortic path although less frequently seen, occurs when the RCA originates from the left sinus or when the entire LCA originates from the right sinus. However, this path is not seen when LAD has an isolated ectopic origin.

The pre-aortic course runs between the aorta and pulmonary artery. This terminology refers to the fact that an anomalous RCA or LAD or LCA (never LCX) crosses the aorto-pulmonary septum or space, sub-epicardially.

When an intra-septal course is taken, it is mainly located inside the upper, anterior inter ventricular septum. This path is mostly intra-mural (intra-myocardial). The intra-myocardial trunk often produces 1-2 septal perforators that indicate the trans-septal portion of the arterial trunk.
In the pre-cardiac path, the route is characterized by sub-epicardial location on the anterior wall of

the infundibulum. This path may be taken by all the vessels except the LCX artery. It is commonly seen in patients with Tetralogy of Fallot in which the LAD has ectopic origin from the right sinus and courses anterior to the hypoplastic, stenotic infundibulum. The anomalous pre-cardiac vessel frequently gives rise to infundibular, but never septal branches.

Single Coronary Artery

This pattern exists when a single aortic ostium provides for all of the coronary blood flow, (Allen and Snider, 1966; Glover et al., 1982; Hillestad and Eie, 1971; Sharbaugh and White, 1974). The reported incidence of single coronary artery is approximately 0.024%, (Shirani and Roberts, 1993).

In most cases of a single left coronary artery, detailed anatomic study reveals the presence of a small ostium in the right cusp that leads to a conal branch. In cases of single right coronary artery, no coronary, however small, is seen to originate from the left cusp. In considering the classification, indeed, the anomalous vessel's single proximal trunk should not be designated as RCA or LCA according to the cusp of origin. Rather, it should be considered a mixed trunk, which gives rise to left and right branches, identified according to their respective areas of distribution. The criteria for classification of a single coronary artery may be based on any of the following sinuses of origin - right or left anterior, posterior or ectopic, in which case it may arise from the ascending aorta, pulmonary artery, or a systemic artery. The vessel may follow any of the paths described previously.

2.4.2. ANOMALIES OF INTRINSIC CORONARY ARTERY ANATOMY

The classification of anomalies into the category of "intrinsic arterial anatomy" appears to combine a variety of occurrences that include those with a clinical association and those that are of anatomical interest.

The presence of *congenital ostial stenosis or atresia* in the LCA, LAD, RCA or LCX artery may be included in this group, secondary to which coronary ostial dimple, coronary ectasia or aneurysm may be noted. Others within this group include, *absent coronary artery, coronary hypoplasia, intramural coronary arteries* or the presence of *muscular bridges.* In some cases, a *subendocardial* coronary course may persist or *coronary crossing* may occur.

With regards to the LAD and its branching patterns, an anomalous origination of posterior descending artery from this main branch or one of its septal branches may occur. In some cases, the LAD may be "absent" due to the persistence of a split pattern either by a bifid arrangement or a main branch with an equal sized septal branch, (Gorlin, 1976).

Congenital ostial stenosis or atresia

The literature contains reports of coronary arteries that have become stenosed because of a membrane or fibrotic ridge located at or near the aortic orifice of an otherwise normal heart. This condition may be associated with tangential origin of the coronary artery. In extreme cases, LAD and RCA may be affected. This anomaly characteristically presents with one or more of the following anatomical features:

(a) Proximal occluded artery has a larger diameter than the intermediate segments,

(b) One or more collateral connections are present,

(c) Proximal anatomy of the occluded vessel has a blind pouch that adjoins an aortic sinus,

(d) The site of ostial atresia is sometimes recognizable as a dimple in the related aortic sinus, (Angelini et al., 1999).

The term *coronary ostial dimple* has been used to describe an anomalous finding of a depression in the wall of the aorta at the sinus deprived of functional coronary origination and which has embryogenetic implications. *Coronary ectasia* on the other hand, is defined as localized dilations in the otherwise normal sized coronary artery. In terms of its anatomical characteristics, ectasia may be divided into primary and secondary classes. In primary ectasia, a localized segment has disproportionately larger diameter in comparison to neighboring segments. Morphologically, the increase in diameter is greater than 50% with respect to normal. Secondary ectasia is characterized by diffuse coronary dilation. In such instances, the coronary artery diameter is actually appropriate for the increased flow and is excessive only with regard to a nutrient myocardial flow, in which case, the coronary flow velocity is actually increased.

Absent coronary artery

Although the literature reports rare occurrences where an absent coronary artery was seen to manifest clinically, the absent coronary artery has never been established as a specific congenital entity, (Allwork, 1987).

Coronary Crossing

Epicardial coronary arteries are not known to cross one another. Only a few angiographic reports describe crossing of adjacent branches, usually at the sub-epicardial level. There are no anatomic reports to corroborate these angiographic appearances. The crossing arteries are usually the obtuse marginal branches. This anomalous arrangement is known to affect secondary branches. Rarely, they may give rise to clinical difficulties during identification of a branch to be grafted.

Absent LAD

An "absent" LAD is referred to when the normal morphology is not present. Such a pattern may be the result of the proximal anterior descending artery or a supernumerary aortic ostium giving rise to a single large first septal branch that produces most of the secondary branches, leaving a small, or absent mid distal sub-epicardial LAD. In another instance, the proximal LAD may split into two smaller branches that run parallel to each other along the anterior inter-ventricular area. Occasionally, a large diagonal artery may originate high from the proximal LAD, run parallel to it and give off secondary branches in which case a residual LAD with a limited territory of supply remains. Or, a highly dominant RCA may give rise to most of the branches and thereby reduce the contribution of the LAD.

The intra-mural LAD

The LAD may pursue a sub-endocardial course after penetrating myocardial layers. In this way, LAD may even reach the anterior portion of the right ventricular cavity.

Anomalous patterns of the posterior descending artery

Occasionally, the posterior descending artery comprises two segments. One originates normally from the RCA, near the crux in the upper portion of the inter-ventricular groove. The other originates from the mid RCA, close to the acute margin of the heart and reaches the distal posterior portion of the inter-ventricular groove.

In other instances, LAD or LCX artery may supply part or all, of the posterior descending artery territory, causing it to appear "split". Although this anomaly is an anatomical curiosity, it may become clinically significant, should there be a need for surgical manipulation of the posterior descending artery.

2.4.3 ANOMALIES OF TERMINATION

Termination patterns often depict some pattern of anastomotic connection or collateralization. A pattern subject to an anomalous characterisation, may result from inadequate arteriolar or capillary ramifications or fistulous formations.

Fistula's may form from any of the branches, usually the RCA, LCA or conus artery and distribute to any of the following cardiac areas – the right ventricle, right atrium, coronary sinus, superior vena cava, pulmonary artery, pulmonary vein, left atrium or left ventricle.

Coronary Fistulae

Occasionally, the arterioles that arise from the continuous tapering of the coronary arteries maintain small communications with the sinusoidal trabecular spaces. A noticeable

communication between a coronary artery and a cardiac cavity, or a coronary artery and any segment of systemic or pulmonary circulation is termed a "coronary fistula".

Early literature reports, (Baroldi, 1966), indicate two types of communications that exist under normal conditions in the human heart and which serve as alternatives to normal drainage into the coronary venous system. These are known as the a*rterio-sinusoidal vessels* or a*rterio-luminal vessels* and the *Thebesian veins* which are direct communications between a coronary vein and the cardiac cavity.

Fistulous-coronary connections on the other hand, usually involve structures that adjoin coronary arteries, like the veins and cardiac chambers. Less frequently, in otherwise normal hearts, a coronary fistula may drain into an extra-cardiac structure such as the pulmonary artery and its main branches or the superior vena cava. The pulmonary connections are small, numerous and not clinically significant, Angelini et al., 1999).

THE CORONARY COLLATERALS

"Anastomoses in the heart are universal and abundant"

Louis Gross, 1921

That inter-arterial anastomoses between branches of the coronary arteries exists in the ischemic heart, has been well established, (Fulton, 1965). However, their existence and functional significance in the normal heart, has in the past been a topic of repeated debate. Today, the general acceptance of their existence has been embraced by interventional cardiologic advances, which exploit the auxiliary capacity of the collateral circulation.

2.5.1. HISTORICAL OVERVIEW

In bringing into question the existence of the collateral circulation of the heart, Thebesius, Haller, Morgagni and Senac in the 18th century established by means of careful dissection, the anatomy of the inter-arterial connections. However, in the mid to late 1800's, their existence was denied by Hyrtl (1855) and Henle (1866). Based on ligation experiments in dogs, Conheim and Von Schulthess-Rechberg (1881) concluded that the coronary arteries were indeed "end arteries" and that if any communication existed, it was only at a capillary level. Although the findings of these researchers were "confirmed " by others, the body of opposition increased, (Fulton, 1965).

By the beginning of the 20th century, the development and improvement of injection methods gave rise to investigations that were able to demonstrate anastomoses by means of stereo-

arteriography. At the same time, using a much more penetrating injection method, Spalteholz (1907) presented a detailed account of the coronary anastomoses. Gross (1921) confirmed and re-enforced these findings by means of radiographic and photographic demonstrations, (Figure 36). His observations were based upon a study of 100 "normal" hearts, the findings of which were confirmed by Crainicianu in 1922 and Campbell in 1929. Gross (1921) concluded, that anastomoses existed between right and left coronary arteries both in their capillary and pre-capillary distribution. That these anastomoses in turn existed between the branches of each coronary artery and in addition, connections between the coronary arteries and vessels from the adjacent and attached organs were shown to occur. In short, he pronounced that anastomoses in the heart were "universal and abundant".

As interest in the use of injection methods for the study of coronary artery anatomy grew, Schlesinger (1938) employed the technique of using lead-agar injection, radiography (after the heart had been exposed) and dissection. This investigative technique grew in popularity, favored by its simplicity, (Fulton, 1965) and yielded extensive research in the investigation of coronary artery disease. It is interesting to note though, that Schelsinger's statement proclaiming the coronary arteries to be true "Conheim end-arteries", without anastomotic connections, would follow the dynamic statements of Gross (1921), Spalteholz (1907) and others, demonstrating otherwise.

In 1952, Schlesinger's associates, after having investigated 1000 hearts maintained the same conclusion. Fulton, (1965) questioned the relationship between the "Schlesinger-method" of investigation and the results obtained, (Figures 37 and 38). In his challenging statement, Fulton offers the frank acknowledgment by Blumgart et al., (1950) that the limitations of the

"Schelsinger-method" be understood, when its results are being interpreted. In 1965 however, Fulton presented his classic description of the morphology and morphometry of the coronary collaterals.

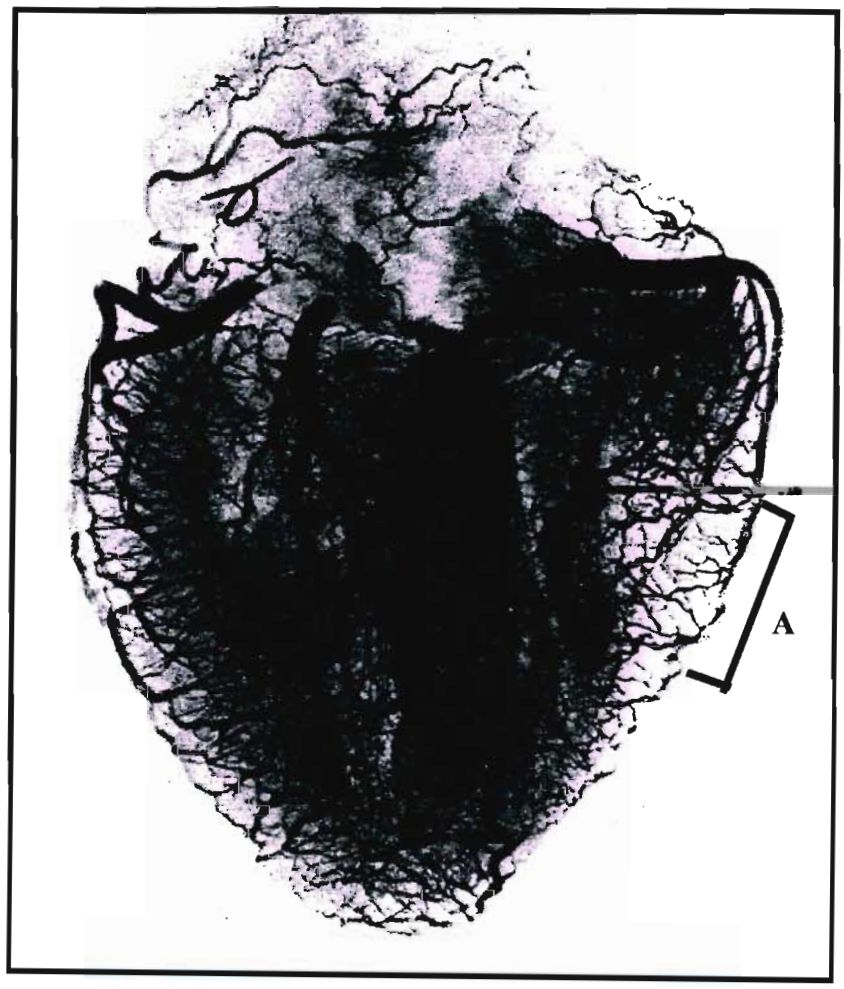


Figure 36: Angiographic demonstration of inter-arterial anastomoses

(Adapted from Gross, 1921)

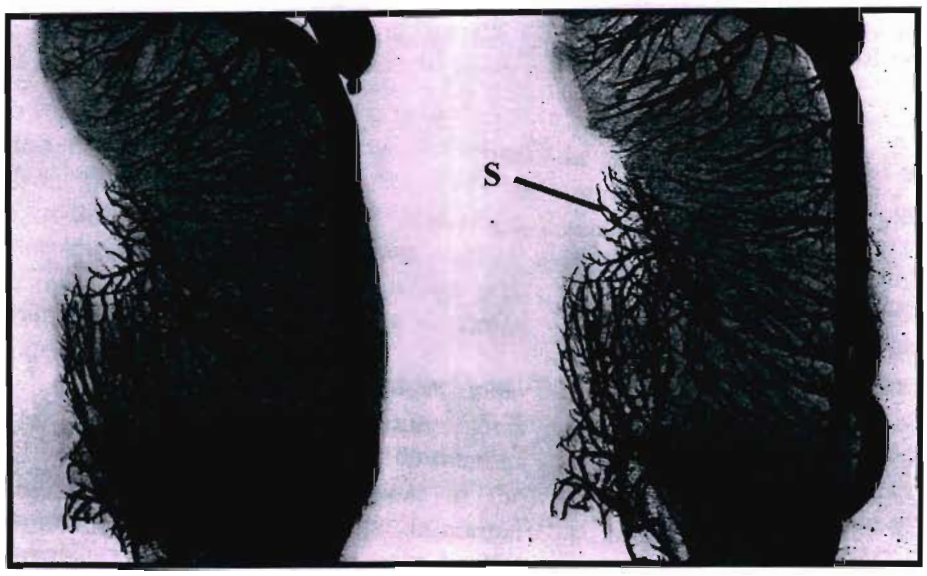


Figure 37: Septal arterial branches and Inter-septal anastomoses

(Adapted from Fulton, 1965)

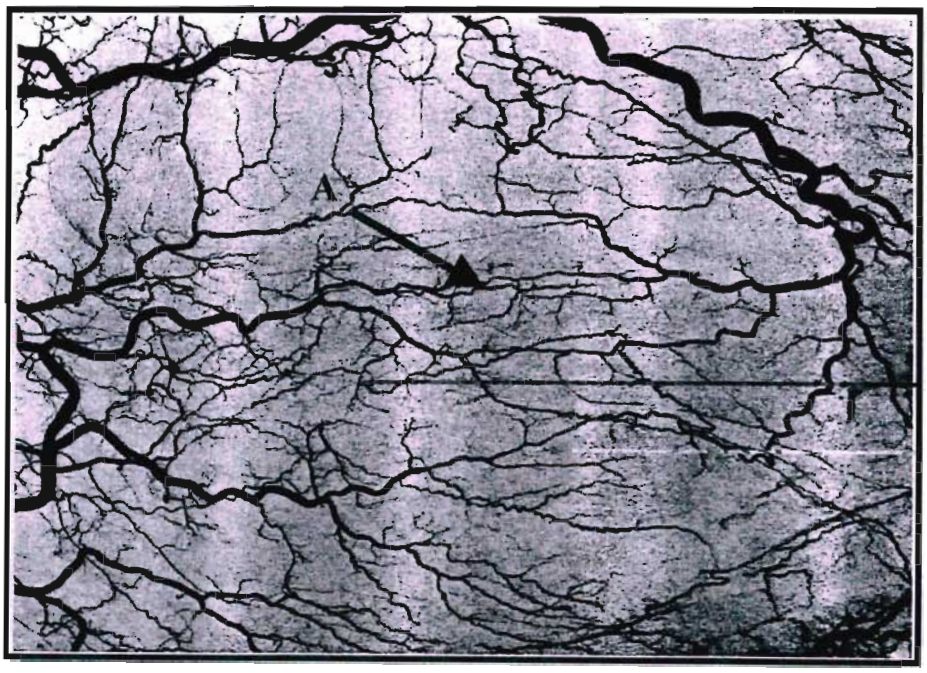


Figure 38: Magnified image of fine inter-arterial connections

(Adapted from Fulton, 1965)

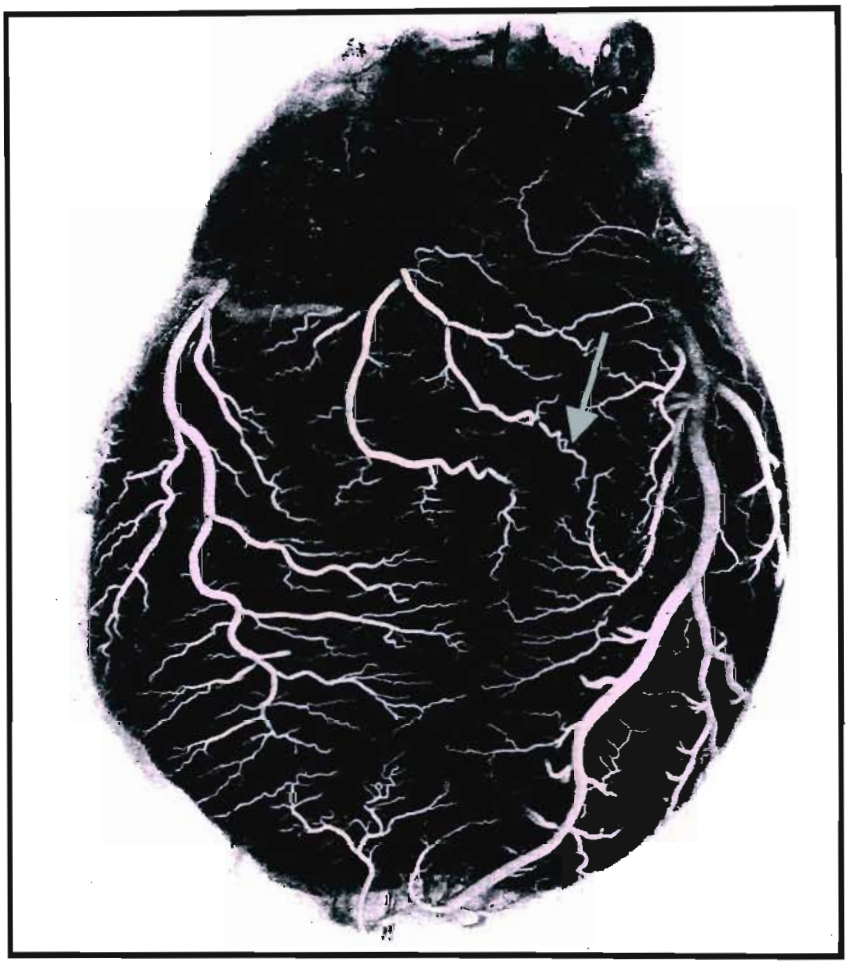


Figure 39: Formation of an intrinsic anastomotic pattern

(Adapted from Gross, 1921)

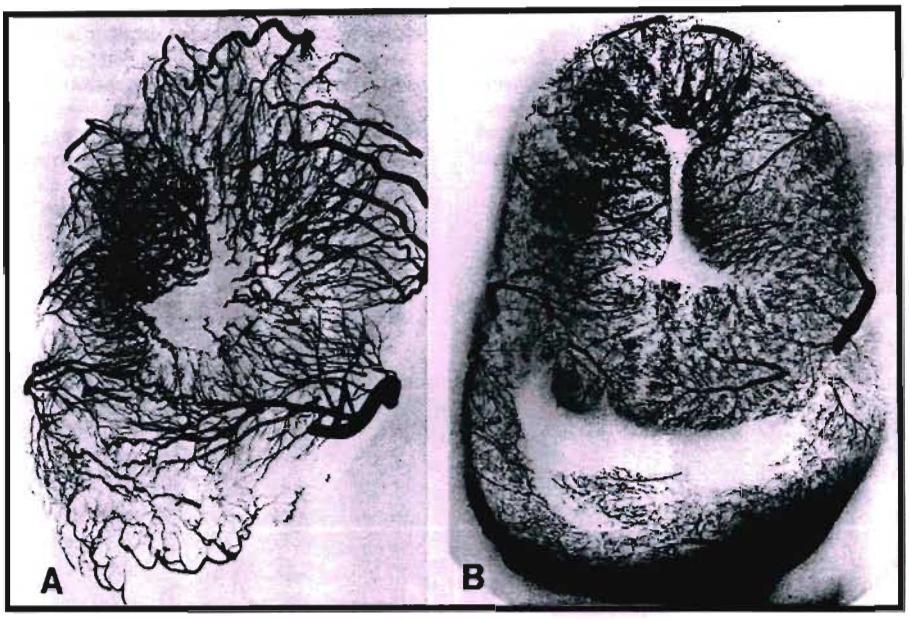


Figure 40: Detailed demonstration of deep collaterals

(Adapted from Fulton, 1965)

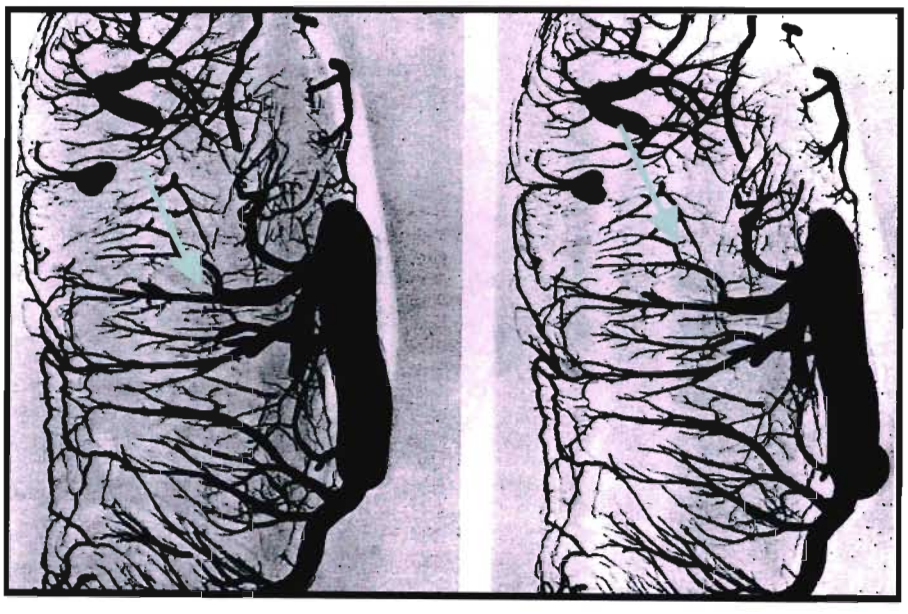


Figure 41: Detailed demonstration of deep septal collaterals

(Adapted from Fulton, 1965)

2.5.2. THE ANATOMY OF THE CORONARY COLLATERALS

A review of the literature on the anatomy of the coronary collaterals highlights significant contributions by Gross, (1921); James, 1961; Fulton, (1965) and Paster, (1977).

Morphometry and Classification

Fulton's, (1965) description of his findings reflects the integral statement by Gross, (1921), that " the anastomoses in the heart are universal and abundant"

He describes atrial anastomoses as an arterial network of fine vessels that exist between the left atrial wall and atrial appendage. These communications exist with less constancy on the right side. The vessels range from 20 to 100 microns in diameter and are abundant in the inter-atrial septum.

Ventricular anastomoses exists in the anterior wall of the right ventricle, which is often found to be the site for extensive, small-scale connections between RCA, LAD and conus artery. Similar anastomoses can be found in the posterior wall of the right ventricle, but less frequently, due to wide variation in the distribution of major coronary branches, (Fulton, 1965), (Figures 40 and 41).

Anastomoses have also been described on the epicardial surface of the right ventricle, near the apex, although these superficial anastomoses are seldom seen at other sites of this territory and rarely on the left ventricle, except at the apex, (James, 1961).

The superficial anastomoses at the apex are commonly made up of 1-2 connections of 50-300 microns in diameter. They are seen to connect the terminal branches of LAD, right marginal artery and PDA. Deeper communications link the superficial arteries with sub-endocardial plexus of the right and left ventricles at the apex. Communications in the atrio-ventricular groove, of dissectible size in ventricular hypertrophy and coronary artery disease, have been described between right and left coronary arteries, (Gross, 1921 and Fulton, 1965); (Figures 37 and 38).

Deep ventricular anastomoses are known to develop in the inter-ventricular septum. The communicating arteries tend to follow the general direction of the septal branches of the LAD. They have been noted to sweep backwards and slightly downwards as they traverse the septum, whereas those located just under the endocardium of the left ventricle, run at right angles to this direction, (Fulton, 1965).

The areas of anastomotic communication in the inter-ventricular septum depend upon distribution of coronary arteries supplying it, and may show considerable variation. However, septal anastomoses are numerous and measure up to 300 microns in diameter. In ischemic hearts, these anastomoses are said to have the potential to enlarge to "tremendous dimensions", (Fulton, 1965).

A network of channels in the deeper zone of the left ventricle forms a sub-endocardial plexus. The collaterals in this region have measured between 100-300 microns. The vessels tend to run within the substance of the trabeculae carnae. To some extent, the distinctness of the plexus may depend on the prominence of these muscular ridges.

The internal network is supplied by branches with an average 200 microns in diameter. They are said to arise from the epicardial branches and run through the ventricular wall at right angles to

the surface, (Gross, 1921). In ischemic situations, the vessels have been observed to be large and the thinness of the ventricular wall at that stage has been noted to favor the extensive communications from the main coronary artery branches, (Paster, 1977).

The sub-endocardial plexus of the right ventricle is less prominent than that of the left. The thinness of the walls makes the distinction between deep and superficial divisions less visible. However, in the hypertrophied state, the vessels of the sub-endocardial plexus is said to increase in size, forming a clearer separation. In this way, they present a pattern similar to that of the left ventricle.

An important contribution to the sub-endocardial plexus of the right ventricle may also be made by septal branches that stem from the LAD artery. Fulton, (1965) confirmed that one of these branches crosses the moderator band (as illustrated by Campbell in 1929) and provides an important contribution to the right ventricle during ischemic damage to the chamber.

The papillary muscles are supplied in a similar fashion as the trabeculae carnae. These vessels, frequently found in the base of the papillary muscles measure up to 300 microns. Finer communications have also been frequently found, (Fulton, 1965). Intra-mural communications in the left ventricle, other than septal vessels and those of the sub-endocardial plexus are not frequently found and are almost never of large caliber. In Fulton's, (1965) recordings, they have been reported to be no less than 100 microns.

Fulton's presentation and re-enforcement of the morphology of the coronary collaterals inspired the works of a few others. In 1961, James published a detailed general description of the

topographic anatomy of the coronary collaterals in an effort to enhance the appealing concept of clinical manipulation of these vessels.

In another significant contribution, James, (1961) defined coronary anastomoses into arbitrary divisions of non-exclusive, overlapping categories. However, each division was intended to be separately useful, " relative to certain aspects of the pathogenesis of ischemic heart disease and the pathophysiology of the coronary collateral circulation".

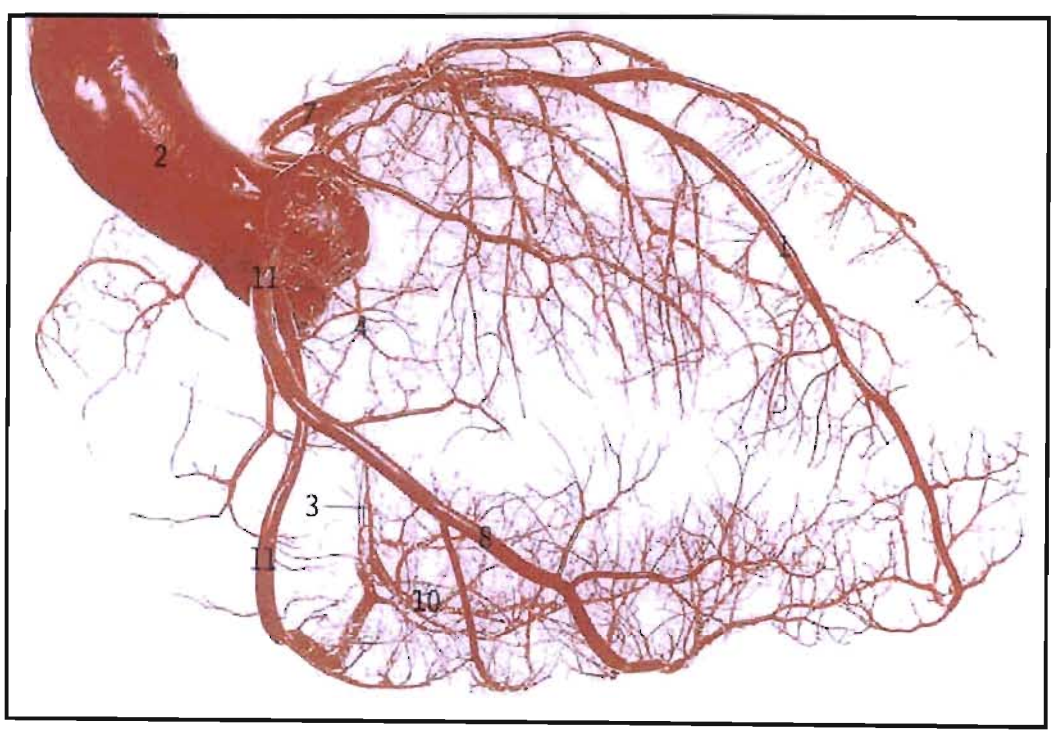


Figure 42: Arterial cast of the coronaries

(Adapted from Abrahams et al., 1998)

2.5.3. CLASSIFICATION OF CORONARY ARTERIAL ANASTOMOSES

Intra-coronary anastomoses

These are represented by connections between one part of the coronary artery to another segment of the same artery. Communications are usually short and several millimeters in length. Functionally, they form effective "bridging" at points of occlusion, (Figure 42).

Intra-coronary anastomoses are generally, expanded vasa vasora that form a cuff around the narrowing vessel. This often creates a "star-burst" appearance on arteriograms. In other instances, these connections may occur between arterial branches, rather than vasa vasora.

Inter-coronary anastomoses

These occur when an anastomosis connects any two separate arteries. The connection is a direct communication, usually slender, straight or gently curving vessels with a diameter not more than 100 microns. The vessels are known to increase in size and tortuosity with occlusive disease.

Trans-atrial anastomoses

The atrial circulation has been described as being particularly suitable for collateral connections. According to James, (1961) atrial arteries are epicardial and therefore subject to less intra-myocardial compression.

The atria and their arteries are located directly above the RCA and LCX artery leaving an open possibility for direct communication. The maximum resistance to atrial branch filling is asynchronous to that of ventricular branch filling and therefore, flow occurs at different times. James, (1961) notes, that although trans-atrial anastomoses are impressively abundant at post-mortem, they are not easily arteriographically visualized in vivo. His reasons seem to indicate limitations on demonstrative techniques and the possibility of interference by extra-cardiac anastomoses.

Trans-septal anastomoses

These are known to occur through atrial and ventricular septa, connecting 2 major coronary artery trunks that occupy the interventricular sulci. It is important to note that this collateral circulation is reported to pass through the septum and in addition to supplying some vessels to the septum itself, it provides an integral contribution to the accessory supply of the free walls adjacent to either sulcus, (James, 1961).

Ventricular epicardial anastomoses

They connect the major coronary arteries and are not subject to normal myocardial systolic compression. Represents an abundant and impressive circulation, especially in the ischemic heart, (Paster, 1977).

Sub-endocardial anastomoses

Small terminal vessels found in all cardiac chambers. They exhibit a typical right angle course from the parent trunks. These have been described in detail in the review of Fulton's, (1965) study.

Anastomoses via specific arteries

Arteries of significance include the conus artery (which provides an important anastomosis between right and left coronary arteries, over the pulmonary conus), sino-atrial and atrio-ventricular nodal arteries, and Kugel's artery (important in trans-atrial anastomotic circulation).

Extra-cardiac anastomoses

These vessels represent connections between the branches of the coronary arteries and those from surrounding organs and structures.

2.5.4. KUGEL'S ARTERY

Kugel in 1927, called attention to a conspicuous artery that produced a free anastomosis between right and left coronary arteries. The artery was described to run in the inter-auricular septum for part of its way and occasionally give off branches to the aortic cusp of the mitral valve. The artery, because of its constant occurrence and situation, was called the "arteria anastomotica

angularis magna", know today as "Kugel's artery".

In order to appreciate the anatomy of this vessel, an understanding of the relationship of the inter-auricular septum to it surrounding structures needs to be reviewed.

The posterior portion of the inter-auricular septum sits on top of the inter-ventricular septum, dividing anteriorly to form a "Y". The stem of the "Y" is represented by the inter-auricular portion posteriorly. The 2 wings of the "Y" form the anterior walls of both atrial appendages and enclose the root of the pulmonary artery and the aorta. The right wing of the "Y", has inserted into it, the aortic cusp of the mitral valve. It is the left wing of the "Y", together with the stem, which in the great majority of cases, carries the *arteria anastomotica angularis magna*, (Kugel, 1927).

Kugel's artery may be described in 3 general groups according to its anastomotic pattern. In the first instance, it forms a simple anastomosis between the LCX or its branches and the *posterior portion* of the RCA and its branches. In the second, it forms a simple anastomosis between the LCX or its branches and the *anterior portion* of the RCA and its branches. In the third, it is represented by *diffuse anastomoses* between branches from the anterior portions of the LCX and RCA and the posterior portion of the LCA.

The first description is most commonly seen. The artery arises from the LCX or its branches, 1-2 cm from the origin and passes directly posterior into the wall of the left atrial appendage. The course is described as being a tortuous one, passing medially within the anterior wall of the right atrial appendage, close to the insertion of the aortic leaflet of the mitral valve. It continues backwards in the inter-auricular septum (stem of "Y"), for its entire length. It unites near the crux

directly, or through intermediary smaller branches with the posterior portion of the RCA. Small twigs from the auricular branches of both coronary arteries also anastomose with this vessel. In cases where the LCA gives off the obtuse marginal artery and passes beyond the crux to the right side of the heart, Kugel's artery is known to re-enter the LCX posteriorly. Sometimes, when the SA nodal artery arises from the LCX, Kugel's artery is given off from the SA nodal branch or forms part of it.

In the 2nd description, Kugel's artery forms and anastomosis between the anterior portion of the RCA. Here again, the vessel follows a similar course as described above. However, at the point of reaching the crux of the heart, it is known to turn abruptly on itself in an anterior direction and re-trace its course through the stem of the "Y", passing within the anterior wall of the right atrial appendage to anastomose with the anterior portion of the RCA or its branches.

The 3rd description occurs less frequently. In this case, Kugel's artery arises as in the previous 2 descriptions, but soon, breaks up into a number of branches. As these branches approach the stem of the "Y", they anastomose with branches arising from the RCXA through the anterior wall of the right atrial appendage and by branches coming anteriorly through the inter-auricular septum of the crux (which may arise from the RCA or LCA).

Kugel and Gross, (1926) noted that when the blood vessels supplied the aortic leaflet of the mitral valve, they almost always arose from the *"arteria anastomotica angularis magna"*. When the valve leaflet is not vascularised, branches arise from the artery and extend to the base of the aortic cusp of the mitral valve where they terminate.

In its course through the anterior and medial walls of the left atrial appendage, Kugel's artery passes intimately near the left and right posterior aortic cusps. In doing so, it sends off branches that extend to the upper limits of the valve flaps to dichotomize at the base to send fine branches along the line of closure. The right anterior cusp is usually supplied by myocardial branches of the LCX in this area, but may also receive supply from Kugel's artery. Kugel's artery also shares a close relationship to the posterior and left lateral surfaces of the aorta. Here, it may send anastomosing branches to the RCA and LCA.

The anatomical value of Kugel's artery, when present, is recognized. In addition, clinically, the intimate relationships it shares with the aortic and mitral valves, commisures and base of the aorta suggests that it may well play a part in the pathogenesis of lesions in the area or provide additional blood flow when needed.

2.5.5. THEBESIAN VESSELS

Interest in the circulation of the heart walls was stimulated by publications by Raymond Vieussens in 1706 and Thebesius in 1708. Their documentation of the channels connecting the coronary vessels directly to the chambers of the heart resulted in the understanding of the significance of theses communications and the general acceptance of the so-called "Thebesian vessels".

Although little was known of their anatomical relationships, or of the exact part they played in the coronary circulation, their importance in playing a role in the circulation of the heart itself was obvious, (Wearn, 1928). Thebesius (1708) raised the question as to why the "Creator had placed these small vessels in the walls of the heart", and then answered it by saying that " they made

possible, a continuous blood flow in the heart by serving as an exit at the beginning of systole". After having answered the question, Thebesius gave " due credit and much praise to the Creator for having such foresight to anticipate the need and usefulness of these little vessels", (Wearn, 1928).

Although these vessels have since become known as the "Thebesian vessels", it was really Vieussens who first discovered their presence. Of greater importance though, was the fact that Vieussens used and injection method via the arteries, and Thebesius injected the veins, yet both described the same openings. According to Wearn, (1928) these findings were subsequently followed by investigations confirming (Lancisi 1740, Senac, 1749; Langer, 1880) and denying the presence of these vessels (Cruveilhier, 1834, Theile 1843; Lannelongue, 1867).

In 1898, Pratt suggested that the Thebesian veins were connected directly with the coronary veins and indirectly with the arteries through capillaries. Moreover, he explained the lack of infarcts in markedly sclerosed hearts, by the presence of the Thebesian circulation.

The embryology of the Thebesian vessels have not been studied extensively, although the literature suggests them to be the remains of the primitive, inter trabecular circulation of the embryo, (Wearn, 1928). According to Wearn, (1928), Minot (1900) found that in the earliest stage, the trabeculae of the heart was made up of muscle cells covered with endothelium, but without capillaries of their own. He believed that the "sinusoidal" circulation of the embryonic heart was sufficient to nourish the trabecular muscle.

However, Grant and Regnier (1926) reported that the sinusoids were not initially connected to the

heart, but grew into them at a later stage. As the coronary veins grew and branched into the myocardium and provided evidence showing direct connection, other than through the capillary bed, between the coronary arteries and the chambers of the heart. In addition, connections between larger coronary veins and the Thebesian veins were demonstrated. The study also showed that although the size and number of vessels varied between sides, as much as 90% of the arterial flow may escape via the Thebesian vessels.

An investigation by Archie in 1974 however, declared the openings in the atrial and ventricular walls to be an unlikely source of supply to the subendocardium because of inappropriate pressure differences in both systole and diastole. Furthermore, Griggs et al., (1972), commented that if these vessels were a source of additional supply, then the subendocardium, instead of being the myocardial area most vulnerable to ischemic damage, would be best protected.

It is interesting to note, before dispelling the possibility of any positive role these Thebesian vessels may have to play in the coronary circulation, to consider the findings based on case reports in Wearn's (1928) study. The reports describe two instances of post mortem findings showing complete closure of both coronary arteries at the aortic orifices. In each case, a heart without openings for the coronary arteries had maintained a sufficient circulation that enabled the individual to survive and earn a living for a significant number of years. Wearn (1928) postulates that the existence of the Thebesian vessels, "being the only other entrance to the coronary circulation", was responsible for the nourishment necessary in maintaining the myocardium.

He further supports these claims by pointing out how often, individuals that live comparatively

healthy lives present with total occlusion of the coronary arteries at post mortem, showing that when allowed sufficient time to adapt, the Thebesian vessels can take over the function of coronary arteries. Needless to say, with these contrasting points of view being presented, the obvious issues therefore, of where and how these hearts get sufficient circulation to function so efficiently, remains unclear.

EXTRA-CORONARY COLLATERALS

"Healing is a matter of time..... but sometimes it is a matter of opportunity."

Hippocrates (460-375 BC)

2.6.1. INTRODUCTION

For most conventional intra-cardiac operations, maintaining a quiet, dry operative field is essential. Cardiac arrest may be achieved by cross-clamping the aorta, thereby interrupting coronary flow. Sometimes however, the heart continues to beat or fibrillate despite aortic cross-clamping. This suggests that anoxia of the myocardium cannot be achieved when coronary flow is interrupted or that there may be supplementary flow from another source, (Brazier and Hottenrot, 1974).

Further evidence suggesting the possibility of an extraneous flow contribution may be observed during valve replacement or coronary artery revascularisation. Occasionally, outpouring of arterial blood from the coronary ostia occurs when the aorta is opened, or when the coronary artery is incised. This persists in the arrested heart, "despite adequate venting of the left ventricle and decompression of the coronary sinus to prevent retrograde flow", (Brazier and Hottenrot, 1974). In addition, it is not uncommon to observe that some patients maintain excellent ventricular function, despite complete occlusion of all 3 major coronary arteries.

The possibility of the existence of an extra-coronary collateral circulation was postulated by Thebesius in 1708, (Bloor and Liebow, 1965), in his description of the *venae cordis minimae* -

small openings in the atrial and ventricular walls. During a study of these Thebesian vessels, Langer (1880), described branches of the coronary arteries anastomosing with vessels in the mediastinum, parietal pericardium, diaphragm and hila of the lungs.

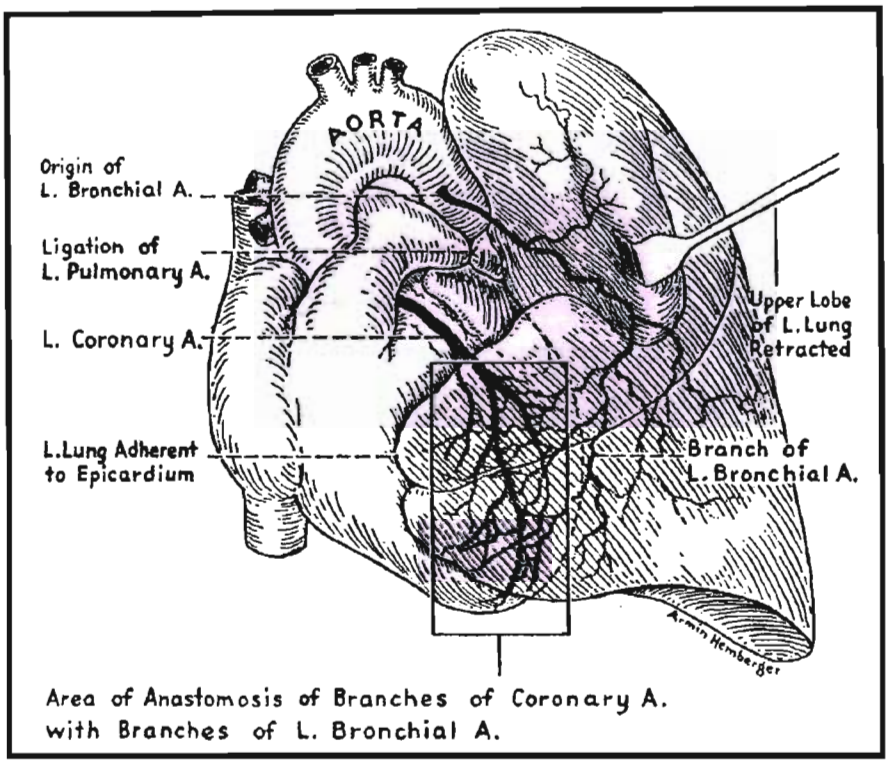


Figure 43: Demonstration of coronary to bronchial anastomosis

(Adapted from Moritz et al., 1932)

The literature reveals little, if any, detailed description of extra-cardiac sources of coronary artery anastomoses. Reports by Gross (1921), Spalteholz (1924) and Karsner (1933) have made reference to the extra-cardiac coronary anastomoses.

2.6.2. ANATOMY OF EXTRA-CORONARY COLLATERALS

In 1926, Woodruff documented the anastomosis of branches of the coronary arteries with the vasa vasora of the ascending aorta. Wearn's (1928) investigation corroborated these findings when he observed that upon injecting the coronary arteries with India ink, the vessels in the ascending aorta filled with injection mass, (Figure 43).

Robertson, in 1929 highlighted the importance of the arteries supplying the fat pads of the heart in the presence of coronary artery disease. In addition, he too discussed the anastomoses of the coronary arteries with the peri-adventitial vessels of the ascending aorta.

In 1932, Hudson et al., embarked on an investigation in order to define the origin and extent of the extra-cardiac anastomoses. Their report noted the common sites of emergence of the extra-cardiac branches of the coronary arteries to be around the root of the aorta, the base of the pulmonary artery, pulmonary veins, ostia of the superior and inferior vena cavae and in the inter-vascular pericardial reflections.

According to Hudson et al., (1932). The vessels emerging around the root of the aorta are said to be contributions from the coronary arteries close to their origin, and extensions of the vessels within the pericardial fat pads to the adventitia of the ascending aorta. These vessels are reported to extend from the aortic ring to the diaphragm and occasionally, for a short distance, below the diaphragm. In addition, connections with the pericardial, mediastinal and diaphragmatic vessels exist.

Communicating vessels around the pulmonary artery adhere to the wall of the vessel. They are demonstrated by anastomoses between the coronary branches from the conus, pericardial reflections around the pulmonary artery, mediastinal and bronchial arteries and sub-epicardial fat pads around the base of the heart.

A significant anastomotic plexus exists around the pulmonary veins. They emerge at the site of the pericardial reflections around these veins and anastomose with branches of the pericardial, bronchial and mediastinal arteries. Larger, more numerous vessels are located around the ostia of the vena cavae, closely applied to the adventitia. However, their course and number are not constant.

The contribution of the auricular branches of the right coronary artery to extra-cardiac vessels has been well described by both Gross, (1921) and Hudson et al., (1932). They usually consist of two large branches from the proximal RCA, which arborise around the ostia of the vena cavae after supplying the auricular wall. The larger and more constant of the two, form an annulus of fine branches around the ostium of the superior vena cava.

According to Hudson et al's., (1932) observations, an extensive network of extra-cardiac coronary anastomoses exists over the entire surface of the parietal pericardium. The vessels communicate with the pericardiacophrenic branch of the internal thoracic arteries as well as with the small anterior branches of the thoracic aorta. The principle anastomoses of the pericardial vessels with the coronary arteries however appear to be in the region of the pericardial ostia of the pulmonary veins and vena cavae.

Anastomotic vessels in the diaphragm spread in a fan shaped manner over each dome,

communicating with the coronaries via small vessels in the adventitia of the inferior vena cava and terminal portions of the pericardiacophrenic arteries. The auricular branches from both coronary arteries provide a significant contribution to the vessels in the hila of the lungs. Branches from the pericardial fat extend out along the pulmonary arteries and veins, over the pleural surfaces of the lungs and along the bronchi.

The mediastinal, esophageal and tracheal vessels are also known to communicate with the coronary arteries. These vessels are small anterior branches of the thoracic aorta and anastomose freely with the bronchial, phrenic and intercostal branches of the internal thoracic arteries. In Fulton's, (1965) study, mediastinal connections of considerable size, up to 800 microns at the terminal diameter, were observed. Although these larger vessels were found in cases of coronary artery disease, they were not restricted to those cases. In addition, Gross (1921), Robertson (1929) and Hudson et al., (1932) state suggest the presence and caliber of extra-coronary collaterals to be greater in persons of advanced age.

THE INFLUENCE OF AGE CHANGES IN THE BLOOD SUPPLY TO THE

"A man is as old as his right coronary artery"
Gross, 1921

2.7. CORONARY ARTERIES: AGE INFLUENCE

Oertel, in 1921, highlighted and rendered the clinical importance of age related changes to a general anatomical principle upon which depends the metamorphosis of the individual from birth to senility and the development of disease.

His conclusions provided the following interpretations;

(a) Organs which normally exhibit a developmental cycle of changes in cell elements and tissue organization undergo corresponding functional modifications.

(b) Pathological, anatomical and functional changes must therefore be interpreted in conformity with and to an age period.

(c) Anatomical and functional expressions of a disease vary in one and the same organ according to its construction and composition during an age period, (Gross, 1921).

Based on this general concept of age influence, one can appreciate similar changes in the vasculature of the heart. According to the findings by Gross (1921), the cardiac vessels show clearly, the undeniable effect of age periods in producing effects anatomically, physiologically and pathologically.

Gross, (1921), quoting Bizot (1837) showed that in the embryo, both ventricles are initially about the same thickness. At birth, the left is slightly thicker and thereafter outgrows the right so that it eventually becomes preponderant. He adds further that according to Valentin, (1844) and Beneke, (1878), there is a greater increase in size on the left side until the later decades of life, when this growth reverses, (Gross, 1921).

Based on Müller's (1877-1881) study of 1481 hearts, Gross, (1921) concluded that the turning point in growth of the heart is at the 7th decade in men and 8th decade in women. In addition, sub-epicardial fat was found to begin at the base of the heart and follow the RCA to the apex and then the LCA and related structures.

Gross (1921), upon examining a series of hearts at various decades, noted that the circulation in an average heart at birth is equally divided and fails to show septal anastomoses. In addition, he observed that the luminal size is uniform and the vessels, except for its distal segments, lacks tortuosity. There are no visible *arteria telae adiposae.*

According to his records, the average heart in the 1st decade showed the main coronaries to pursue a straight and uniform course. The septal anastomoses are not visible and there is no sign of *arteria telae adiposae.* In the 2nd decade however, the distribution of blood begins to be slightly more marked on the left side, yet the vessels have not as yet begun to become tortuous. Upon stereoscopic visualization, fine septal anastomoses can be observed. At this stage, stray branches to the fatty tissue may be observed in the coronary furrows parallel to the main arteries.

A definite sign of left dominance appears in the 3rd decade. The caliber of the septal anastomoses increases, as does the degree of tortuosity. As is anticipated, the *arteria telae adiposae* become abundant. These chances are reported to be remarkably progressive in the 4th to 6th decades of life. From the 7th to 8th, and especially in the 8th decade, the complicating feature of arteriosclerosis appears. However, by this stage, the preponderance of anastomoses may seem to compensate for the diminishing capacity of the normal circulation to effectively vascularise the myocardium, (Gross, 1921); (Figures 44 and 45).

This theory may be supported by case findings that relate the individual's asymptomatic status of coronary arteries during life and the unauthenticated findings of severely compromised vessels at necropsy. One can only conclude that with the progression of age, and the concurrent development of anastomoses, the heart can and does receive a considerable vascular reserve from these collaterals whose functional possibilities increase in direct proportion, (Gross 1921). There is thus, an undeniable functional significance in progressive evolutionary changes.

On the subject of the *arteria telae adiposae,* Gross (1921) points out that the development of fat vessels should not be seen as a secondary mechanism to functional need of the myocardium for he has demonstrated their constant and dynamic appearance proportional to age, irrespective of pathological lesion. However, he agrees that pathological interference of a coronary artery can lead to the enhancement of these vessels both quantitatively as well as qualitatively.

The degree of tortuosity of coronary arteries Gross (1921) sees as a general expression of advanced age. He attributes this characteristic to the histological deterioration of the vessel wall and relative shrinkage and eventual atrophy of the myocardium in the later decades of life.

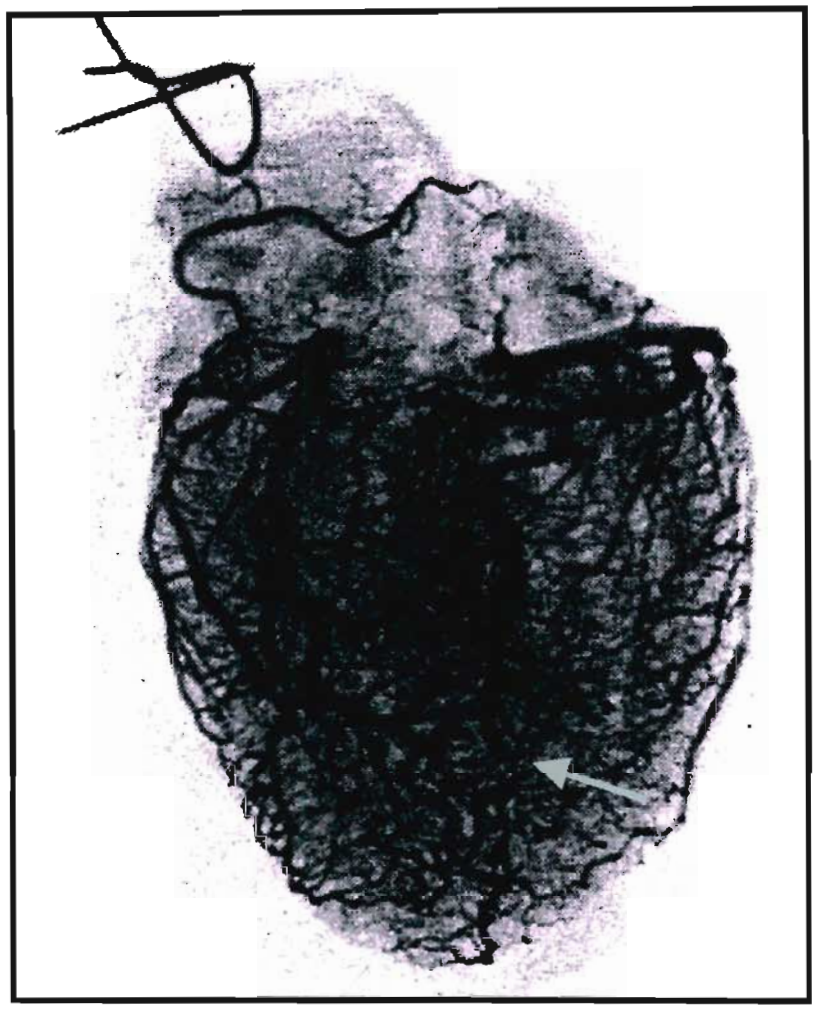


Figure 44: Extensive arterial anastomoses with a heart of advanced age

(Adapted from Gross, 1921)

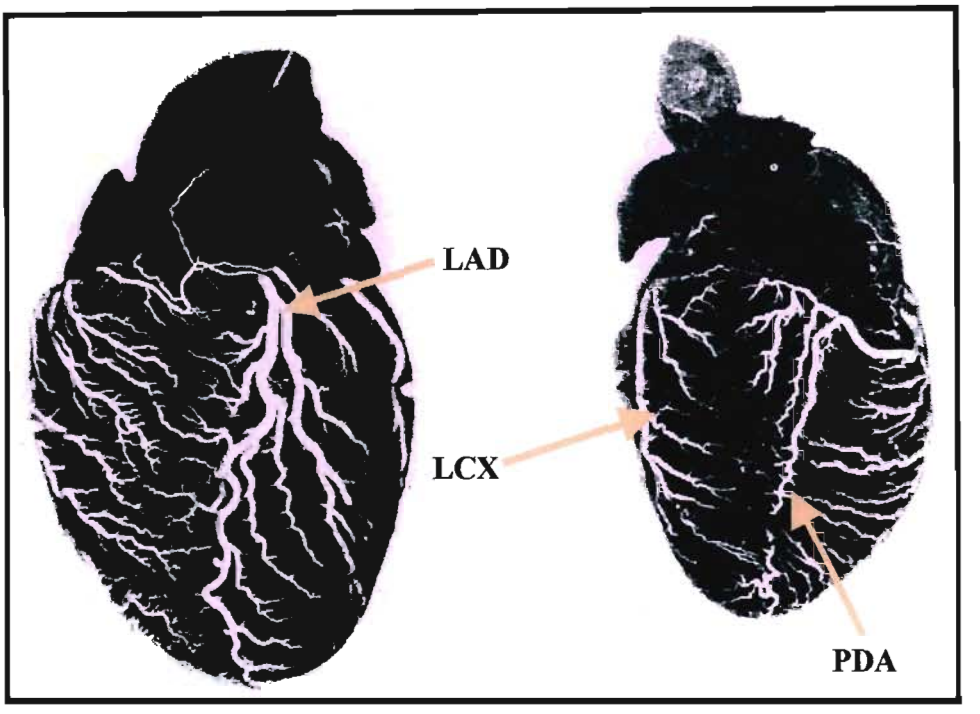


Figure 45: Arteriographic images of coronary arteries in a fetal specimen

(Adapted from Gross, 1921)

MYOCARDIAL BRIDGES AND THE INTRA-MURAL CORONARY ARTERY

"It is a melancholy reflection that a shift of a few millimeters in the anatomical course of the main coronary branches would have resulted in practical immunity from the most common form of coronary vascular accident...This reflection gains a certain piquancy through the investigations of Chase and de Garis (1939) who found that while the gorilla and gibbon have an epicardial network of main coronaries, the coronary arteries of the chimpanzee and to a lesser extent, the orangutan tend to run a mural course. Using an outmoded form of expression, it would seem that, in this respect at any rate, we are descended from the wrong type of ape."
Geiringer, (1950)

2.8.1. INTRODUCTION

Although major branches of the human coronary arteries are known to maintain a predominantly sub-epicardial course, it is not unusual for a major coronary artery branch to follow an intra-myocardial course, either by travelling a significant length within the myocardium or beneath an arrangement of muscular slips. In attempting to afford some definition to this variation in coronary artery disposition, the terms *mural coronary*, *intramural coronary* and *intra-myocardial coronary* have frequently been used.

The literature, since the early1700's contains casual accounts of this exception to the normal sub-epicardial position of the coronaries. Reyman, in 1737 reported his observation of the left coronary artery frequently submerging beneath a layer of thin myocardial fibres for a distance before resurfacing to maintain its usual course.

Other anatomists such as Crainicianu (1922) and Spalteholz (1924) mentioned the occurrence of myocardial bridges, although providing only brief descriptions of their observations, whilst Gross

(1921), Cohn (1931) and Bianchi (1904) made no mention of this entity at all. It was not until 1950 however that a detailed account of the mural coronary was presented by Geiringer.

Standard texts define the myocardial bridge as "*an anatomic arrangement in which an epicardial artery tunnels beneath myocardial fibres, for a limited segment, to return to the epicardium distal to the bridge*", (Angelini et al., 1983). In some instances, the vessel may maintain an *intracavitary* course often within the right ventricle and although rarely, an *intraseptal* route may be taken.

To further elucidate the concept of arterial disposition, the following classification serves as a basis for description of their course. Polacek and Zechmeister, (1968) suggested mammalian hearts to be of three main types: Type A hearts, which includes those of the hamster, squirrel, rat, guinea pig and rabbit, exhibit coronary arteries which assume an entirely intra-myocardial course. Type B hearts, those of goat, sheep, dog, cat and man, contain coronaries that are predominantly epicardial, but exhibit myocardial bridges quite frequently. In Type C (horse, cow, pig), the coronaries are entirely epicardial.

The most frequent observation of myocardial bridges has been in relation with the LAD artery, particularly along its middle segment. The fibres that cover the artery are said to be extensions of the fibres arising from the infundibular territory, crossing the artery in a somewhat perpendicular course, Angelini et al., (1983). Considering the depth of the anterior interventricular groove of the heart as an influence on the epicardial position of the LAD artery, Geiringer (1950) observed that on occasion, the vessel may be deeply entrenched for a significant part of its course, by an extent to which the artery becomes encapsulated as it were to assume and intramuscular position.

Although in a strict anatomical sense, the artery is indeed still epicardial, from a physiological standpoint, it is classified as mural. It is not uncommon for branches of the LAD artery to deviate to an intra-myocardial position and when termination of the main vessel occurs in a bifurcation, it is not unusual for one of the branches to pursue an intra-mural course.

The arrangement of muscle fibres around arteries situated in the atrioventricular groove appear in a scattered pattern, often originating from the atrial myocardium. The arteries are loosely enclosed in myocardial loops and are most frequently associated with the terminal RCA and LCX arteries. The obtuse marginal and diagonal branches on the other hand, tend to traverse the left ventricular myocardium by penetrating the ventricular wall, often without ever re-surfacing.

The incidence and arrangement of myocardial bridges have been reported by several authors, (Angelini et al., 1983; Polacek, 1961; Zapedowski, 1965). Myocardial bridges are present in at least 60% of hearts and range from 5.4 to 85.7%. Generally, a myocardial bridge is recorded to be about 2.3-42.8 mm in length, (Kosinzki and Grzybiak, 2001) and between 1-4 mm thick, Angelini, (1983). The bridges vary in number from 1 to 6, (Bezerra et al., 1987) and the angle between the long axis of the vessel and muscle fibres is recorded between 5 and 90 degrees, (Kosinzki and Grzybiak, 2001).

The use of angiography in the visualization of intra-mural arteries was first introduced by Portsmann and Iwig., in 1960. Although no confirmation by surgical or anatomic means was afforded, the authors considered systolic narrowing to be evidence of myocardial bridging. In further reports of canine experiments, "sudden bending" and "rigidity" were considered anatomic features indicative of a coronary artery passing beneath the myocardium.

In later attempts at establishing distinct anatomic markers, Eliska et al., (1968) suggested that kinking of the coronary artery during systolic compression was likely to indicate that the artery may be crossed by either muscle fibres or a cardiac vein. To date, angiographers tend to agree that systolic narrowing of a coronary segment suggests myocardial bridging.

Although no anatomical study of significance may be found in the literature, angiographic descriptions of intra-mural coronaries are available. Observers have documented that at the site of systolic narrowing, the coronary artery is likely to undergo a sharp angulation before and after the bridged segment, (Angelini et al., 1983).

The incidence of angiographically demonstrable myocardial bridges is low. Various reports have recorded values ranging from 0.5% to 4.5 % of patients undergoing selective coronary angiography, (Angelini et al., 1983; Venkateshu et al., 2000).

In a retrospective cineangiographic study attempting to correlate the frequency of myocardial bridges in various clinical settings, only 5.5% of a total of 1100 images observed, showed bridging of the LAD artery and only 1 suggested bridging of the posterior branch of the RCA. The study continued to record incidences in patients with fixed coronary artery lesions and those without. Results showed a lower incidence of bridging in those with fixed lesions (4.3% vs 8.6%). Systolic narrowing patterns of the LAD artery were also observed in patients with systolic overload, especially in the presence of hypertrophic cardiomyopathy (16%) and in about 11%, indication of myocardial bridging was observed in patients with chest pain but with no fixed lesions. In addition, a higher incidence of bridging was shown to occur in males than in females.

Angelini et al., (1983) presents the following list of factors that influence the manifestation of an intra-mural artery:

a) *The orientation of the coronary arteries and the myocardial fibres*, especially in the case of the LAD, where fibres that have a perpendicular course are more subject to extrinsic compressive forces.

b) *In the presence of increased amount of adipose tissue or loose connective tissue*, as often found around the right coronary artery, the compressive force exerted by the myocardial bridge is decreased.

c) *The length and thickness of the muscle bridge*

d) *Where there is an aortic outflow tract obstruction* and the systolic tension that develops is higher than the intra-coronary arterial pressure.

e) *The artery wall tone* under the influence of administered vasodilators.

f) *The presence of a fixed obstruction in the proximal segment* of the coronary artery, where the compressive forces are not affected even though there is a decrease in distal intra-coronary pressure.

"If nature had only one fixed standard for the proportions of the various parts, then the faces of all men would resemble each other to such a degree that it would be impossible to distinguish one from another..."

Leonardo Da Vinci (1452 – 1519)

2.9. CORONARY ARTERIES: ETHNICITY AND SEXUAL DIMORPHISM

It is difficult to trace when the concept of ethnicity from an anatomic perspective was acknowledged. While socialists continue to caution any inference toward racial distinction, the scientific notion of ethnicity as an influencing factor in creating a predisposition for anatomic variation cannot be ignored.

The standard anatomic texts, while substantively clear, invariably present descriptions that appear to be widely based on data gathered from a predominantly, if not exclusively, Caucasian population. Although in terms of its anatomic value, such information makes for interesting study, its clinical impact, when measured against a diversity of target groups, may be limited.

The literature reports as such are scarce. Although anatomical accounts remain insignificant, a few clinically oriented commentaries have been published. Kurjia et al., (1986), reported on coronary artery variation in the native Iraqi population. The study, based on the arterial classification proposed by Schlesinger (1940), documented variations in arterial patterns in terms of preponderance and the origin of coronary branches. The authors point out that significant variation between their findings and which is reported in the literature exists.

It is a wide spread clinical impression that individual of Asian origin are among those most frequently subject to coronary artery disease. Studies conducted by Dhawan and Bray (1995) in the UK have not only supported these impressions, but have also confirmed a difference in size with regards to coronary artery diameter between Asians and Caucasians. When compared to Caucasians, Asians tended to have smaller vessel dimensions. Studies by Vanker et al., (2000) highlighted similar differences based on morphometric studies of the internal thoracic arteries (ITA). When compared to South African Whites and Blacks, Indians were shown to have the smaller ITA diameters on both right and left sides.

In 1995, Sahni and Jit studied the origin and size of coronary arteries in North-West Indians within the Chandigarh zone in India. Results of their investigation showed coronary artery sizes to have significant correlation between body size, heart weight and age and that the size of the vessel increased with an increase in age. Furthermore, when compared with data given in the Western literature, coronary diameters were significantly smaller.

In an investigation of coronary artery dimensions in young males (mean age 32 ± 8 years), Litovsky et al., (1996) measured the effect of age, race, body surface area, heart weight and atherosclerosis between Black and White groups within the USA. They showed that Blacks had larger coronary arteries than Whites and that in addition to body surface area and age, race was an independent predictor of lumen area.

Although not much has been published in terms of coronary arteries and the influence of sex, it is apparent from the results of a few reports that differences between sexes exist. O'Connor et al., (1996) in a study examining the effect of coronary artery diameter in patients undergoing coronary

artery bypass surgery, highlighted the association between arterial diameter and peri-operative mortality. After controlling for differences in age and body size, results showed that sex remained an important predictor of coronary artery size. Women were found to have smaller coronary arteries than men and as such were at a greater risk of post-operative mortality.

CORONARY ARTERY HISTOLOGY AND ATHEROSCLEROSIS

"I was making a transverse section of the heart pretty near its base, when my knife struck against something so hard and gritty, as to notch it. I well remember looking up to the ceiling, which was old and crumbling, conceiving that some plaster had fallen down. But on further scrutiny, the real cause appeared: the coronaries were become bony canals!"

Edward Jenner, 1799

2.10.1. HISTOLOGIC FEATURES OF THE NORMAL CORONARY ARTERY

The histological picture of the coronary arteries is similar to that displayed by most arteries in that it has a concentric arrangement of an intimal layer, a middle layer and an outer layer. The intimal layer or tunica intima, is composed of a layer of endothelial cells along with a subendothelial layer of connective tissue and smooth muscle cells. The endothelial cells are arranged longitudinally, in relation to the artery and are attached via occluding junctions and gap junctions, (Alexander et al., 1998). The intimal layer is separated from the media by the internal elastic lamina, a fenestrated sheet of elastic tissue.

The media is made up predominantly of smooth muscle cells and connective tissue. In the coronary arteries, there is a higher proportion of muscle than elastic tissue, (Baroldi, 1983). The media may consist of up to 40 layers of smooth muscle tissue and ranges in thickness from 125μm to 350μm, (Waller et al., 1992). Varying amounts of collagen and elastic fibers are also present within these layers.

The adventitial layer consists of collagen and elastic tissue, along with vasa vasora, nerves and lymphatics. The bundles of collagen are arranged longitudinally. According to Likoff et al., (1972), this arrangement combined with the somewhat "loose" consistency of the adventitia, influences changes in the diameter of the coronary arteries. The adventitia is said to range from 300μm to 500μm in thickness.

2.10.2. ATHEROSCLEROSIS AND CORONARY ARTERY ANATOMY

Coronary artery disease is by far one of the most widely considered clinical subjects. Whilst investigations aimed at understanding the mechanisms involved in plaque formation and localization, and therapeutic possibilities continue to gain favor, there are also certain anatomical concepts worth considering. Fulton's (1965) account of the coronary arteries offers the following thought of interest, that " no matter what may be the true aetiology of atherosclerosis it is evident that anatomical or dynamic factors must play a part in determining the localization of the lesions in the heart. For the distribution of coronary atherosclerosis, though widespread and variable, is not entirely haphazard."

The extent of atherosclerotic changes that occur in an artery may range from stiffening of the vessel to diffuse or segmental dilatation or narrowing, (Gorlin, 1976); (Figures 46 and 47). The most significant, however, is the degree of atherosclerosis that ultimately impedes flow through the vessel. With regards to the coronary arteries, this disease may involve the entire epicardial length of the vessel. Under most circumstances however, such lesions tend to be confined to specific areas of predilection and is essentially segmental, (Gorlin 1976). The sites of localization

appear to be based on hydraulic factors subject to physiological and anatomical properties of the arteries.

The major curves of the RCA and LCX are said to be particularly prone to lesions as is the point of division of the LCA. The sites of origin of the first septal and diagonal branches from the LAD are considered uncommonly susceptible areas. Gorlin, (1976) goes on to describe an association between the branching formation and localization of lesions. He adds that vessels are usually spared segmental lesions in portions of the vessels beyond major bifurcations. This is commonly seen in the PDA, the distal branches of the LAD and terminal diagonal branches and also in the LCX system. It is interesting to note that when the marginal branches are single and appear to be straight, they are found to be free of atheromata.

Much consideration appears to have been given to the branching pattern and length of the left main stem in terms of lesion distribution. Fulton, (1965) found such involvement to be located in an area of around 1cm in length, beginning about 2cm from the point of bifurcation.

In an attempt to explain this occurrence, Roberts, (1986) commented on the influence of mechanical stresses, anatomically determined by the length of the main trunk. He maintained that the maximum stress appeared to fall on the LAD just before it anchors by its first septal perforator. This portion of the artery, he termed the "pathological neck" and further added that a shorter length presented a predisposition to plaque distribution.

Fulton, (1965) discussed the influence of diminished caliber resulting from branching. He confirmed that whilst severe atherosclerosis was largely confined to the main coronary vessels and

their major epicardial branches, the smaller branches, save for their points of origin from a diseased parent vessel, were relatively if not totally free from lesions.

In addition to what has been described as far as the branching patters and morphometry of the coronaries are concerned, it is both anatomically and clinically interesting to consider what influence an anomalous path taken by an epicardial artery may have on the predilection of atherosclerosis.

Studies on the intra-mural coronary arteries seem indicate that such a disposition may offer a degree if not total immunity from sclerotic lesions, (Fulton, 1965; Geiringer, 1951; Polacek, 1961 Angelini et al., 1983). Although a report by Edwards et al., (1956) cast doubt on this "protective" effect of the myocardial tissue, there appears to be stronger support in favor of intra-mural vessels being relatively free from atherosclerosis.

Whilst the influence of an intra-mural position in protecting a coronary artery from atherosclerotic changes is remarkable, there is no definitive account of the mechanism involved in this occurrence. Fulton, (1965) suggests a relationship between lower gradients of pressure during systole between the arterial lumen and the external layer resulting from peripheral myocardial compression. In addition, he adds that it may perhaps be related to the lower degree of shearing stresses during this cardiac phase where in the intra-mural position the artery may be splinted as opposed to lying "unsupported" in an epicardial position. With the understanding of course that in such a position, the degree of kinking and buckling that accompanies each cardiac contraction is great.

Angelini et al's., (1983) detailed review on myocardial bridges appears to support Fulton's, (1965) theories on the factors surrounding the systolic wave pattern. He adds, that by compressing an arterial segment, myocardial bridges may indeed oppose the detrimental effect of systolic action.

His comments are supported by evidence obtained from in-vivo animal studies using rabbits, whose major coronary arteries are typically intra mural. Conducted by Ying-han, in 1978, the study involved inducing atherosclerosis in the experimental group by means of cholesterol ingestion. Results showed that although sclerotic changes were recorded in the aorta, the proximal coronary arteries remained free of disease, even when extensive lesions occurred in the subendocardial arteries. Polacek and Zechmeister (1968) observed similar results in cholesterol-induced dogs, whose anatomical arrangement of the coronaries resembles that of man's.

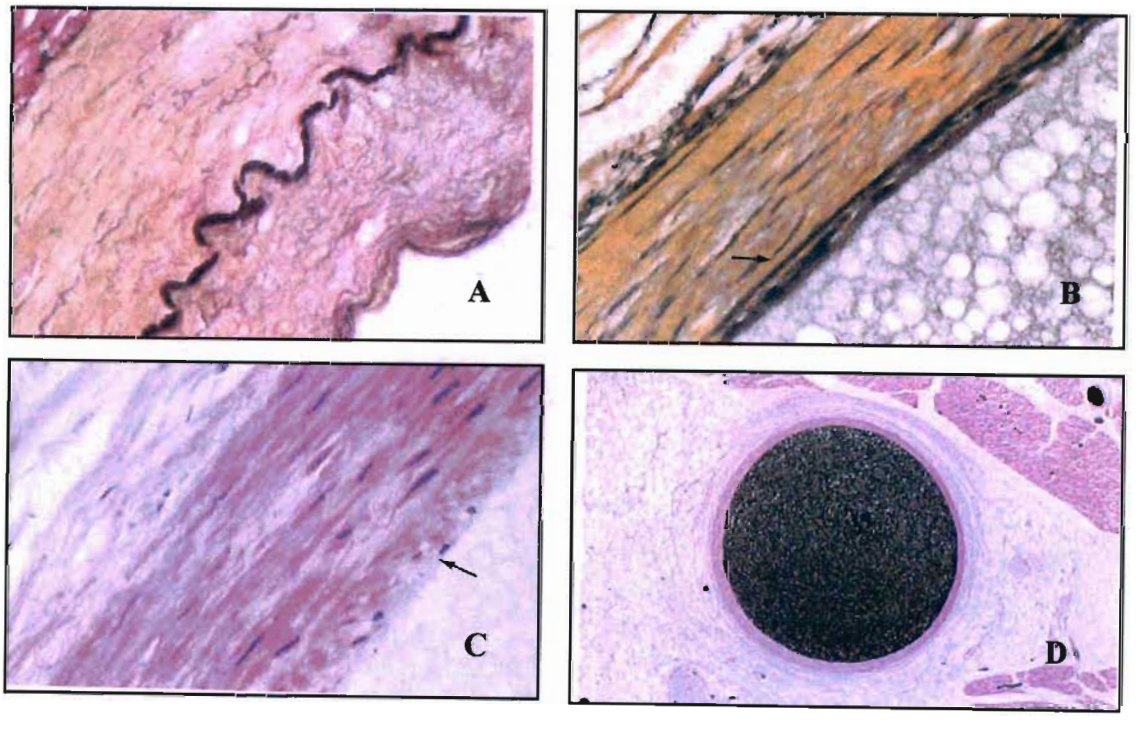


Figure 46: Histological appearance of a normal coronary artery (A, B, C, D)

(Adapted from Fareer-Brown, 1977)

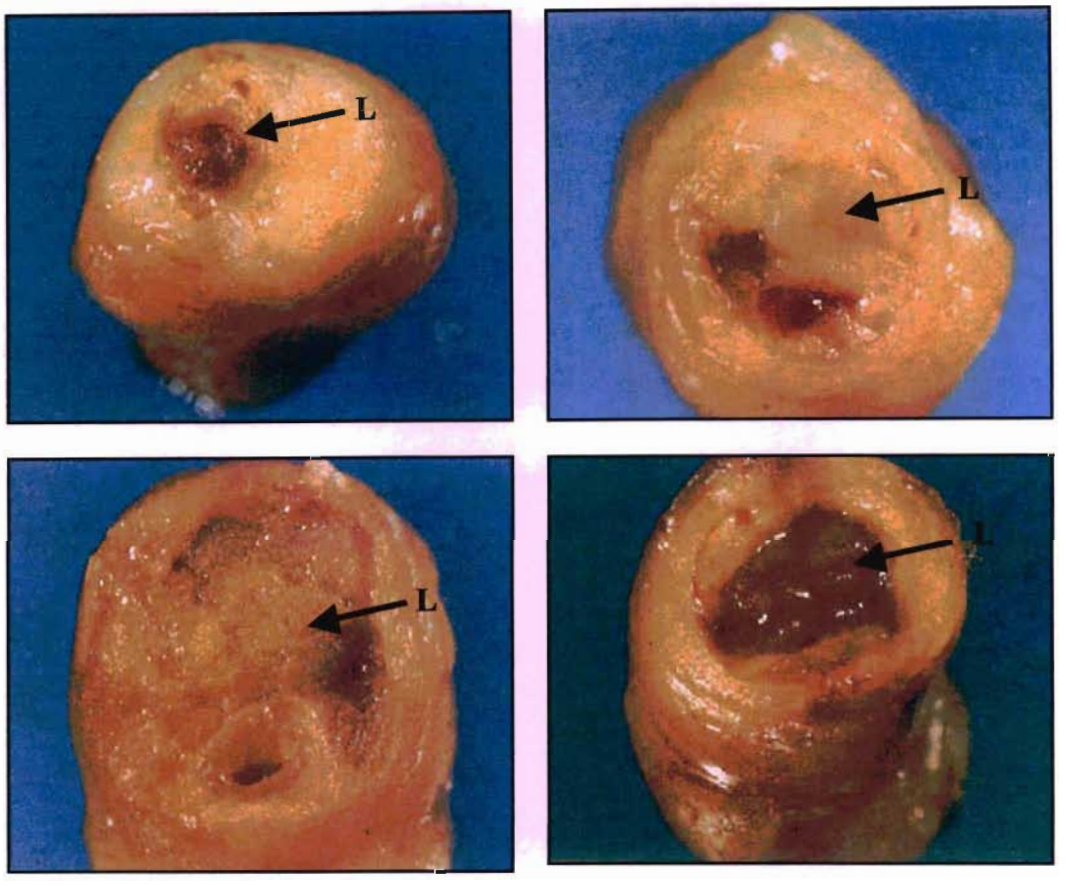


Figure 47: Gross pathologic appearance of a diseased coronary artery

(Adapted from Fareer-Brown, 1977.)

CHAPTER III

MATERIALS AND METHODS

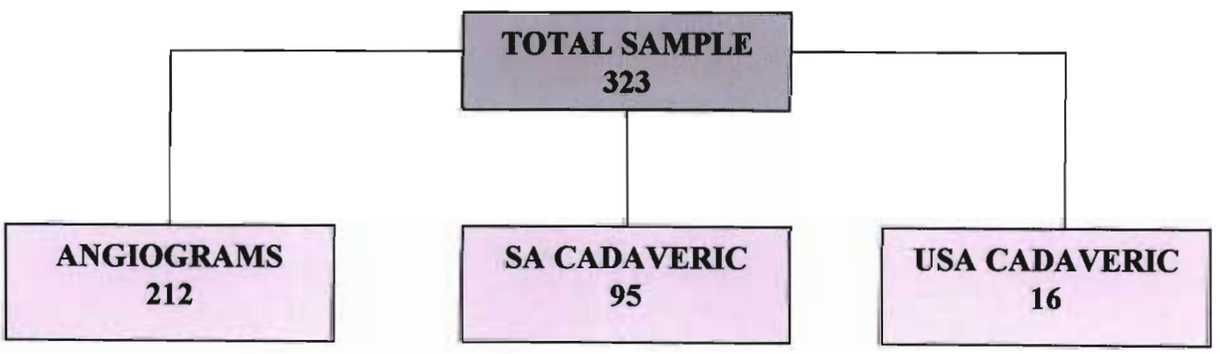
"For my judgment is that it is much better that you should learn the matter of cutting by eye and touch than by reading and listening. For reading alone never taught anyone how to sail a ship, to lead an army, nor to compound a medicine, which is done rather by the use of one's own sight and the training of one's own hands"

Jacobus Sylvius, (1635)

ANATOMICAL INVESTIGATION

3.1 ANATOMICAL INVESTIGATION

A total of 323 sets of coronary arterial patterns were studied. The sample consisted of 212 angiograms and 95 cadaveric dissections of hearts obtained within a South African group and 16 fresh tissue dissections of hearts obtained within a North American group. Ethics approval was obtained from the University of Durban-Westville, Durban SA, (ETHICS NUMBER 01061) and from the University of Louisville, Louisville KY, USA, (Appendix F).



3.1.1. HARVESTING OF POST MORTEM AND CADAVERIC HEARTS

A standard dissection procedure was employed in the harvesting of fresh, post-mortem and cadaveric adult specimens. The middle mediastinum was exposed, using an anterior approach into the thoracic cavity. The sternal plate was removed via separation of the sterno-clavicular joint and each costochondral junction. This was followed by a combination of a vertical and two

infero-lateral "Y" incisions to the anterior wall of the pericardial sac. The heart was freed superiorly by a horizontal cut transecting the superior vena cava, the ascending aorta and pulmonary trunk. The pulmonary veins were transected along with the inferior vena cava in order to free the heart from its lateral and inferior attachments.

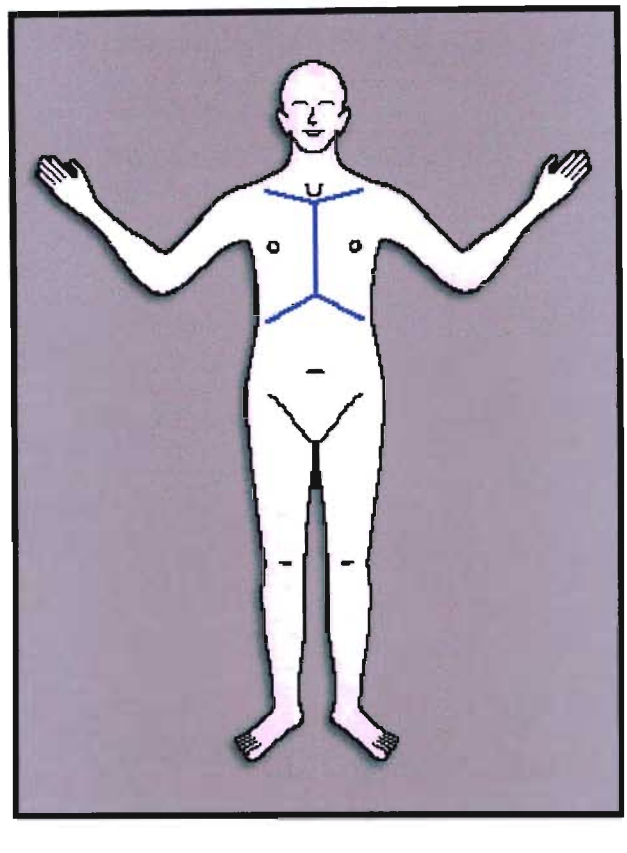


Figure D: Diagram showing dissection incision lines

3.1.2. INJECTION PROCEDURE

The harvested specimen was washed under running water in order to displace loose clots and blood within the chambers and great vessels. The coronary ostia were then cannulated and a 5% Ammonia solution was injected into the coronary arteries in order eliminate remaining luminal debris. A mixture of 20% Barium in latex was then injected into the coronary arteries under

controlled pressure. The hearts were then placed in 10% formaldehyde for 24 hours to allow the latex solution to set. The coronary arteries were exposed using a micro-dissection technique under magnification.

3.1.3. DISSECTION PROCEDURE

"Extreme precautionary measures were taken during the harvesting procedure in order to prevent biohazard contamination of the laboratory facility and maintain personal barrier protection of the investigator. A double pair of latex gloves, protective surgical mask, rubber boots, plastic eye shields and a disposable plastic apron were used at all times. As standard procedure, harvested specimens were subjected to extensive washing under cold, running water until free of fluid and clotted blood and loose tissue debris prior to any further investigation

3.1.3.1 Dissection of the Right Coronary System

The RCA was located in the region of the posterior inter-ventricular groove. The RCA was then traced from this posterior end, along a superior dissection plane within the coronary groove. As the artery very often lay embedded within a prominent fatty pad, the depth of the dissection was maintained at a superficial plane until the location of the vessel was more definite. The RCA was exposed up to its ostial origin.

3.1.3.2. Dissection of the Left Coronary System

The main trunk of the LCA was palpated at the left coronary sinus by a lateral separation of the pulmonary trunk and left atrial appendage. Once the main trunk had been located, the pulmonary

trunk was transected at a level 2cm above the superior limit of the infundibulum. This maneuver allowed a wider access to the dissection of the LCA and LAD.

The adventitia and epicardial fat was then removed in order to expose the left main trunk. Dissection continued along an inferior plane along the length of the LAD using deliberate outward movements of the scissors. The myofascial connections surrounding the LAD were further dissected using the tip of a size 10 blade after applying the required degree of tension necessary to define the plane. In order to prevent unnecessary damage to the underlying myocardium, the movements of the blade were limited to the direction of the fascial fibres within the plane.

Two approaches were used in the dissection of the LCX artery. In the first approach, the artery was traced from its point of origin within the "pulmono-auricular" cave and exposed to its point of termination. The alternate dissection approach involved locating the LCX at the posterior inter-ventricular groove and then dissecting along the course of the artery within the coronary groove.

3.1.4. MORPHOMETRIC ANALYSIS

Of the total sample of South African cadaveric hearts, 83 specimens were found to be suitable and subsequently subjected to further morphometric analyses. The coronary arteries of each heart were investigated under magnification and the following were recorded:

1. **The length of the LCA**

   Measurements were recorded in centimeters from the external aortic origin to its point of branching.

2. The termination pattern of the LCA

Three types of termination patterns were indicated. A bifurcation pattern was recorded in the event of the LCA branching into 2 components – the LAD and LCX arteries. A trifurcation pattern was recorded in the presence of an additional central branch - the ramus marginalis artery and a quadrification pattern was determined where there were 2 additional midline branches.

3. Origin of the LAD and LCX

The origin of the LAD and LCX were recorded in terms of their origin via independent aortic ostia or as branches from the LCA.

4. Presence and anatomy of the ramus marginalis artery

The ramus marginalis artery was identified as a central branch arising from a trifurcation of the LCA. The incidence of its presence was recorded and its cardiac course and branches were described.

5. The origin of the conus artery

The conus artery was identified as the first branch of the RCA. The origin of the artery was examined in terms of a high origin from the RCA when the branch take-off was at the angle of the RCA root and aortic wall and separate origin from the right aortic sinus via an independent ostium.

6. Measure of arterial dominance

Arterial dominance was measured by the origin of the posterior inter-ventricular artery. Origin of the artery from the LCX indicated left dominance, right dominance when it originated from the RCA and co-dominance when the posterior inter-ventricular groove received 2 branches from both the RCA and LCX artery.

7. The path taken by the LCX

The course of the LCX was determined through dissection along its main trunk. The arterial path was traced to determine whether it passed through the atrio-ventricular groove or failed by ending short of the groove along the obtuse margin of the left ventricle.

8. The branching pattern of the LAD

The LAD was examined for the persistence of a bifid pattern. A true bifid pattern was illustrated in the presence of 2 equal sized vessels descending on either side of the anterior inter-ventricular groove.

Table 2: Key for morphometric analyses of the coronary arteries

ANATOMICAL FEATURE	KEY
Length of the LCA	LCA/cm
Termination pattern of the LCA	TP/LCA
Origin of the LAD and LCX	LAD/LCX
Presence of the ramus marginalis	RM
High or Separate origin of the conus artery	CONUS/ho & CONUS/so
Arterial dominance	DOMINANCE
Path taken by the LCX	LCX/path
Branching pattern of the LAD	LAD/bifid

Results were recorded and confirmed by 2 independent observers. The age, sex, ethnicity and height for each specimen were recorded on a corresponding data sheet.

3.1.5. ANGIOGRAM SELECTION AND ANALYSIS

Two hundred and twelve angiograms of coronary arteries were randomly selected from the South African clinical groups of three cardiologists positioned at the St Augustine's and Entabeni Hospitals, in KwaZulu-Natal.

The arterial patterns were studied in the RAO, LAO and lateral projections according to the system described by Raphael et al., (1980). In each case, the coronary trees were analyzed by direct observation with an emphasis on each of the following anatomical features:

1. The termination pattern of the LCA
2. The origin of the LAD and LCX arteries
3. Presence of the ramus marginalis artery

4. A high or separate origination of the conus artery

5. The arterial dominance

6. The branching pattern of the left LAD artery

Table 3: Key for angiographic analyses of the coronary arteries

ANATOMICAL FEATURE	KEY
Termination pattern of the LCA	TP/LCA
Origin of the LAD and LCX	LAD/LCX
Presence of the ramus marginalis	RM
High and Separate origin of the conus artery	CONUS/ho & CONUS/so
Arterial dominance	DOMINANCE
Branching pattern of the LAD	LAD/bifid

The origin of each main trunk was observed, additional anomalies were recorded and the resulting anatomical pattern was described. Results were recorded on a corresponding data sheet with information detailing ethnicity, age and sex.

3.2. INVESTIGATION OF MYOCARDIAL BRIDGES

3.2.1. ANGIOGRAPHIC ANALYSIS

A series of 100 post-operative surgical reports of patients undergoing coronary artery bypass procedures at the St Augustine's and Entabeni Hospitals in KZN, were selected over a period between 1999 and 2001. The subjects were selected from the patient groups maintained by three clinicians within the selected hospital areas.

In each case, the position of the LAD was recorded as described during surgical presentation. The corresponding angiogram of each patient was then accessed and analyzed. In each case, the appearance of the LAD was studied on the angiogram, first upon direct observation and second, based on a measure of its "degree of straightness".

Each angiogram was viewed in the RAO projection at 030, CAUD 015 in MONOPLANE. The "LCA-LAD" segment was selected and the point of origin of the first septal perforator was marked. Based on a total length calculation, the LCA-LAD segment was divided into proximal, middle and distal thirds. Since the proximal and middle thirds were segments most frequently subjected to surgical manipulation, the study focussed its observations on these segments.

In order to establish a scientific validation of the degree of tortuosity determined from this standard observation, the following program was developed:

This program calculated an indicator which was called the 'tortuosity index' = SLENGTH (curve 1 NCURVES) for each of NCURVES. This index was a measure of the 'tortuosity' or the amount of twisting or deviation from a straight line or so called normal curve.

The measure used was computed as follows:

- For any curve made up of NPOINTS, number of the pair-points (X(1),Y(1)), (X(NPOINTS),Y(NPOINTS)) the Euclidean distances was computed between successive pairs of points and totaled as the 'tortuous' distance.
- This was then normalized (i.e. divided) by the distance of the extreme points, which is the shortest (straight-line) path between the end points.
- This measure allowed comparison of these indices for paths of varying extreme distances in a consistent way, so that if there was no variation from a straight line, the index should = 1, no matter how long the path.
- Large deviations from 1, implied pronounced tortuosity. Although a number of alternate measures could have been devised, with equally informative indicators, in view of standardization, this study applied the program as described.

By applying the test for deviation, the morphology of the LAD artery was determined. Using an index of 1 as the definitive for the measure of "straightness" along any length of the LAD artery, a

parameter was set between 1 and 1.5 to include all results with calculated values within this range as "angiographically straight". Any value greater than 1.5 was indicative of a tortuous vessel.

3.2.2. CADAVERIC INVESTIGATION

A series of 20 barium injected post-mortem hearts, with confirmed myocardial bridges over the LAD were selected. The arterial tree of each heart was x-rayed in the AP and Lateral projections under high exposure. A detailed micro-dissection of the LAD under magnification followed. A further dissection of the intra-mural LAD segment was conducted. Gross findings were compared with the radiographic results.

3.3. INVESTIGATION OF THE EXTRA-CORONARY COLLATERALS

3.3.1. SPECIMEN SELECTION

A specimen selection was employed, using fresh, lightly embalmed cadavers of advanced age with no signs of cardio-thoracic exploration. This criteria was based upon studies that suggested the presence of ECC's to be related to the progression of age, (Gross, 1921; Moritz, 1932; Brazier et al., 1975) at which stage coronary artery insufficiency is a common factor and the known fact that surgical exploration may give rise to artificially created collaterals.

3.3.2. LABORATORY EQUIPMENT

The lab facility included an x-ray table, C-arm x-ray machine, endoscope facility that constituted a camera, 30° endoscope, light source attachment with 80% brightness and television monitor. A video machine was connected to the x-ray monitor in order to dynamically record the filling of the coronary trees.

A 4 -5 mm Thru-Lumen Fogarty^{R} embolectomy catheters was used for the infusion of the injection material. The device consisted of a double lumen catheter with a latex balloon at the distal end. One lumen was used for the balloon inflation and was accessed through the gate valve at the proximal end. The thru-lumen was used for the infusion of injection material. A removable

thin steel wire, modified with a slight curve at the distal end and a 90° bend on the proximal end was used to provide sturdiness to and facilitate manipulation of the catheter during insertion.

3.3.3. INJECTION PROCEDURE

The injection material consisted of a lead-oxide Microfil^{R} solution. A mixture of 10 ml Diluent, 10 ml Clear Microfil^{R} and 10 ml Lead-Oxide powder was combined and 0.1 cc of catalyst was added just prior to the injection. Previous studies, including those by Schaper, (1988) and Fulton, (1965) indicated the use of barium-sulphate and bismuth-oxychloride as radio-opaque substances of choice. A confirmatory test was conducted in order to compare the degree of radio-opacity of the substances employed under standard investigations.

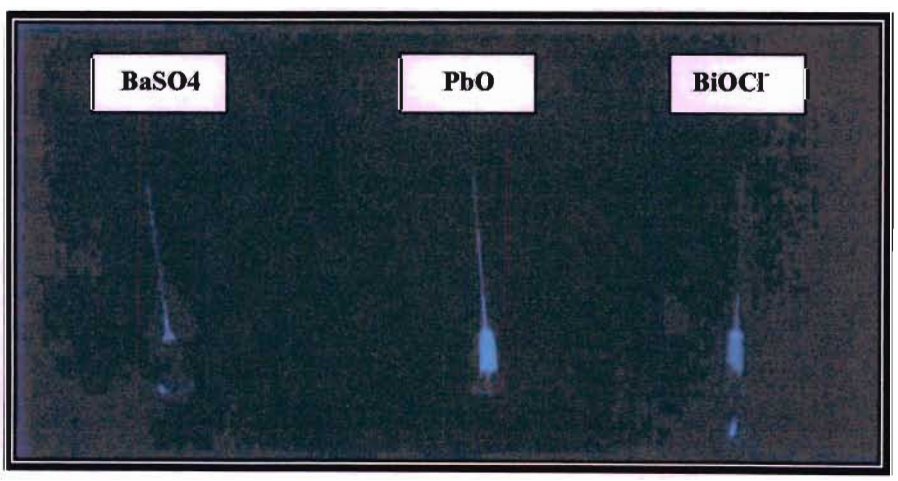


Plate 1: Test for radio-opacity of selected injection media

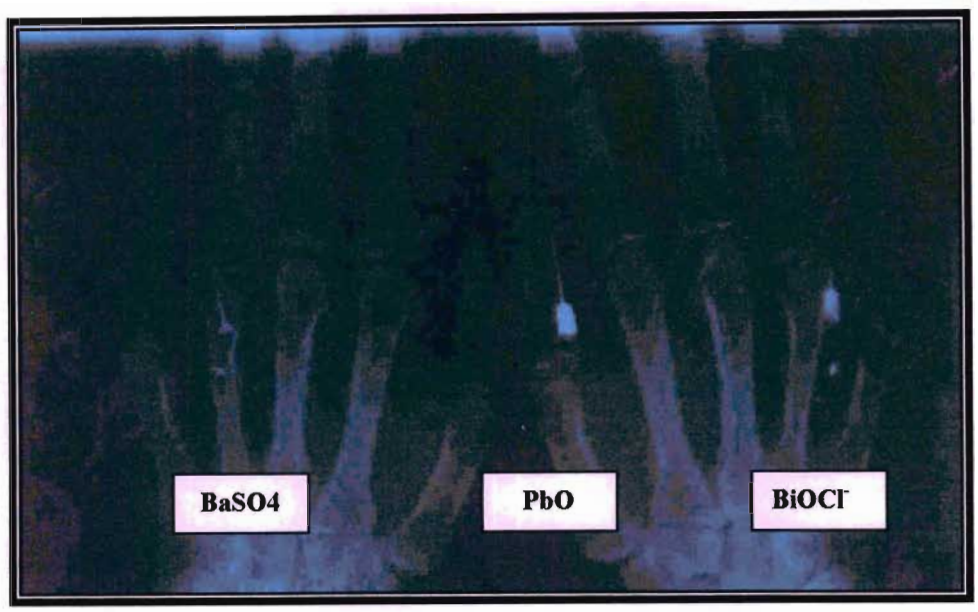


Plate 2: Test for radio-opacity of selected injection media against soft tissue

The radio-opacity of the injection material was confirmed with an image taken against soft tissue. Since lead-oxide was shown to have the highest degree of opacity, it was chosen as the medium of choice in this investigation, (Figures 48 and 49).

Dissection instruments included a battery operated hand- saw with a curved oscillating blade, micro-dissecting instruments and 4X magnification surgical loops. The measuring instruments included calibrated metric rods ranging from 0.2 mm to 2.0 mm in diameter, fine suture silk and a surgical caliper.

3.3.4. DISSECTION PROCEDURE

The right and left common carotid and subclavian arteries were exposed and transected to create access pathways into the ascending aorta. The vessels were then flushed using a constant flow of water under pressure via an irrigating cannula and drained by suction. Clots that were adherent to the aortic walls were removed endoscopically by means of specialized forceps.

3.3.4.1. Location and Cannulation of Coronary Ostia

The endoscope was advanced through either the right or left common carotid artery, depending on which vessel provided the easier access and best view of the aortic root. The aortic root was further surveyed to determine the total number of coronary ostia. The right coronary ostium was identified with a direct and a "straight-up" view, holding the endoscope in the normal position. The left ostium was best visualized by rotating the endoscope and the camera, thereby producing a 60 degree angled approach.

The catheters were then passed through one of the access routes and the ostia were cannulated. The balloon was immediately inflated using the indicated amount of water and the system was secured. A volume of 5-10 cc of injection material was infused at a pressure of 200-250 mmHg into the right and then the left coronary ostium.

3.3.4.2. Radiographic Identification

Filling of the coronary tree was observed fluoroscopically and radiographs were recorded from antero-posterior, right and left oblique and lateral views. Vessels containing injection material outside the cardiac shadow were identified radiographically as ECC's. The injection material was allowed to set at 15 degrees Celsius for approximately 5 hours.

3.3.4.3. Gross Inspection and Dissection

The superficial and extrinsic muscles of the anterior thoracic wall were reflected. The sterno-clavicular and sterno-costal joints were dislocated. The sternum was removed leaving the internal thoracic arteries on the surface of the fibrous pericardium. The right and left antero-lateral quadrants of the rib cage were removed to create wider access.

The surrounding structures, including the fibrous pericardium, internal thoracic arteries, distal trachea and hila of the lungs were inspected for injection material. A longitudinal incision was made into the anterior fibrous pericardium to gain access into the pericardial cavity. The superficial aspect of the heart and areas around the great vessels and diaphragm were inspected. A longitudinal incision into the posterior wall and to the left of the fibrous pericardium allowed access to the oesophageal area.

3.3.4.4. Morphometric Analyses by Micro-Dissection

ECC's were identified and traced to their sites of origin with the aid of micro-instruments and surgical loops. The course and anatomy were described. The following data was recorded for the ECC's identified using suture cotton and a surgical caliper to determine length and by direct optical comparison using calibrated metric rods for external diameter:

1. The site of origin to the coronary collateral component
2. The number of main ECC branches
3. The length of the main ECC branch
4. The external diameter of the main ECC branch
5. The number of secondary branches
6. The external diameter of the secondary branches

Table 4: Key for ECC morphometry

ANATOMICAL FEATURE	KEY
The site of origin to the coronary collateral component	CC
Number of main ECC branches	#1°B
Length of main ECC branch	L 1°B
External diameter of main ECC branch	D 1°B
Number of secondary branches	#2°B
External diameter of secondary branches	D 2°B

The area of location, according to sites of ECC's described by Hudson et al., (1932) was recorded.

The probable incidence of ECC's in each location and their mean dimensions were calculated.

HISTOPATHOLOGICAL INVESTIGATION

3.4. HISTOLOGICAL PROCEDURE

Histology of the coronary arteries was limited to an evaluation of the pathological status of the LAD in relation to its myocardial position. Cadaveric hearts with confirmed intra-mural LAD's were selected for histo-pathological screening. In each specimen, a 15mm block of vessel was harvested from three myocardial locations. A segment of length 15mm each was harvested from the pre-mural, intra-mural and post-mural areas.

Table 5: Key for LAD artery segments

LAD SEGMENTS	KEY
Pre-mural: 15mm length of segment before myocardial bridge	LAD I
Intra-mural: 15mm length of segment beneath myocardial bridge	LAD II
Post-mural: 15mm length of segment after myocardial bridge	LAD III

The tissue was then fixed, sectioned and prepared using the standard procedure for hemotoxylin and eosin staining. A series of 5 sections from each segment (Total n = 435) were screened for evidence of pathology and verified under microscopy by a qualified histopathologist.

A grading system was employed in order to define the degree of atherosclerosis. In each section, changes to the vessel intima were noted in order to determine the degree of atherosclerotic progression. A classification was based as follows:

1. In terms of slight intimal alteration resulting in an irregular border, the disease was regarded as "mild", (GRADE 1+).

2. A thickened intima with hyperplasia of at least 1mm thickness was considered to be reflective of "moderate" disease, (GRADE 2+).

3. The presence of advanced hyperplasia with plaque formation and localisation was categorised as "severe" disease (GRADE 3+).

Results were graded and subjected to further gross and statistical analyses.

The evaluative approach of this study incorporated a combination of interpretation by means of anatomical description and statistical estimation.

CHAPTER IV

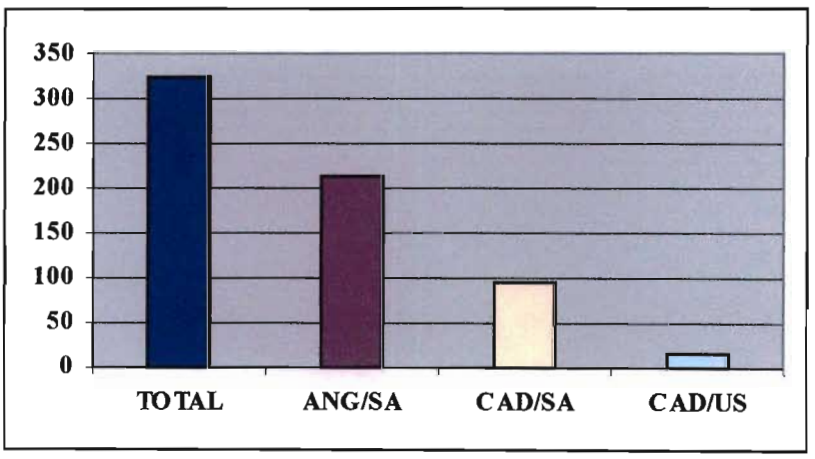
RESULTS

"Make it rather your serious endeavour not only to acquire accurate book knowledge of each bone but also to examine assiduously with your own eyes the human bones themselves"
Galen, (130-200)

CORONARY ARTERY ANATOMY

4.1.1. SAMPLE DISTRIBUTION

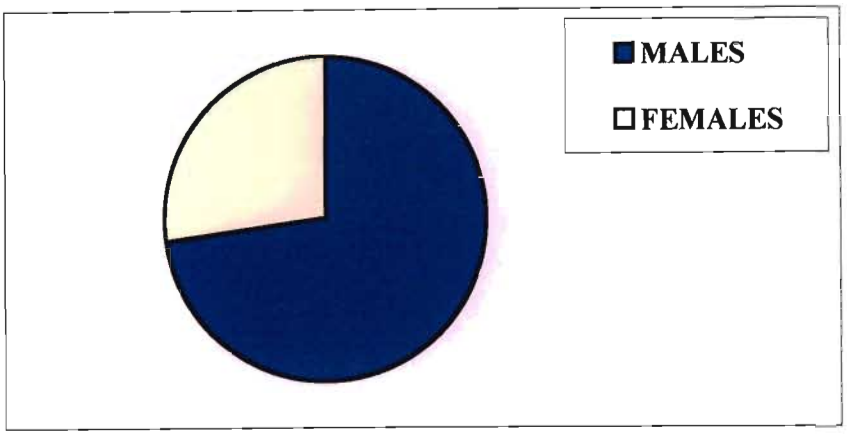
A total of 323 sets of coronary arterial patterns were studied. The sample consisted of 212 angiograms and 95 cadaveric dissections of hearts obtained within a South African group and 16 fresh tissue dissections of hearts obtained within a North American group, (Graph 1).



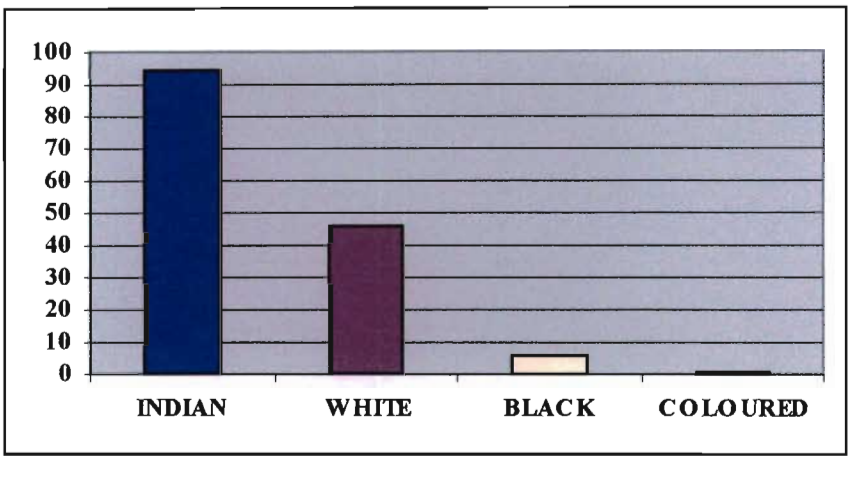
Graph 1: Sample Distribution for coronary artery patterns (n=323)

The angiogram group (ANG/SA) consisted of data obtained from 2 banks clinical centres: Entabeni Hospital (ENTAB) and St Augustine's Hospital (SAN).

The ENTAB group consisted of a total of 147 angiograms. The group distribution was as follows:



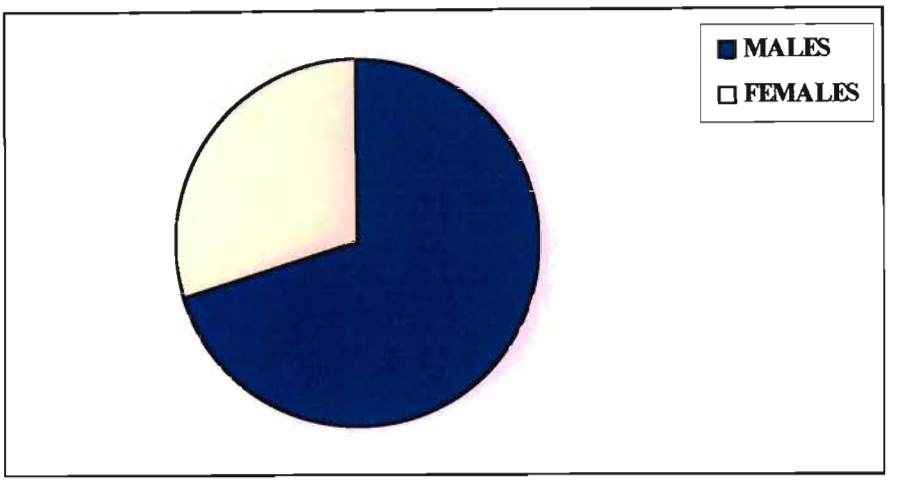
Graph 2: Sex distribution for Entabeni Group (n = 147)



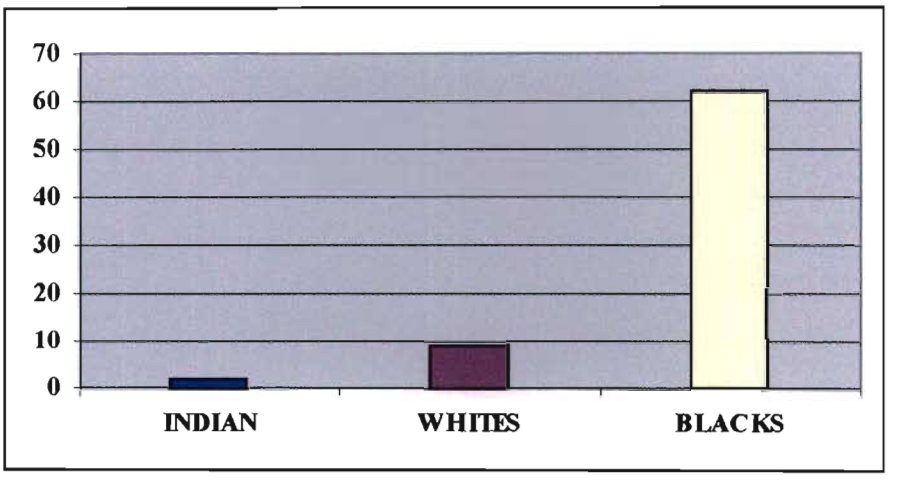
Graph 3: Ethnic distribution for Entabeni Group (n = 147)

The SAN group consisted of a series of 65 angiograms. The angiograms were randomly selected with no record of sex, age or ethnicity. Analysis was applied only in terms of variation.

The cadaveric group included 3 sets of data (n = 102). The first set of data (n = 83) was obtained from post-mortem cadavers within a South African population and included sex, age, ethnicity and height recordings. The sample distribution for this group was as follows:



Graph 4: Sex distribution for cadaveric group (n = 83)



Graph 5: Ethnic distribution for South African cadaveric group (n = 83)

The second set of data included random dissections of hearts from within a South African population (n = 12). In this series, no record of the sex, age, height or ethnicity was included. The third set of data was recorded included fresh tissue

dissections of coronary arteries from within a North American population (n = 7). In this series, no record of the sex, age or height was included. All the specimens were from a White group.

4.1.2. GROSS ANATOMY

Coronary Artery Origination

A right and left coronary system was recorded in 100% of specimens studied, (Plates 6, 7, 8, 9, 13, 18 and 35). The coronary ostia were located in a standard pattern along the line of the sino-tubular junction, (Plate 3). There was no measurable difference between the positions of each ostium in relation to the aortic sinuses although the left ostium appeared somewhat higher than that of the right.

In 14.5% of the total number of cadaveric dissections analysed (n=83) a left main trunk was absent. In this instance, the LAD and LCX arteries originated independently from the left aortic sinus.

In the total sample of 212 angiograms studied there was 1recorded case of an anomalous origin of the left coronary artery from the pulmonary artery.

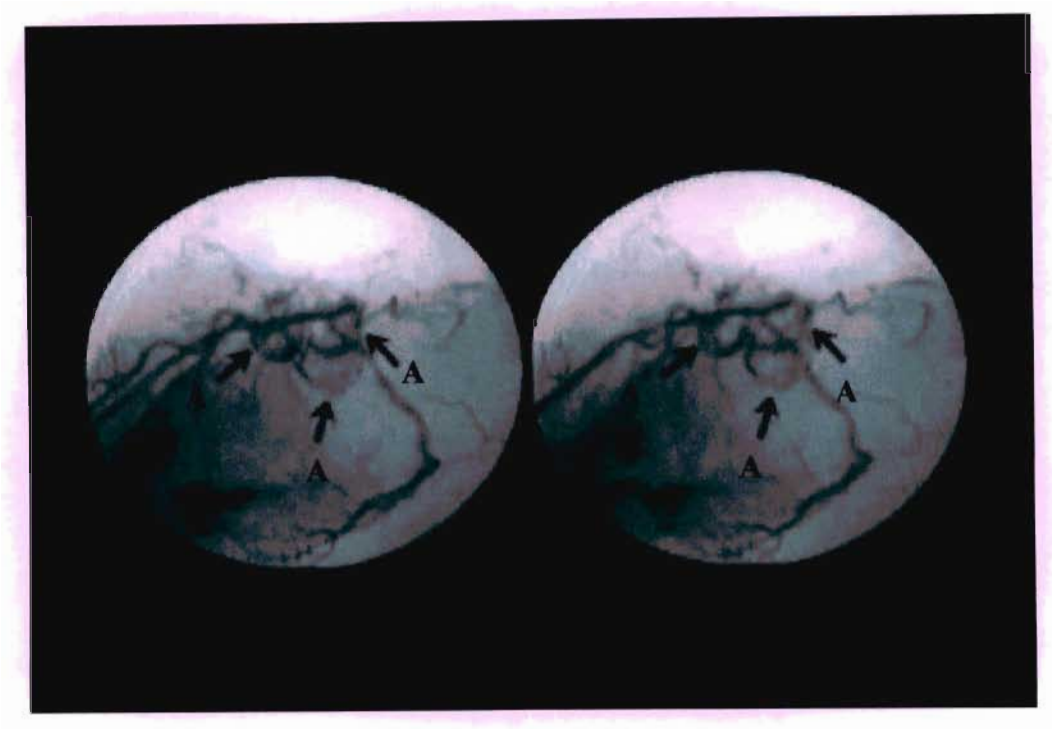


Plate 3: Aortic sinuses and coronary artery origination

The Right Coronary Artery and Branches

The RCA was traced along its path in the right atrio-ventricular groove. As the RCA descended along its course in this groove it was invested in a substantial but loose collection of fatty tissue with an average circumferential thickness of 9mm. The proximal 2.5cm of the artery inclined to the left of the groove along the right wall of the infundibulum, (Plate 4).

An incidence of 2.5% of bridging of the RCA was recorded. The pattern of bridging ranged from a series of 3 to 4 slips with an average width of 3mm to complete investment of the entire length of the artery up to the acute margin of the right ventricle.

The posterior atrio-ventricular course of the RCA was observed in 100% of dissections. The PDA was recorded as a branch of the RCA arising in the vicinity of the crux in 80.7%. A single branch pattern was recorded in 78% and a double branch pattern in 22% (n=83).

The conus artery was a constant and maintained the anastomotic ring with a corresponding left branch around the proximal infundibular trunk. The conus artery arose as the first branch of the RCA in 77.2%. Its origin from the RCA at the angle between the main trunk and the aortic wall was recorded in 19.2% and regarded as a high conus origin. In 3.61% the conus arose via a separate ostium directly from the ascending aorta.

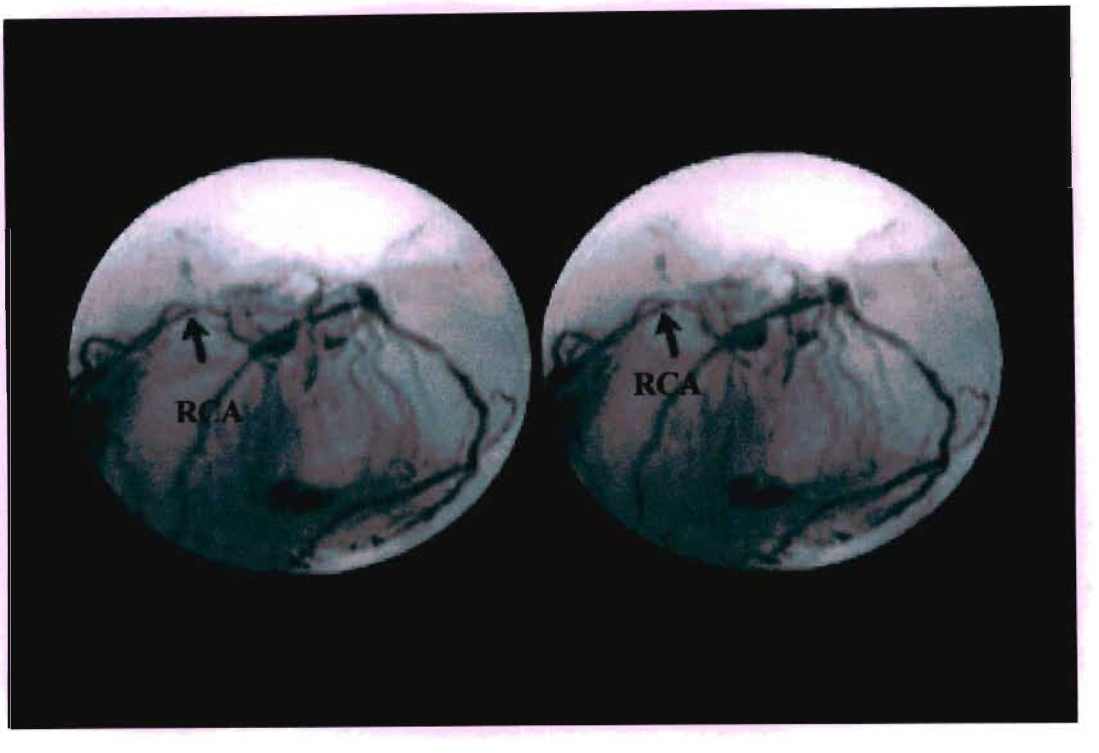


Plate 4: Angiographic demonstration of the RCA

The Left Coronary Artery and Branches

The LCA was located in a fossa between the pulmonary trunk and left atrial appendage in 85.5% of dissections. An average measurement of 0.82 cm of length of the LCA was recorded. There were no recorded branches from the LCA during the course of this length. The artery was maintained within this fossa by a surrounding layer of dense and fatty connective tissue, which extended from a combination of adventitia of the posterior wall of the pulmonary trunk and epicardium of the anterior wall of the left atrium.

The LCA terminated by means of a bifurcation in 86.7% and trifurcation in 13.3% of cases. In the case of a bifurcating pattern, the left and right divisions were identified as the LAD and the LCX respectively, (Plates 5a and 5b). Where the LCA terminated in a trifurcation, the central division was identified as the ramus marginalis.

An incidence of 13.3 % of the presence of this branch was recorded. The ramus marginalis descended along the obtuse margin of the left ventricle and ended approximately in line with the middle third of the LAD. In no case was this branch observed to extend beyond this region.

In the presence of a bifurcation pattern, the right division when identified as the LAD, continued down a path a mean distance of 1.6cm to the left of the inter ventricular groove in 52%, within the inter ventricular groove in 31.1% and in 16.9% as a bifid trunk to the left and right of the inter ventricular groove. The main branches of the LAD were identified in all cases as diagonal branches to the left, with a mean count of 3 and septal perforators to the right, usually 2 significant sized vessels.

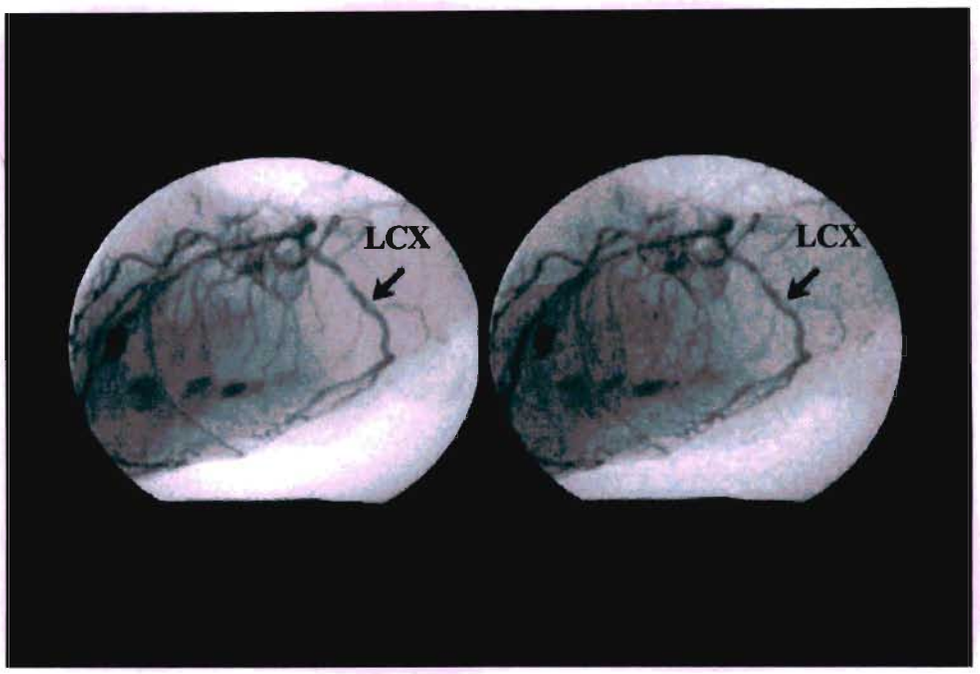


Plate 5 (a): Angiographic demonstration of the LCX artery

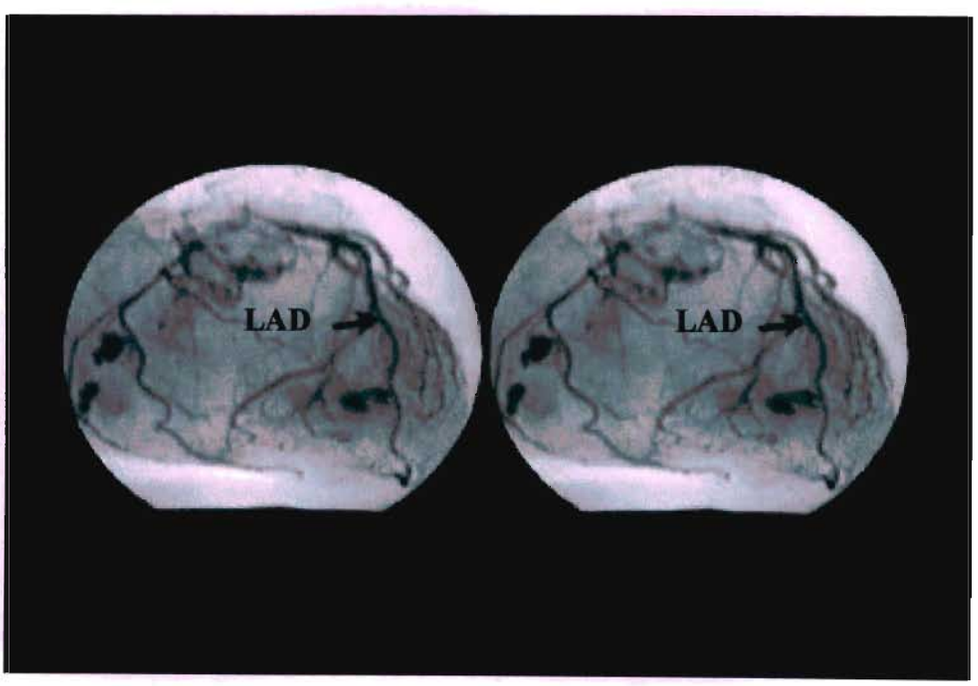


Plate 5 (b): Angiographic demonstration of the LAD artery

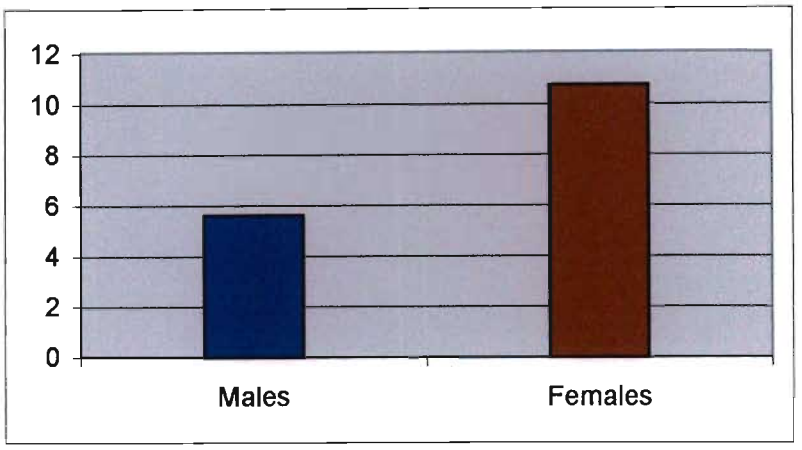
A varying pattern of myocardial investment of the LAD was observed in 50.6% of hearts. Myocardial bridges were recorded over the proximal third of the LAD in 21.4%, over the middle third in 80.9% and distal third in 11.9%. The LAD was intra myocardial in 11.9% and in 14.3% the bridge extended over 2 segments. Detailed description of myocardial bridges in relation to the LAD are discussed in section two of this chapter.

In the bifurcation pattern of termination of the LCA, the left division was identified as the LCX. As the LCX traveled laterally from its site of origin, it adhered to the inferior surface of the left atrial appendage by means of a film of epicardial tissue. The artery continued posteriorly within the coronary groove in 72.3% to reach the crux and give off a PDA in 6% of hearts. In 12%, the LCX gave off a smaller PDA equal in size to the posterior ventricular contribution from the RCA.

In the remaining 27.7% of hearts, the LCX failed to pass along the coronary groove, but instead, ended abruptly along the obtuse margin of the left ventricle. The arterial pattern usually consisted of a single main trunk with a random distribution of secondary branches along the delineated cardiac territory.

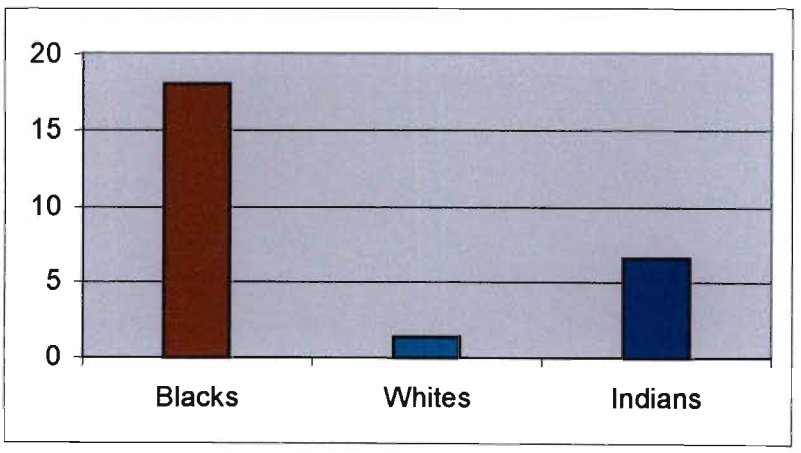
Incidences of findings with regards to sex and ethnicity are presented in a series of tables and graphs that follow.

Results are reported for a combination of data recorded from cadaveric dissections and analysis of angiograms. A total of 226 arterial patterns were selected for gender analysis and consisted of 161 males and 65 females.



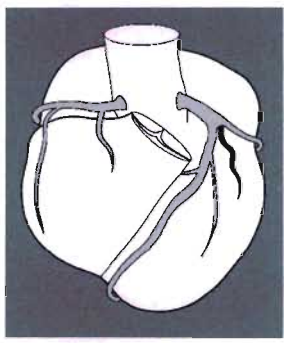
Graph 6: Sex distribution (n = 226)

For evaluation between ethnic groups, the total sample consisted of 231 where Blacks constituted 68, Whites 67 and Indians 96. Where analysis was limited to cadaveric hearts, the group distribution for selected patterns was as follows: sex distribution 77 (52 males and 25 females) and ethnic distribution 83 (61 Blacks, 20 Whites and 2 Indians).



Graph 7:Sex distribution (n = 226)

Table 6: Recorded incidences of the presence of the Ramus Marginalis

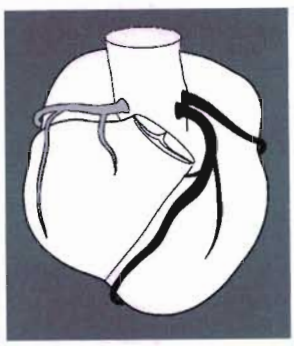


PRESENCE OF RAMUS MARGINALIS

VARIABLES	SAMPLE SIZE	INCIDENCE %
SEX	226	
Males	161	5.6
Females	65	10.7
ETHNICITY	231	
Blacks	68	18.0
Whites	67	1.4
Indian	96	6.6

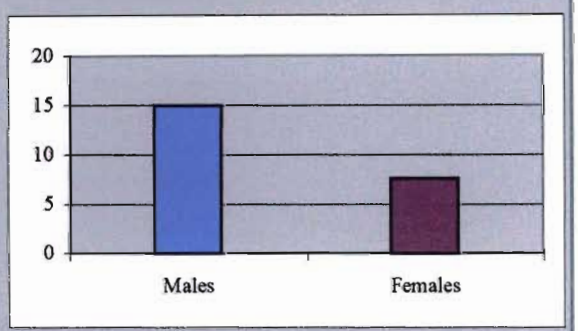
The ramus marginalis is found to be present in 10.7% of females and 5.6% of males. Mean incidence was highest in Blacks (18.0%), followed by Indians (6.6%) and then Whites (1.4%), (Table 6 and Plates 15, 16, 17 and 23).

Table 7: Recorded incidences of separate origin of LAD and LCX arteries



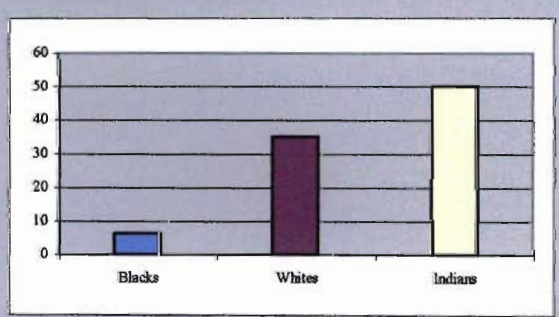
SEPARATE ORIGIN OF LAD & LCX

VARIABLES	SAMPLE SIZE	INCIDENCE %
SEX	77	
Males	52	15%
Females	25	7.6%



Graph 8 (a): Separate Origin of LAD- Males Vs Females

ETHNICITY	83	
Blacks	61	6.5%
Whites	20	35%
Indians	2	50%



Graph 8 (b): Separate Origin of LAD- Males Vs Females

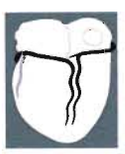
An incidence of 15% of separate origin of LAD and LCX, (Plate 19) was recorded in males and 7.5% in females. Pearson chi squared was significant at $p = 0.03$. Amongst ethnic groups, Indians were found to have an incidence of 50%, Whites, 35% and Blacks, 6.5% of separately originating LAD and LCX arteries.

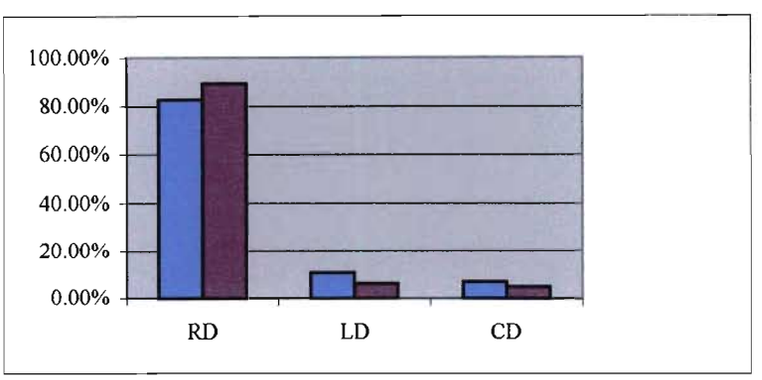
ARTERIAL DOMINANCE

Of a total of 226 arterial patterns studied, 85.9% showed a tendency for right dominance. The incidences for left dominance were 8.3% and co-dominance, 5.7%., (Plates 20, 21 and 26)

Amongst males, 82.6% of arterial patterns were right dominant, 10.5%, left dominant and 6.8% co-dominant. Females had recorded incidences of 89.2%, 6.1% and 4.6% for each dominance type, respectively.

Table 8: Recorded incidences of arterial dominance

INCIDENCE OF DOMINANT PATTERN			
ARTERIAL PATTERN (N = 226)		**SEX** Male (N =161)	Female (N = 65)
	Right Dominance	82.6 %	89.2%
	Left Dominance	10.5%	6.1%
	Co-Dominance	6.8%	4.6%

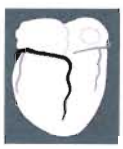


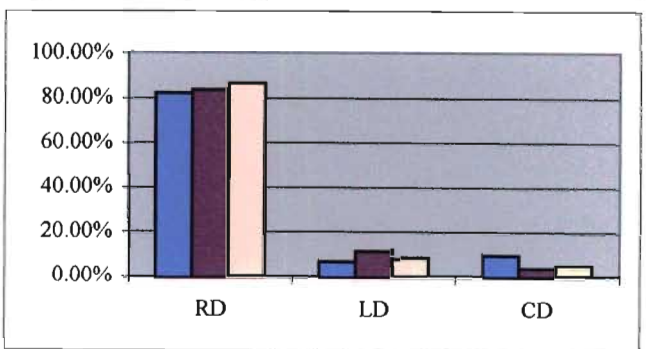
Graph 9 (a): Incidence of dominant pattern

Amongst the ethnic groups studied, right dominant hearts were recorded in 82.3% of Blacks, 83.6% of Whites and 86.4% of Indians. Left dominant patterns were observed in 7.3% of Blacks, 11.9% of Whites and 8.3% of Indians. In each of these

groups, co-dominance was recorded with the following percentages: Blacks, 10.3%; Whites, 4.5% and Indians, 8.3%.

Table 9: Recorded incidences of arterial dominance between ethnic groups

INCIDENCE OF DOMINANT PATTERN			
ARTERIAL PATTERN	**ETHNICITY**		
	Blacks	Whites	Indians
Total Sample (N = 231)	68	67	96
 Right Dominance	82.3%	83.6%	86.4%
 Left Dominance	7.3%	11.9%	8.3%
 Co-Dominance	10.3%	4.5%	5.2%



Graph 9 (b): Incidence of dominant pattern amongst ethnic groups

No significant p-values were recorded for sex, age, ethnic group or height when measured against arterial dominance.

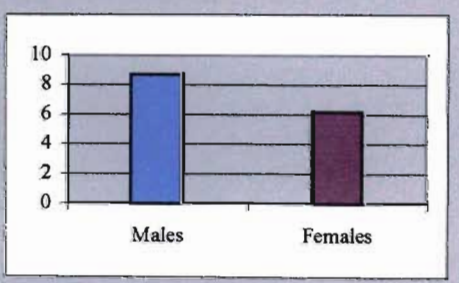
THE BIFID LAD

A bifid LAD was recorded in 8.7% of males and 6.2% of females. The incidence recorded for Blacks was 17.6%, Whites, 4.5% and Indians, 6.3%., (Plates 24, 28).

Table 10: Recorded incidences for the presence of a bifid LAD

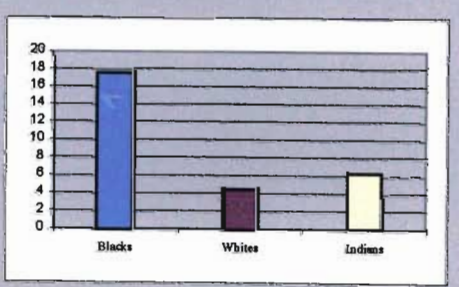
PRESENCE OF BIFID LAD

VARIABLES	SAMPLE SIZE	INCIDENCE %
SEX	**226**	
Males	161	8.7
Females	65	6.2



Graph 10 (a): Presence of a bifid LAD – Males Vs females

ETHNICITY	**231**	
Blacks	68	17.6
Whites	67	4.5
Indians	96	



Graph 10 (b): Presence of a bifid LAD – Ethnicity

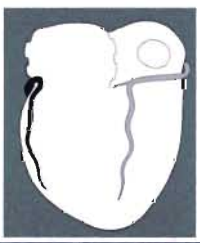
A p value of 0.003 was recorded for the presence of bifid LAD between ethnic groups.

ANOMALOUS PATH TAKEN BY THE LCX

In 28.8% of males, the LCX was observed in an anomalous path, failing to continue within the left atrio-ventricular groove, (Plate 22, Table, 11). The incidence recorded in females was 24.0%.

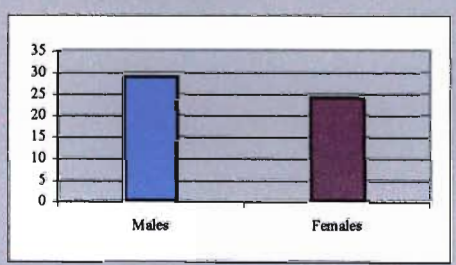
Blacks showed an incidence of 29.5% whereas Whites and Indians had recorded incidences of 20% and 50% respectively. The p Value was significant at 0.04 for ethnicity.

Table 11: Recorded incidences of an anomalous path taken by the LCX



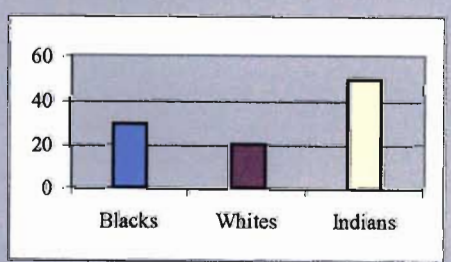
ANOMALOUS PATH TAKEN BY LCX

VARIABLES	SAMPLE SIZE	INCIDENCE %
SEX	**77**	
Males	52	28.8
Females	25	24.0



Graph 11 (a): Anomalous path of LCX artery – Males Vs females

ETHNICITY	**83**	
Blacks	61	29.5
Whites	20	20.0
Indians	2	50.0



Graph 11 (b): Anomalous path of LCX artery – Ethnicity

ORIGIN OF THE CONUS ARTERY

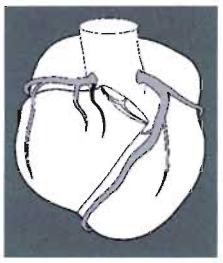
An incidence of 21% in males and 16% in females was recorded for a high origin of the conus artery, Plates 7, 14 and Tables 12, 13).

The incidences for Blacks, Whites and Indians were 19.7%, 10% and 100% respectively.

Pearson chi squared was significant at $p = 0.009$ for ethnicity.

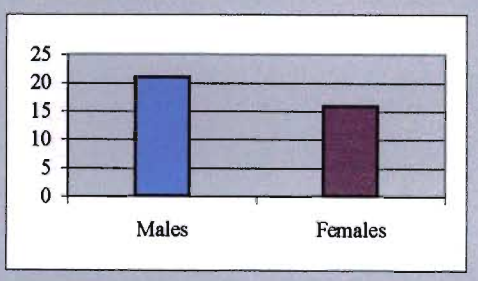
In 3.8% of males and 4.0% of females, the conus artery was observed to originate from an independent ostium. This separate origin of the conus artery was observed in 4.9% of Blacks only.

Table 12: Recorded incidences of a high origin of the conus artery



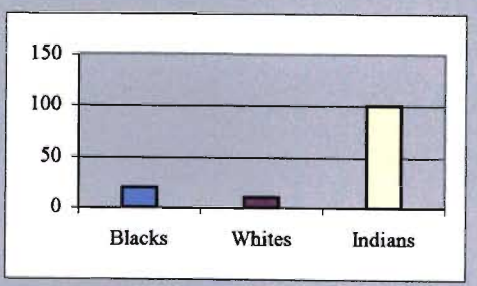
HIGH ORIGIN OF CONUS ARTERY

VARIABLES	SAMPLE SIZE	INCIDENCE %
SEX	**77**	
Males	52	21
Females	25	16



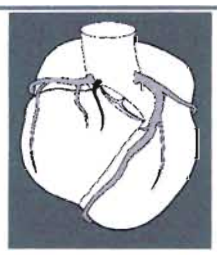
Graph 12 (a): high origin of conus artery – Males Vs females

ETHNICITY	**83**	
Blacks	61	19.7
Whites	20	10.0
Indians	2	100.0



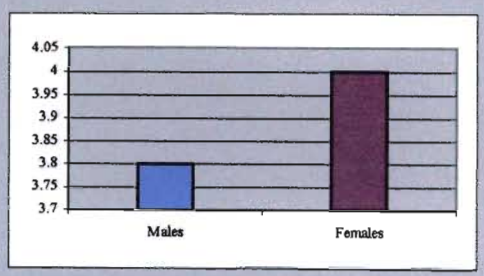
Graph 12 (b): High origin of conus artery – Ethnicity

Table 13: Recorded incidences of a separate origin of the conus artery



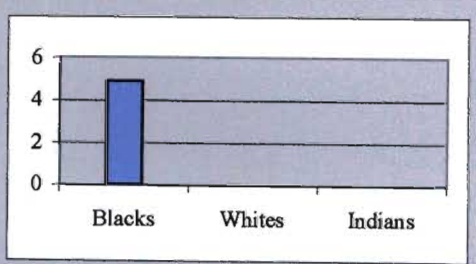
SEPARATE ORIGIN OF CONUS ARTERY

VARIABLES	SAMPLE SIZE	INCIDENCE %
SEX	77	
Males	52	3.8
Females	25	4.0



Graph 13 (a): Separate origin of couns artery – Males Vs females

ETHNICITY	**83**	
Blacks	61	4.9
Whites	20	0
Indians	2	0



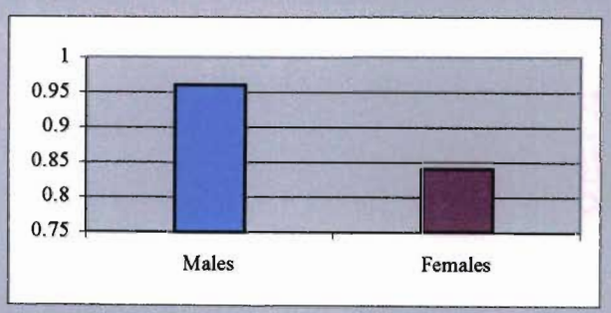
Graph 13 (b): Separate origin of couns artery – Ethnicity

LENGTH OF THE LEFT MAIN TRUNK

The mean length of the LCA recorded for males was 0.96cm and 0.84cm for females. Results of the T-test were approaching significance at the 5% level ($p > 0.05$), (Plate 7 and Table 14).

Table 14: Recorded incidences of the length of the left main in sexes

LENGTH OF LEFT MAIN TRUNK		
SEX	**Sample Size**	**Mean Length (cm)**
Total	77	0.82
Males	52	0.96
Females	25	0.84

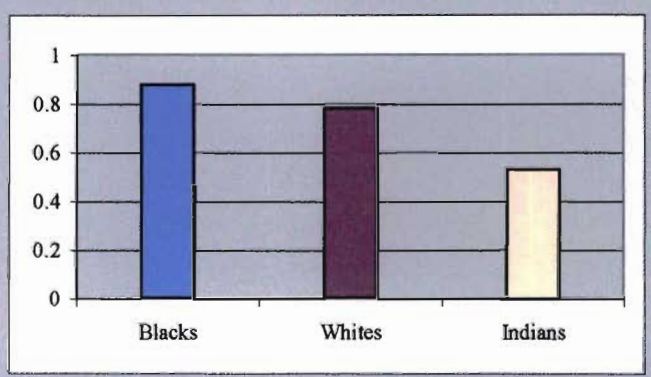


Graph 14 (a) : Length of the left main trunk – Males Vs females

A mean length of 0.88cm was recorded for Blacks. The LCA within the White group measured 0.78cm and within the Indian group, the LCA was found to be 0.53cm. Statistical significance exists between ethnic groups with regards to LCA length.

Table 15: Recorded incidences of the length of the left main in ethnic groups

LENGTH OF LEFT MAIN TRUNK		
ETHNIC GROUP	**Sample Size**	**Mean Length (cm)**
Total	83	0.82
Blacks	61	0.88
Whites	20	0.78
Indians	2	0.53



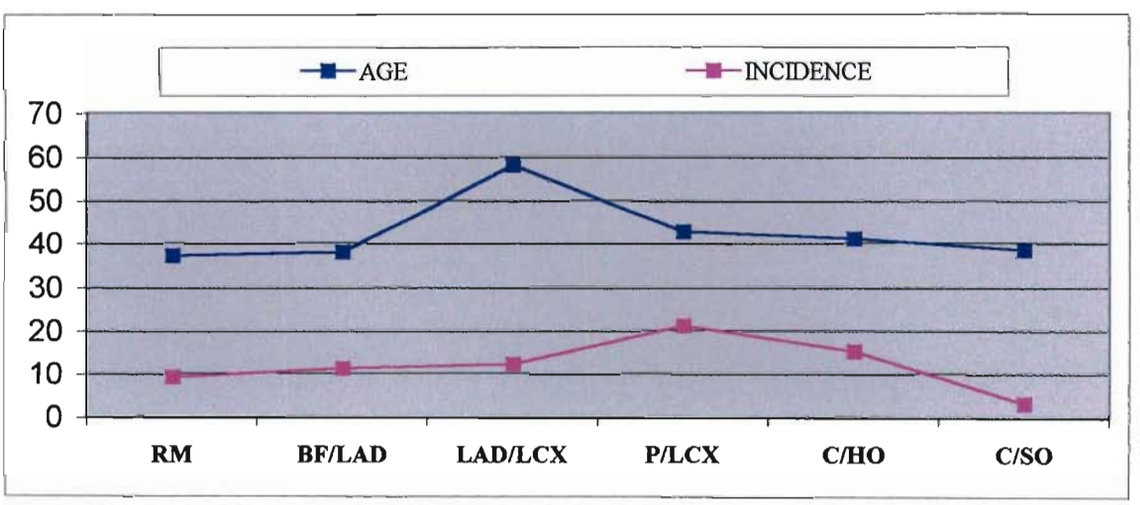
Graph 14 (b): Length of the left main trunk - Ethnicity

AGE CORRELATION ON INCIDENCES OF FINDINGS

Incidences for each of the variables were correlated against age. Statistical analysis showed no correlation between age and the presence of anomalies.

Table 16: Age correlation on incidences of findings

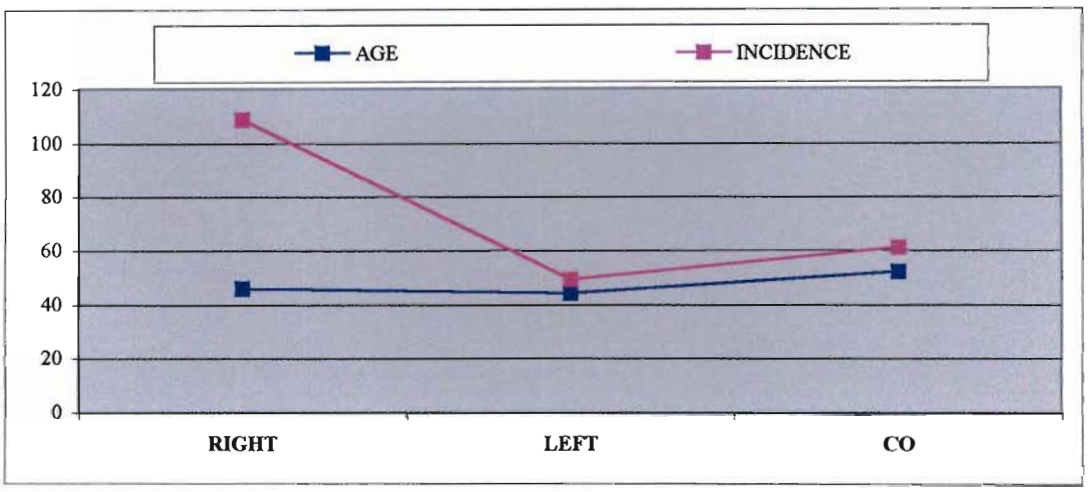
AGE CORRELATION ON INCIDENCES OF FINDINGS							
	TOTAL	**RM**	**BF/LAD**	**LAD/LCX**	**P/LCX**	**C/HO**	**C/SO**
Mean	46.4	37	37.8	58.17	42.5	41.0	38.33
N	77	9	11	12	21	15	3
Std D	20.2	13.9	12.6	27.03	0.07	14.7	18.6



Graph 15: Age correlation on incidences of findings

Table 17: Age correlation on arterial dominance

AGE CORRELATION ON ARTERIAL DOMINANCE				
	TOTAL	**RIGHT**	**LEFT**	**CO**
Mean	46.4	45.73	44.0	52.0
N value	77	63	5	9
Std D	20.2	20.2	22.8	20.1



Graph 16: Age correlation on arterial dominance

Statistical analysis showed no correlation between age and arterial dominance.

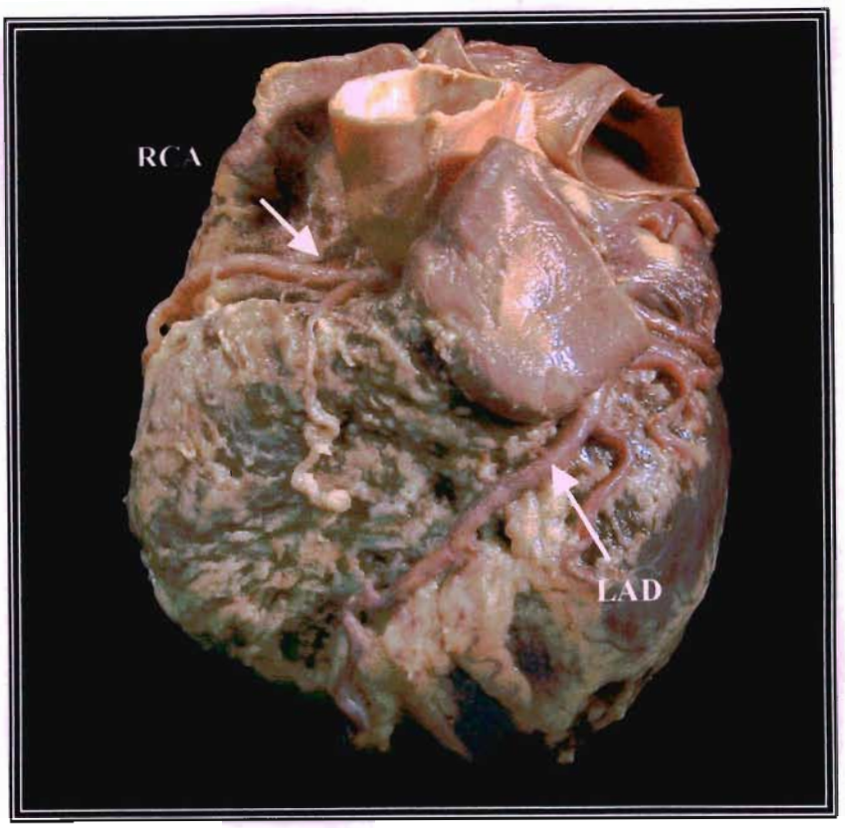


Plate 6: Anterior view of the LAD and RCA

Plate 6 presents an antero-lateral view of the heart orientated at 90° to the right of the anatomical plane. The transected pulmonary trunk is seen as the most anterior great vessel with the ascending aorta behind and a little to the right of it. Posterior to the ascending aorta, lies the superior margin of the right atrial appendage to the right and the lumen of the left superior pulmonary vein to the left. The RCA is clearly visible as it descends along area for the right atrio-ventricular groove. Its first branch, the conus artery is seen arising close to the origin of the RCA and passing toward the left to form an anastomosis on the anterior wall of the infundibulum. This dissection clearly demonstrates the suspended position of the RCA within the right atrio-ventricular groove. In an un-dissected heart, the atrioventricular groove is filled with a significant amount of loose fatty connective tissue, giving the appearance of the RCA coursing within the groove. The LAD is also clearly visible in this view. A large diagonal branch is seen passing along the antero-lateral surface of the left ventricle and smaller ventricular branches appear along the distal segment of the LAD. The ventricular branches are shown in a typical location within a flattened fat pad. Both the LCA and the LCX are obscured by the lateral displacement of the pulmonary trunk.

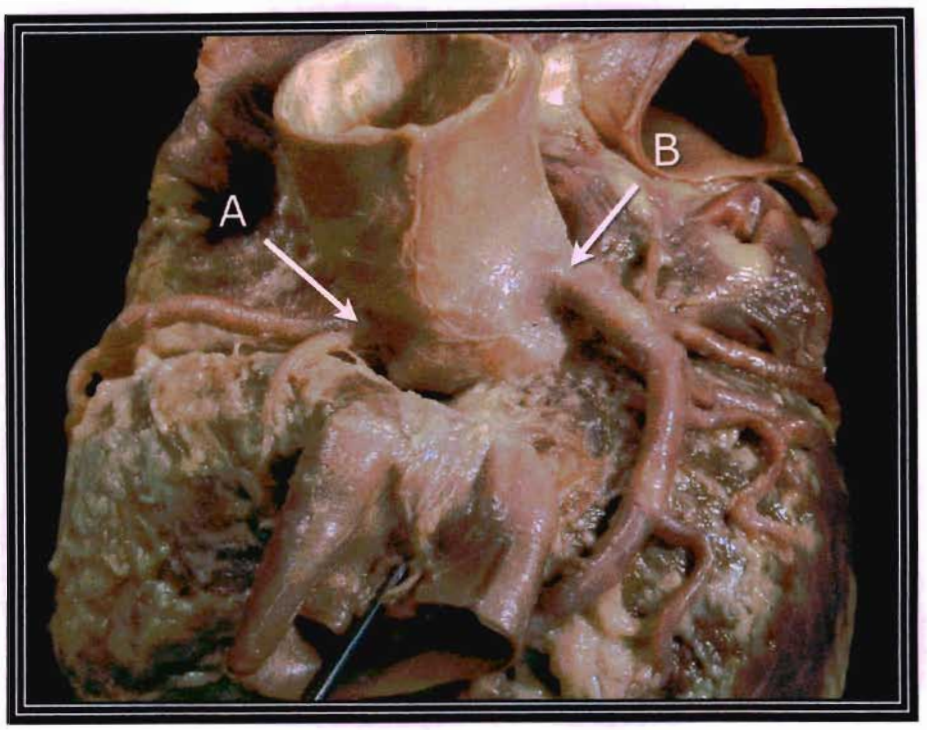


Plate 7:Antero-superior view of the LCA (B) and RCA (A)

The heart is orientated at 90° to the right of the anatomical plane. The pulmonary trunk has been retracted anteriorly to expose the LCA. The LCA bifurcation is visible in line with the apex of the left atrial appendage. The LCX is shown passing posteriorly along the coronary groove. The LAD is shown curving along the infundibular margin with 2 lateral diagonal branches, exhibiting a fair degree of tortuosity.

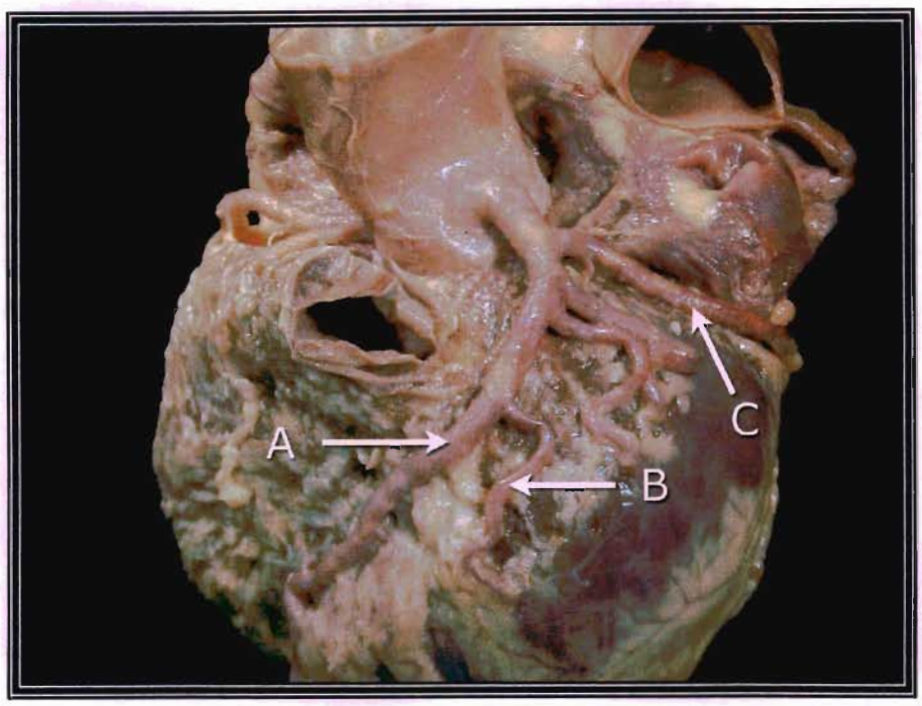


Plate 8: Antero-lateral view of the LCA system
(A: LAD, B: diagonal branch, C: LCX)

The heart is orientated to bring the anterior LCX into focus. The first branch of the LCX passes superiorly to supply the left atrium. At this point, the LCX is somewhat anchored in a loop by this atrial branch and by the epicardial tissue of the left atrial appendage. In this specimen, no branches are seen arising from the LCX. The LCX passes downward and posteriorly, hugging the lower limit of the left atrium as it continues toward the crux.

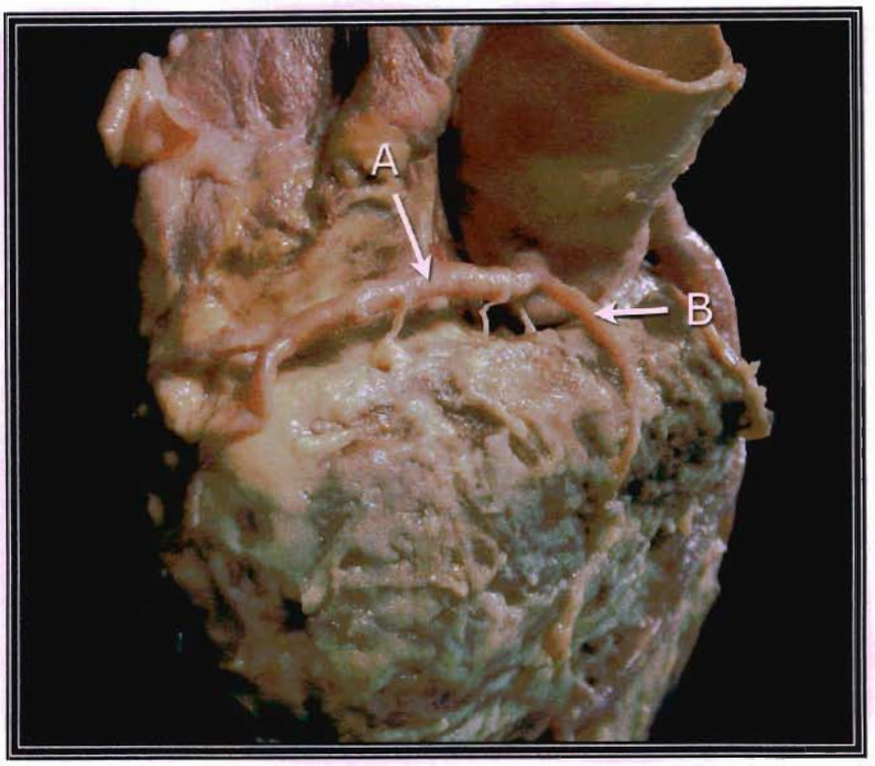


Plate 9: Right lateral view of the RCA (B) and its conus branch (B)

In this view, the heart is orientated with the apex at 90° to the surface. In this position, the right atrium is held upright so that the depth of the right atrio-ventricular groove is decreased. The distance between the inferior surface of the RCA and the sulcus is demonstrated. The RCA is seen passing downward and posteriorly as it tapers toward the crux. The extent of the conus branch is also seen clearly in this view.

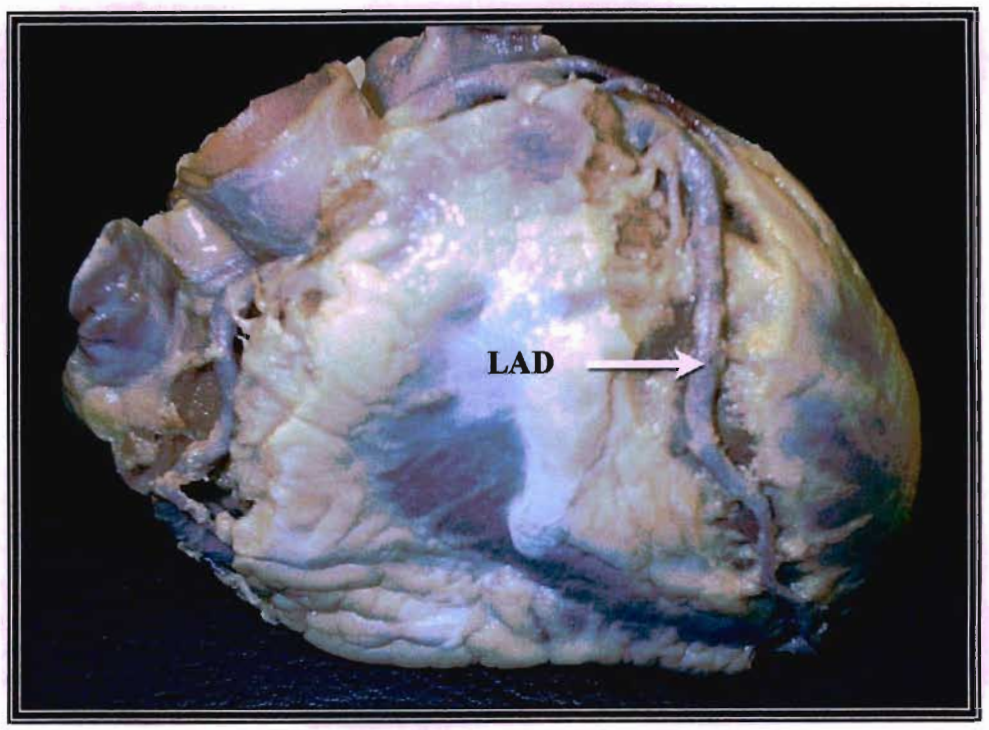


Plate 10: Anterior view of the LAD within the inter ventricular groove

The heart is placed with the diaphragmatic surface on the table. The LAD is positioned to the left of the anterior inter-ventricular groove. A distinct tapering of the LAD is demonstrated in this heart. The distal segment appears to increase in tortuosity which is typical for this portion of the vessel.

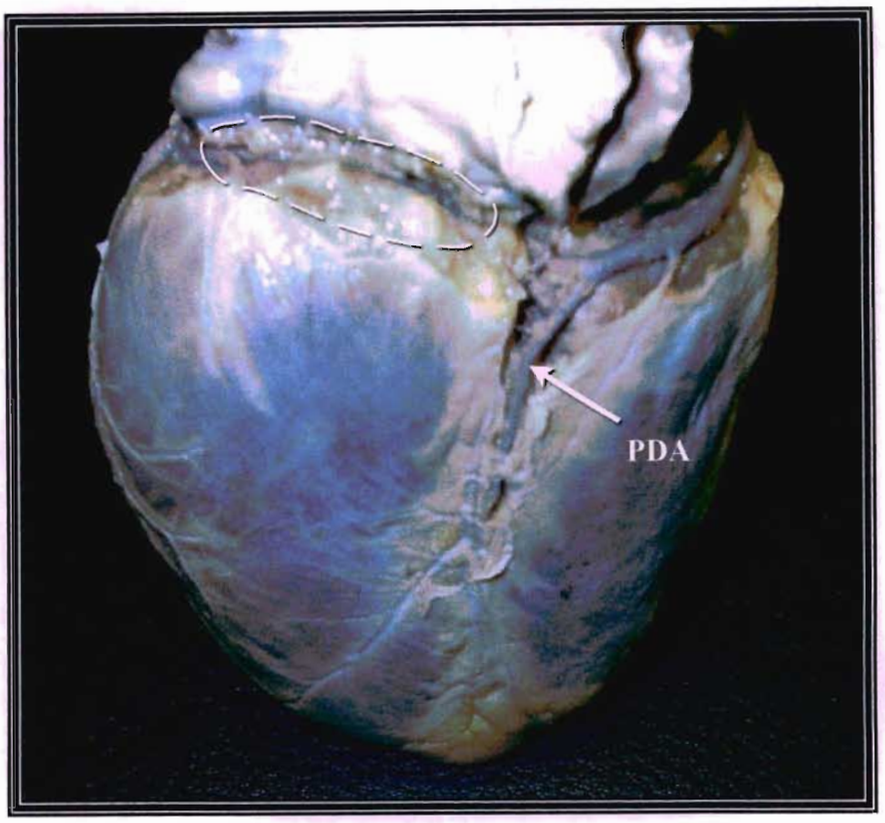


Plate 11: Posterior view of the PDA with absent posterior LCX artery

This is a posterior view of the heart oriented at 90° to the surface. The posterior wall of the left atrium is seen as the portion superior to the coronary groove. The RCA appears to the left and shows the formation of the PDA. In this dissection, the dense epicardium was removed in order to demonstrate the origin of the PDA at the crux and its course down the posterior inter-ventricular groove. In its posterior course, the distal portion of the RCA maintains closer contact with the ventricular wall.

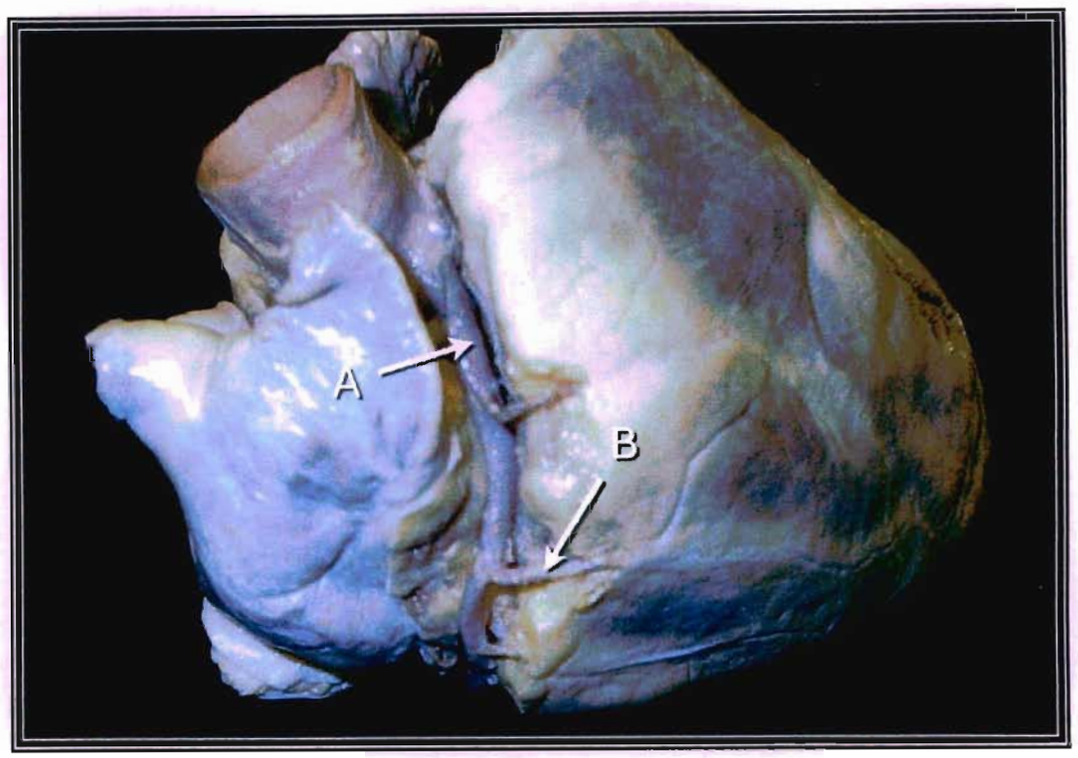


Plate 12: Anterior view of the RCA (A) and ventricular branch (B) in the right atrio-ventricular groove

The RCA has been exposed within the atrio-ventricular area. The RCA in this position is anchored via ventricular branches to the atrial wall of the right ventricle. The lowest ventricular branch runs along the acute margin of the heart. It is fairly small in this specimen and runs within a significant amount of fat.

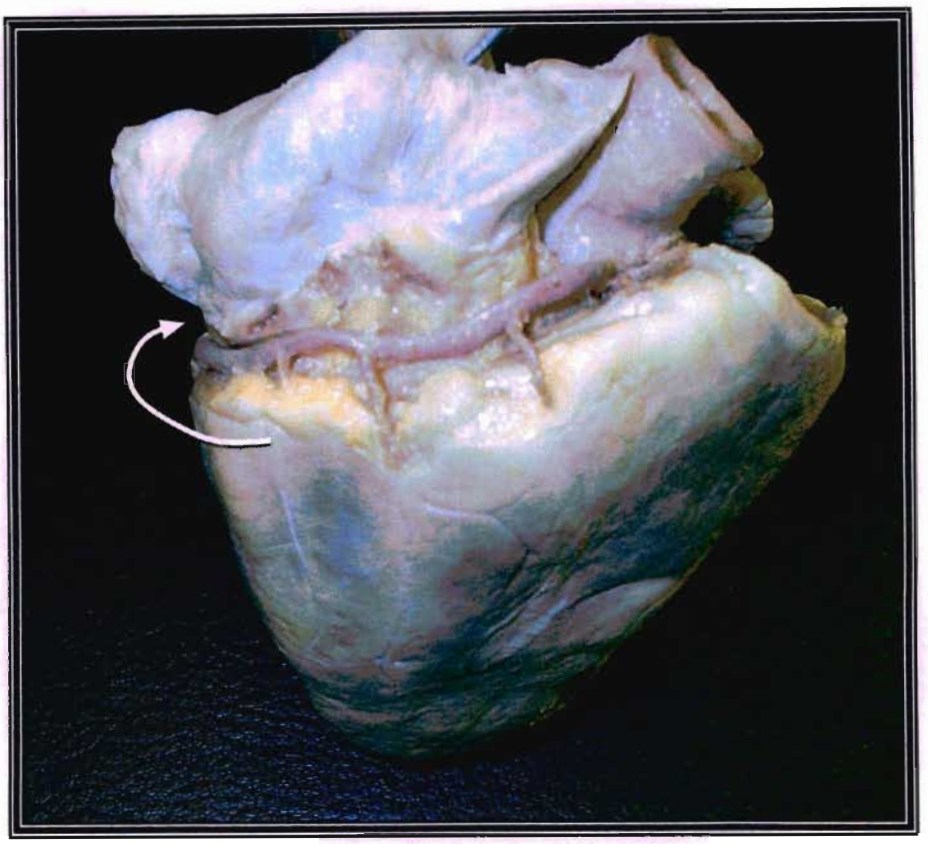


Plate 13: Posterior course of the RCA indicated by a curved arrow

The heart is orientated to demonstrate the atrial and ventricular surface with the apex placed at 90° to the table. The RCA is visible from its emergence and along its length in the atrio-ventricular area with ventricular branches anchoring the artery to the right ventricular wall.

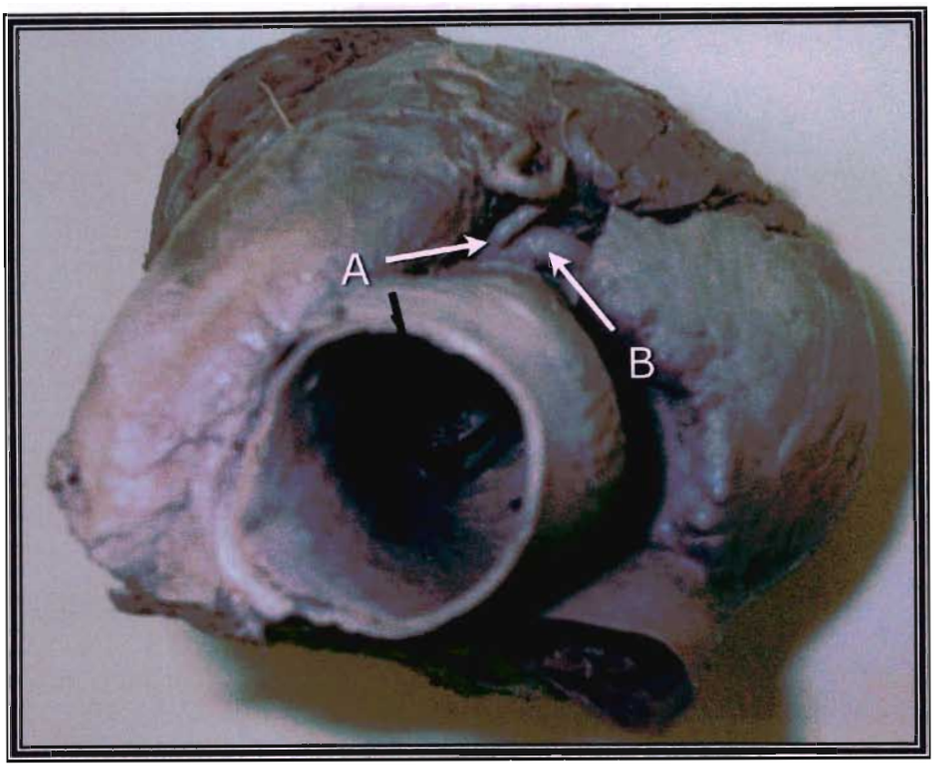


Plate 14: Superior view of a separate conus artery (A) and RCA (B) origination

The heart is orientated in the anatomical position with a view from above. The aorta has been transected with an angle looking down the left ventricle. The aortic cusp is visible within the lumen on the right posterior wall. To the left of the aorta is the pulmonary trunk, passing backwards. The RCA may be observed as it emerges from the aorta to pass beneath the apex of the right atrial appendage. The conus branch of the RCA is seen arising from the aorta. It passes to the left in a tortuous course across the infundibulum.

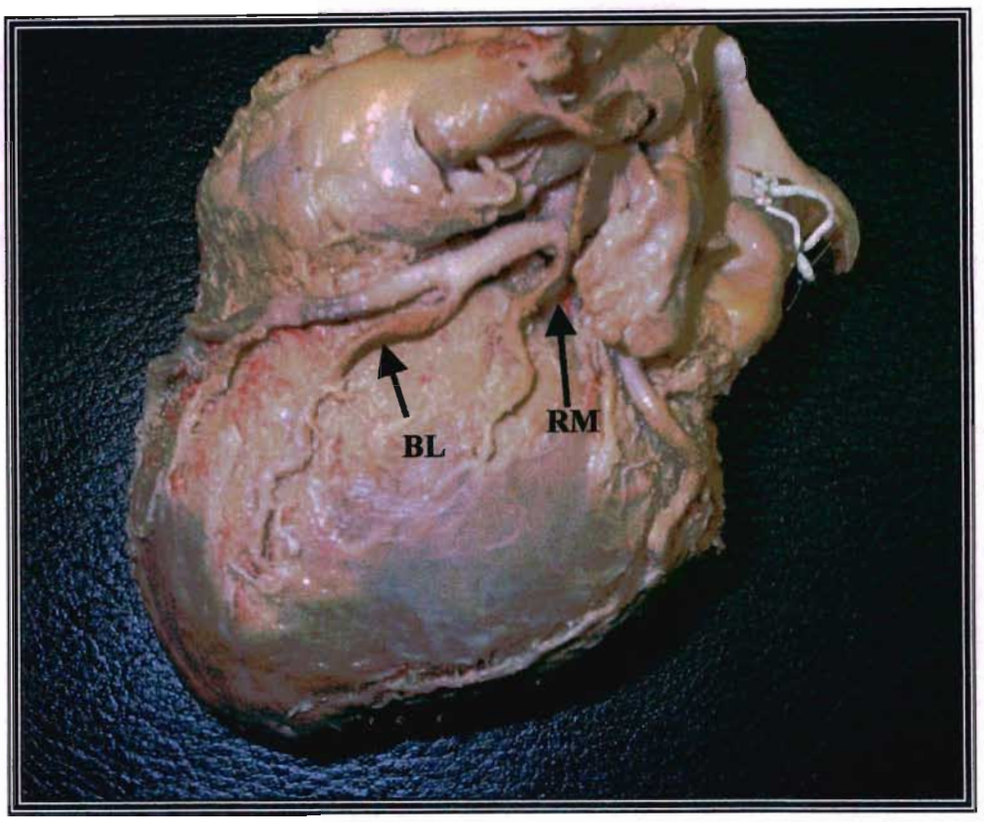


Plate 15: Antero-lateral view of bifid LAD (BL) and ramus marginalis (RM)

The heart is placed on its diaphragmatic surface with an anterior view of the left ventricle. The LAD is shown passing superiorly and inferiorly to the right of the heart. The first diagonal branch is visible. The second diagonal branch is of a size comparable to the middle LAD. When filled, this branch may be equal in size to the LAD. The LCX is seen to the left of the heart as it emerges from beneath the left atrial appendage. The LCX in this heart fails to reach the crux and deviates in its course to follow a path along the obtuse margin of the left ventricle.

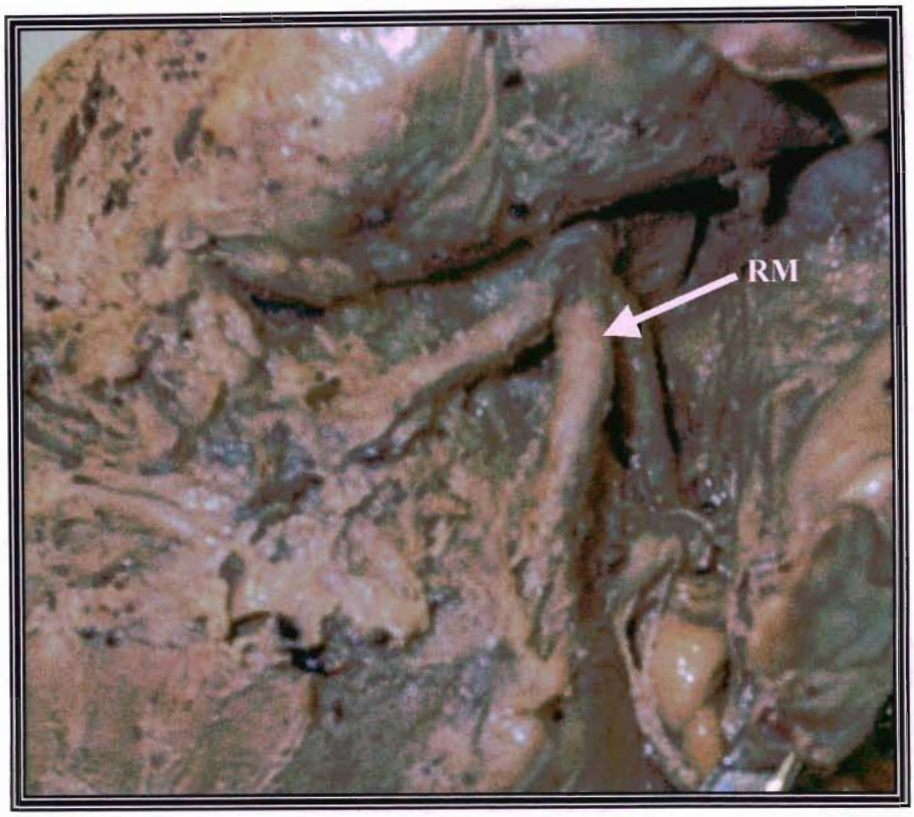


Plate 16: Lateral view of a trifurcation pattern of termination of the LCA showing ramus marginalis branch (RM)

The heart is being viewed with the focus on the area between the pulmonary trunk and the left atrial appendage. The pulmonary trunk lies superiorly at an angle. Behind it, lies the ascending aorta, with its root visible below the lower margin of the pulmonary trunk. The LCA can be seen emerging from the aortic sinus. The pattern of branching is in the form of a trifurcation. The branch to the right continues as the LAD, to the left, the LCX and in the center, the ramus marginalis.

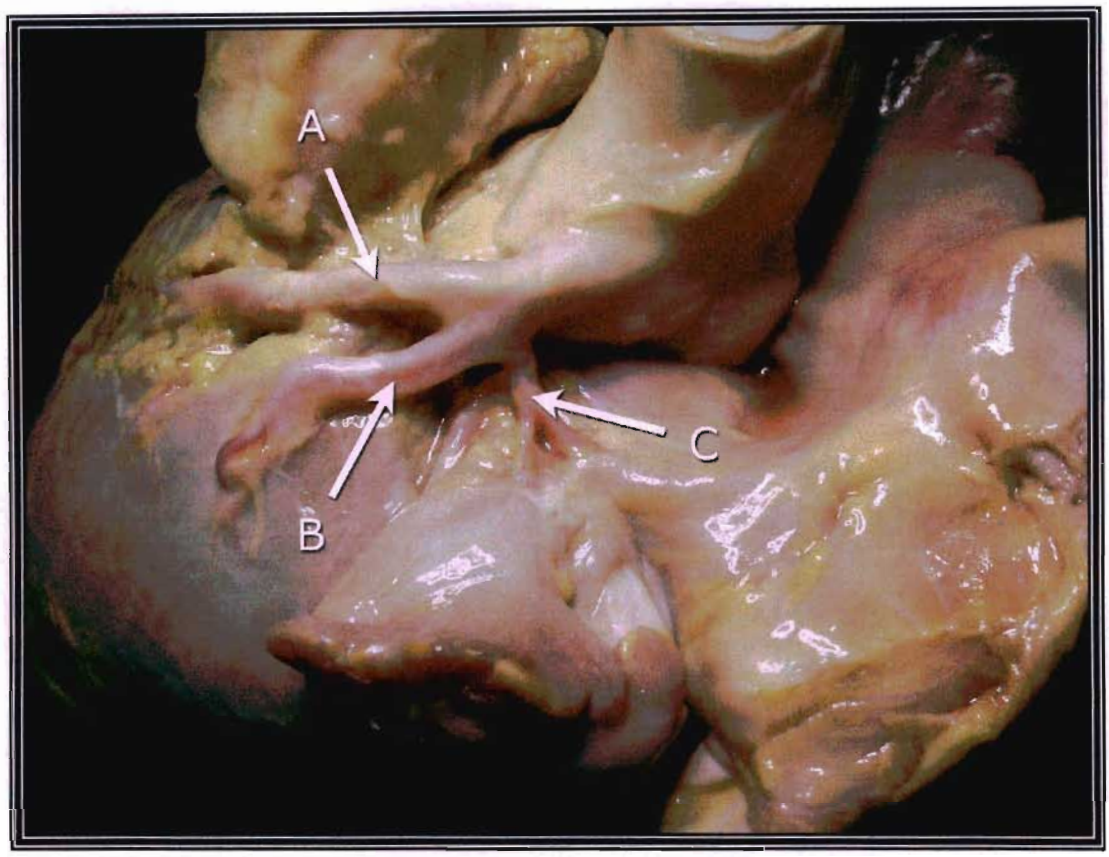


Plate 17: Lateral view of ramus marginalis (B), LAD (A) and LCX (C) arteries

The heart is being viewed with the focus on the area between the pulmonary trunk and the left atrial appendage. The LCA can be seen emerging from the aortic sinus. The pattern of branching is in the form of a trifurcation to give rise to the ramus marginalis (B), the LCX (C) and the LAD (A).

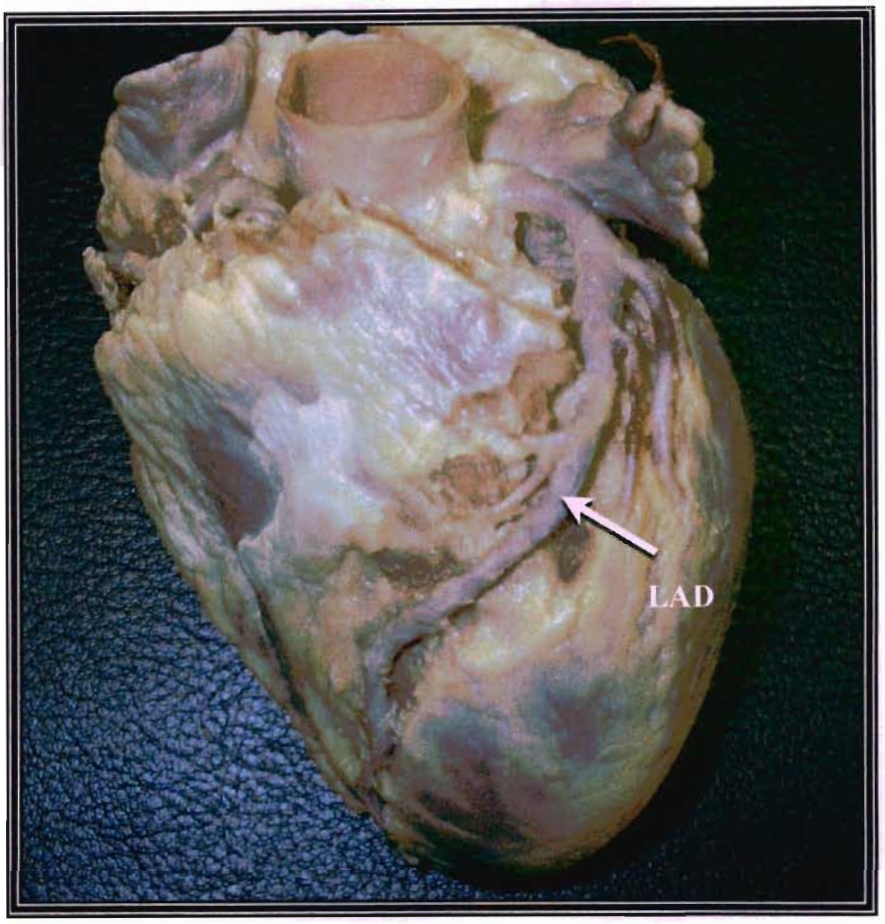


Plate 18: Anterior view of an epicardial position of the LAD

The heart has been orientated at 90° to the right. The anterior inter-ventricular grove is in focus. The LAD is shown passing down the groove. To the left are two diagonal branches and to the right, a bifid septal perforator.

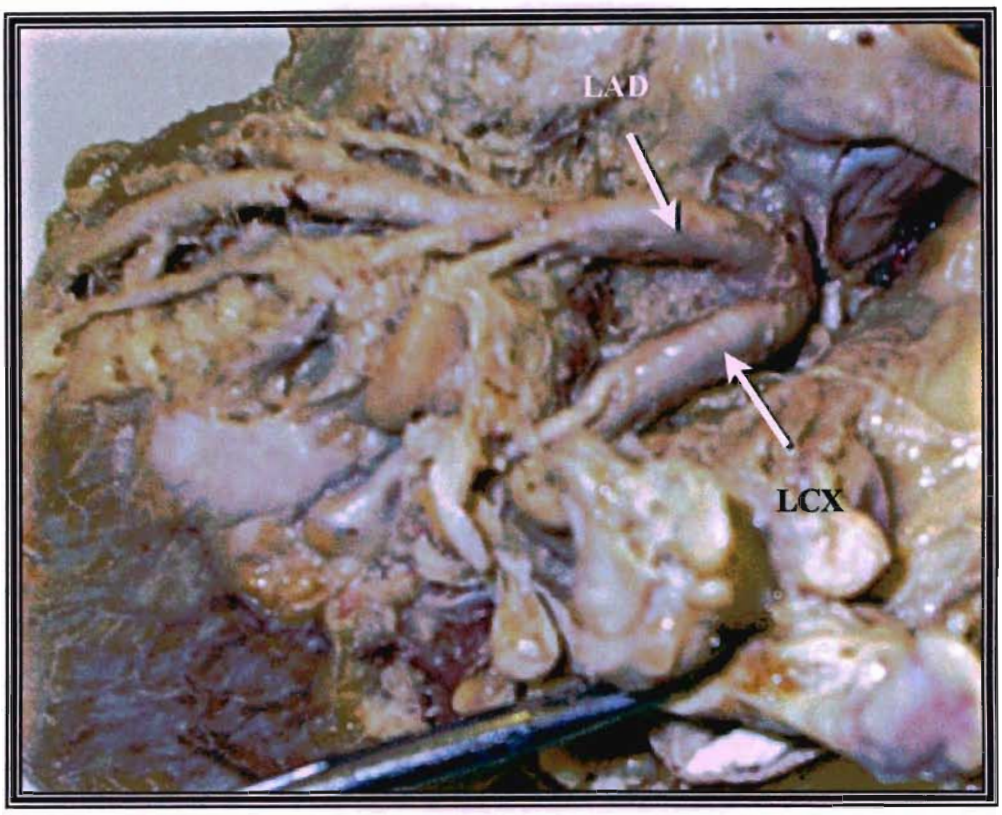


Plate 19: Separate origin of the LAD and LCX

The heart has been rotated at 160° to the right. The left atrial appendage has been retracted to expose the origin of the LAD and LCX. Both these vessels are shown to arise from separate ostia. The LAD is the vessel passing superiorly and to the right. The LCX passes below to continue along the obtuse margin of the left ventricle.

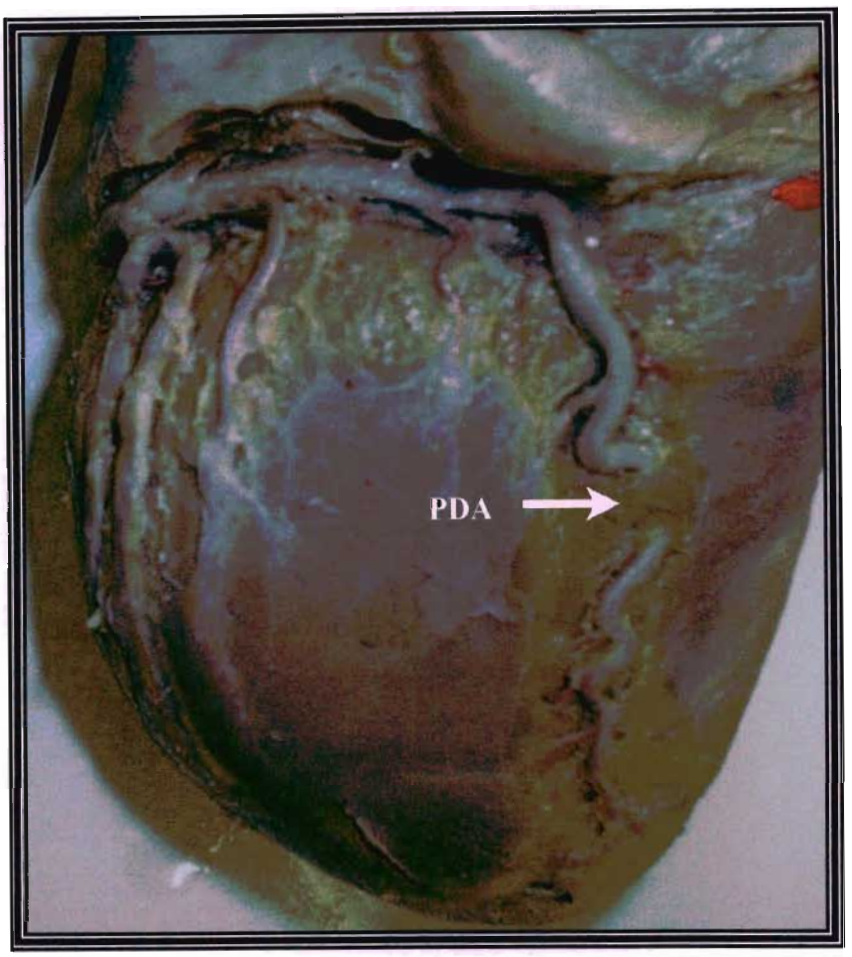


Plate 20: Posterior view of a bridged PDA in a left dominant heart

The heart has been orientated with the posterior ventricular surfaces in focus. The LCX is seen passing from in front and continuing along the coronary groove. Three obtuse marginal branches descend along the left ventricle. The first 2 to the right come off a common trunk. The LCX continues beyond the crux to form the PDA. A myocardial bridge is seen passing across a part of the middle segment of the PDA.

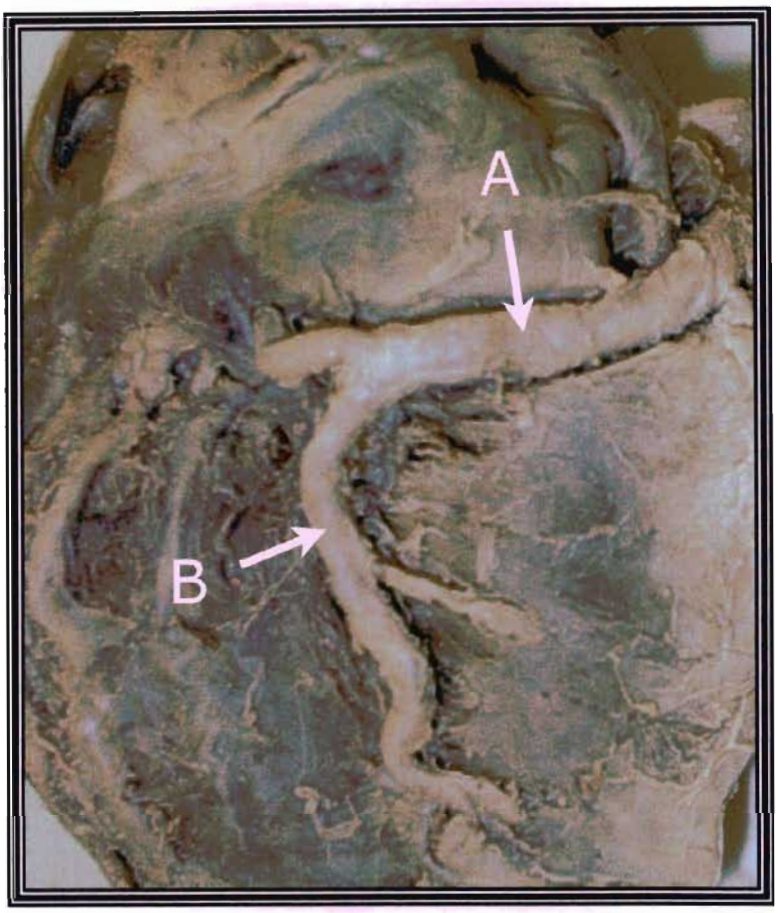


Plate 21: Posterior view of the RCA (A) and PDA (B) in a right dominant heart

The heart has been orientated with the posterior ventricular surfaces in focus. A very large RCA is seen superiorly on the left of the picture. The RCA (A) continues in the atrio-ventricular groove, down the coronary groove beyond the crux to form the PDA (B). The PDA is comparable in size to the RCA. A smaller component of the RCA continuation is seen passing superiorly from the crux.

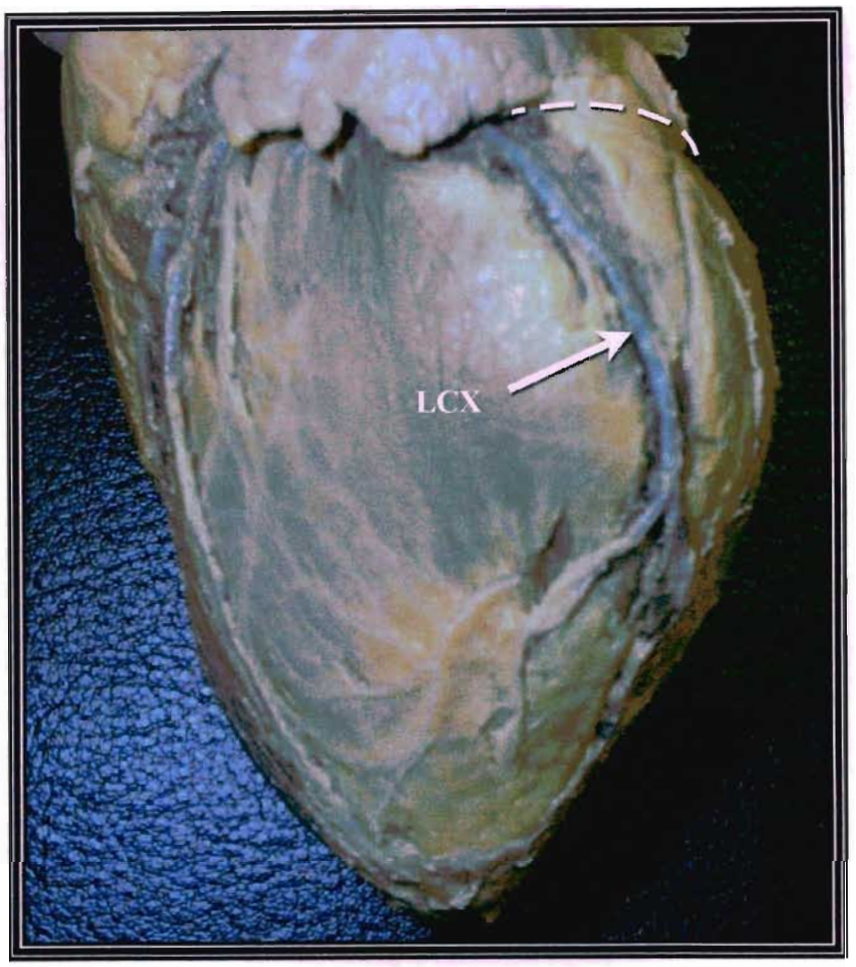


Plate 22: Anomalous path of the LCX artery ending along the obtuse margin

The heart has been placed on the surface with the right atrium and the right portion of the right ventricle at 180° to the surface. The apex is pointing inferiorly to show the obtuse margin of the left ventricle. The LCX is seen passing down this margin as it emerges from beneath the inferior margin of the left atrial appendage. In this heart, the LCX fails to reach the crux and there is no note of any subsidiary branch coursing the coronary groove.

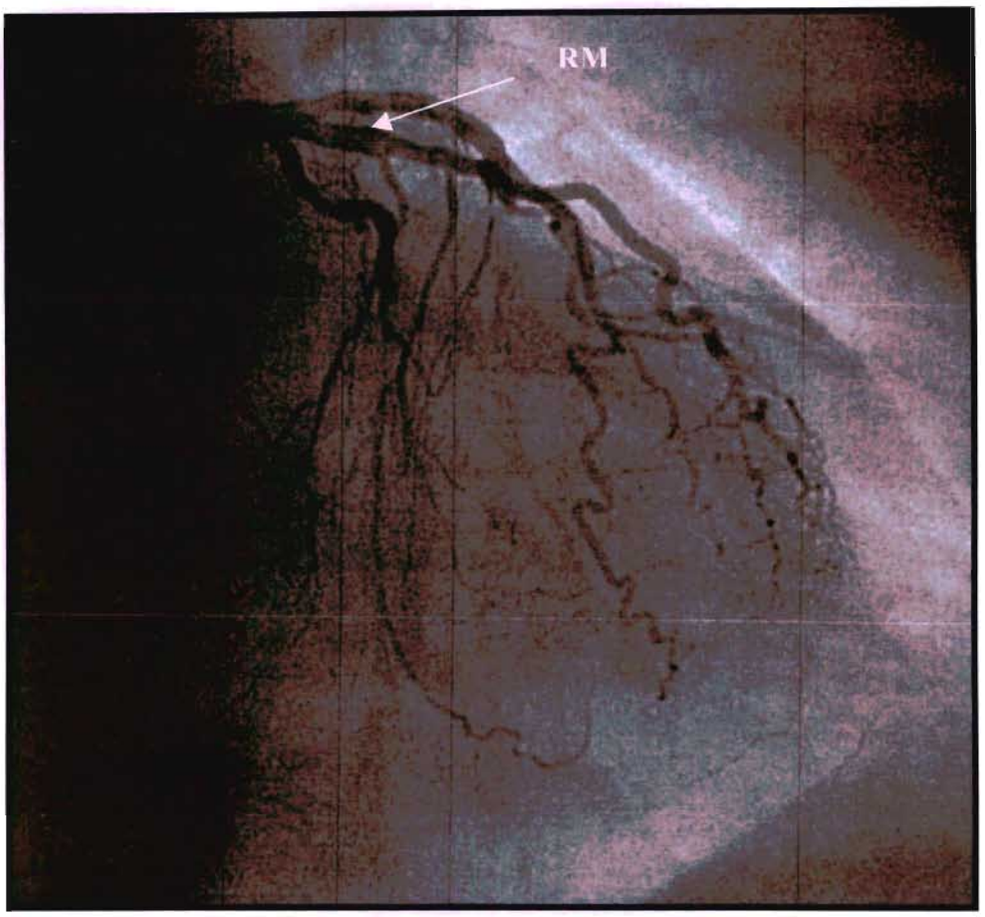


Plate 23: Angiographic demonstration of LCA trifurcation and ramus marginalis branch (RM)

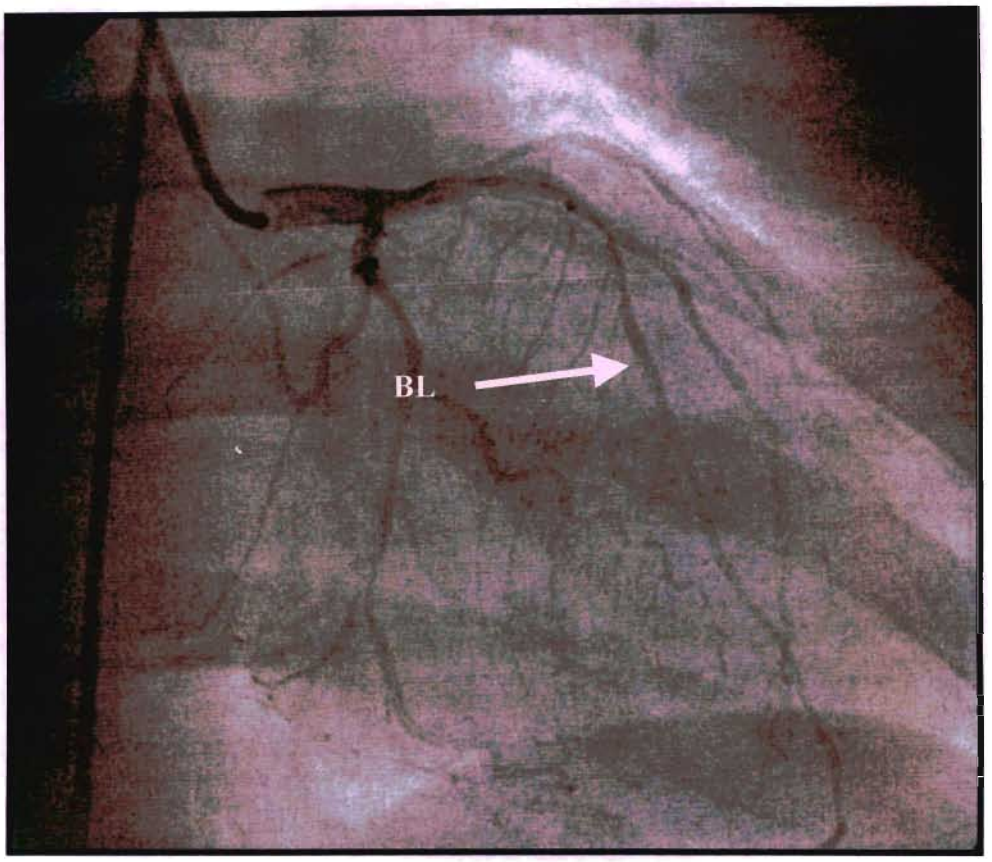


Plate 24: Angiographic demonstration of a bifid LAD (BL)

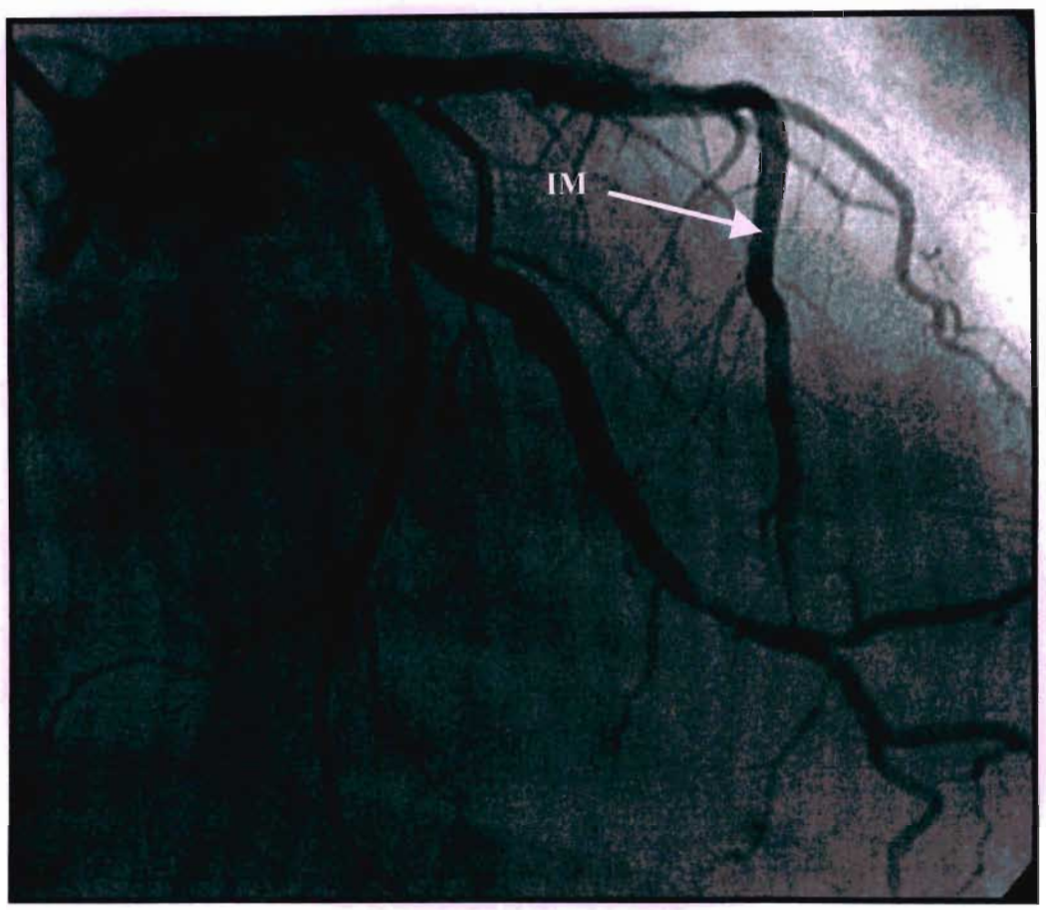


Plate 25: Angiographic demonstration of an intra-myocardial artery (IM)

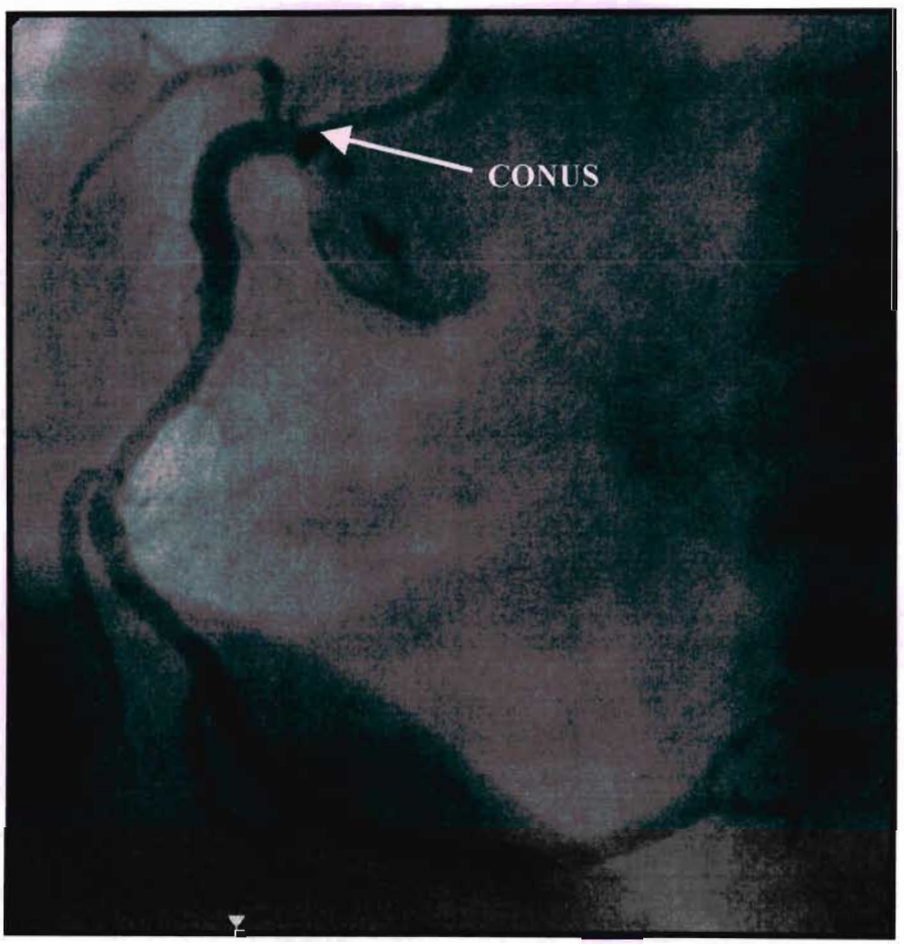


Plate 26: View of the RCA with high origin of conus artery

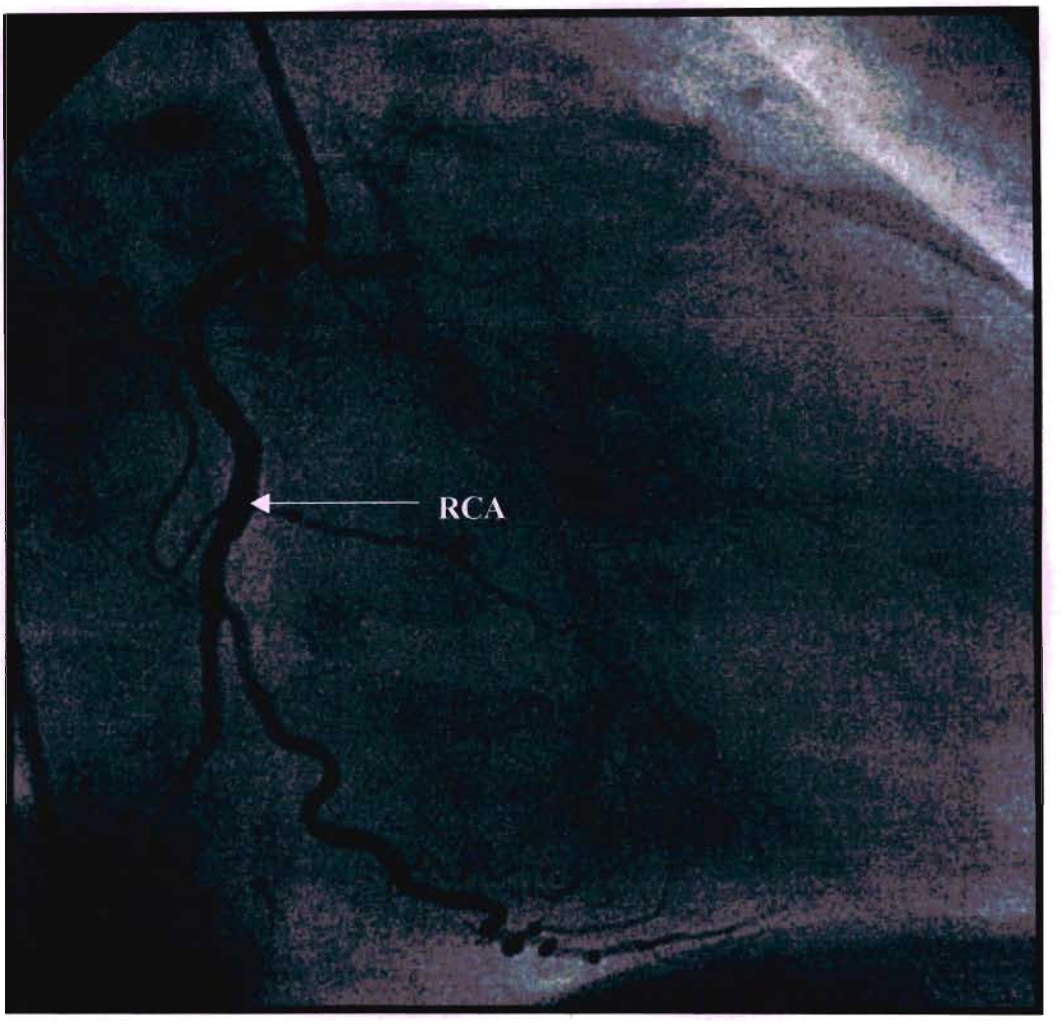


Plate 27: Angiographic demonstration of a dominant RCA

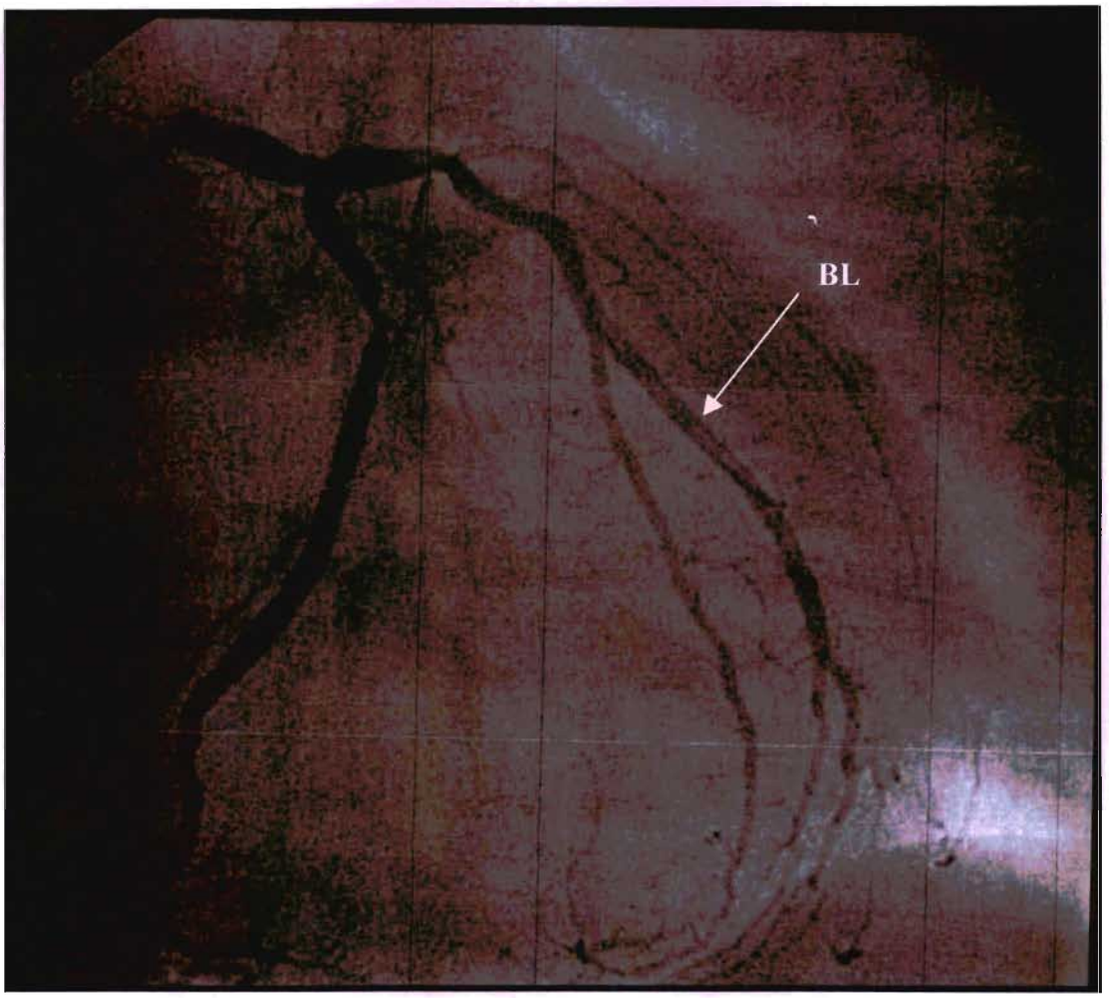


Plate 28: Angiographic demonstration of a bifid LAD (BL)

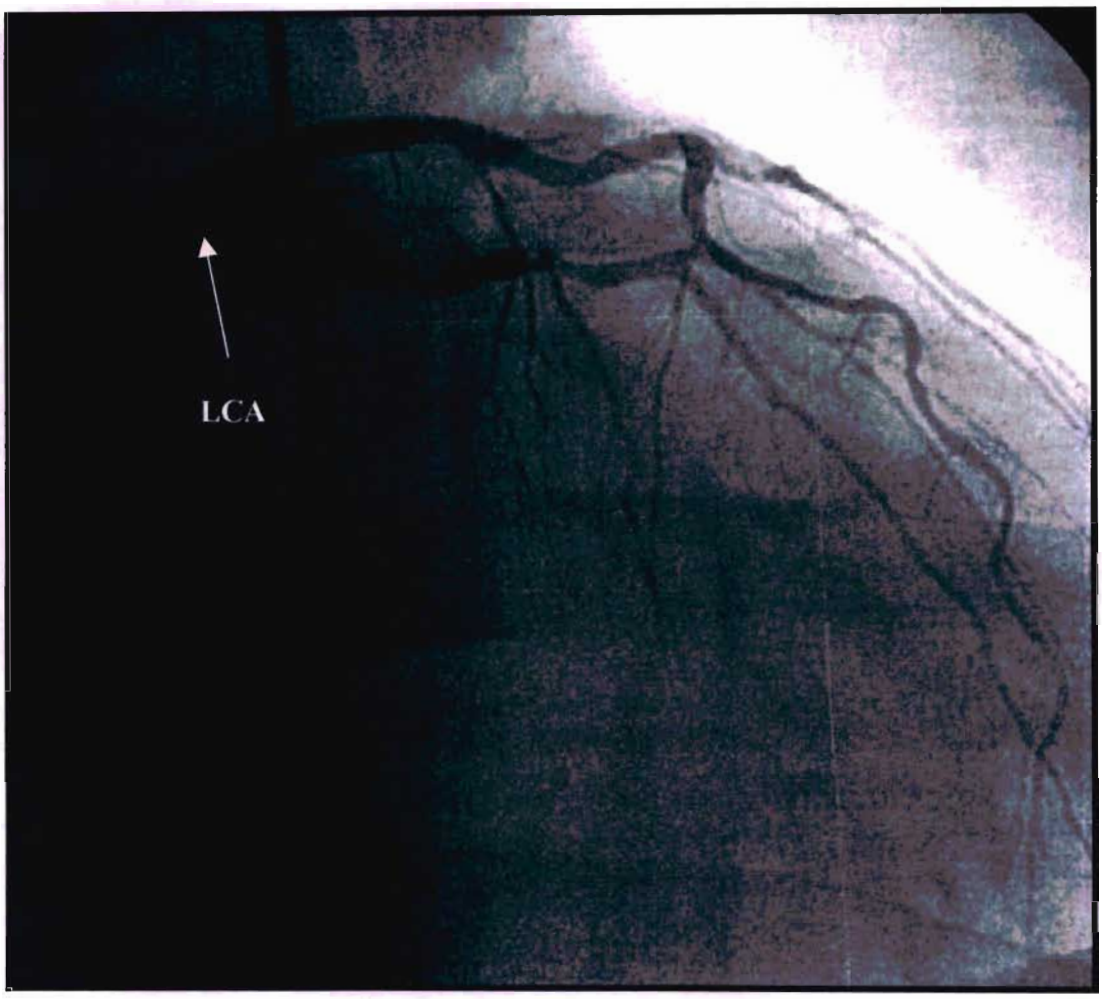


Plate 29: Angiographic demonstration of a short stemmed LCA

MYOCARDIAL BRIDGES

4.2.1. SAMPLE DISTRIBUTION

Of the total sample (n = 323) a sample of 144 specimens of South African origin, were analysed with regards to myocardial bridging. Of the total sample, 72 specimens indicated bridging. In this group of bridged vessels, (n=72), 2 subgroups were identified: a cadaveric group (n=44) and an angiographic group (n=28).

With regards to the LAD in particular, a correlative analysis was conducted in order to determine its morphology using 100 angiograms and 20 selected cadaveric dissections, (Figure 50).

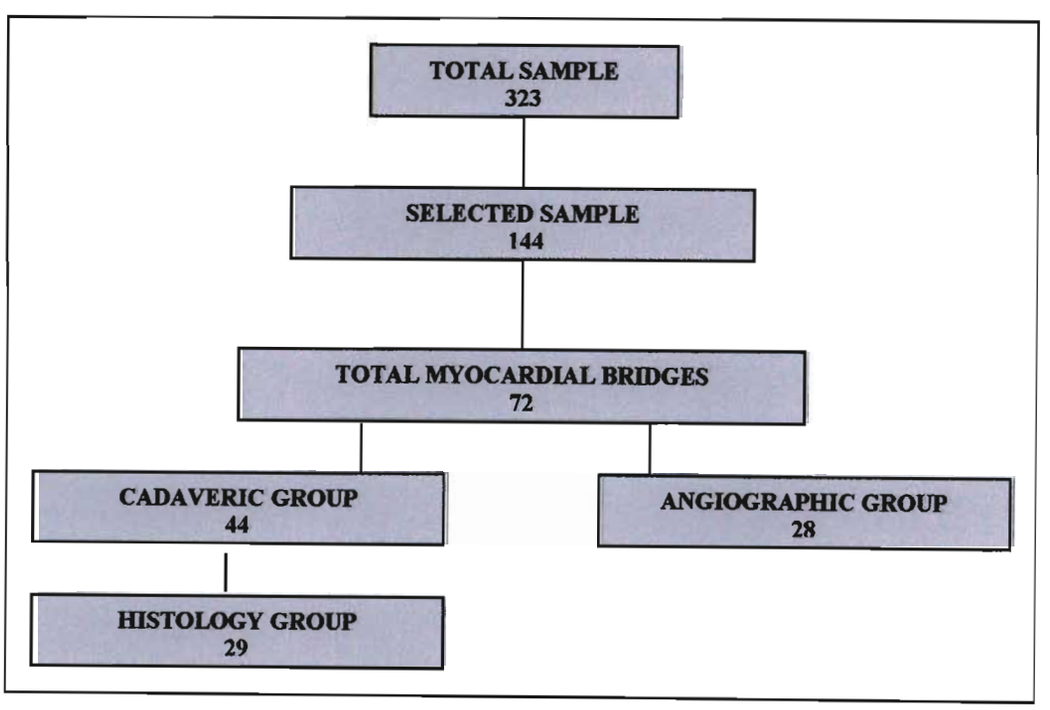


Figure 48: Flow chart of sample distribution for myocardial bridge study

4.2.2. RECORDED INCIDENCES AND REPORT OF FINDINGS

Myocardial bridges were observed in 44 of the 83 cadaveric cases selected, resulting in an incidence of 53% of the total cadaveric population studied. From the analysis of the surgical reports, an incidence of 28% of LAD's was shown to be deep to muscle. When combined, the overall incidence for the presence of myocardial bridges was 39% (73 counts for n=183) in the population studied. In terms of ethnicity, a 30% incidence was recorded amongst Indians, 14% amongst Whites and 56% amongst Blacks.

Bridges were recorded over the RCA, LAD and LCX. Micro-dissection of the myocardial arrangement revealed a series of three predominant patterns, (Plates 30, 31, 32, 33, 34 a, b, c, d).

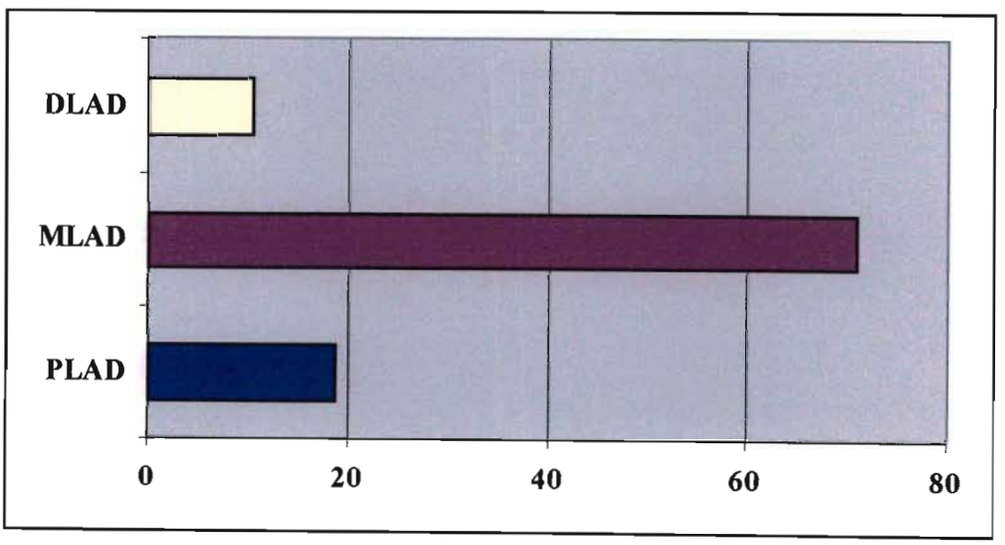
Myocardial fibres were observed to form loops over the arteries in 16.3%. In the second pattern, the bridge passed over the artery along a diagonal plane of parallel fibres. This arrangement occurred in 73.5% of hearts. In the remaining 10.2%, the arteries maintained an intra-myocardial position, (Figure 52).

An incidence of 2.5% of bridging of the RCA was recorded. The pattern of bridging ranged from a series of 3 to 4 slips with an average width of 3mm to complete investment of the entire length of the artery up to the acute margin of the right ventricle.

In the total sample of 44 hearts with myocardial bridges, a total of 48 counts of bridges were recorded in relation to the LAD. The counts were recorded over three segments of the LAD. An incidence of 18.75% was seen over the proximal third of the LAD, 70.8% of the bridges recorded appeared to lie over the middle third and 10.4% over the distal of the vessel. The LAD was intra myocardial in 10.2% and in 14.3% the bridge extended over 2 segments. Incidences of findings and mean measurements for bridges over the LAD are tabulated below.

Table 18: Incidences of myocardial bridges on the LAD

LAD SEGMENT	INCIDENCE %	MEAN LENGTH OF MB (mm)
Proximal LAD	18.8	10.73 [2.3 – 16.0]
Middle LAD	70.8	20.02 [0.9 – 46.0]
Distal LAD	10.4	7.66 [0.7 – 23.5]



Graph 17: Incidence of myocardial bridges on LAD artery segments

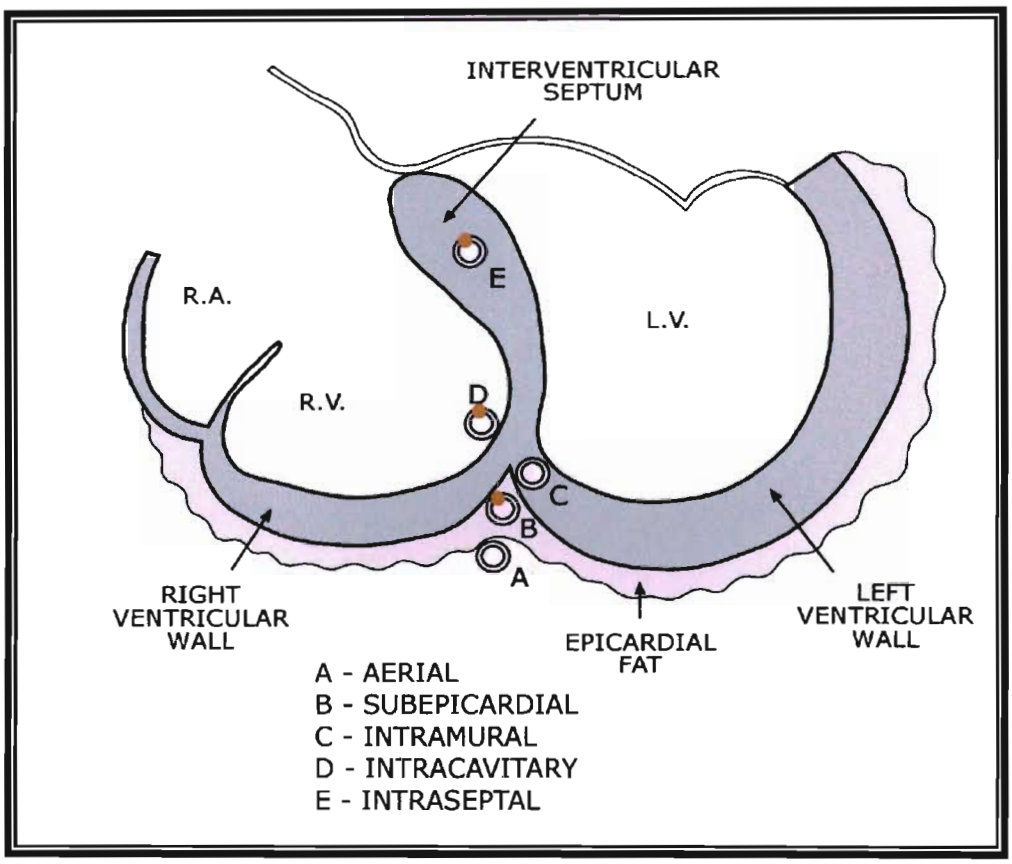


Figure 49: Cardiac positions of the LAD artery

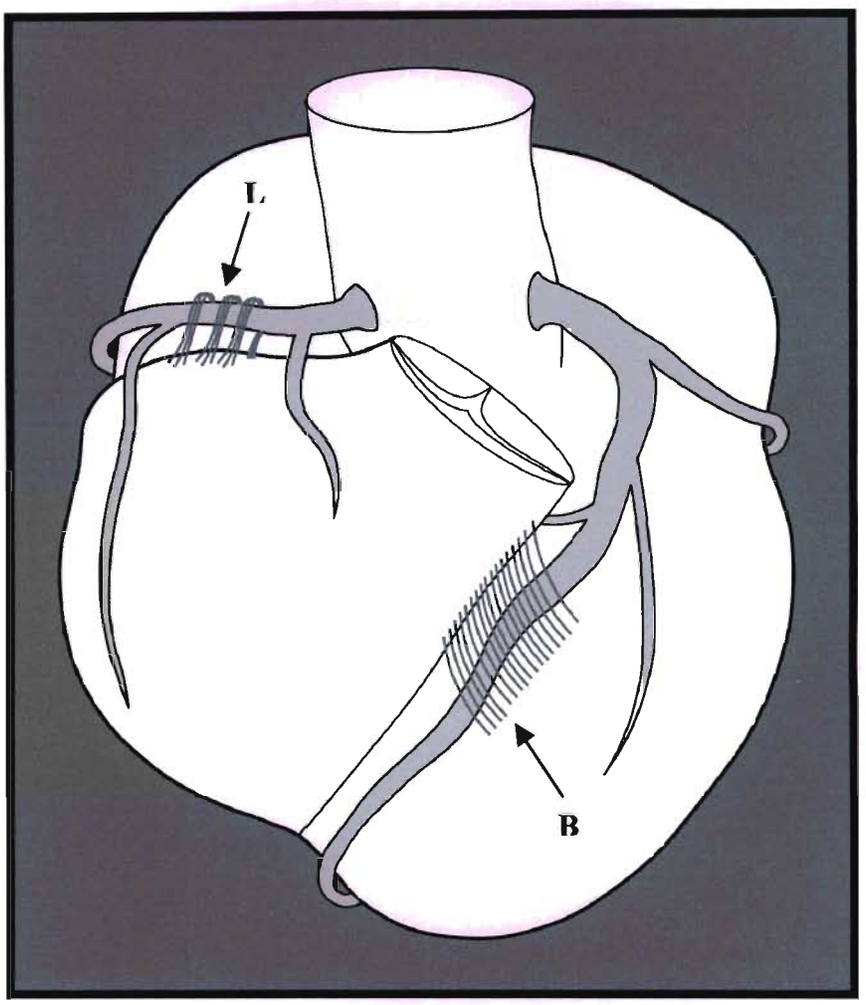


Figure 50: Myocardial bridge patterns showing loops (L) and parallel arrangements (B)

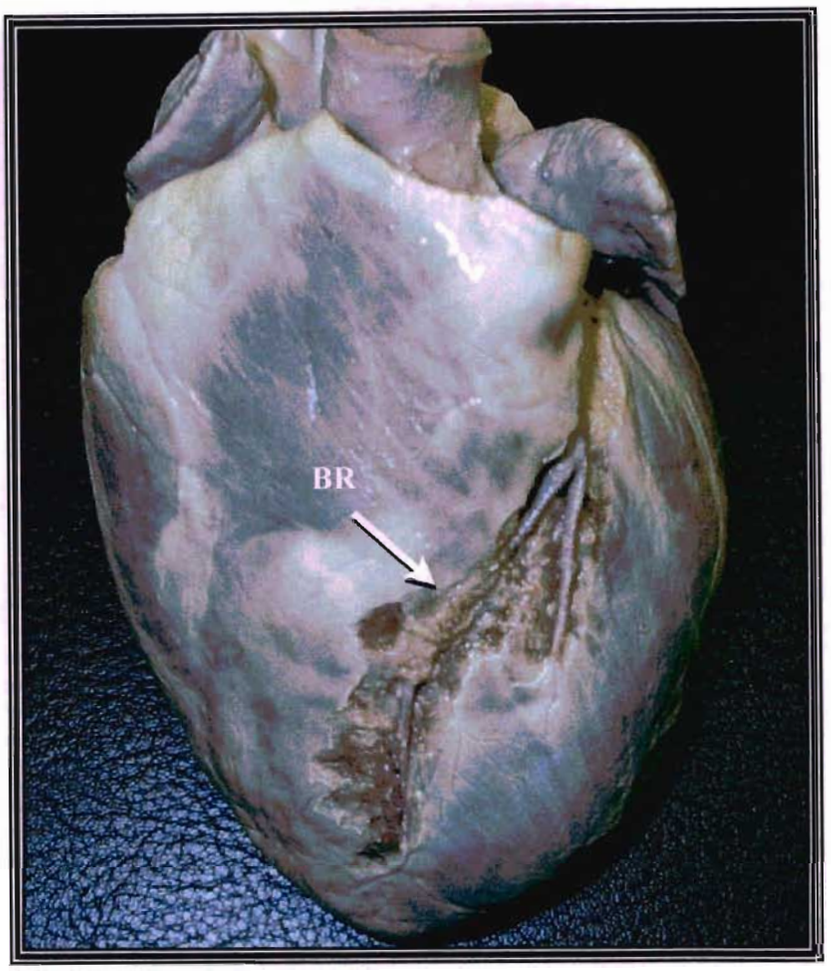


Plate 30: Bifid LAD showing bridged right branch (BR)

The heart is placed with the diaphragmatic surface on the table and tilted at 90° to the right. The anterior inter-ventricular groove is in focus. The LAD appears bifid in its middle segment. As the LAD components pass inferiorly, a segment of the right branch is wrapped in a loop of myocardial fibres. The post mural segment tapers as it continues toward the apex.

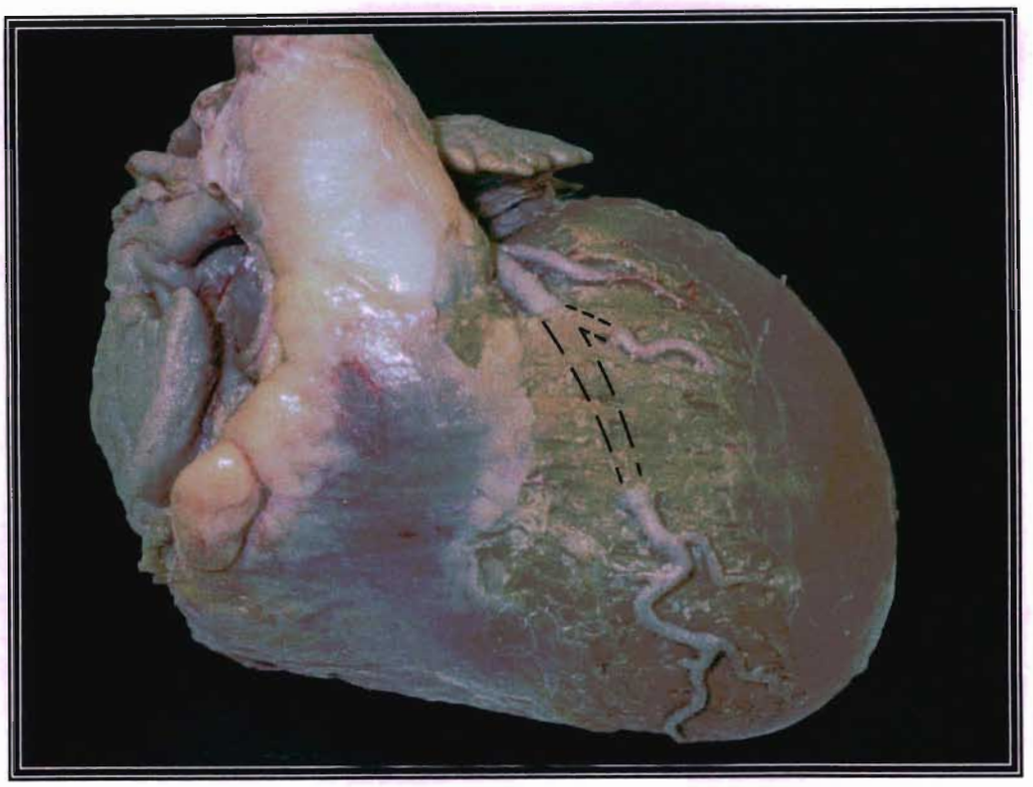


Plate 31: Myocardial Bridge over middle portion of the LAD artery indicated along dotted lines

The heart is orientated in the anatomical position. The LAD is seen as it hooks around the root of the pulmonary trunk. In its course down the inter-ventricular groove, it is covered by an arrangement of parallel myocardial fibres across parts of the proximal and distal segments. Two superior diagonal branches are seen. The lower one is bridged proximally. Two smaller diagonal branches arise from the post mural segment of the LAD. The LAD tapers as it continues posteriorly and to the right of the apex.

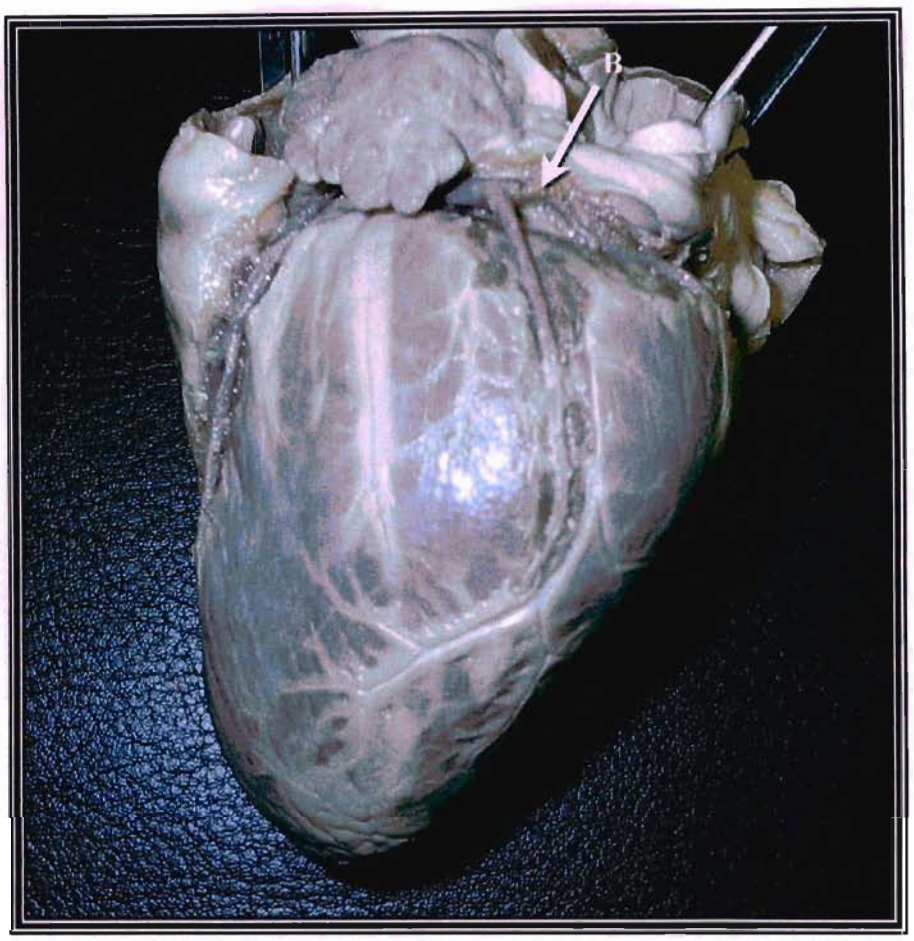


Plate 32: Myocardial loop arrangement over LCX (B)

The heart has been placed on the surface with the right atrium and the right portion of the right ventricle at 180° to the surface. The apex is pointing inferiorly and the obtuse margin of the left ventricle is facing anteriorly. The proximal segment of the LCX is seen emerging from beneath the left atrial appendage. Its large obtuse marginal branch descends along the ventricular surface while the LCX continues along the coronary groove toward the crux. The segment of the LCX is drawn to the pulmonary wall of the left atrium by a loop of myocardial fibers. The LAD is seen passing along the left border of the right ventricle.

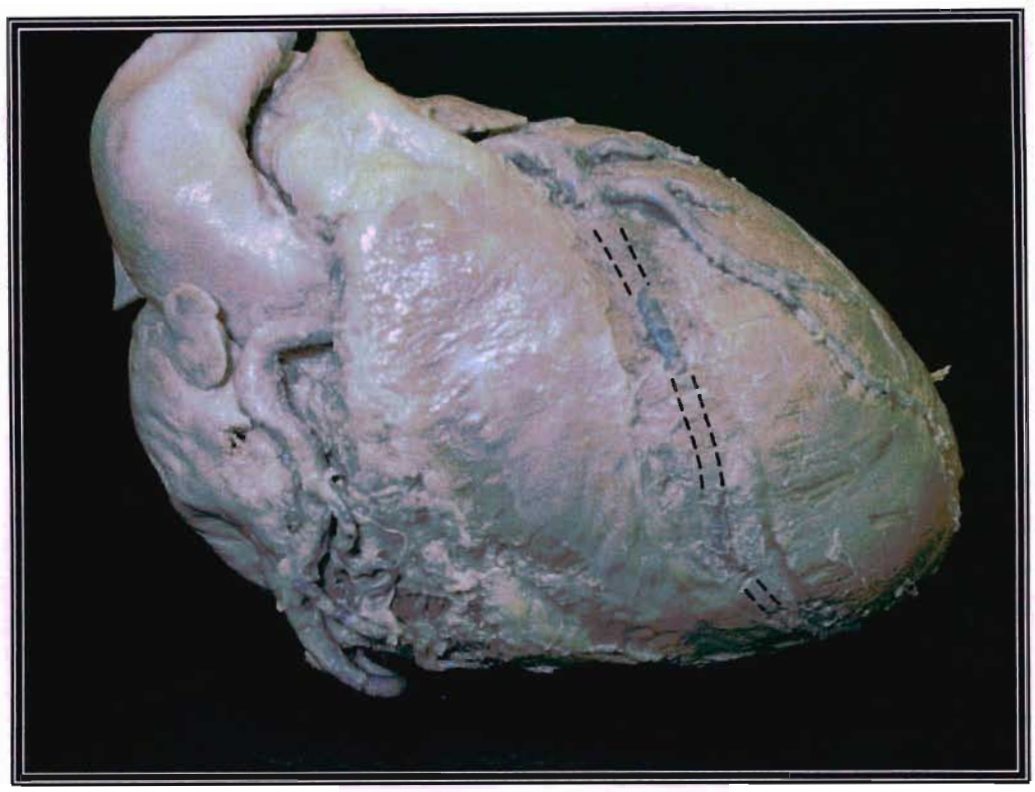


Plate 33: Myocardial bridge pattern over middle and distal LAD indicated along dotted lines

The specimen is being viewed in the anatomical position. The aorta is seen arching over the pulmonary trunk on the right. The RCA is displaced outward in the right atrio-ventricular groove. The LAD is in focus passing down the anterior inter-ventricular groove. In this specimen, the LAD is bifid. The right component of the vessel is bridged in two places: over the proximal and upper distal segments.

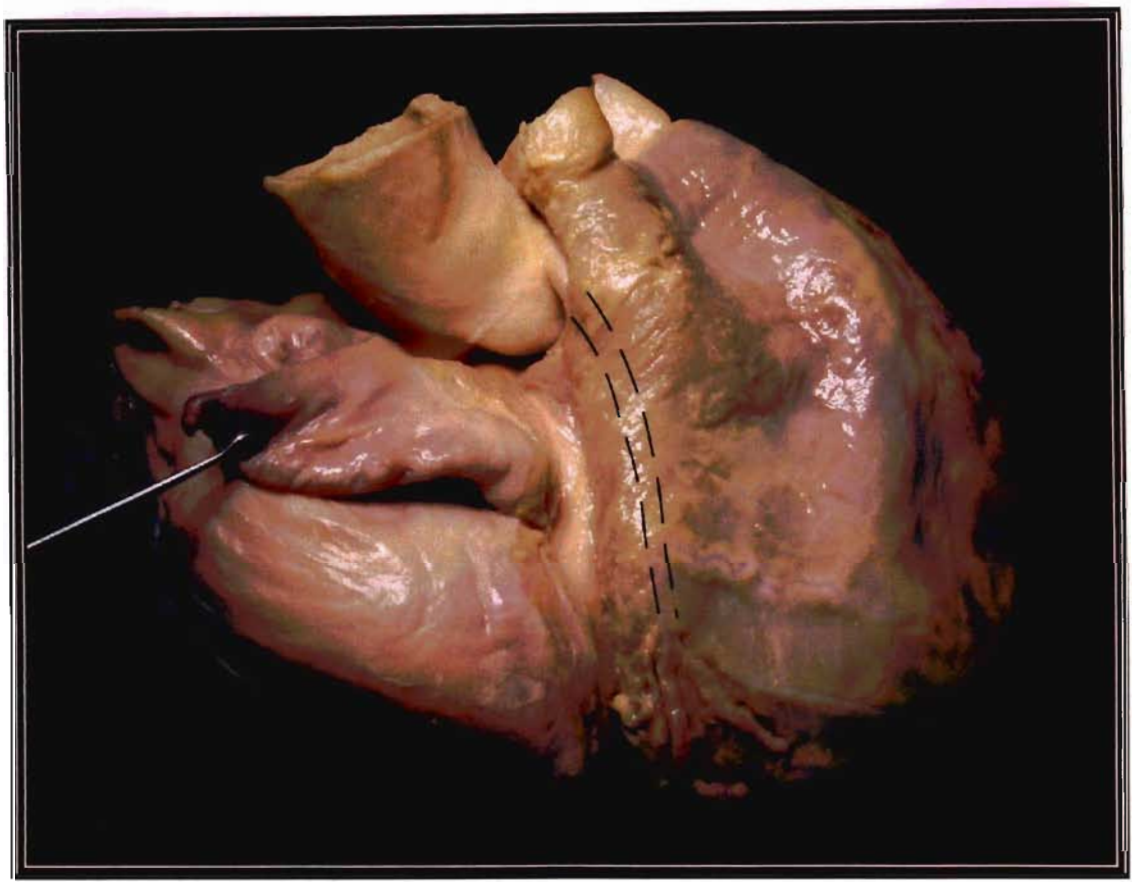


Plate 34 (a): Intra-mural RCA – Anterior view

The heart is being viewed anteriorly in the anatomical position. The right atrial appendage has been retracted to expose the right atrioventricular groove. Between the right atrium and right ventricle lies the root of the aorta, superiorly. The right main trunk is partially visible as it extends downward from the aorta. There appears to be no vessel occupying the atrioventricular sulcus.

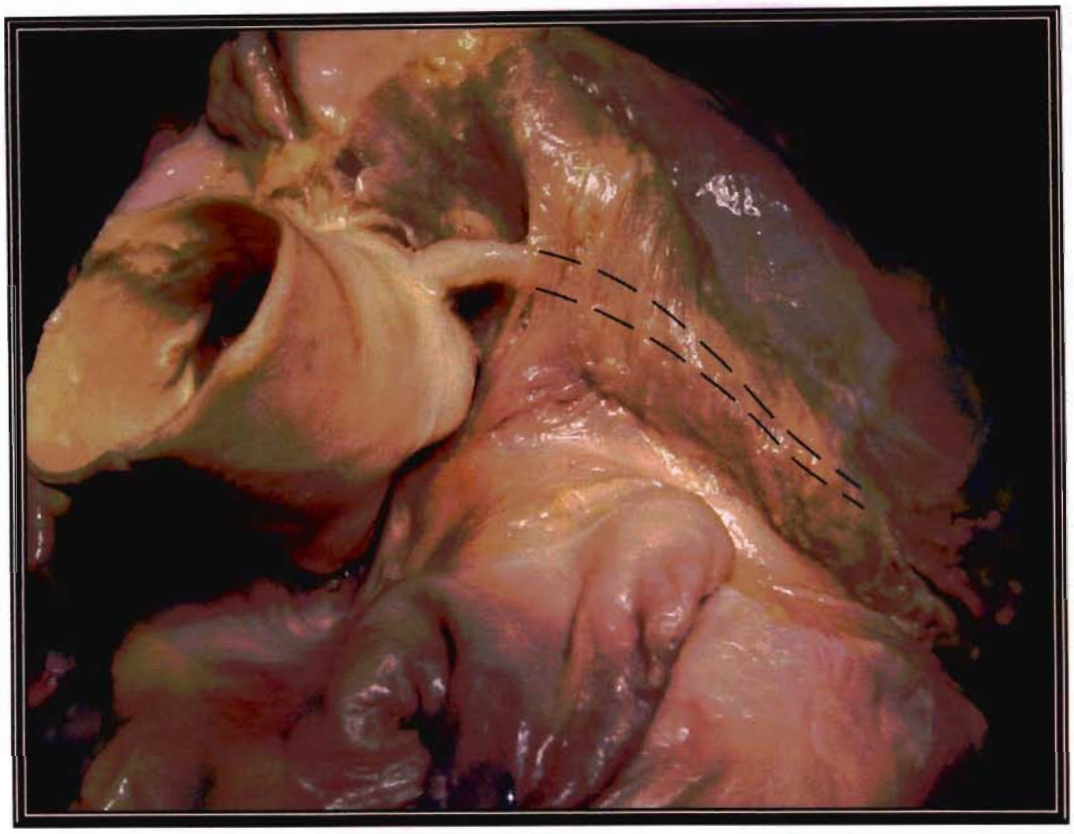


Plate 34 (b): Intra-mural RCA – Horizontal view

The heart is being viewed with the right ventricle in a superior position, the right atrium directed inferiorly and the atrioventricular groove lying in a horizontal plane across the picture. The aortic stump has been displaced to the left of the image to show the RCA arching outward from the aortic sinus. The RCA the passes within the wall of the infundibulum and is completely invested in a layer of myocardium(shown in dotted lines). It re-emerges from its intra-mural position in the region of the acute margin of the heart to give off the right marginal artery.

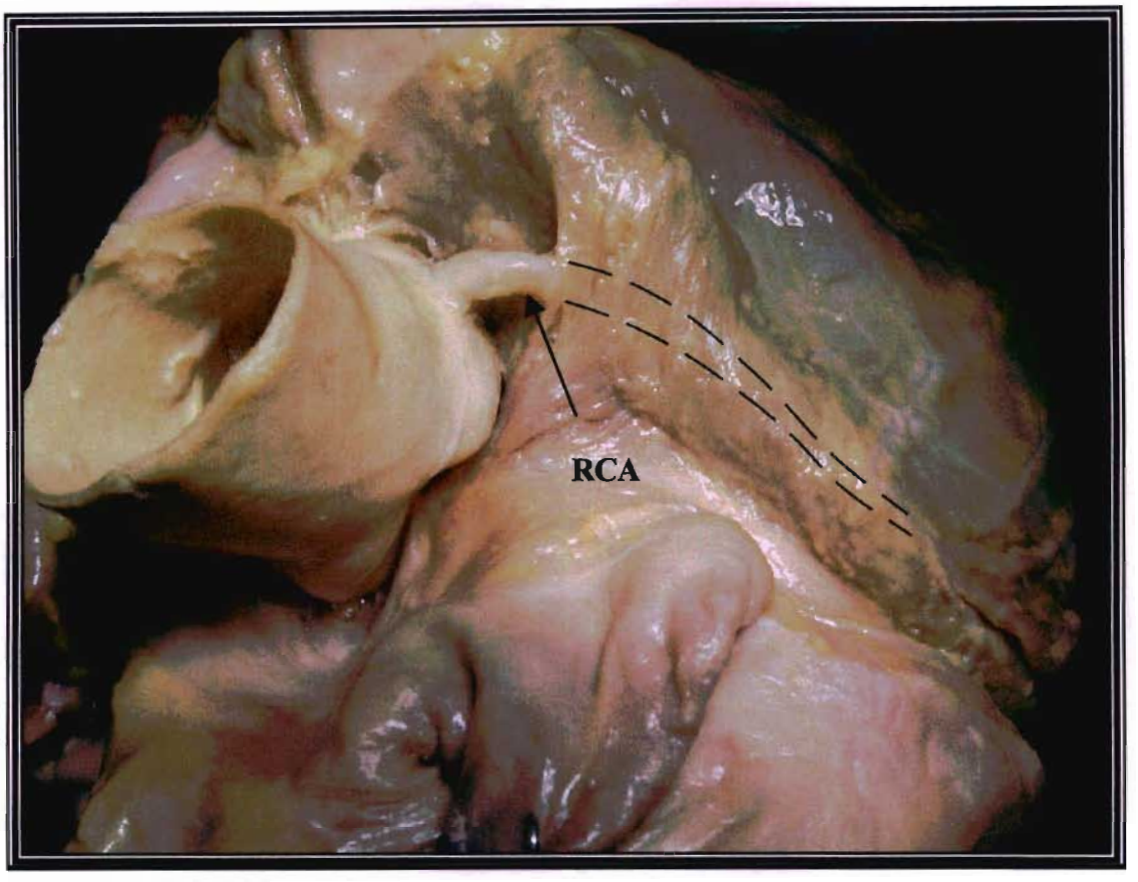


Plate 34 (c): Pre-mural segment of intra-mural RCA

The heart is being viewed in an oblique plane. The RCA is seen as it descends into the myocardial cave (shown in dotted lines) along the right ventricular border.

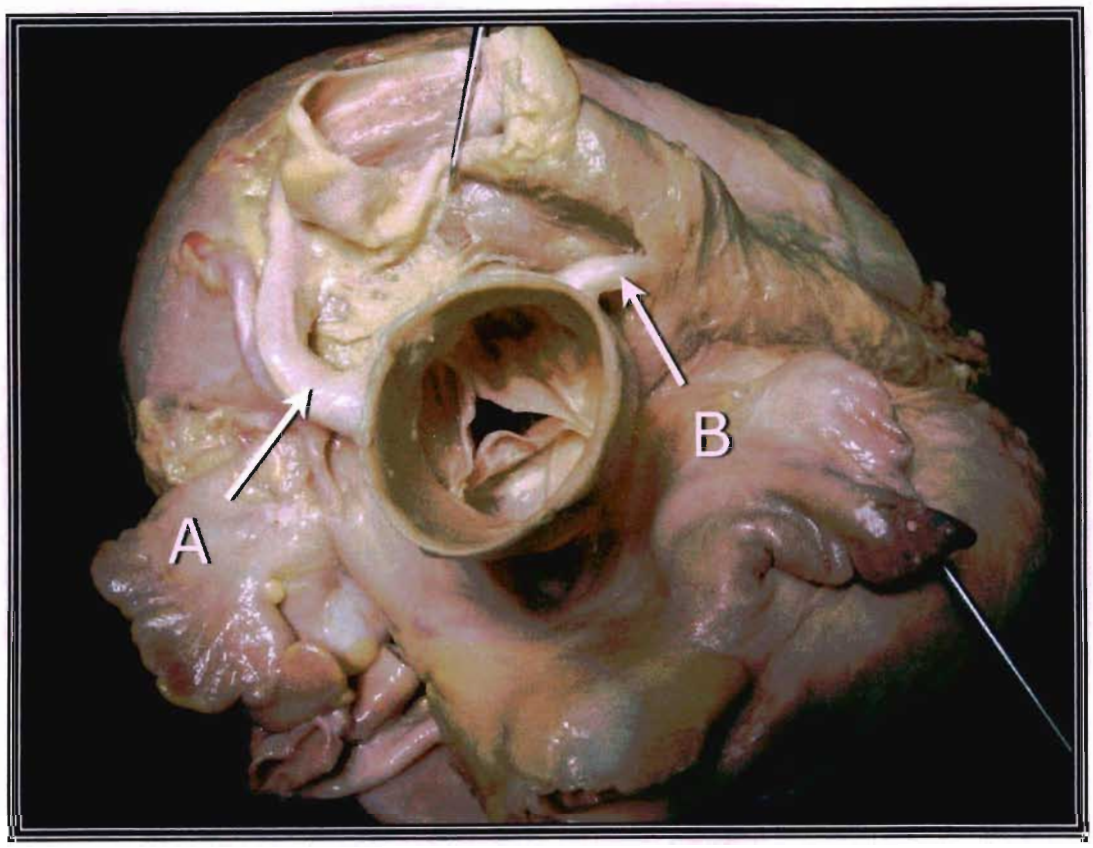


Plate 35:Origin of the LCA (A) and RCA (B) on a superior view

The heart is being viewed from a superior position. The left (A) and right (B) proximal coronary arteries are shown arising from the aortic sinuses. The LCA is shown, continuing along an epicardial course and the RCA is shown along an intra-mural course.

ANGIOGRAPHIC INVESTIGATION OF INTRA-MURAL LAD

Further mathematical analysis of the 28 angiograms of patients positively identified with myocardial bridges associated with LAD revealed the following:

1. A positive correlation between a straight appearance of the LAD on angiogram and deep myocardial position upon surgical observation

2. The average calculated TDX was 1.147

3. The highest recorded TDX was 1.373 and lowest TDX was 1.045

Results showed that 100% (n=28) of angiographically demonstrated LAD's had TDX values within the predicted range (TDX < 1.5) for straightness, (Tables 20.1 to 20.28)

In the cadaveric investigation of 20 hearts with intra-mural LAD's the segment of the vessel under the bridge appeared "straight" upon dissection when compared with the post and pre-mural segments, (Table 19).

Analysis of post-mortem angiograms corresponded with the results from dissection. The LAD appeared "straight" upon morphological observation, (Plates 36, 37, 38, 39).

Table 19: Intra-mural LAD – Surgical and Angiographic correlation indices

SURGICAL POSITION	STRAIGHTEST SEGMENT	TDX VALUE	SEX/ETH
Deep to fat and muscle	Proximal and Middle LAD	1.067	WM
Deep to muscle	Middle LAD	1.146	IM
Deep to fat and muscle	Middle LAD	1.196	IM
Deep to muscle	Middle LAD	1.146	IM
Deep to fat and muscle	Middle LAD	1.196	IF
Deep to fat and muscle	Middle LAD	1.106	IM
Deep to muscle	Middle LAD	1.184	IM
Deep to muscle	Middle LAD	1.079	IM
Deep to muscle	Middle LAD	1.323	IM
Deep to muscle	Middle LAD	1.045	IM
Deep to muscle	Middle LAD	1.075	WM
Deep to muscle	Middle LAD	1.162	IM
Deep to muscle	Distal LAD	1.108	IM
Deep to muscle	Proximal and Middle LAD	1.137	IM
Deep to muscle	Proximal and Middle LAD	1.103	WM
Deep to muscle	Middle LAD	1.104	WF
Deep to muscle	Proximal LAD	1.191	IF
Deep to muscle	Proximal LAD	1.261	IM
Deep to muscle	Middle LAD	1.373	IF
Deep to fat and muscle	Middle LAD	1.119	IF
Deep to fat and muscle	Middle LAD	1.129	IF
Deep to fat and muscle	Middle and Distal LAD	1.204	WM
Deep to muscle	Middle LAD	1.063	IM
Deep to muscle	Middle LAD	1.165	IM
Deep to muscle	Middle LAD	1.195	IM
Deep to muscle	Proximal LAD	1.103	IM
Deep to muscle	Proximal LAD	1.156	IM
Deep to muscle	Middle LAD	1.124	IM
Deep to muscle	Middle LAD	1.103	IM
Deep to muscle	Middle LAD	1.112	WM

TABLE20.1: WHITE MALE / 78 YRS

Surgical Description	LAD Deep to muscle
TDX Value	1.067 IDX
Straightest Segment	MID LAD

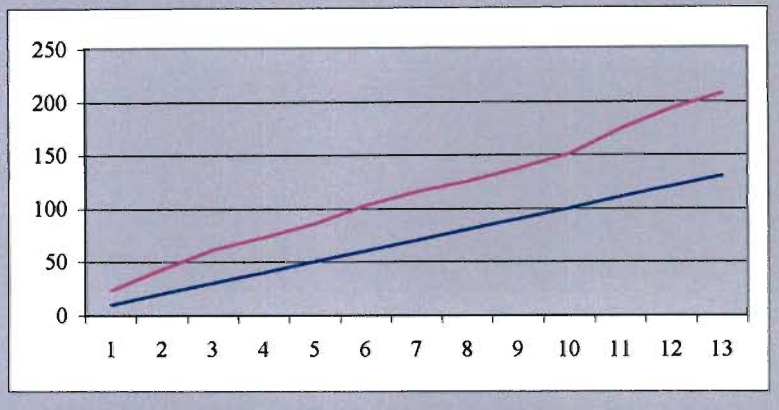


TABLE 20.2: INDIAN MALE / 55 YRS

Surgical Description	LAD Deep to muscle
TDX Value	1.146 IDX
Straightest Segment	MID LAD

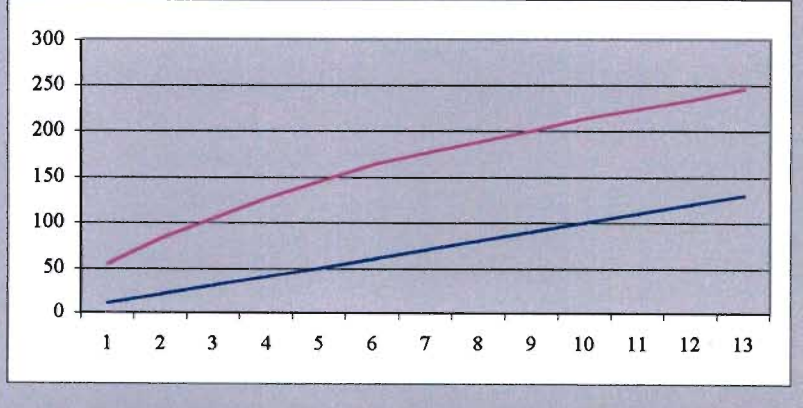


TABLE 20.3: INDIAN MALE / 60 YRS

Surgical Description	LAD Deep to muscle
TDX Value	1.196 IDX
Straightest Segment	MID LAD

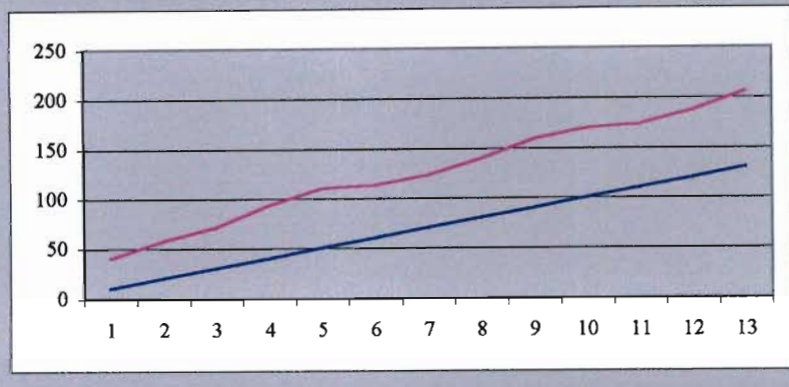


TABLE 20.4: INDIAN MALE / 54 YRS

Surgical Description	LAD Deep to muscle
TDX Value	1.106 IDX
Straightest Segment	MID LAD

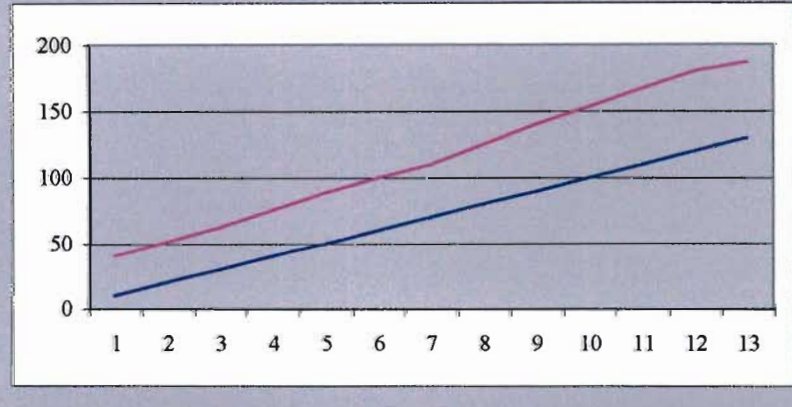


TABLE 20.5: INDIAN FEMALE / 60 YRS

Surgical Description	LAD Deep to muscle
TDX Value	1.184 IDX
Straightest Segment	MID LAD

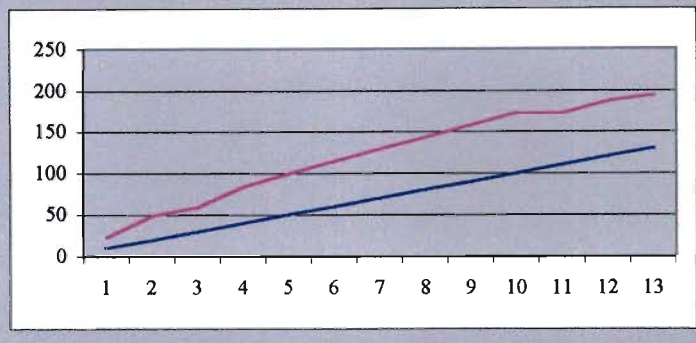


TABLE 20.6: INDIAN MALE / 47 YRS

Surgical Description	LAD Deep to muscle
TDX Value	1.079 IDX
Straightest Segment	MID LAD

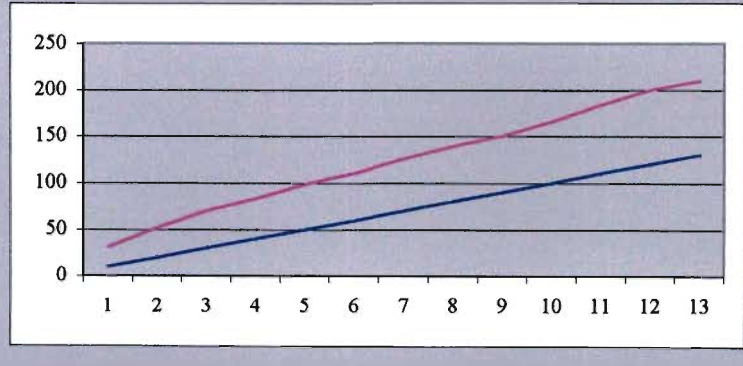


TABLE 20.7: INDIAN MALE / 74 YRS	
Surgical Description	LAD Deep to muscle
TDX Value	1.324 IDX
Straightest Segment	MID LAD

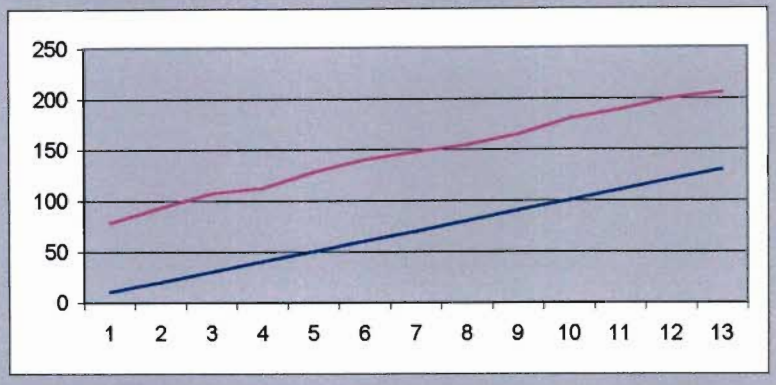


TABLE 20.8: INDIAN MALE / 61 YRS	
Surgical Description	LAD Deep to muscle
TDX Value	1.045 IDX
Straightest Segment	MID LAD

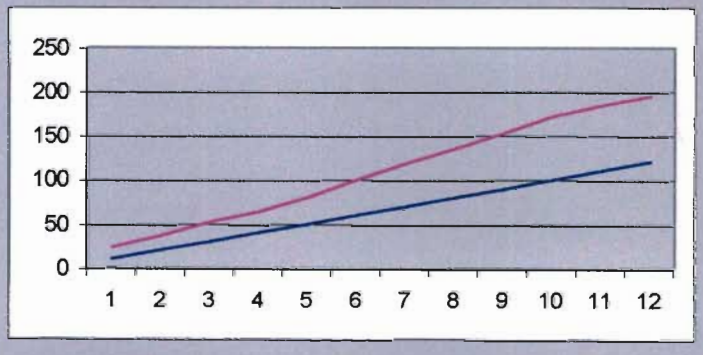


TABLE 20.9: INDIAN MALE / 50YRS	
Surgical Description	LAD Deep to muscle
TDX Value	1.075 IDX
Straightest Segment	MID LAD

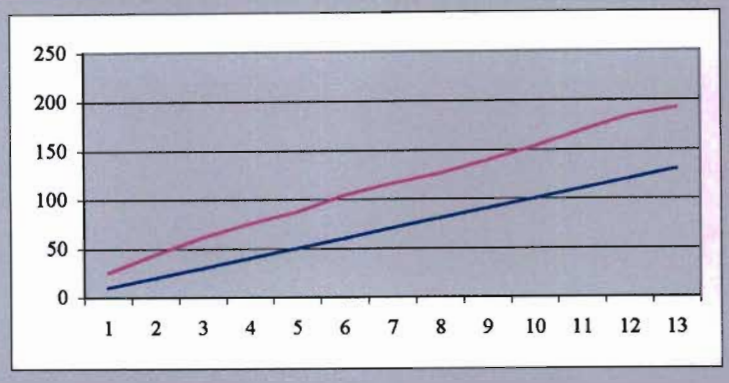


TABLE 20.10: INDIAN MALE / 62 YRS	
Surgical Description	LAD Deep to muscle
TDX Value	1.162 IDX
Straightest Segment	MID LAD

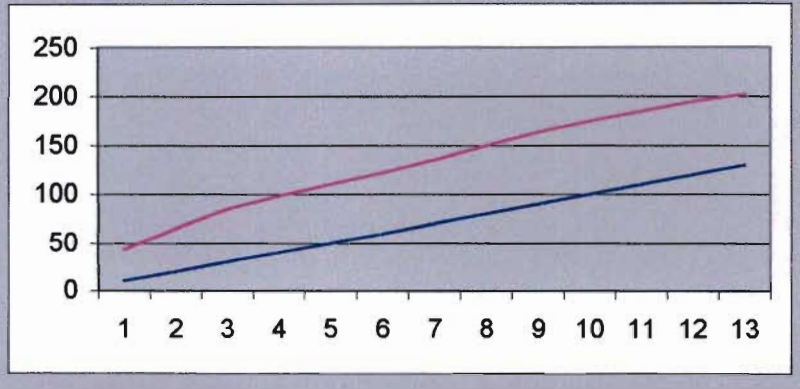


TABLE 20.11: WHITE MALE / 64 YRS	
Surgical Description	LAD Deep to muscle
TDX Value	1.108 IDX
Straightest Segment	MID LAD

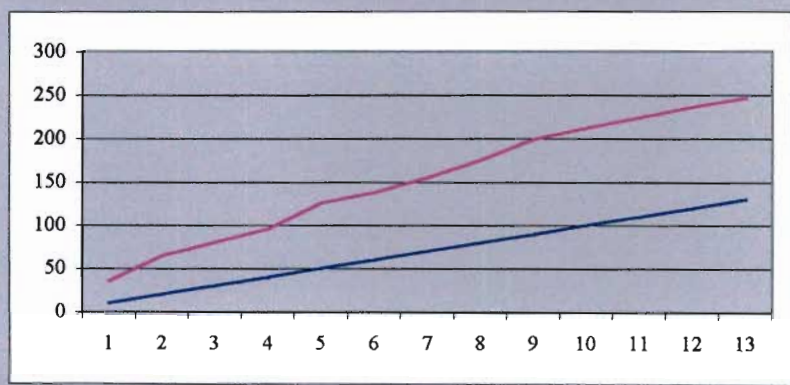


TABLE 20.12: INDIAN MALE / 63 YRS	
Surgical Description	LAD Deep to muscle
TDX Value	1.137 IDX
Straightest Segment	MID LAD

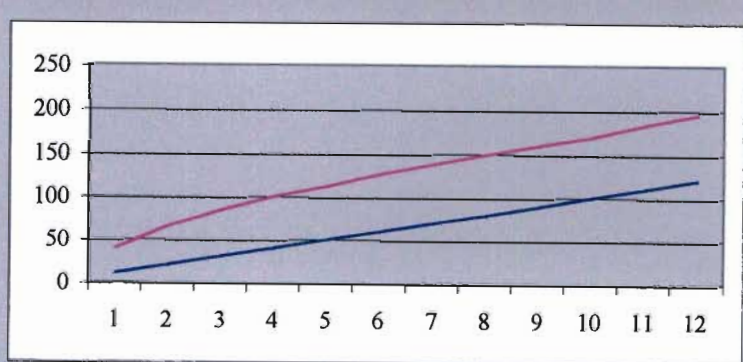


TABLE 20.13: INDIAN MALE / 56 YRS	
Surgical Description	LAD Deep to muscle
TDX Value	1.103 IDX
Straightest Segment	MID LAD

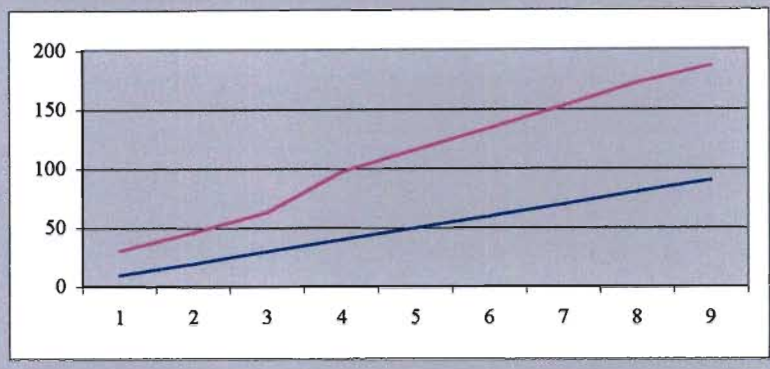


TABLE 20.14: INDIAN MALE / 63 YRS	
Surgical Description	LAD Deep to muscle
TDX Value	1.104 IDX
Straightest Segment	MID LAD

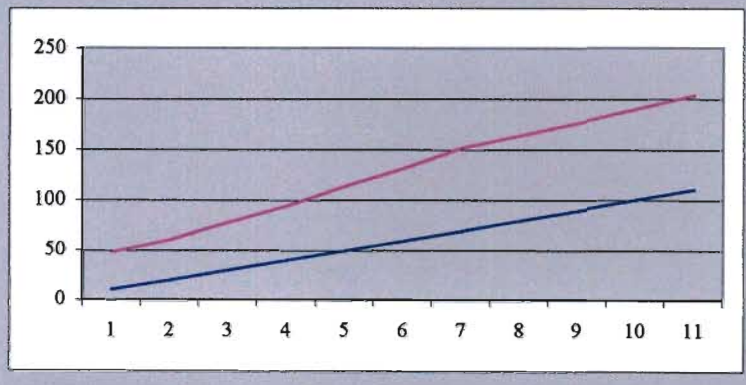


TABLE 20.15: WHITE MALE / 80 YRS	
Surgical Description	LAD Deep to muscle
TDX Value	1.191 IDX
Straightest Segment	MID LAD

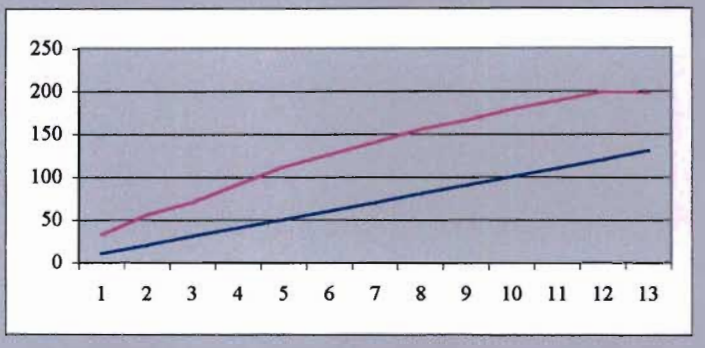


TABLE 20.16: WHITE FEMALE / 72 YRS	
Surgical Description	LAD Deep to muscle
TDX Value	1.261 IDX
Straightest Segment	MID LAD

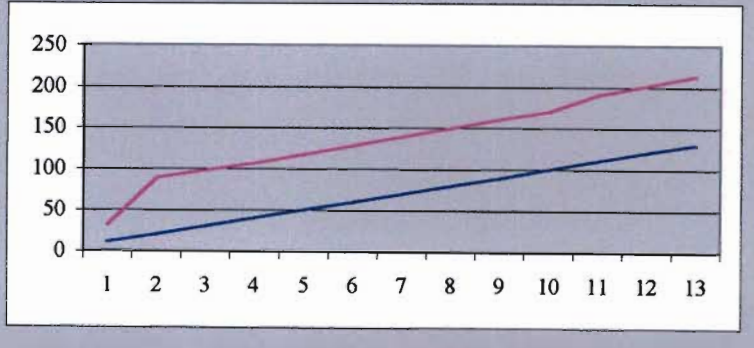


TABLE 20. 17: INDIAN FEMALE / 65 YRS	
Surgical Description	LAD Deep to muscle
TDX Value	1.373 IDX
Straightest Segment	MID LAD

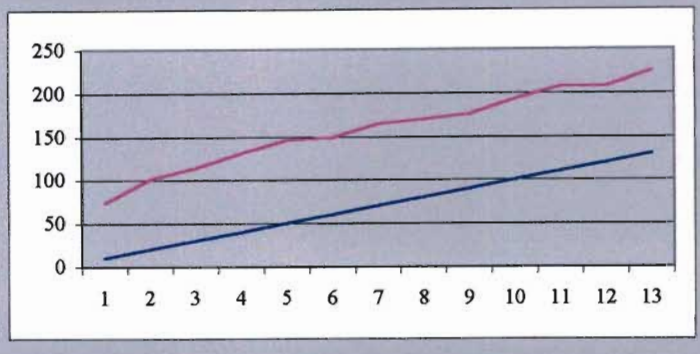


TABLE 20.18: INDIAN MALE / 77 YRS	
Surgical Description	LAD Deep to muscle
TDX Value	1.119 IDX
Straightest Segment	MID LAD

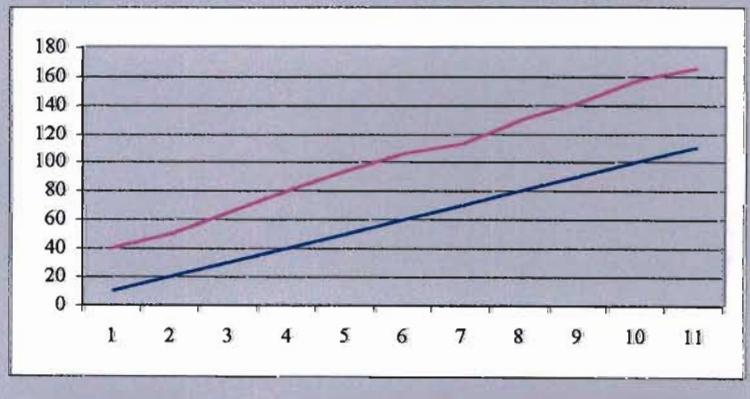


TABLE 20.19: INDIAN FEMALE / 69 YRS

Surgical Description	LAD Deep to muscle
TDX Value	1.129 IDX
Straightest Segment	MID LAD

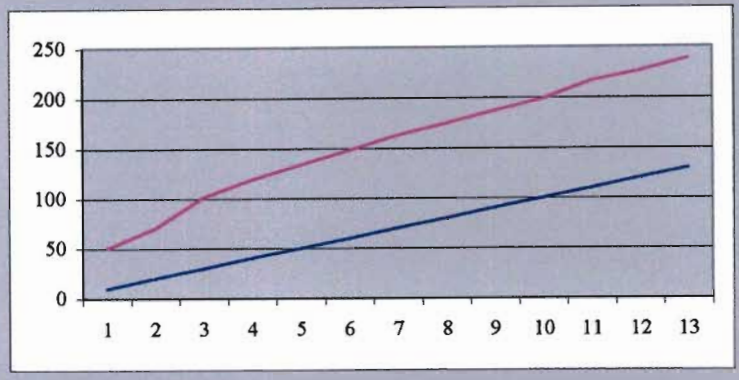


TABLE 20.20: INDIAN FEMALE / 64 YRS

Surgical Description	LAD Deep to muscle
TDX Value	1.204 IDX
Straightest Segment	MID LAD

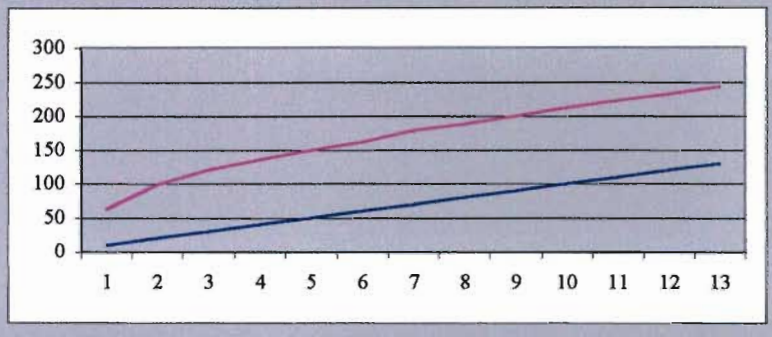


TABLE 20.21: INDIAN FEMALE / 64 YRS	
Surgical Description	LAD Deep to muscle
TDX Value	1.063 IDX
Straightest Segment	MID LAD

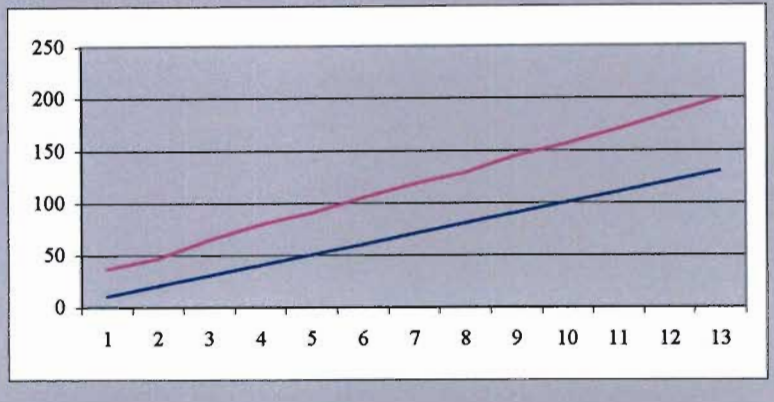


TABLE 20.22: WHITE MALE / 64 YRS	
Surgical Description	LAD Deep to muscle
TDX Value	1.165 IDX
Straightest Segment	MID LAD

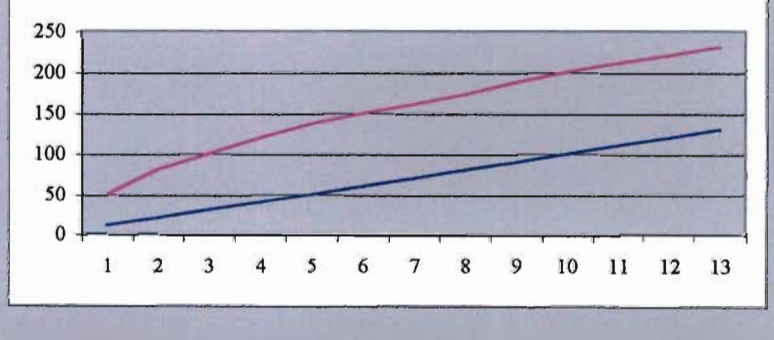


TABLE 20.23: INDIAN MALE / 55 YRS

Surgical Description	LAD Deep to muscle
TDX Value	1.195 IDX
Straightest Segment	MID LAD

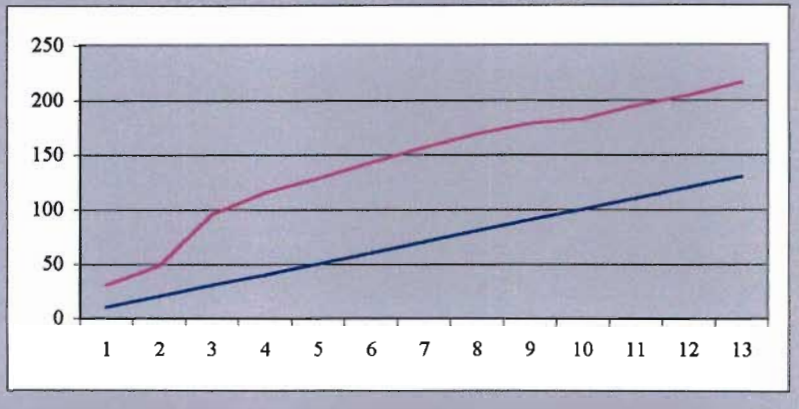


TABLE 20.24: INDIAN MALE / 58 YRS

Surgical Description	LAD Deep to muscle
TDX Value	1.103 IDX
Straightest Segment	MID LAD

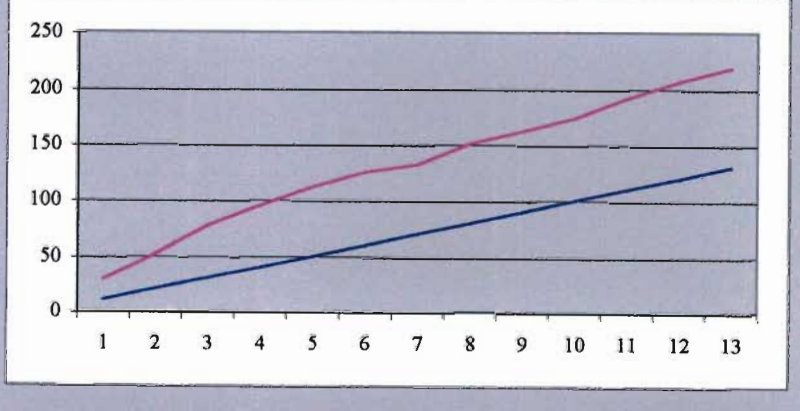


TABLE 20.25: INDIAN MALE / 64 YRS	
Surgical Description	LAD Deep to muscle
TDX Value	1.156 IDX
Straightest Segment	MID LAD

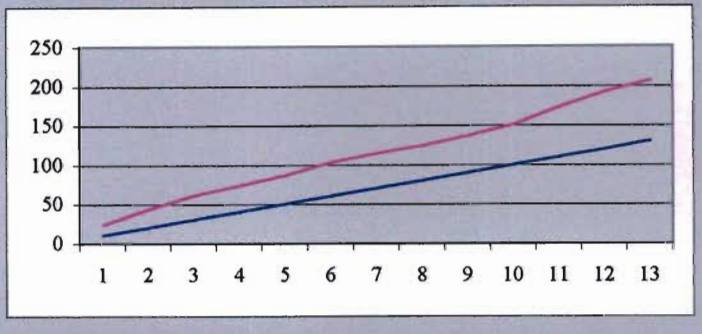


TABLE 20.26: INDIAN MALE / 54 YRS	
Surgical Description	LAD Deep to muscle
TDX Value	1,124 IDX
Straightest Segment	MID LAD

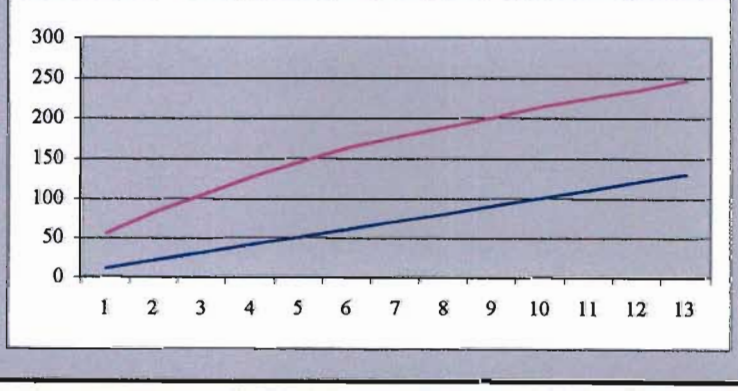


TABLE 20.27: INDIAN MALE / 47 YRS	
Surgical Description	LAD Deep to muscle
TDX Value	1.103 IDX
Straightest Segment	MID LAD

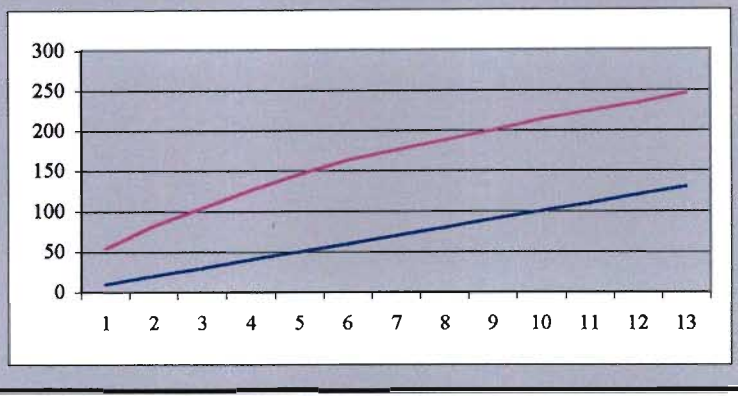
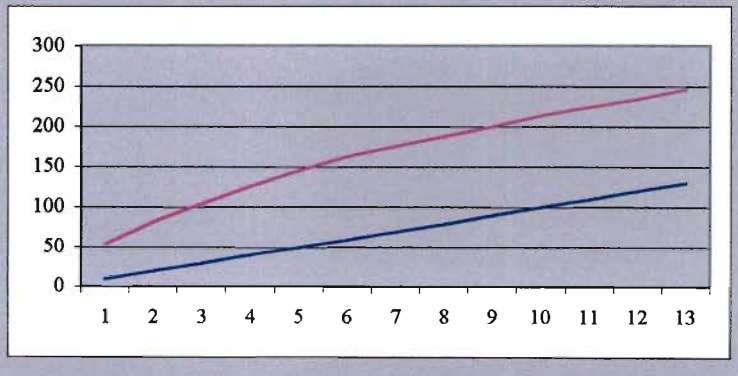


TABLE 20.28: WHITE MALE / 68 YRS	
Surgical Description	LAD Deep to muscle
TDX Value	1.112 IDX
Straightest Segment	MID LAD



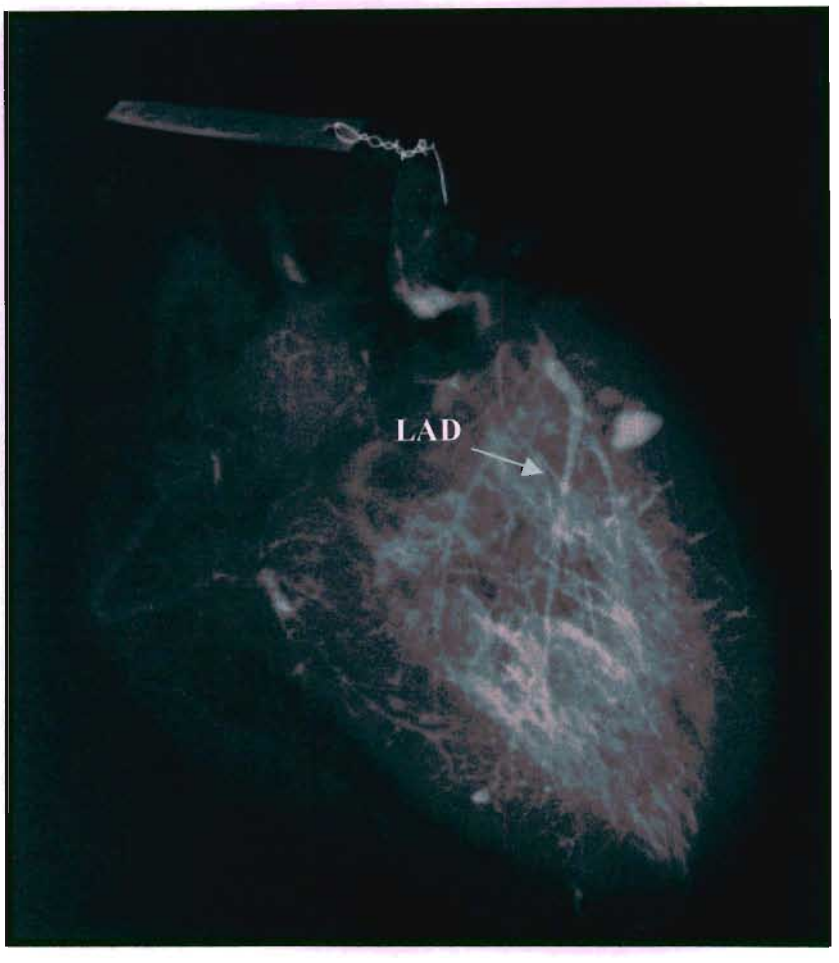


Plate 35: Cadaveric angiogram showing LAD

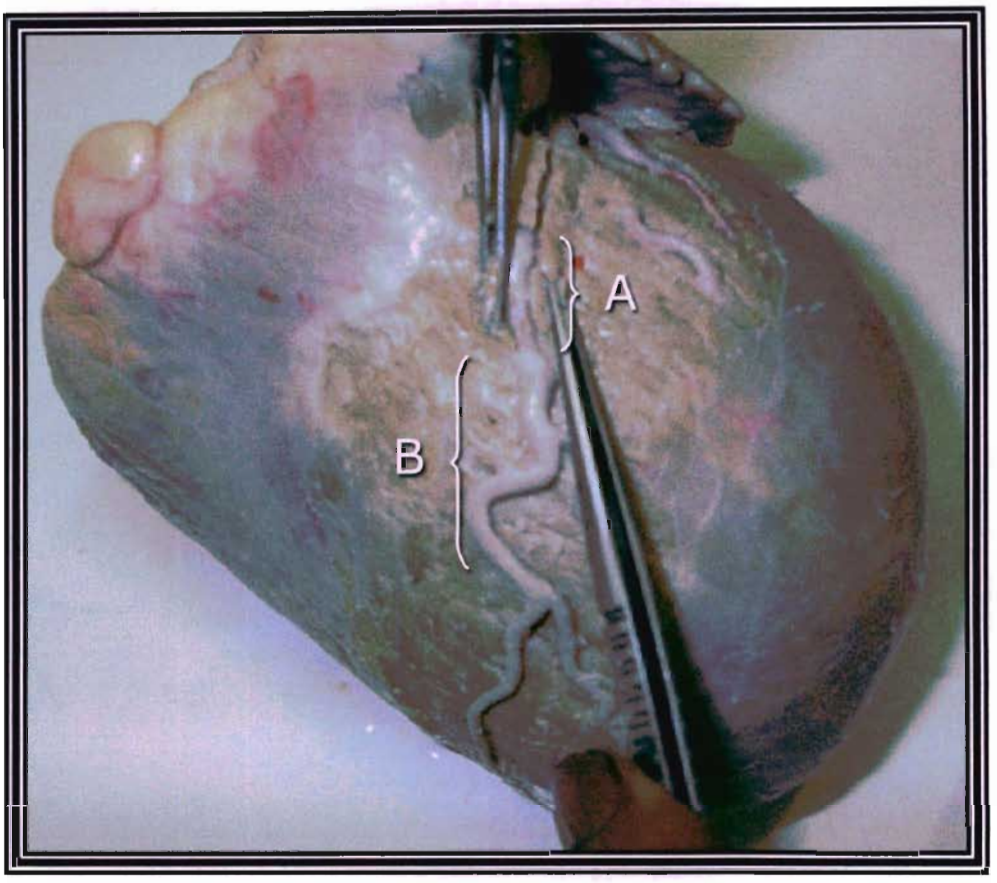


Plate 36: Exposure of intra-mural LAD (A) and epicardial LAD (B)

The heart has been placed in the anatomical position with a 45° tilt to the right. The intra-mural segment of the LAD has been exposed. The intra-mural segment is visibly less tortuous than the post-mural segment.

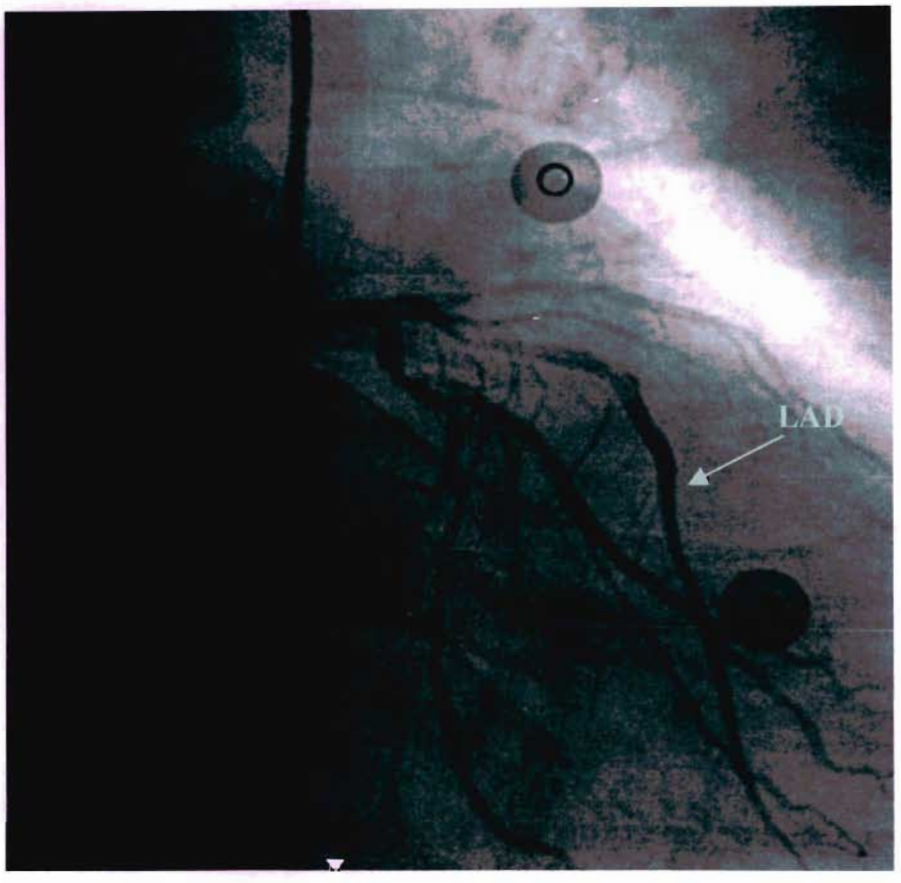


Plate 37: Angiographic demonstration of a straight LAD

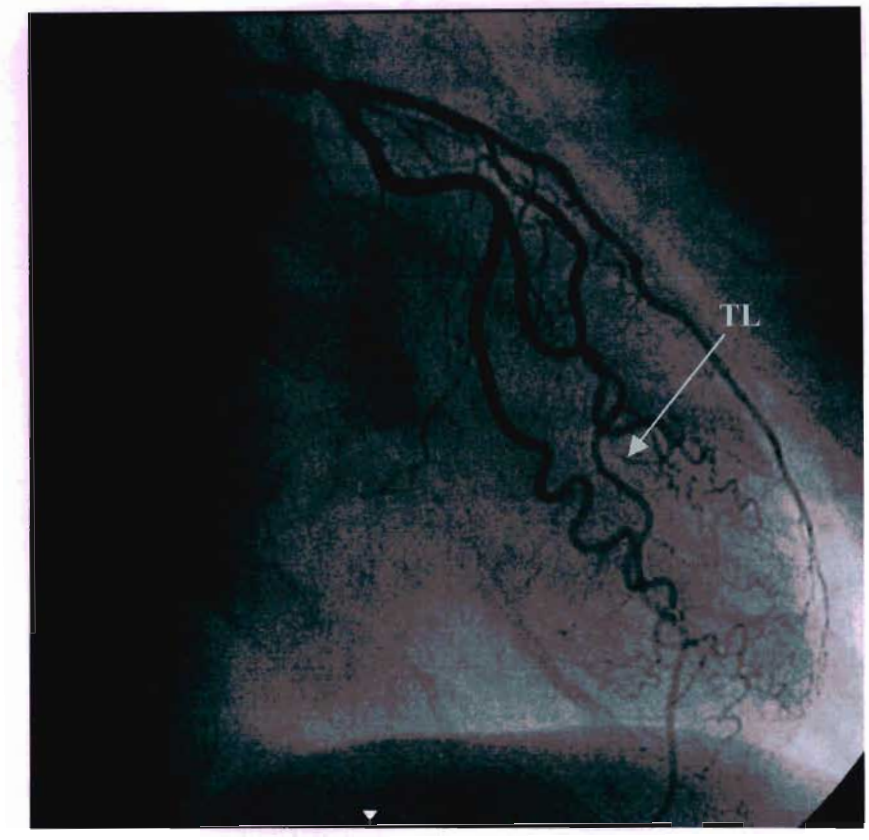


Plate 38: Angiographic demonstration of a tortuous LAD (TL)

EXTRA-CORONARY COLLATERALS

4.3.1. SAMPLE DESCRIPTION

A total of 9 cadavers were studied. The group consisted of 6 females and 3 males, with an age range from 65 to 83 years.

4.3.2. LOCATING ACCESS ROUTES FOR CANNULATION

Successful endoscopic location of the coronary ostia was achieved via the right common carotid artery in 100% of cases investigated.

4.3.3. CANNULATION AND FLUOROSCOPY

In 44% of cannulations, the left coronary ostium was difficult to locate due to a position significantly above the sino-tubular junction. In un-obstructed ostia, the right appeared more rounded whilst the left, was characteristically slit-like.

In instances where cannulation was difficult due to the location of the ostia, the procedure was modified by replacing the original guide wire with thin guitar wire that extended past the luminal opening at the Fogarty tip. A 90° bend was added to the proximal end of the wire to allow for greater control and guidance into the ostia. At the distal end, the wire was bent to approximately 20° close to the balloon area to allow easier approach to the openings.

Visualization of possible collateral vessels was achieved by means of the fluoroscopic mechanism employed. Identification of vessels outside the cardiac shadow was positive for presence of ECC connections on gross inspection in 100% of cases.

4.3.4. DISSECTION TECHNIQUE

The standard dissection technique was successfully applied in all cases.

4.3.5. GROSS INSPECTION

On gross observation, 67% of specimens showed extensive vascularization of the fibrous pericardium via coronary collaterals. In each of these cases, the epicardial fat pads around the root of the aorta, pulmonary trunk and main coronary artery branches were also highly vascular, (Plates 40, 41a, 41b, 42, 43).

ECC connections were found to exist most frequently around the ascending aorta (AA), followed by the superior vena cava (SVC), inferior vena cava (IVC); pulmonary trunk (PT); left pulmonary veins (LPV's); right pulmonary arteries (RPA's); right pulmonary veins (RPV's) and left pulmonary arteries (LPA's).

4.1.1. DETAILED OBSERVATION AND MORPHOMETRIC ANALYSIS

ECC's usually present an arterial pattern consisting of one or two main stems with secondary anastomotic branches. The average external diameter of an ECC was measured to be 0.6mm (0.4-0.7), average length 52.5mm (18-83), with at least 5 secondary branches (3-9) of external diameter, 0.3mm (0.2-0.5), Table 21).

Collateral connections were observed between the vasa vasora of the ascending aorta and the RCA, conus and LCX arteries respectively, (Figure 53).

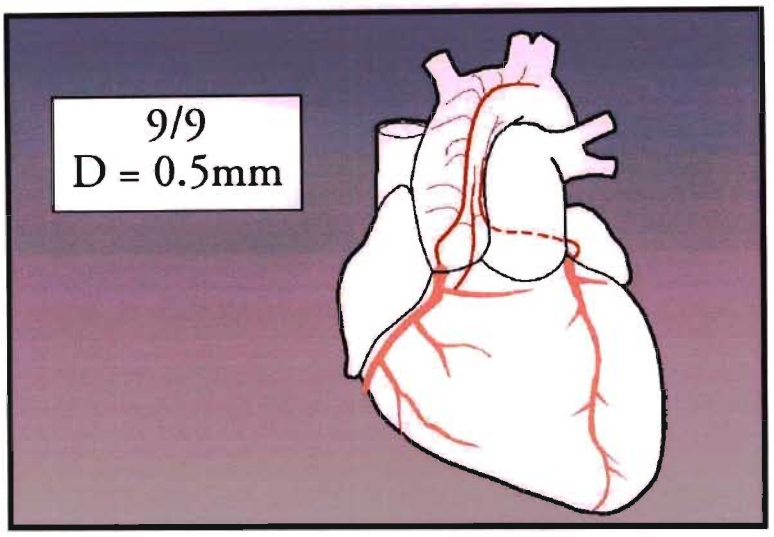


Figure 51: Incidence and morphometry of aortic ECC's

The adventitial vessels of the pulmonary trunk (PT) anastomosed with branches from the conus and LCX artery.

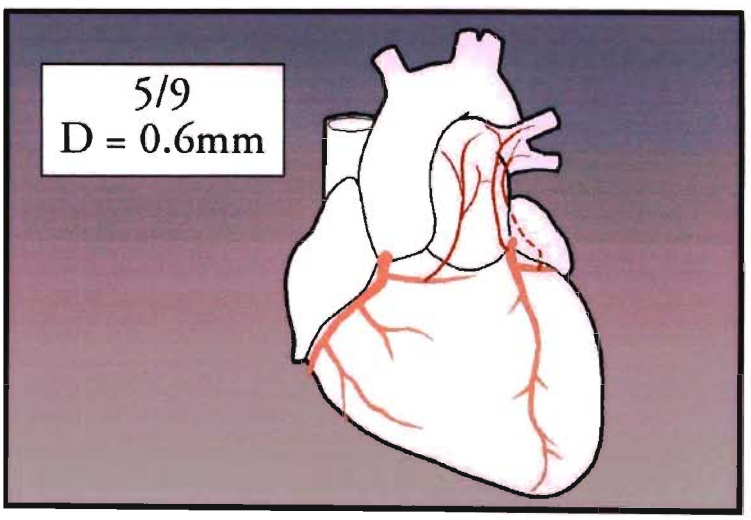


Figure 52: Incidence and morphometry of pulmonary ECC's

Connections were seen between the RPA's and RCA whereas vessels from the LAD and LCX anastomosed with those around the LPA's, (Figure 54).

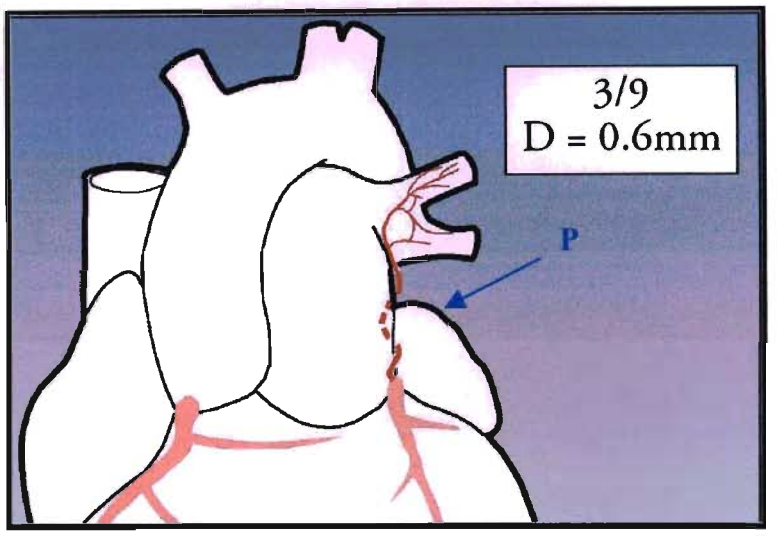


Figure 53: Incidence and morphometry of left pulmonary ECC's (P)

The right atrial branches created anastomoses with the vasa vasora of the RPV's and the LPV's received collaterals from left atrial (LA) and LCX arteries, (Figures 55 and 56).

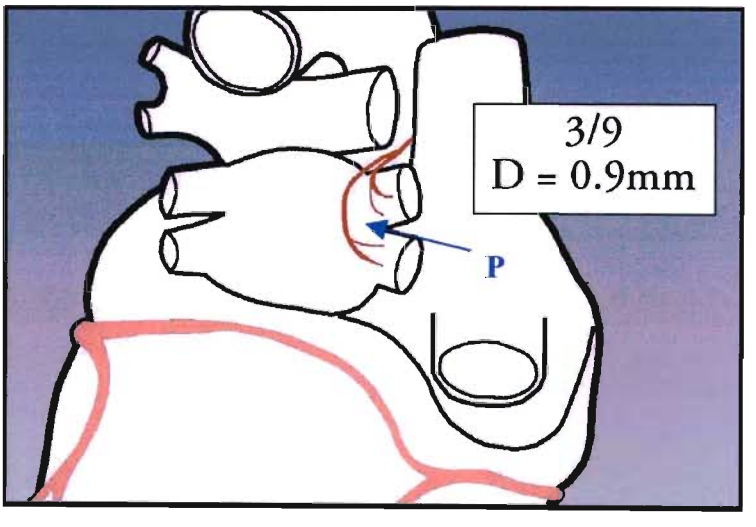


Figure 54: Incidence and morphometry of ECC's to right pulmonary veins (P)

The vessels around the IVC originated from the right atrial (RA), RCA and LCX arteries whereas those around the SVC originated from RA and LCX arteries, (Figure 57).

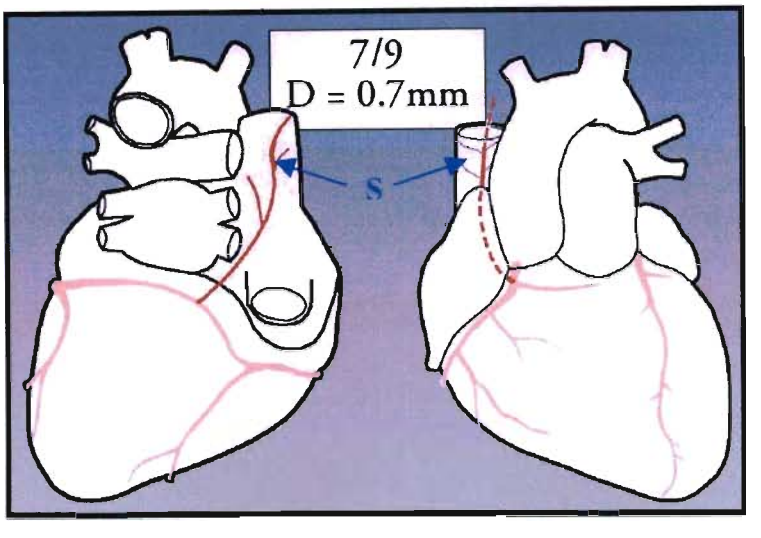
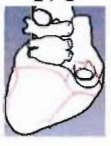
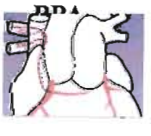


Figure 55: Incidence and morphometry of ECC's to the SVC

In addition to this usual distribution of ECC's, the following isolated cases were observed: bronchial collaterals, tracheal vessel communication, left internal thoracic to RC artery anastomosis and LAD to inferior thyroid artery connection. In the case of the inferior thyroid artery collateral, an extensive anastomosis between the pericardiocophrenic, bronchial, tracheal, pulmonary and aortic vessels were also noted.

TABLE 21: ECC MORPHOMETRY

ECC	Origin	Mean Diameter (mm) Main Branch	Mean Length (mm) Main Branch	Mean # 2° Branches	Mean Diameter (mm) 2° Branches
AA 	RCA, Conus, LCX	0.5	87	8	0.3
SVC 	RAA, LCX	0.7	41	5	0.2
IVC 	RAA, RCA, LCX	0.4	59	6	0.2
PT 	Conus, LCA	0.6	81	2	0.4
LPV 	LAA, LCX	0.6	30	3	0.7
RPV 	RAA	0.9	18	2	0.2
	RCA	0.4	22	4	0.2
LPA 	LAD, LCX	0.6	83	9	0.5

Connection between the inferior thyroid artery and the LAD

A description of an anastomotic connection between the inferior thyroid artery and the LAD was observed in one specimen. On further inspection of the anterior ventricular anastomoses between LAD and left marginal branch, an anastomotic connection was traced from its anastomosis with a branch of the left marginal to a point of anastomosis at the origin of the LCA.

In this area, a cruciate pattern of anastomoses was observed. The lower left branch from the L marginal joined a lower right branch from the fat over the PT to form a union at an area to the left of the LCA origin, between the ascending aorta and PT. The upper right branch joined the upper left branch and continued to the right, beneath the ascending aorta. The branch then travelled underneath the aortic arch and then ascended along the trachea from the left principal bronchus, diagonally across the anterior surface of the trachea and then along the right margin of the trachea. It measured 24.4 cm in length and 1.8 mm diameter throughout its course.

As it travelled across the trachea, it anastomosed with branches from the tissue surrounding the trachea. It anastomosed with a branch arising from the right tracheal region as well as the right pericardiocophrenic artery. At a point 1 cm from the carina, an anastomosis was recorded between the vessel and a right bronchial branch. The anomalous vessel continued to terminate within the tissue of the posterior lobe of the thyroid gland, closely related to an anomalous inferior thyroid artery.

The inferior thyroid artery arose from the first part of the subclavian artery in isolation as the first ascending branch, bilaterally. On the right, the vertebral, ITA and thyro-

cervical trunk of 2 branches was identified. On the left, the ITA and thyro-cervical trunk originated from a common trunk.

On the right side of the trachea, the anomalous branch contributed to an extensive anastomosis between the pericardiocophrenic, bronchial, tracheal, pulmonary and aortic vessels.

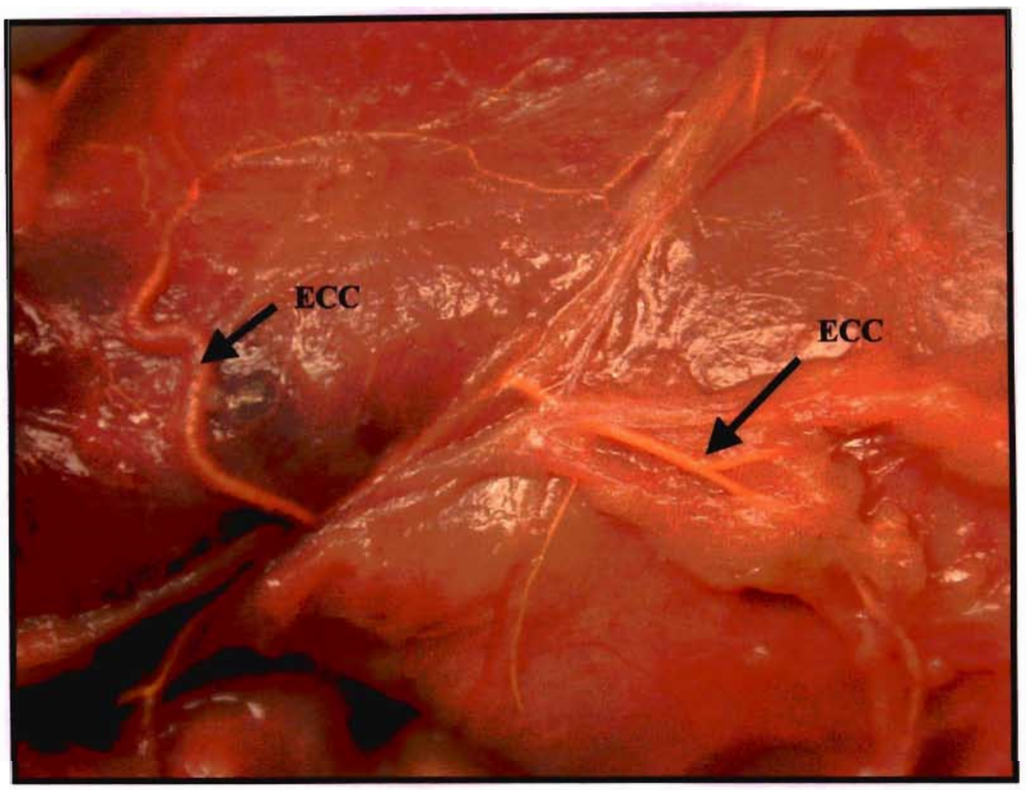


Plate 39:Demonstration of aortic ECC's

In this view, the aorta has been orientated to lie horizontally across the plane. The ECC's are shown as the PbO injected vessels that extend across the aortic wall.

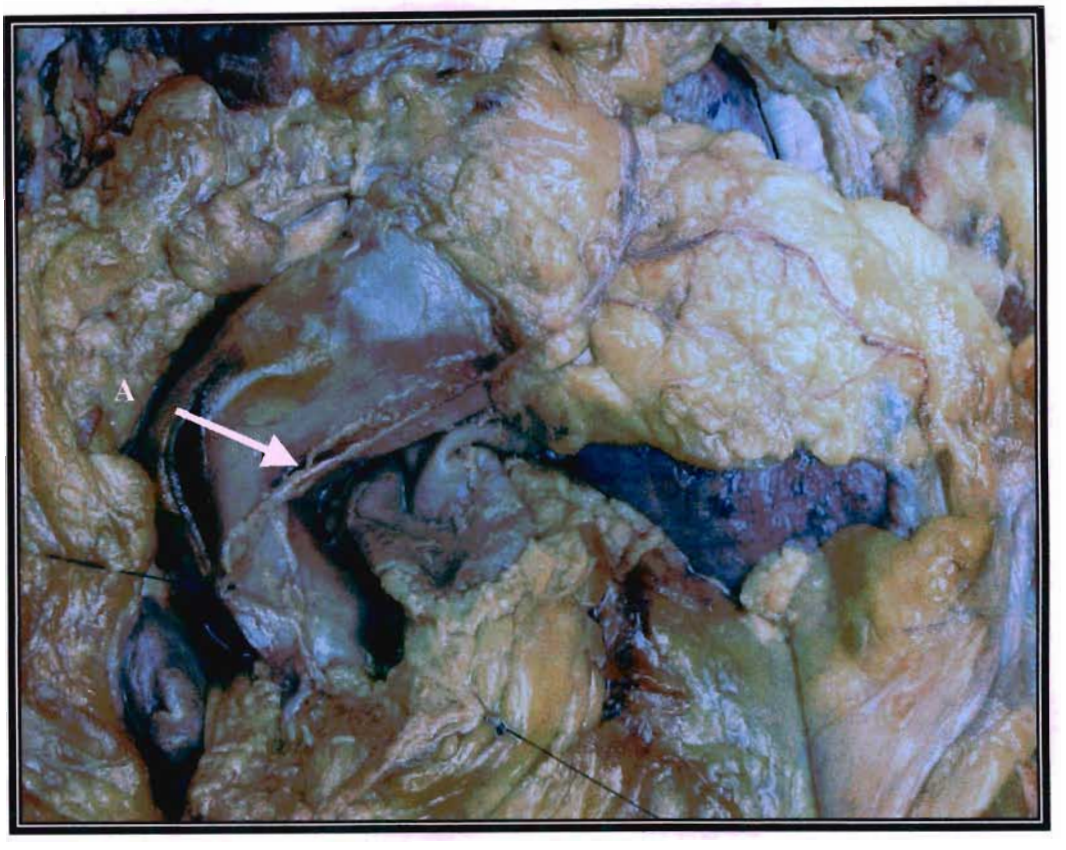


Plate 40: ECC branch (A) to ascending aorta and pericardial fat

The superior limit of the heart is in focus with the ascending aorta in the centre of the picture. A fine vessel can be seen running superiorly along the surface of the aorta. The fat has been reflected to the left to lie over the left lung.

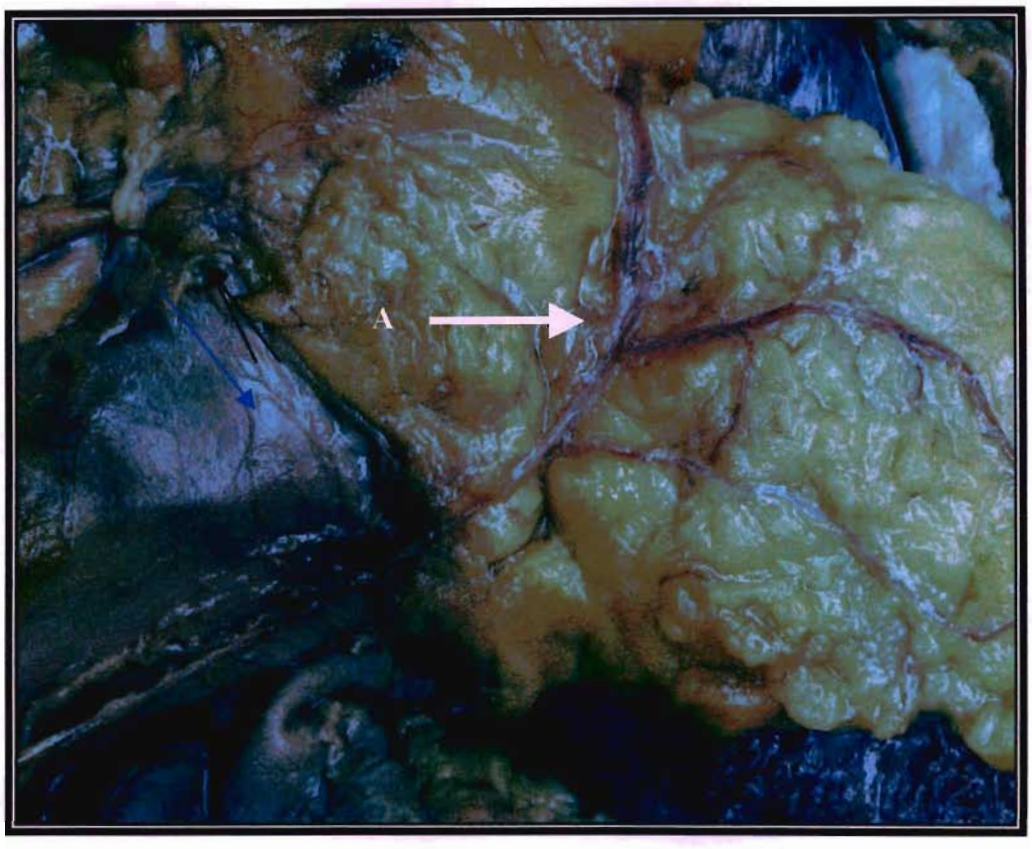


Plate 41: ECC branch (A) to pericardial fat – enlarged image

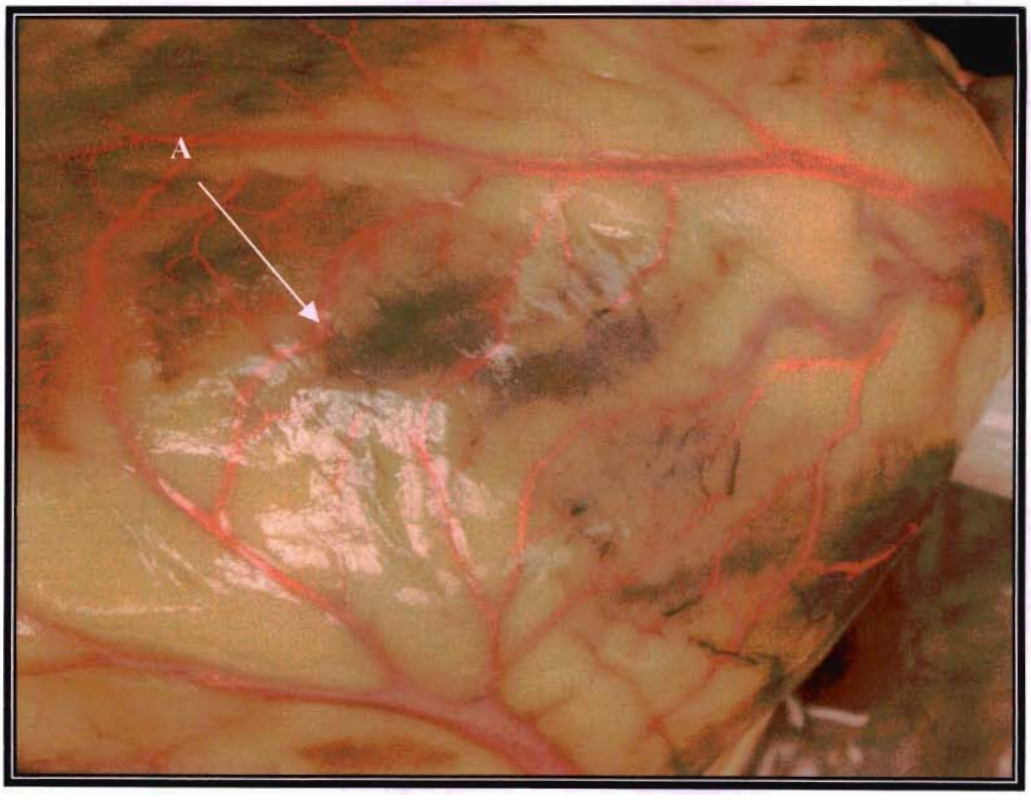


Plate 42: Inter-coronary connections (A)

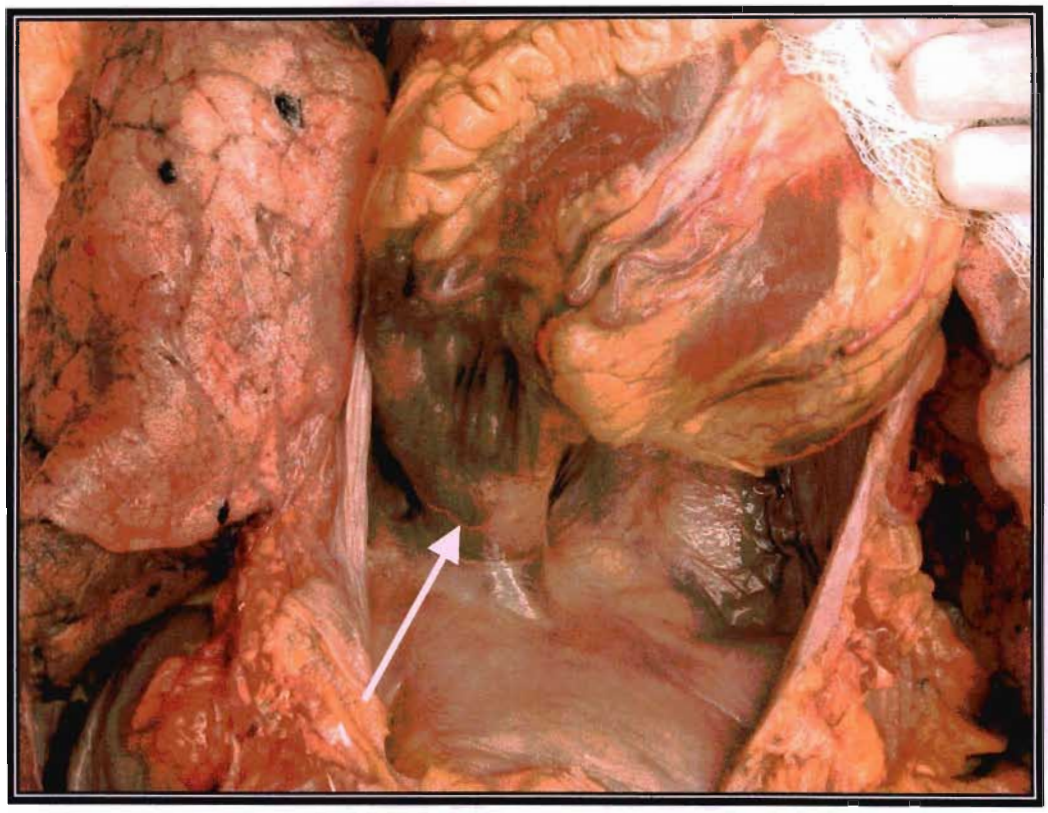


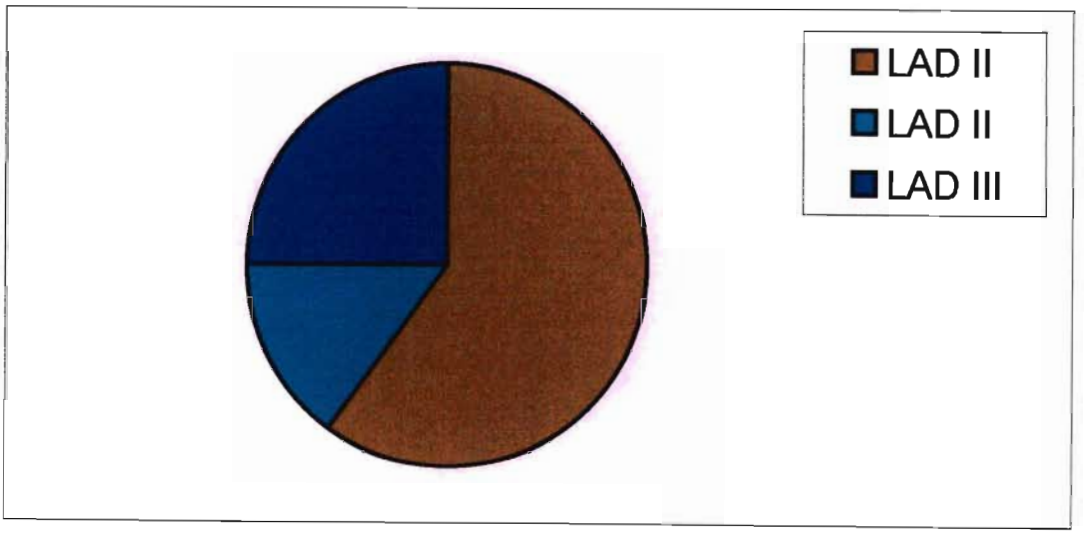
Plate 43: ECC (A) from vasa vasora of the inferior vena cava

4.4. HISTOPATHOLOGICAL ANALYSIS

The overall incidence of intimal hyperplasia and atherosclerotic changes in the LAD segments analysed was 68.96% (n=20). Of the 20 LAD segments that were identified with having sclerotic changes, 12 were counted in the LAD I group 3 from the LAD II group and 5 from the LAD III group, (Table 22).

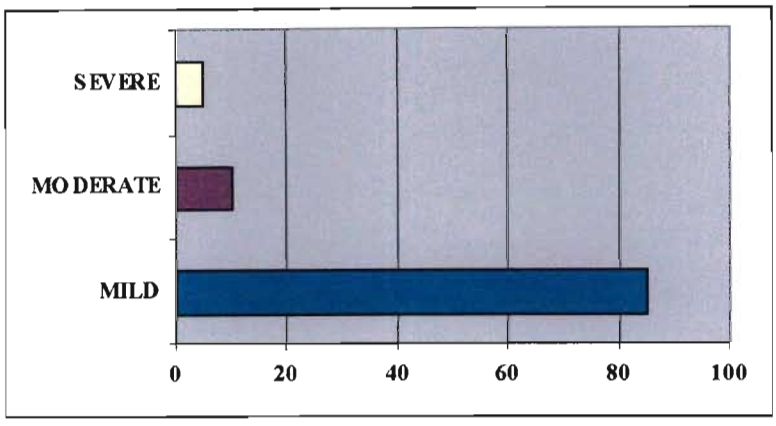
Table 22: Extent of Atherosclerosis in LAD Segments

SEGMENT	EXTENT OF ATHEROSCLEROSIS			
	MILD	MODERATE	HIGH	%INCIDENCE
LAD I	10	1	1	60
LAD II	3	0	0	15
LAD III	4	1	0	25



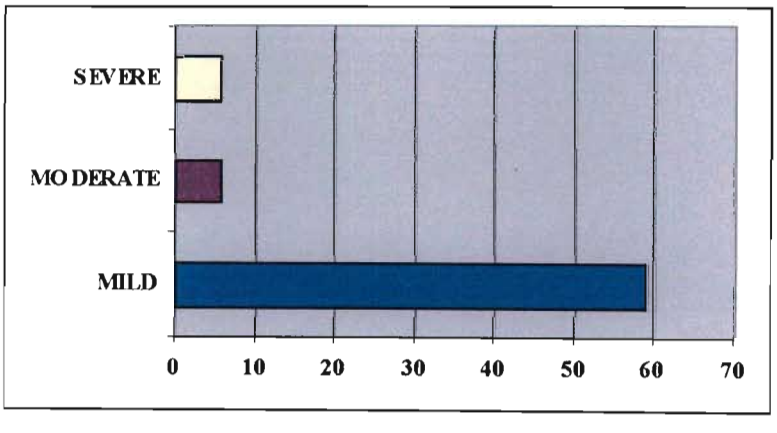
Graph 18: Extent of atherosclerosis in LAD segments

When analysed in terms of severity, 85% showed alteration to the intima indicating very early stages of the arterial disease process. In 10%, there were signs of plaque formation with moderate progression and only 5% (1 case) showed extensive disease, (Graph 19).



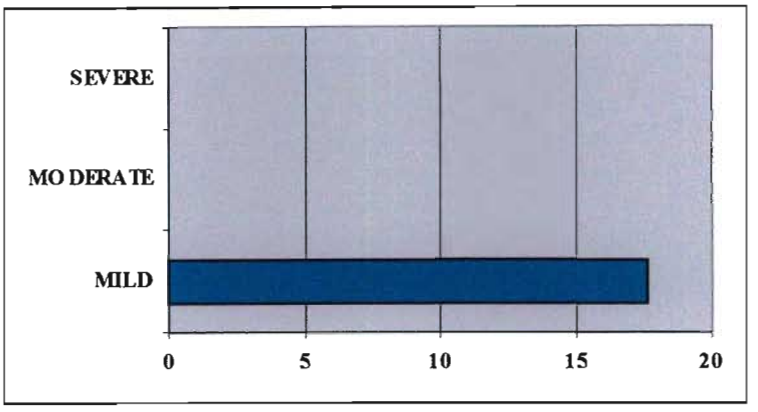
Graph 19: Degree of atherosclerosis in LAD segments

In the analysis of the arterial segments, the pre-mural portion (LAD I) recorded an incidence of 70.5% with observable signs of intimal changes, (Plates 44, 47, 50, 52). The extent of disease ranged between 5.8% in the high and moderate categories to 58.8% in the mild group, (Graph 20)..



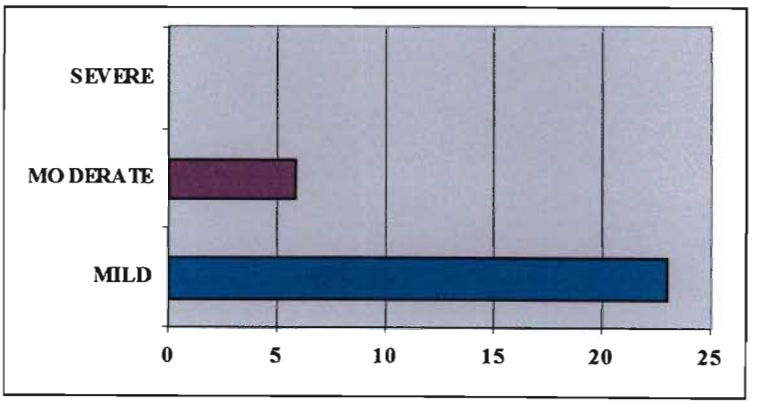
Graph 20: Degree of atherosclerosis in pre-mural LAD segment

The intra-mural portion of the artery (LAD II) showed only mild signs of intimal changes with a recorded incidence of 17.6%., (Plates 45, 48, 57, 53, 55, 56, 57 and Graph 21)



Graph 21: Degree of atherosclerosis in the intra-mural LAD segment

The post-mural portion (LAD III) recorded incidences of 23.5% and 5.8% in the mild and moderate categories respectively, (Plates 46, 49, 54 and Graph 22).



Graph 22: Degree of atherosclerosis in post-mural LAD segment

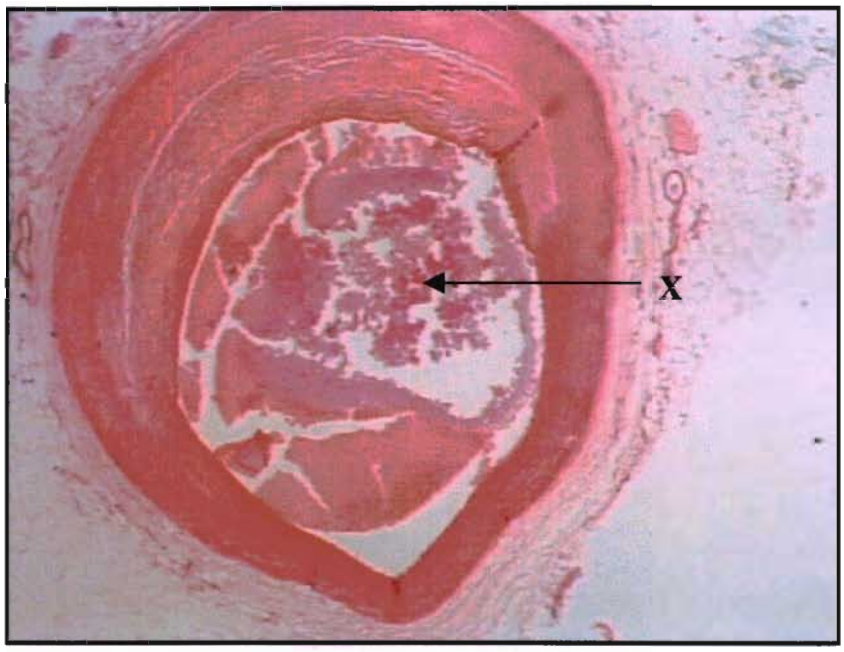


Plate 44: Pre-mural LAD (1) segment showing extensive disease

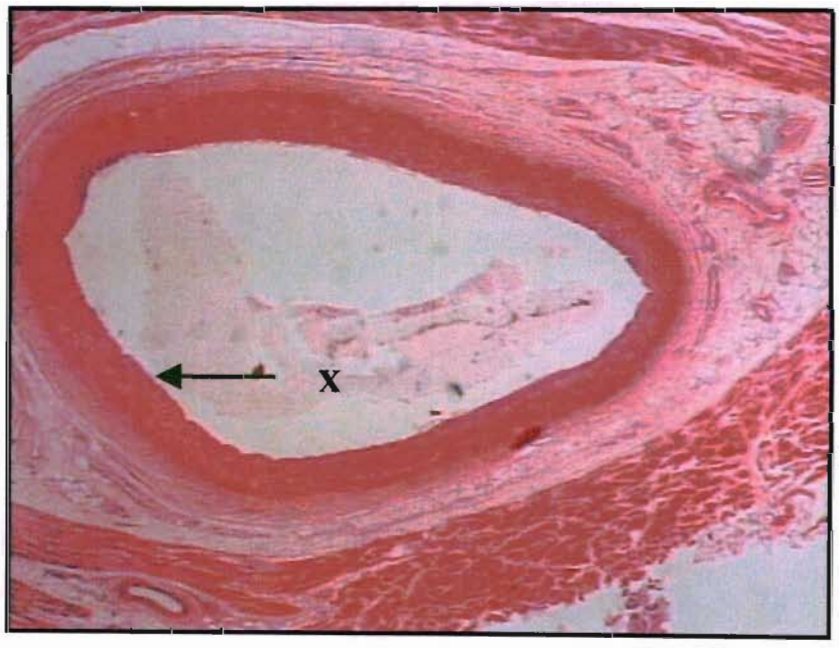


Plate 45: Intra-mural LAD (1) segment showing very mild intimal irregularity

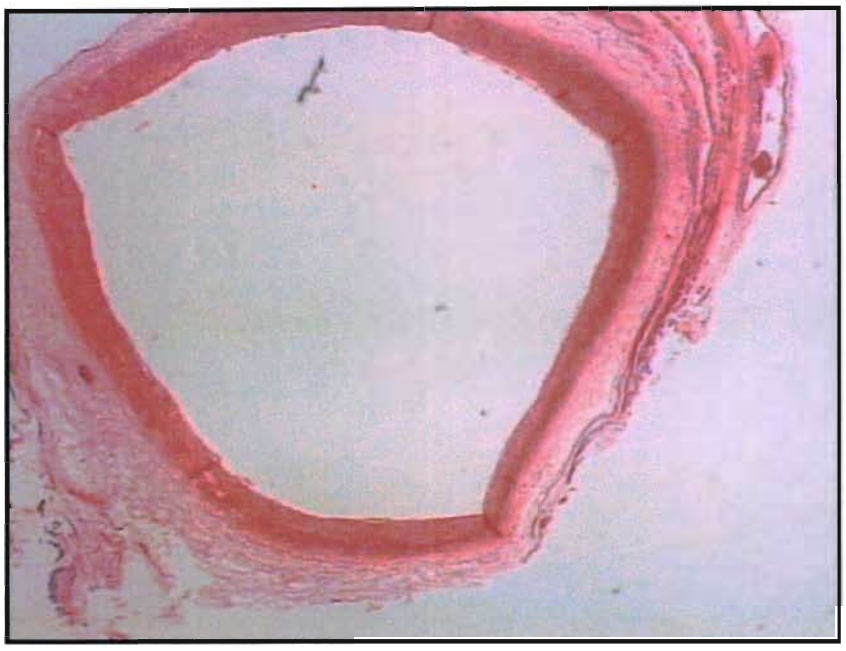


Plate 46: Post-mural LAD (1) segment showing very mild intimal irregularity

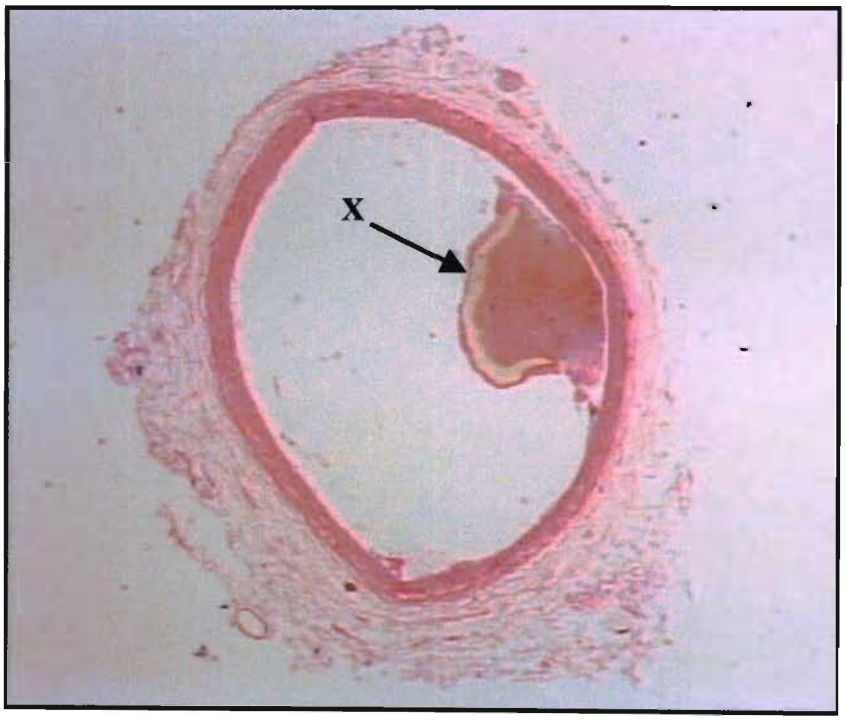


Plate 47: Pre-mural LAD (2) segment showing advanced plaque formation

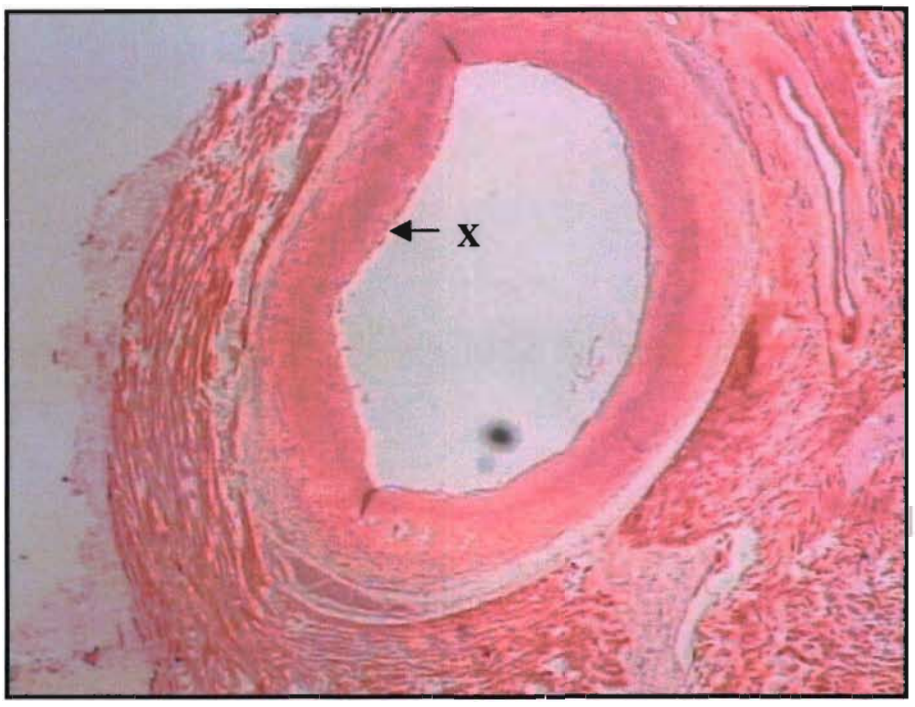


Plate 48: Intra-mural LAD (2) segment showing no signs of atherosclerosis

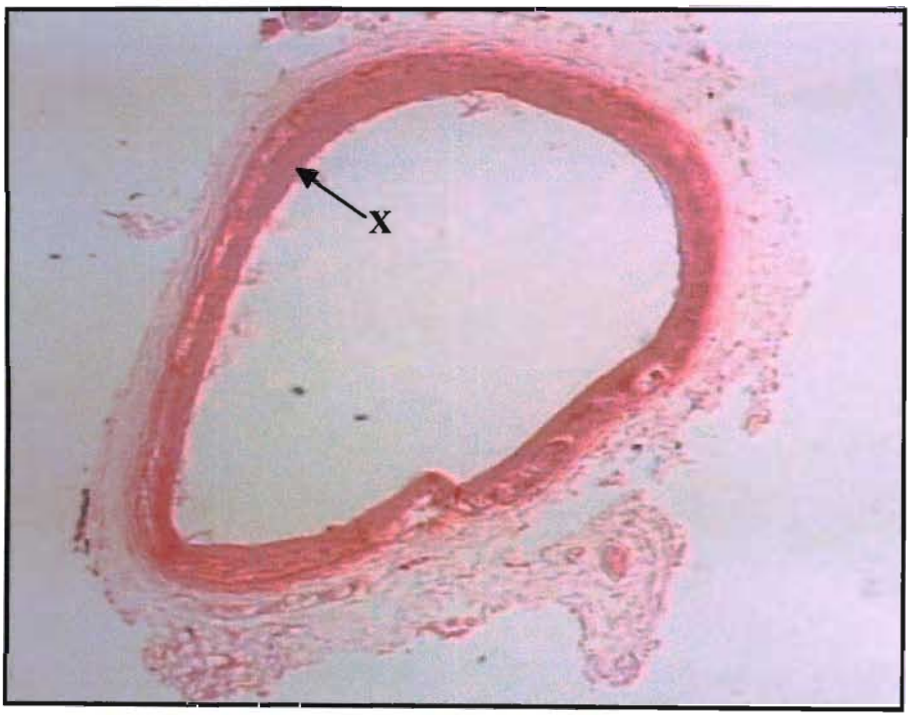


Plate 49: Post-mural LAD (2) segment showing no signs of atherosclerosis

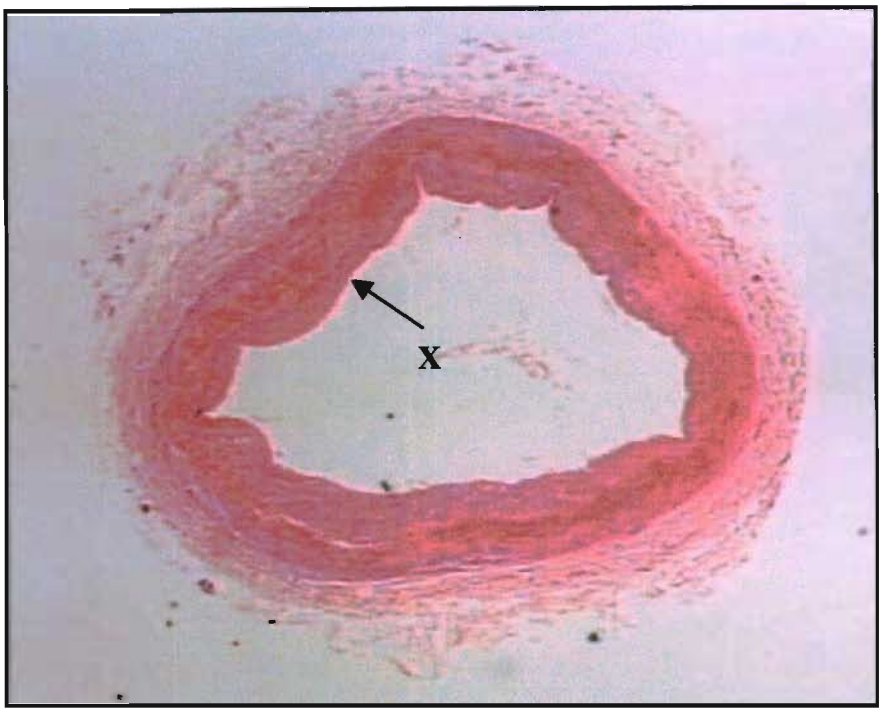


Plate 50: Pre-mural LAD (3) segment showing intimal hyperplasia

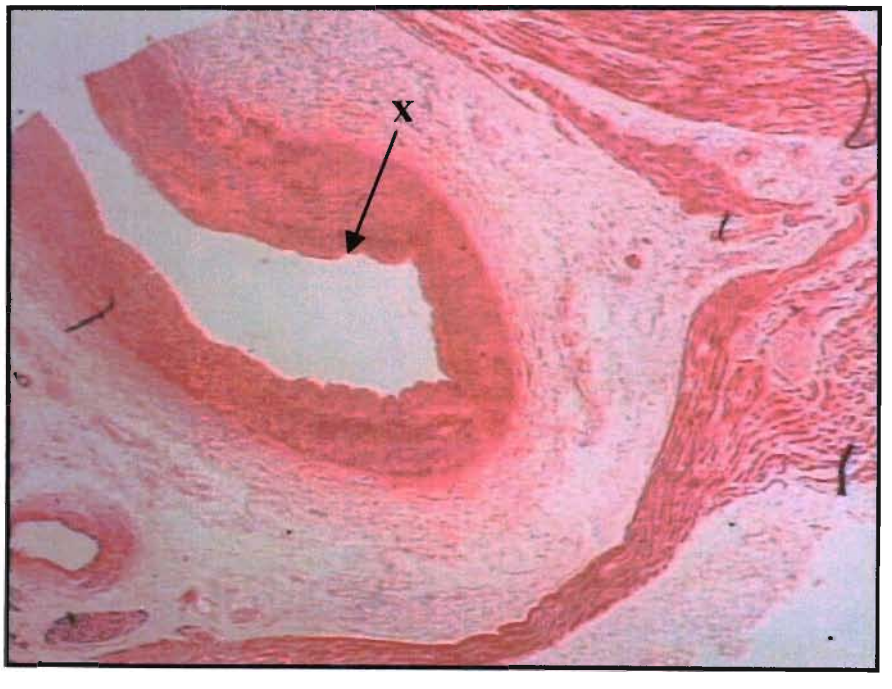


Plate 51: Intra-mural LAD (3) segment showing intimal hyperplasia

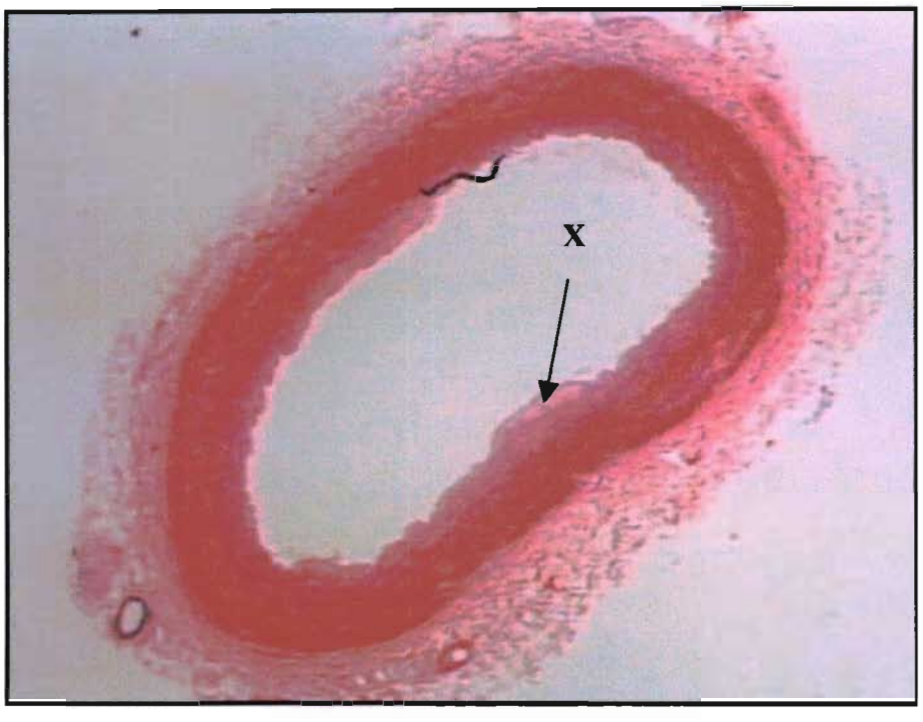


Plate 52: Pre-mural LAD (4) segment showing intimal hyperplasia

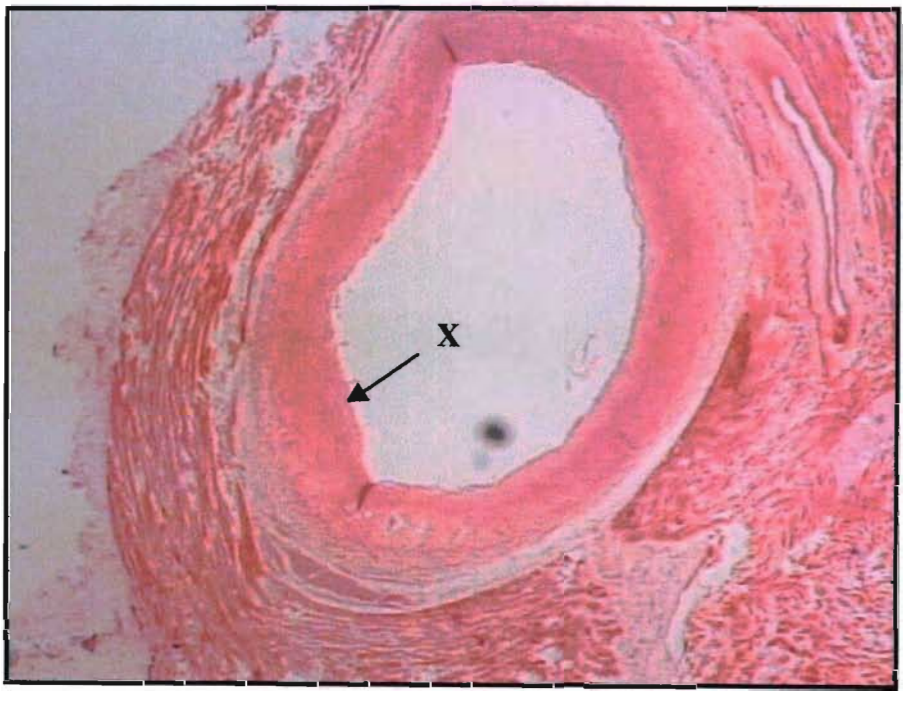


Plate 53: Intra-mural LAD (4) segment showing no signs of intimal hyperplasia

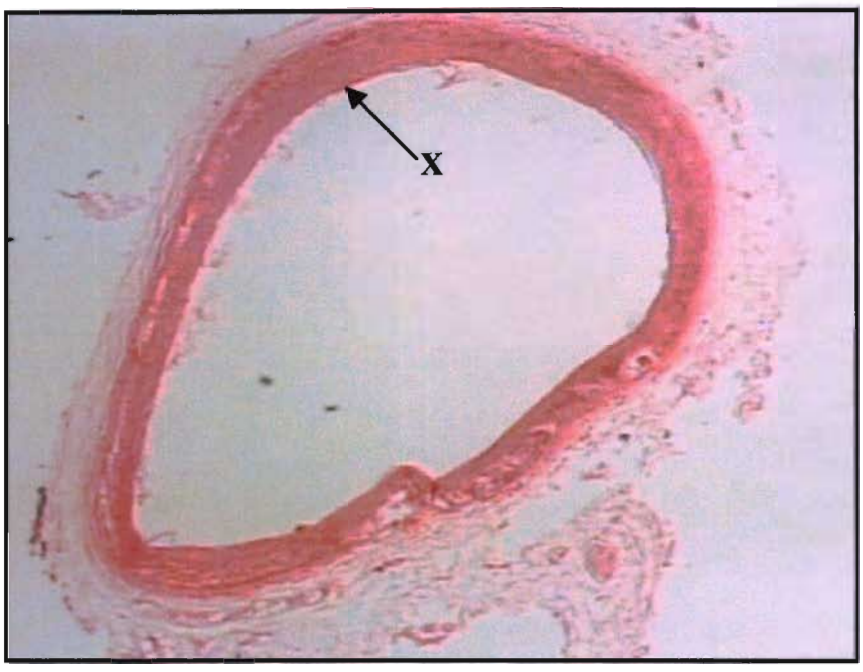


Plate 54: Post-mural LAD (4) segment showing no signs of intimal hyperplasia

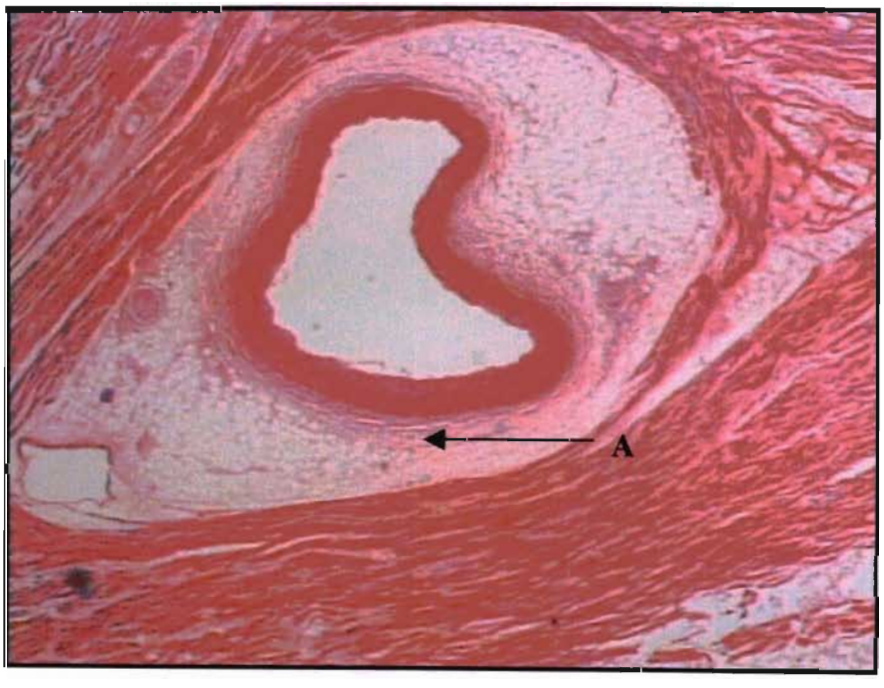


Plate 55: Intra-mural segment showing extensive surrounding layer of adipose tissue

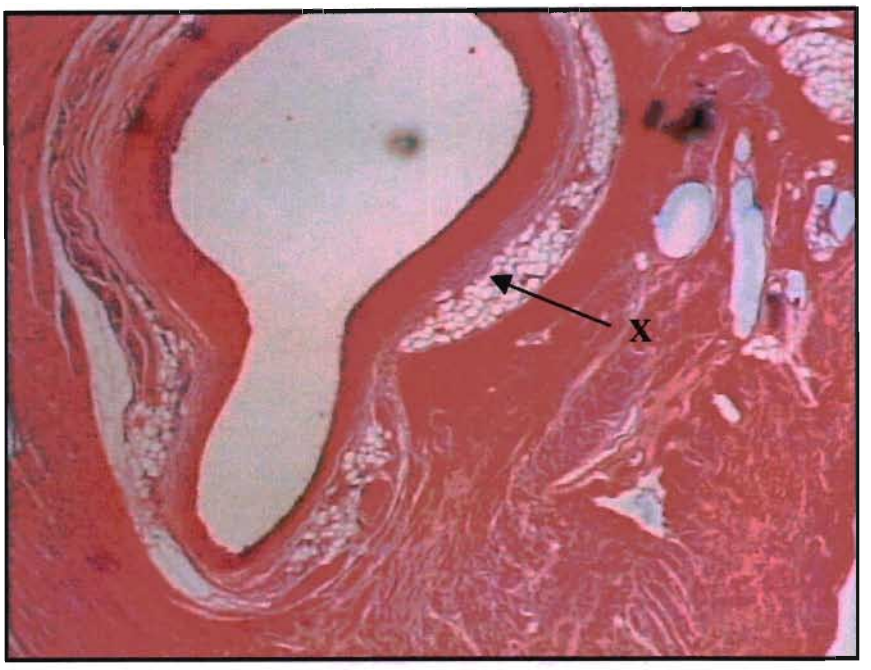


Plate 56: Intra-mural segment showing minimal surrounding layer of adipose tissue

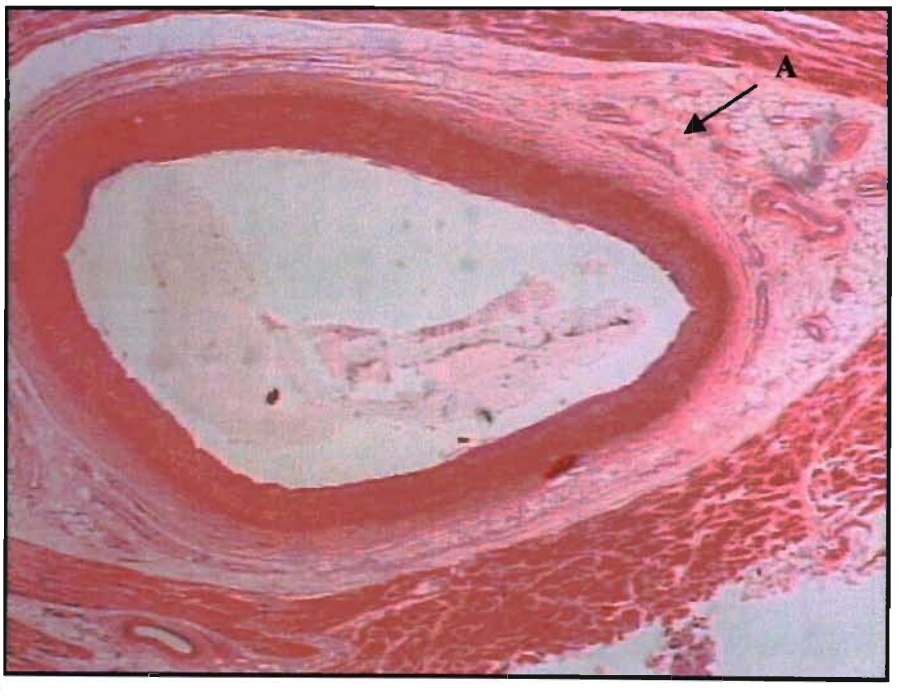


Plate 57: Intra-mural segment showing minimal surrounding layer of adipose tissue

CHAPTER V

DISCUSSION

"Anatomy is the only solid foundation of medicine; it is to the physician and surgeon what geometry is to the astronomer. It discovers and ascertains truth, overturns superstition and vulgar error and checks the enthusiasm of theorists and sects in medicine, to whom, perhaps more of the human species have fallen a sacrifice than to the sword itself or to pestilence"

William Hunter (1750)

GROSS ANATOMY OF THE CORONARY ARTERIES

5.1.1. CORONARY ARTERY ANATOMY

Coronary artery anatomy continues to sustain considerable interest amongst investigators, (Angelini, 1999; James 1961; Fulton, 1965; Allwork 1987; Kosinski and Grzybiak, 2001; Smith, 1962). Although there has been an appreciable level of standard anatomical discussion, the main research incentive appears to have been largely a factor of clinical necessity rather than anatomical curiosity.

The literature dedicated to the study of the coronary arteries have come from applying a dynamic approach in appreciating aspects of its morphology, histology and pathology. Documentation of such findings has resulted in seminal contributions by a key group of scientists, (Gross, 1921; Schlesinger, 1940; Fulton, 1965 and James, 1961) whose efforts continue to form a nucleus from which potential investigations are supplemented.

In its clinical approach, this study focused primarily on a selection of coronary arterial features that appeared to warrant further investigation within the South African population, (Tables 6, 7, 8, 9, 10, 11, 12, 13, 14 and 15).

1. The presence of the ramus marginalis,
2. Separate origination of the LCX and LAD,
3. Bifid pattern of LAD,

4. Myocardial bridging of the vessels,
5. Anomalous path of the LCX,
6. High and separate origin of the conus branch,
7. Arterial dominance and
8. The length of the LCA, were documented.

5.1.2. THE STUDY GROUP

The sample selected for this study presents an interesting construction of sub groups that appear to have assumed a distribution pattern of some clinical and social relevance. The cadaveric group consisted predominantly of Black South African hearts, (Appendix C2). There was a very small number from the Indian population, (Appendix C2) and a small but significant number from the White population, (Appendix C2).

Indeed, any attempt to afford a purposeful explanation in support of such sample distribution can only be speculative. Specimens were harvested from post-mortems conducted at medico-legal facilities and obtained under ethical guidelines from unclaimed victims of un-naturally caused deaths. Although a dependable resource, the increasing restrictions that continue to be placed upon the management of such facilities has significantly limited the number of specimens dedicated for use in this study.

The group investigated in the angiogram analysis presented quite the opposite picture, (Appendix E1). In this sample, Indian South Africans formed the predominating group. Whilst Whites made up a significant portion, the number of arteriographs obtained from Black patients was minimal, (Appendix E1). Besides the fact that this patient profile may

well be reflective of a social disposition, the fact that Indian's present by far with the highest rates of heart disease, is strikingly evident, (Dhawan and Bray, 1995; Ranjit et al., 2001).

The cadaveric profile for the investigation of the ECC's was determined from specimens obtained within the United States, (Table 21). The cadavers were Caucasian with advanced age and larger body size.

The observations reported in this study on the anatomy of the coronaries have been based not only on statistical evidence but also to an equally significant extent on dissection experience. Such understanding of anatomical structures and formations cannot be simply appreciated by a few random dissections. Indeed, the essence of true comprehension lies within the fundamental task of adept inspection and acquired familiarity.

5.1.3. EVALUATION OF THE CORONARY ARTERIES

The standard anatomy of the coronary arteries was confirmed as described in the literature reviewed, (Williams et al.1989; Angelini et al., 1999; Alexander et al., 1998; Gross, 1921); James, 1961). Right and left systems were identified and appeared somewhat consistent in their distribution. Branching patterns varied, but in no way unique to the already existing list of coronary artery anomalies.

The main coronary trunks were found to be unbalanced in their positions of origin from the sino-tubular region, the left often situated slightly higher than the right. These results compared favorably with reports by Turner and Navaratnam, (1996). Although data

regarding ostial locations in relation to the aortic sinuses are available, the factors promoting the positioning of the ostia remain unclear.

The anatomical construction of the heart itself favors this arrangement. As shown in (Plates 6 and 7), when viewed with the right anterior ventricular surface in front, this disposition of the coronaries is clearly demonstrable. It is interesting to note that the coronary ostia in addition, vary in shape. During the post-mortem endoscopic visualization of the ostia, it was noted that the left usually appeared "slit-like", whilst the right was more rounded.

The widespread use of coronary arterial catheterization for diagnostic and therapeutic purposes has brought about an increased awareness of coronary artery anomalies. In the event of cannulating coronary arteries during cardiac catheterization, (Muriago et al., 1997) knowledge of such variance in ostial location may be technically valuable in achieving successful intra-vascular advancement of a catheter. In addition, from a clinical standpoint, the shape of the lumen may well influence the degree of occlusion that atheroclerosis may have in the event of it being present.

Variation in origin and branching pattern of coronary arteries are of interest to the anatomist and clinically important to the interventional cardiologist and cardiothoracic surgeon, (Abedin and Goldberg, 1978).

In considering the early branching patterns of the right and left main trunks, two points of variability are brought into focus; a). The pattern of terminating pattern of the LCA and b). The origin of the conus branch from the RCA.

5.1.4. BRANCHING PATTERNS OF THE LCA - THE RAMUS MARGINALIS

In the South African series, the branching pattern of the LCA was observed according to trifurcation and bifurcation modes of termination.

The bifurcation pattern appeared more frequently with an incidence of 86.7%, (Tables 6 and 7). The ramus marginalis branch was observed in the remaining 13.3%. With the relative infrequency of its occurrence, the ramus marginalis has not been afforded much attention in the anatomical literature. Its presence however has been mentioned in terms of its clinical relevance in surgery and diagnostic imaging, (Ochsner and Mills, 1978 and Alexander et al., 1998).

The ramus marginalis may be identified as the central branch of the LCA that arises between the LCX and LAD arteries. As it descends onto the anterior ventricular surface, it arches slightly away from the myocardium, where it is suspended in a pad of fatty tissue. When present, the ramus marginalis engages the role of the main obtuse marginal artery in supply to the upper surface of its left ventricular territory. The artery is often limited in its length, ending short of the obtuse margin, in a characteristic tortuous taper, (Plates 16, 17 and 23).

There are seldom any significant branches but fine branches appear to arise from its right border to join the diagonal branches of the LAD artery in their supply. It is usually equal in size to the LCX and LAD, but may in some cases exceed the external diameter of the LCX artery.

The recorded incidence of the ramus marginalis appears to fall within the range of 7 to 15% as reported by Ochsner and Mills (1978). The authors suggest that when identifying the artery on angiogram, it may be helpful to consider that the vessel may be seen to arise higher than the LAD and LCX when viewed in the LAO position. Gorlin (1976) raised an interesting correlation regarding the take off points of secondary coronary branches and their association with evidence of atheroma. He concludes that early and frequent branching within a short space may be an influencing factor in the predilection of arterial disease. The likelihood of a ramus branch being prone to disease in an already compromised arterial system is therefore high and has been confirmed as such during the course of dissection within this study.

The ramus marginalis may be present twice as often in females than in males, (10.7% Vs 5.6%). With regards to its ethnic distribution, the mean incidence is highest amongst Blacks (18.0%), followed by Indians (6.6%) and then Whites (1.4%), (Table 6).

Tobias and Allen (1988) described the presence of a "diagonal" artery that occurs more frequently amongst African natives than Caucasians from within the same area. It appears likely that the authors may indeed have described what is being regarded as the ramus marginalis. If this is so, then their findings corroborate those of this study in highlighting this ethnic association. There is an obvious disparity however in the application of the nomenclature, which draws back to the point of the dissociation between anatomical and clinical descriptions with regards to the ramus marginalis. In view of what has already been discussed, it may be appropriate therefore to formalize the status of the ramus marginalis within the anatomical literature.

The termination pattern of the LCA when it occurs as a bifurcation is well documented and commonly seen. In this series, an incidence of 86.7% suggests concurrence with that reported in the literature, (Williams et al., 1989). In very rare occasions, the LCA may end in a quadrification. In the total bank of 323 arterial patterns studied, 3 cases of such a pattern were noted. Although this incidence may appear insignificant, when compared to the less than 1 in 2000 cases reported in the literature (Ochesner and Mills, 1978) of LCA quadrification, its occurrence is at least reason for note.

5.1.5. CORONARY ARTERY MORPHOMETRY AND THE LENGTH OF THE LCA

The morphometry of the coronary arteries has during recent years received more attention, (Alexander et al., 1998; Dhawan and Bray, 1995 and Kronzon et al., 1974). Having developed from the clinical interest in patient profiles, there have been few subsequent reports highlighting ethnic variation in terms of coronary artery dimension, (Dhawan and Bray, 1995 and Kurijia et al., 1986).

The clinical impression in South Africa supports the notion that the size of the vessels varies between the major patient groups within this population. In a report by Vanker et al., (2000), the morphometry of the internal thoracic arteries (ITA's) was shown to vary between patients. When compared, White South Africans had larger ITA's than Indian South Africans. The study highlighted the clinical correlation that where arterial size was smaller, surgical results and indications were not always optimal.

Dhawan and Bray (1994) having been motivated by similar clinical issues, have documented findings of their studies within the United Kingdom where the patient profile compares

favorably with that within the South African population. Their investigation confirmed Indian coronary arteries to be smaller than those patients of European descent.

The group distribution for this study illustrated a very interesting clinical picture. In the angiogram group, the predominating population was Indian. When compared by percentage, this group formed almost two thirds (64%) of the total, confirming the known high incidence of cardiovascular disease within this community. In all patients presenting for diagnostic cardiology, cardiovascular disease was confirmed, with 2 and 3 vessel disease in most cases. Such being the pathologic profile, it was not possible to achieve any valuable calculation of luminal diameter from these angiograms. Similarly, any attempt at measuring coronary diameters would not have achieved scientific validation. All further morphometric analysis was therefore limited to the cadaveric group and directed by the reviewed commentaries along the lines of clinical relevance.

The length of the left main trunk was investigated and compared within the selected population. These were the only measurements conducted under standard dissection.

The mean length of the LCA was recorded to be 0.82cm and ranged between 0.4 -2.4 cm. The mean length of the LCA recorded for males was 0.96cm and for females, 0.84cm. Results of the T-test approached significance at the 5% level ($p > 0.05$). However, the mean indicated that the LCA was larger for males, (Tables 14 and 15; Graphs 14a and14b).

When evaluated in terms of ethnicity, the highest mean length was recorded in the Black population (0.88cm). The LCA within the White group measured 0.78cm and within the Indian group, the LCA was found to be 0.53cm, (Table 15). Statistical significance existed

between ethnic groups with regards to LCA length. Indians were shown to have the shortest left main trunk.

The left main stem is known to range in length from 1 to 4mm, (Alexander et al., (1998). Findings of this study appear to fall below the reported range within the literature. Muriago et al., (1997) believe that a shorter main stem may pose problems in coronary angiography or during surgical procedures in which cannulation is indicated. The author suggests that high branching of the left main stem tends to promote unwanted lodging of the catheter in one of the branches and reduce the flow to other branches. Results of this study highlights an important clinical concept and indicates the need for further morphometric evaluation of coronary artery dimensions, especially within the South African Indian group.

5.1.6. SEPARATE ORIGIN OF THE LAD AND LCX

In most hearts, a left main trunk (LCA) is an easily identifiable structure. The absence of secondary branches of the LCA suggests that the role of the LCA in the coronary arterial formation may indeed be only to supply a common trunk for the origin of the LCX and the LAD. That perhaps being its sole purpose, in the event that the LCX and LAD should arise via separate ostia from the aortic sinus, the LCA would be considered absent.

In about 14.5% of the population studied, the LAD and LCX arose independently from the aorta. The incidence appeared slightly higher in males than in females, 15% Vs 7.5%, (Table 7; Graphs 8a and 8b; Plate 19). A significant difference between ethnic groups was noted. Indians were found to have the highest recorded incidence (50%), followed by Whites (35%) and then Blacks (6.5%).

When compared with reports in the literature regarding independently originating LAD and LCX arteries, (James, 1961 and Schlesinger, 1940), an incidence of 14.5% in this series is significantly high. A comparison of incidences for this anomaly is illustrated in the table below.

Table 23: Separate origin of LAD and LCX arteries – Inter-population comparison

Reference	Location	% Incidence
James, (1961)	USA	0.9
Schlesinger, (1940)	USA	0.2
Velican, (1981)	Hungary	3
Kurjia, (1986)	Iraq	0.8
Chaudry, (1965)	Pakistan	1.8
Donaldson et al., (1982)	England	0.1
Present Study	**South Africa**	**14.5**

In discussing the occurrence of this arterial pattern, Angelini et al., (1999) made a point of categorizing such a find as anomalous since its occurrence was recorded in less than 1% of the population studied. If Angelini et al.'s (1999), basis for classification were applied to the findings in this study, then it would appear that separate origination of LAD and LCX arteries in the absence of a left main trunk would be a "normal" rather than anomalous occurrence within the South African population. Even in the event of excluding the results from the Indian group due to the small number, the 35% in Whites and 6.5% incidence amongst Blacks tend to support this assumption in classification.

Interventional cardiologists need to be aware of the incidence of such an arterial pattern when evaluating patients within this population.

5.1.7. THE BIFID LAD ARTERY

The LAD and LCX were observed further in terms of their course. The course of the LAD appeared to be constant along its descent between the right and left anterior ventricular walls. Its position however appeared rather inconsistent when observed in association with the anterior inter-ventricular groove, (Table 10; Graphs 10a, 10b; Plates 24, 28).

In at least 52% of cases, the LAD was observed to pass to the left of the groove and within the groove as the established "anterior inter-ventricular artery" in only 31% of hearts. In 17%, a single artery was replaced by a bifid structure with right and left components.

In a confirmed bifid LAD, the components are almost always equal in size and both pass along a forward and downward incline in close proximity to each other. In this way, a true bifid LAD may be distinguished from a dominant first diagonal branch. It was interesting to observe that the right component was usually covered by a bridge of myocardium, arranged in a paralleled fashion along the upper part of its course, (Plates 24 and 30).

The literature reports an incidence of approximately 4% of LAD bifurcation (Alexander et al., 1998), which is significantly lower than the 17% recorded in this study. When considered clinically, such an early bifurcation of the LAD may not be conducive to good revascularization of the vessel, (Ochsner and Mills, 1978). In addition, it may be important

to anticipate that in the presence of a bifid LAD, the right branch has a high possibility of being intra-mural. Since the determination of such a location is difficult from angiographic observation, knowledge of this anatomical arrangement may prove valuable.

There was no significant difference between sexes with regards to the presence of a bifid LAD. However, presence of a bifid LAD varies significantly between ethnic groups. In this study, Blacks were found to have the highest recorded incidence (17.6%) followed by Indians (6.3%) and then Whites (4.5%), (Table 10; Graphs 10a, 10b).

5.1.8. ANOMALOUS PATH OF THE LCX ARTERY

When reviewed within the standard anatomical literature (Williams et al., 1989; Moore, 1992, McMinn, 1987), the LCX artery appears to have earned its designation by its tendency to course through the left atrio-ventricular or coronary groove, which occupies the superior circumflex area of the heart. In tracing its course through anatomical dissection however, it is evident that the LCX sometimes fails to fulfill its anticipated course. In this event, it descends instead along the obtuse margin of the heart, short of the posterior left ventricular surface without reaching the vicinity of the crux., (Plate 22 and Table 11).

Anatomically, such a path taken by the LCX may be considered anomalous. An incidence of 27.7% of such a course was recorded within this study group.

There has been one report of this anomalous path of the LCX. Kumar in 1989, after dissecting 132 hearts, found only 1 heart in which the LCX deviated from its course in the

left atrioventricular groove by passing inferior to the sulcus and failing to reach the vicinity of the crux.

The author's description corresponds with the observations of this study and the resulting course and distribution pattern of the corresponding vessels are comparable. The close to 28% incidence in this study however differs significantly from the less than 1% find in Kumar's, (1989) investigation.

Kumar, (1989), citing Jain and Hazary (1958) continues to report that such an incidental course of the LCX may resemble the pattern commonly identified in "buffalos, goats, sheep and dogs". He subsequently adds that such anatomical display may indeed support the theory of human beings having evolved from quadrupeds. Interesting. Though there is uncertainty in how one would correlate his theory with the high incidence within the experimental population of the present study.

Even more confounding, is that Edmunds, (1997) in his book "Surgical Anatomy of the Heart" makes a point when introducing the LCX of mentioning that in 85 to 95% of patients, the LCX terminates at the obtuse margin of the heart.

Such disparity appears to support a need for further understanding and in some way, to create a convergence between descriptions of similar structures and concepts within the clinical and anatomical literature.

5.1.9. ARTERIAL DOMINANCE

Introduced by Schlesinger (1940), the term dominant is used to indicate the areas of the heart supplied by each artery. Although the left system is known to supply a greater mass of myocardium than the right, it is not usually "dominant", (Allwork, 1987). The dominant coronary artery is that which gives rise to the posterior descending artery, coursing the posterior interventricular groove.

Nerantzis et al., (1996) however, brings a different perspective to the issue of dominance. The authors consider that beyond the "usual" measure of arterial dominance lies the concept of "real" dominance. They propose, that when the RCA extends beyond the crux to the left circumflex territory after having given off the PDA, the coronary system is said to be one of "real right dominance". In this instance, the extension of the RCA is responsible for supplying the posterior papillary muscle. Clinically, this may explain why certain lesions of the RCA may result in severe mitral dysfunction.

The arterial supply to the posterior ventricular area is most frequently a contribution from the RCA. In the cadaveric investigation, the RCA was found to be the arterial source of the PDA in 80.7%. Further observation of the PDA in each case revealed a single branch pattern in 78% and a double branch pattern in 22% of the hearts studied, (Tables 8 and 9; Graphs 9a, 9b).

The variation in morphology of the PDA becomes clinically relevant during interpretation of angiograms. Arsiwala, (1993) described a variant course of the PDA where the artery was found to lie outside the groove after having had an early origination from the RCA. In this

case, it is important to be aware that the PDA should not be mistaken for the acute marginal branch. During the course of reviewing angiograms in this study, there were occasions during which this pattern was observed. Although there was no record of the incidence, based on a clinical impression, a recommendation may be made that if attempting to distinguish between the acute marginal and PD arteries, it may be noted that the acute marginal branch typically arches a short distance from its takeoff point as opposed to the PDA passing without any proximal angulation.

In 6% of cadavers the PDA came off the LCX and in 12%, the LCX gave off a smaller PDA equal in size to the posterior ventricular contribution from the RCA.

In the total combined sample of cadaveric hearts and angiograms, there was an average of 84% incidence of right dominance, 9% incidence of left dominance and 7% incidence of co-dominance.

There was no significant difference in arterial dominance between sexes. Both sexes showed an equal tendency toward each type of preponderance. The right dominant pattern showed the highest occurrence followed by left and then co-dominant systems, (Table 8)

There was no relationship between arterial dominance and ethnicity. Results indicate highest tendency toward right dominance in all three groups studied. Although not significant, there appears to be a higher incidence of left dominance amongst whites and a higher incidence of co-dominance amongst Blacks, (Table 9; Graph 9b). In addition, *p*-values in the table of coefficients indicate that neither sex, age, ethnic group nor height, influence the dominance of an arterial pattern.

The incidences reported in this investigation vary in comparison to the findings reported in the literature. The high incidence of right dominance compares favourably with that recorded by Allwork et al., (1989), Edmunds, (1997); Gross, (1921); Chaudry, (1965); Blumgart et al., (1976) and Kronson et al., (1974). When measured against findings of Kurjia et al., (1986); Schelzinger et al., (1940) and Velican et al., (1981), results were significantly higher. Comparative incidences are presented in the tables below.

Table 24: Review of documented incidences of coronary arterial dominance

Documented Reports	%RIGHTDOMINANCE	%LEFT DOMINANCE	%CO-DOMINANCE
Allwork et al., (1989)	70	15	15
Edmunds (1997)	85-90	-	-
Gross (1921)	92	-	-
Chaudry (1965)	75	13	12
Blumgart et al., (1976)	72	10	18
Kronson et al., (1974)	84	10	3
Kurjia et al., (1986)	46	14	40
Schelzinger et al., (1940)	48	18	34
Velican et al., (1981)	58	15	27
Present Study	**84**	**9**	7

Dominance can be a significant determinant of prognosis in acquired coronary artery disease. In most incidences of left dominance, the RCA is usually small and fails to reach the right margin of the heart. Consequently, an acute, proximal occlusion could have unfavourable effects as the potential for re-opening collateral channels is diminished. Surgically, the pre-

operative indication of dominance may have an influence in the determination of graft placement.

5.1.10. THE ORIGIN OF THE CONUS ARTERY

The branch origin of the conus artery has been a subject of recent clinical interest. As described in the literature review, (Williams et al., 1989) the conus artery usually arises as the first branch of the RCA. The primary role of the conus artery is to supply the conus of the infundibulum, the territory of the pulmonary trunk. In its lateral course across the infundibulum, the branch is said to complete the well-known *"Circle of Vieussens"* by anastomosing with a corresponding branch from the LAD.

Variations in the origination of the conus artery have been documented in this study, (Tables 12 and 13; Graphs 12a, b and 13a, b). In particular, the height of its origin from the RCA and its independent origin from the ascending aorta has been highlighted as a clinically significant pattern when attempting successful cannulation of the right coronary ostium.

This investigation evaluated the artery as a constant, being present in 100% of arterial patterns studied. The conus artery arose as the first branch of the RCA in 77.2%. Its origin from the RCA at the angle between the main trunk and the aortic wall was recorded in 19.2% and regarded as a high conus origin. In 3.61% the conus arose via a separate ostium directly from the ascending aorta, (Tables 12 and 13).

A "high origin" of the conus artery is defined when the takeoff of the artery is at the angle between the RCA and external aortic wall. In this case, the orifice of the branch is separated from the RCA orifice by a thin ridge of arterial wall, (Plate 14).

The incidence of high origin of the conus artery appears to be slightly greater in males (21%) than in females (16%), (Table 12 and Graph 12a).

A positive relationship between ethnicity and high origin of conus artery was identified. An incidence of 100% was recorded amongst Indians, 19.7% amongst Blacks and 10% amongst Whites. Although it appears as a constant occurrence amongst Indians within this group, it is important to note that the sample size for this group in the cadaveric investigation was very small (n =2) and the possibility of there being a change in this reported incidence is high. It should therefore remain as a finding of note rather than one of significance.

Independent origin of conus artery from the ascending aorta appeared to be somewhat uncommon. Although recorded, the incidence amongst males was 3.8% and 4% amongst females, (Table 13 and Graph 13a). Its occurrence was noted in about 5% of Blacks with no observation amongst Whites or Indians. These results differ from reports in the literature. Muriago et al., (1997) reported an incidence of 75%, whereas Schlesinger et al., (1949) and MacAlpine, (1975) recorded this occurrence in 30 to 50% of normal hearts studied.

Correct identification of the origin of the conus branch and its relationship to the infundibulum is said to be an important pre-operative practice and in particular when dealing with congenital anomalies such as Tetralogy of Fallot, (Levin et al., 1981).

There appears to be a degree of a dissension amongst authors. Whilst some are convinced that an independently arising conus artery is a normal if not predominant find, (James, 1961 and Schelsinger, 1940), others refute these statements by the low incidences verified in their studies, (Kurjia et al., 1986 and Donaldson et al., 1982). The incidences reported in this study support incontrovertibly that an independent origin of a conus branch is an anomalous rather than a ubiquitous find.

Table 25: Comparative incidences of an independently originating conus artery

Reference	Location	% Incidence
James, (1961)	USA	50
Schlesinger, (1940)	USA	51
Symmers, (1907)	England	38
Bianchi, (1904)	Italy	33
Kurjia, (1986)	Iraq	7.6
Alexander and Griffith, (1956)	USA	0.3
Donaldson et al., (1982)	England	0.15
Present Study	**South Africa**	**5**

5.1.11. ANOMALOUS ORIGIN OF THE LCA FROM PULMONARY ARTERY

The observation of an anomalous left coronary artery originating from the pulmonary artery in the adult is particularly interesting. Usually encountered in early childhood and occurring in 1 of 300,000 births, its prognosis is poor. Although about 10% may reach adulthood its persistence during this stage is indeed rare. The literature reveals reports of incidental

findings recorded in ages between 15 and 70 years old, (Ortiz de Salazar et al., 1996 and Weber, 2002).

The patient angiogram that was observed in this study displayed an anatomical picture of a coronary vascular pattern typical of left-sided compromise. The RCA was abnormally dilated with signs of advanced collateralisation.

The angiographic picture conformed to descriptions within the literature reviewed, (Oritz de Salazr et al., 1996; Friedman and Child, 1994 and Berdjis et al., 1994). Corrective options for such an anomaly in the adult have been described. In this case, surgical revascularization was indicated. Although clinical improvement was noted, patient follow up is still in progress. The post-operative evaluation of the RCA in terms of arterial re-adaptation therefore has not been fully established.

MYOCARDIAL BRIDGES

5.2.1 INCIDENCE AND ARRANGEMENT OF MYOCARDIAL BRIDGES

There is significant bank of literature regarding myocardial bridges, (Angelini et al., 1983; Bezera et al., 1989; Bloor and Liebow, 1965, Kosinski and Grzybiak, 2001 and Venkateshu et al., 2000). The topic has received a fair amount of discussion, enough to have had established the general anatomy as well as the overall morphometry and arrangement of these bridges.

Myocardial bridges were observed in 44 of the 83 cases selected with a resulting incidence of 53% of the total population studied. These findings compare favourably with the incidence of 55.6% reported by Ferreira et al., (1991), is slightly higher than the 41% recorded by Kosinski and Grzybiak, (2001) and significantly higher than the 14.2% reported by Venkateshu et al., (2000). In this study, bridges were recorded over all the major coronary arteries. However, as anticipated, they were predominantly associated with the LAD.

Micro-dissection of the myocardial arrangement revealed a series of three predominant patterns. These patterns correspond with those already described by Polacek, (1960).

Myocardial fibres were observed to form loops over the arteries in 16.3%. There is no specific prevalence in the number of loops that may be present on a coronary branch. The loops are generally small, ranging between 2 to 3mm in width across the artery.

This bridge pattern is often associated with parts of the vessel that are placed further away from the major myocardial surfaces. The bridge arrangement over the LCX and RCA is typical for this type of pattern. Usually within the atrioventricular grooves, these vessels appear to be somewhat displaced from the muscle by a surrounding layer of fatty connective tissue.

The LAD may also be predisposed to such bridging, however the tendency for this type is usually confined to a small area on the proximal LAD and its main diagonal branch when present.

5.2.2. MYOCARDIAL BRIDGES IN RELATION TO THE RCA AND LCX ARTERIES

Allwork, (1987), is under the impression that coronary branches such as the LCX and RCA are less prone to myocardial bridges due to their course within fascial grooves. In the present study, the low incidences of such bridging associated with these arteries appear to support the author's notion.

However, through the dissection experience during this study, it is clear that the course of these arteries do not shelter them from bridging. Even within the left atrioventricular groove, the LCX artery may be prone to myocardial straps from the left ventricular margin and sometimes from the left atrial appendage. The RCA on the other hand may follow a path inclined toward the infundibular margin. In the event of this occurrence, the RCA may become completely drawn to the right ventricular myocardium by a complete wrap of the infundibular myocardium. Such a case has been documented in this study.

5.2.3. MYOCARDIAL BRIDGE PATTERNS

The pattern of bridging ranged from a series of 3 to 4 slips with an average width of 3mm to complete investment of the entire length of the artery up to the acute margin of the right ventricle. In the second pattern, the bridge passes over the artery along a diagonal plane of parallel fibres. This arrangement occurred in 73.5% of hearts, (Figures 51 and 52). Bezzera et al., (1987) reported similar findings. The pattern is typically associated with the LAD. Extending from the infundibulum, the fibres are drawn across the artery and blend in with the myocardial surface of the left ventricle, (Plate 34a).

5.2.4. MYOCARDIAL BRIDGES IN RELATION TO THE LAD

In the total sample of 44 hearts with myocardial bridges, a total of 48 counts of bridges were recorded in relation to the LAD, (Appendix C3). The counts were recorded over three segments of the LAD. An incidence of 18.75% was seen over the proximal third of the LAD, 70.8% of the bridges recorded appeared to lie over the middle third and 10.4% over the distal of the vessel. The LAD was intra myocardial in 10.2% and in 14.3% the bridge extended over 2 segments. Incidences of findings and mean measurements for bridges over the LAD are repeated in the table below, (Graph 17).

Table 26: Reported incidences and mean lengths for myocardial bridges

LAD SEGMENT	INCIDENCE %	MEAN LENGTH OF BRIDGE (mm)
Proximal LAD	18.8	10.73 [2.3 – 16.0]
Middle LAD	70.8	20.02 [0.9 – 46.0]
Distal LAD	10.4	7.66 [0.7 – 23.5]

These findings are in accordance with those of Ferreira et al., (1991) and confirm that the LAD is most frequently bridged in its proximal and middle thirds. An intra-myocardial course was shown to occur in 10.2% of hearts studied.

In a retrospective cineangiographic study attempting to correlate the frequency of myocardial bridges in various clinical settings, only 5.5% of a total of 1100 images observed, showed bridging of the left anterior descending artery and only 1 suggested bridging of the posterior branch of the right coronary artery, (Ferreira et al., 1991)

DeZwaan and Wellens, (1984) report a case in which surgical approach to connecting a muscular bridge of LAD was complicated, due to perforation of the right ventricle during un-roofing of a segment of intra-myocardial LAD in an unexpected, sub-endocardial location. As no angiographic clues allow this anomaly to be diagnosed before surgery, the anatomical awareness of its morphology becomes pertinent.

5.2.5. MYOCARDIAL BRIDGES - CLINICAL CONSIDERATIONS

In South Africa, clinicians whose patient profiles include predominantly Whites and Indians are aware of morphometric differences as well as the extent of diffuse disease between these two groups. Diffuse disease, with multiple obstructions often necessitates graft placements in recruitable less obstructed segments of the artery. Not infrequently, an intramural portion of the LAD must be exposed in order to obtain an adequately sized segment, free of disease, for bypass. Although we are aware from previous studies (Dhawan & Bray, 1995, Vlodaver et al., 1969, Litovsky et al., 1996) that inter-ethnic and sex differences may exist, this study does not suggest any such differences in the prevalence of myocardial bridges.

As MIDCAB operations begin to replace conventional approaches in coronary revascularization, the identification of arteries through smaller access fields may not as straight forward, (Sachweh et al., 2002). Anatomic variations, as well as vessel displacement in the presence of cardiomegaly may often result in incorrect placement of a graft, especially if the target vessel is unexpectedly located beneath the myocardium.

5.2.6. ANGIOGRAPHIC MANIFESTATION OF THE INTRA-MURAL LAD

With the trend towards minimal access cardiac surgery, pre-operative identification of such anatomical variants of coronary artery disposition becomes increasingly relevant if technical challenges associated with its surgical presentation are to be abated.

The role of angiography in the clinical diagnosis of intra mural coronaries was introduced by Portsmann et al., in 1960. Although no confirmation by surgical or anatomic means was afforded, the authors considered systolic narrowing to be evidence of bridging. In further reports of canine experiments "sudden bending" and "rigidity" were evidenced as angiographic manifestation of an intra mural course.

In later attempts at establishing distinct anatomic markers, Eliska et al., (1968) suggested that kinking of the coronary artery during systolic compression was likely to indicate that the artery may be crossed by either muscle fibres or a cardiac vein.

The design of this investigation aimed at examining the relationship between the angiographic manifestation and anatomical presence of the intra-mural LAD was based largely on a clinical impression of the possibility of this association.

When measured in terms of a tortuosity index, values ranged between 1.045 and 1.373 with a mean of 1.147. According to the mathematical scale applied, all values tended towards 1 and fell within the prescribed range of 1 – 1.5. Results of this investigation showed clearly, that an intra-mural LAD artery has a higher degree of "straightness" than when in an epicardial position, (Tables 20.1 to 20.28).

The findings of this study therefore, indicated a positive correlation between the morphological appearance of a coronary artery in terms of tortuosity and straightness and its cardiac position: whether epicardial or intra-myocardial.

These results of this study support the clinical impression that if the LAD appears "straight" on an angiogram, it is likely to be buried beneath a bridge of myocardium. Furthermore, these findings are supported by the anatomical observation of the relevant segments of the LAD beneath the myocardium. This study in addition, presents evidence that shows the intra-mural segment of the artery to be straight when compared with the more tortuous pre- and post-mural portions.

The approach used in this investigation has not been previously described. Its design may be easily employed in further studies or into order to confirm observations from angiograms. These findings may well contribute to a list of possible factors that suggest myocardial bridging and provide valuable information to the surgeon in the pre-operative patient work-up.

THE EXTRA CORONARY COLLATERALS

5.3.1. ANATOMY OF THE ECC'S

The intrinsic collateral potential of the heart has been a subject of consistent discussion. Authors such as Gross, (1921), Schlesinger, (1940) and Schaper et al., (1988) have been the forerunners in documenting the anatomy of these vessels and contributions by James and Fulton have further enhanced knowledge in this area. Whilst such valuable resource was being maximised in terms of investigative work, very little attention went into exploring the possibility of collateral potentials outside of the cardiac territory. Although the concept of extrinsic collateralisation has received mention in the literature, there has been only one significant attempt at defining the anatomy of extra-coronary collaterals. The study was conducted in 1932 and documented by Hudson et al.

Results of this study corroborate the findings of Gross (1921) and Hudson et al (1932) and confirm the existence and known distribution of ECC's.

In this investigation, it was possible to trace ECC's to their points of origin, count the number of connections, measure the length and external diameter of the main branches as well as their secondary branches.

ECC's usually present an arterial pattern consisting of one or two main stems with secondary anastomotic branches. The average external diameter of an ECC was measured to be 0.6mm (0.4-0.7), average length 52.5mm (18-83), with at least 5 secondary branches (3-9) of

external diameter, 0.3mm (0.2-0.5), (Table 21). The morphometric data presented in this study have not been previously documented in the literature.

This study confirms the role of the vasa vasora in collateral formation. These vessels, although typically very small and often masked by the adventitia through which they pass, have the potential not only to increase in size, but also to provide a network of channels that allow small amounts of blood to flow between the coronary arteries and great vessels, by creating a so-called coronary-systemic link.

Extensive vascularisation of the fibrous pericardium via coronary collaterals and the prevalence of the *arteria telae adiposae* appear to be constant. According to Gross (1921), the development of fat vessels should not be seen as a secondary mechanism to functional need of the myocardium, since they are constant and dynamic and their appearance is proportional to age, irrespective of pathologic lesion. However, he agrees that pathologic interference of a coronary artery can lead to the enhancement of these vessels both quantitatively and qualitatively.

Although only one case of bronchial to coronary artery connection was observed in this series, its frequent occurrence has been previously documented and well described, (Moberg, 1968; Dupont and Riquet, 1991). However, its clinical presentation does remain a rare occurrence. Anatomically, bronchial to coronary artery connections are reported to be present at birth and function in response to pressure gradient. However, when clinically manifested, these connections are thought to have a role in stimulating angina pectoris via coronary steal.

There have been current reports of internal thoracic to coronary artery connections observed during routine coronary angiography. Although their occurrence remains infrequent, (Singh and Varat, 1982) such collateralisation may be a source of potential complication if a surgically selected ITA is to be harvested for coronary grafting. Such possibility of arterial anastomosis may indeed be an added indication for selective ITA angiography as part of the pre-operative work-up.

Although Hudson (1932) and Moberg (1968) allude to coronary communications with tracheal vessels, there is no report of an inferior thyroid artery to coronary artery collateral. This study describes this rare connection and adds to the relatively scarce anatomical data on this subject.

5.3.1. ECC'S – CLINICAL CONSIDERATIONS

Detailed knowledge of ECC's may make possible the development of new strategies to control unwanted flow during cardiac surgery. It is not unimaginable, that interventional catheter techniques could potentially dilate ECC connections to major coronary artery branches. Angiographically directed installation of growth factors to sites of ECC's offers the potential for increasing flow when major coronary arteries are occluded. Patients with severe diffuse coronary artery disease, not amenable to coronary angioplasty or coronary artery bypass graft surgery may benefit from techniques to increase myocardial blood flow through ECC's. In addition, augmentation of extra cardiac anastomoses through surgically created adhesions may be enhanced.

This investigation was successful in its attempt to encourage a revival of interest in the extrinsic coronary collateral resource. Although the sample consisted of only 9 cadavers, its value in this study is measured in terms of the complexity that was involved in conducting such an anatomical investigation. The study design was original in this field of investigation and the set-up may be easily reproduced in order to support on-going studies. This study contributes to the anatomical knowledge on cardiac collaterals. From a clinical standpoint, the results provide a basis from which to encourage interventionalists to explore the possibilities of potentiating the already existing collateral network.

Substantial morphometric data on ECC's will contribute in providing an anatomic basis for the planning and implementation of innovative therapies and techniques, to maximize the role of ECC's.

HISTOPATHOLOGICAL ANALYSIS

6.1. HISTOPATHOLOGICAL ANALYSIS

Results of the histological study confirmed early signs and presence of atherosclerotic disease in more than two thirds of the samples analysed (69% incidence). When measured in terms of severity, the highest percentage (59%) of confirmed pathology was categorised as mild. Only 7% were moderately diseased and a low percentage (3.4%) showed advanced stages of plaque formation, (Table 22 and Graph 18).

There was no ethnic diversity within the selected group. All specimens were harvested from Blacks within the South African population. When combined with an average recorded age of 33 years, these variables fall within the expectancy for the atheroclerotic disease profile, typical for this population group, (Morar et al., 1998).

Results from evaluation of the mural LAD segments presents an interesting platform for discussion of the somewhat endorsed "cardio-protective" property of the Myocardial Bridge.

Signs of disease process were observed in each of the segments analysed. Although the highest recorded incidence was confined to the pre- mural followed by the post-mural segments of the LAD, the intra-mural portion of the artery was not entirely spared from pathology. Even though the extent of disease was limited to minimal disruption of the intimal lamina, the incidence was not far below that recorded in the post-mural segments (15% vs 20%), (Graphs 21 and 22).

Within the arena of debate there appears to be two distinct schools of thought on the protective function of the Myocardial Bridge. Polacek, (1961), Penther et al., (1977) and Polacek and Zechmeister, (1968) maintain that the intra-mural segment of the LAD artery is never affected by atherosclerosis. Edwards et al., (1956) however significantly challenges their findings by his documentation of atheroma within the bridged segments of the LAD artery. Geirenger's, (1950) report appears to be more guarded in its conclusions and although he has not denied the presence of intimal hyperplasia in intra-mural segments, he proclaims that such incidental findings never produce clinically significant atheroma.

This study concurs with that of Edwards et al., (1956) and Geirenger, (1950) in its acceptance that the intra-mural LAD may be subjected to atheroma. The results however do not appear to correspond with those obtained by Edwards with regard to the incidence and severity of disease. When compared, the present study shows incidence of only mild disease as opposed to the high incidences of moderate and severe disease as shown in Table 27, below.

Table 27: Degree of atherosclerosis in the intra-mural LAD segment

Degree of Atherosclerosis	Edwards et al., (1956) (n = 16)	Present Study (n = 29)
MILD	2	3
MODERATE	6	0
SEVERE	8	0

Both Geirenger (1950) in his landmark article and Angelini et al, (1983) in their well - documented review of myocardial bridges afford two elucidating thoughts.

Firstly, the type and pattern of the Myocardial Bridge appears to be associated with its proposed protective effect. The most favourable arrangement is one where the myocardial fibres cross the artery in a parallel fashion for at least 2cm in length. Myocardial loops and thinner bridges appear less effective in its action on the underlying arterial wall and subsequent support of the media.

Secondly, the amount of fatty tissue located between the muscle and the vessel wall tends to influence the supporting function of the myocardial bridge on the media. The greater the amount of fatty tissue surrounding the artery, the greater its tendency to behave like an epicardial vessel and hence, the greater its vulnerability to arterial disease.

Although this study applied a selection criterion based on the length of the bridge when harvesting LAD segments for histopathological investigation, the amount of sub-myocardial fatty tissue could not be pre-measured. However, results of this study appear to support the notion that high amounts of surrounding fatty tissue associated with an intra-mural LAD artery may promote intimal alteration and mild signs of atheroma when present.

Results of this investigation illustrate that the intra-mural LAD artery is relatively protected from vascular disease. It does not however support the theory that in such a sub-myocardial position, the LAD artery is never prone to the damaging effects of atherosclerosis.

The "cardio-protective" effect of a muscular bridge, whilst prevalent, is dependant on the thickness and extent of the bridge itself. Any increased amount of adipose tissue between the artery and the muscle will invariably decrease the protective effect of the muscle on the media by artificially reverting the artery as it were, to an epicardial position.

CHAPTER VI

CONCLUSION

"And indeed, there are several great men whom the study of Anatomy has not only lifted to the recognition of a god, but who are impelled to sing His praise observing with what admirable wisdom and singular providence He has perfected the arrangement of every part"

Rene Descartes, (1596-1650)

6.1. DOCUMENTATION OF ANATOMICAL AND CLINICAL DATA FROM A SOUTH AFRICAN POPULATION

The anatomy of the coronary arteries has been successfully documented and a bank of data, specific for a South African population has been presented. Significant aspects of coronary arterial patterns have been discussed and interpreted in terms of its clinical relevance.

6.2. ANATOMICAL REVIEW OF THE CORONARY ARTERIES

An extensive review of the literature was conducted. The process of evaluation involved research on publications that dated as far back as the 1500's. Seminal contributions were studied in their original text and compared with that of the current literature. The review presented in this dissertation is a composition of information that has been formulated by a combination of standard and surgical anatomy.

Through the dissection experience of more than 150 hearts, this study was able to contribute to the general anatomical description of the coronary arteries. In some ways new perspectives were afforded and in the same faith, already existing concepts were verified.

6.3. ETHNIC AND SEX DIFFERENCES IN CORONARY ARTERY ANATOMY WITHIN A SOUTH AFRICAN POPULATION

Although not statistically significant, mean values for coronary artery size differed between sexes. The findings were similar when evaluated in terms of the coronary artery anomalies studied.

There were significant differences between ethnic groups in terms of the length of the LCA. Mean values showed that Indians had the shortest LCA's when compared with Blacks and Whites.

The highest incidence of the ramus marginalis branch was recorded amongst Blacks.

Separate origin of the LCX and LAD was highest amongst Indians and high in comparison to reports documented in other countries.

A high origin of the conus artery was found to be dominant amongst Blacks. A low incidence of separate origin of the conus from the aorta was recorded in the South African population. These findings are significantly lower than that reported in the literature.

A right dominant system has the highest occurrence within this population. Statistical evaluation confirms that neither sex, ethnicity, age or height influence dominance in a coronary arterial pattern.

The presence and description of the bifid LAD has been recorded. Its occurrence is highest amongst Blacks.

The anomalous path of the LCX has been documented and described. The significantly high occurrence of this disposition of the LCX within the South African population appears to be the highest reported find in the literature.

6.4. ANATOMY OF MYOCARDIAL BRIDGES

The anatomy of myocardial bridges over the major coronary branches has been described. Morphometric analyses have been conducted and incidences have been recorded. There are no observable differences between ethnic groups or sex.

6.5. ANGIOGRAPHIC MANIFESTATION OF THE INTRA-MURAL LAD

Results of this study confirm a relationship between the straight appearance of the LAD on angiogram and its anatomical presence. The findings of this study contribute to the bank of diagnostic indicators that are used to predict myocardial bridges pre-operatively.

6.6. ANATOMY OF THE EXTRA CORONARY COLLATERALS

Extra coronary collaterals have been successfully investigated and observed. Measurements of vessel dimensions and patterns have been recorded.

This study presents an original method for the investigation of these collaterals using technologically advanced materials and equipment.

6.7. HISTOPATHOLOGICAL ANALYSIS OF THE INTRA-MURAL CORONARY ARTERY

Results of this investigation illustrate that the intra-mural LAD artery is relatively protected from vascular disease. It does not however support the theory that in such a sub-myocardial position, the LAD artery is never prone to the damaging effects of atherosclerosis.

The "cardio-protective" effect of a muscular bridge, whilst prevalent, is dependant on the thickness and extent of the bridge itself. Any increased amount of adipose tissue between the artery and the muscle will invariably decrease the protective effect of the muscle on the media by artificially reverting the artery as it were, to an epicardial position.

POTENTIAL FOR CLINICAL APPLICATION

"It is a well-documented fact that successful clinical outcome of diagnostic investigations or procedures conducted within a patient population is influenced by the degree to which the clinician is familiar with the patient profile. Although recognised factors in the management of a patient, ethnicity and sexual dimorphism with regards to the presence of anatomical variation have not been scientifically documented within the South African population. Increased awareness of such anatomical information amongst clinicians has become a valuable tool in the overall

surgical management of the cardiac patient as well as in the acute management of those presenting at trauma units.

The straight appearance of an intra mural coronary artery on an angiogram is a positive contribution to the list of diagnostic indicators in the determination of a deep position of the LAD artery. Such pre-operative information may be of value to the clinician in directing a surgical approach that may best favour clinical outcome. Knowledge of the morphometry of myocardial bridges and bridge patterns may be applied during the operative technique that can therefore be approached with caution so as to avoid unwanted injury to the underlying vessel. In addition, findings of a relatively low incidence of atherosclerosis within an intra mural coronary artery segment may prove useful to the surgeon. Myocardial bridge resection may be indicated in order to recruit more patently viable sections of the artery as a site for graft placement.

The existence of extra coronary collaterals presents challenging and exciting possibilities for interventional cardiologists, surgeons and anatomists. For the surgeon, awareness of these anatomical patterns may be of benefit in preventing unwanted flow and consequent warming of the myocardium during the administration of cardioplegia. As a potential resource for occluded vessels, these collateral channels may be enhanced pharmacologically by induced growth therapy. Alternatively, surgically created pleural-pericardial adhesion appears to provide an additional mechanism for maximising the presence of auxiliary vessels and when naturally functional, the increased flow to the compromised regions of the heart may limit the extent of myocardial damage.

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APPENDICES

APPENDICES

APPENDIX A

A1. SEPARATE ORIGIN OF LAD AND LCX –SEX

Crosstab

			SEX		
			Female	Male	Total
LAD_LCX	Present	Count		1	1
		% within SEX		.9%	.7%
	Not present	Count	40	108	148
		% within SEX	100.0%	99.1%	99.3%
Total		Count	40	109	149
		% within SEX	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	.369[b]	1	.543		
Continuity Correction[a]	.000	1	1.000		
Likelihood Ratio	.628	1	.428		
Fisher's Exact Test				1.000	.732
Linear-by-Linear Association	.367	1	.545		
N of Valid Cases	149				

a. Computed only for a 2x2 table

b. 2 cells (50.0%) have expected count less than 5. The minimum expected count is .27.

A2. SEPARATE ORIGIN OF LAD AND LCX – AGE

Crosstab

			AGEGRP					Total
			31-40	41-50	51-60	61-70	71-80	
LAD_LCX	Present	Count			1			1
		% within AGEGRP			2.1%			.7%
	Not present	Count	11	29	47	51	10	148
		% within AGEGRP	100.0%	100.0%	97.9%	100.0%	100.0%	99.3%
Total		Count	11	29	48	51	10	149
		% within AGEGRP	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	2.118[a]	4	.714
Likelihood Ratio	2.280	4	.684
Linear-by-Linear Association	.017	1	.897
N of Valid Cases	149		

a. 5 cells (50.0%) have expected count less than 5. The minimum expected count is .07.

A3. SEPARATE ORIGIN OF LAD AND LCX – ETHNICITY

Crosstab

			ETHNCITY			
			Black	Indian	White	Total
LAD_LCX	Present	Count			1	1
		% within ETHNCITY			2.1%	.7%
	Not present	Count	7	94	46	147
		% within ETHNCITY	100.0%	100.0%	97.9%	99.3%
Total		Count	7	94	47	148
		% within ETHNCITY	100.0%	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	2.164[a]	2	.339
Likelihood Ratio	2.309	2	.315
Linear-by-Linear Association	1.825	1	.177
N of Valid Cases	148		

a. 3 cells (50.0%) have expected count less than 5. The minimum expected count is .05.

A4. RAMUS MARGINALIS – SEX

Crosstab

			SEX		
			Female	Male	Total
RM	Present	Count	3	4	7
		% within SEX	7.5%	3.7%	4.7%
	Not present	Count	37	105	142
		% within SEX	92.5%	96.3%	95.3%
Total		Count	40	109	149
		% within SEX	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	.959[b]	1	.327		
Continuity Correction[a]	.294	1	.588		
Likelihood Ratio	.876	1	.349		
Fisher's Exact Test				.386	.280
Linear-by-Linear Association	.952	1	.329		
N of Valid Cases	149				

a. Computed only for a 2x2 table

b. 1 cells (25.0%) have expected count less than 5. The minimum expected count is 1.88.

A5. RAMUS MARGINALIS – AGE

Crosstab

			AGEGRP					Total
			31-40	41-50	51-60	61-70	71-80	
RM	Present	Count	2	1	2	1	1	7
		% within AGEGRP	18.2%	3.4%	4.2%	2.0%	10.0%	4.7%
	Not present	Count	9	28	46	50	9	142
		% within AGEGRP	81.8%	96.6%	95.8%	98.0%	90.0%	95.3%
Total		Count	11	29	48	51	10	149
		% within AGEGRP	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	6.080[a]	4	.193
Likelihood Ratio	4.374	4	.358
Linear-by-Linear Association	1.188	1	.276
N of Valid Cases	149		

a. 5 cells (50.0%) have expected count less than 5. The minimum expected count is .47.

A6. RAMUS MARGINALIS – ETHNCITY

Crosstab

			ETHNCITY			
			Black	Indian	White	Total
RM	Present	Count		6	1	7
		% within ETHNCITY		6.4%	2.1%	4.7%
	Not present	Count	7	88	46	141
		% within ETHNCITY	100.0%	93.6%	97.9%	95.3%
Total		Count	7	94	47	148
		% within ETHNCITY	100.0%	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	1.624[a]	2	.444
Likelihood Ratio	2.076	2	.354
Linear-by-Linear Association	.406	1	.524
N of Valid Cases	148		

a. 3 cells (50.0%) have expected count less than 5. The minimum expected count is .33.

A7. CONUS – PRESENCE IN SEX, AGE & ETHNICITY

Crosstab

			SEX		
			Female	Male	Total
CONUS	Not present	Count	40	109	149
		% within SEX	100.0%	100.0%	100.0%
Total		Count	40	109	149
		% within SEX	100.0%	100.0%	100.0%

Chi-Square Tests

	Value
Pearson Chi-Square	.[a]
N of Valid Cases	149

a. No statistics are computed because CONUS is a constant.

Crosstab

			AGEGRP					
			31-40	41-50	51-60	61-70	71-80	Total
CONUS	Not present	Count	11	29	48	51	10	149
		% within AGEGRP	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%
Total		Count	11	29	48	51	10	149
		% within AGEGRP	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%

because CONUS is a constant.

Chi-Square Tests

	Value
Pearson Chi-Square	.[a]
N of Valid Cases	148

a. No statistics are computed because CONUS is a constant.

Crosstab

			ETHNCITY			
			Black	Indian	White	Total
CONUS	Not present	Count	7	94	47	148
		% within ETHNCITY	100.0%	100.0%	100.0%	100.0%
Total		Count	7	94	47	148
		% within ETHNCITY	100.0%	100.0%	100.0%	100.0%

A8. DOMINANCE - RELATION TO SEX

Crosstab

			SEX		
			Female	Male	Total
DOMINANC DOMINANCE	Co-dominant	Count	1	4	5
		% within SEX	2.5%	3.7%	3.4%
	Left	Count	3	13	16
		% within SEX	7.5%	11.9%	10.7%
	Right	Count	36	92	128
		% within SEX	90.0%	84.4%	85.9%
Total		Count	40	109	149
		% within SEX	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	.760[a]	2	.684
Likelihood Ratio	.808	2	.668
Linear-by-Linear Association	.630	1	.427
N of Valid Cases	149		

a. 3 cells (50.0%) have expected count less than 5. The minimum expected count is 1.34.

A.9 DOMINANCE – RELATION TO AGE

Crosstab

			AGEGRP					Total
			31-40	41-50	51-60	61-70	71-80	
DOMINANC DOMINANCE	Co-dominant	Count			1	4		5
		% within AGEGRP			2.1%	7.8%		3.4%
	Left	Count	2	4	7	2	1	16
		% within AGEGRP	18.2%	13.8%	14.6%	3.9%	10.0%	10.7%
	Right	Count	9	25	40	45	9	128
		% within AGEGRP	81.8%	86.2%	83.3%	88.2%	90.0%	85.9%
Total		Count	11	29	48	51	10	149
		% within AGEGRP	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	8.773[a]	8	.362
Likelihood Ratio	10.171	8	.253
Linear-by-Linear Association	.008	1	.931
N of Valid Cases	149		

a. 8 cells (53.3%) have expected count less than 5. The minimum expected count is .34.

A10.DOMINANCE – ETHNCITY

Crosstab

			ETHNCITY			
			Black	Indian	White	Total
DOMINANC DOMINANCE	Co-dominant	Count		5		5
		% within ETHNCITY		5.3%		3.4%
	Left	Count	1	8	7	16
		% within ETHNCITY	14.3%	8.5%	14.9%	10.8%
	Right	Count	6	81	40	127
		% within ETHNCITY	85.7%	86.2%	85.1%	85.8%
Total		Count	7	94	47	148
		% within ETHNCITY	100.0%	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	4.139[a]	4	.387
Likelihood Ratio	5.758	4	.218
Linear-by-Linear Association	.114	1	.735
N of Valid Cases	148		

a. 4 cells (44.4%) have expected count less than 5. The minimum expected count is .24.

A11.BIFIDLAD – SEX

Crosstab

			SEX		
			Female	Male	Total
BIFIDLAD	Present	Count	2	5	7
		% within SEX	5.0%	4.6%	4.7%
	Not present	Count	38	104	142
		% within SEX	95.0%	95.4%	95.3%
Total		Count	40	109	149
		% within SEX	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	.011[b]	1	.916		
Continuity Correction[a]	.000	1	1.000		
Likelihood Ratio	.011	1	.916		
Fisher's Exact Test				1.000	.605
Linear-by-Linear Association	.011	1	.916		
N of Valid Cases	149				

a. Computed only for a 2x2 table

b. 1 cells (25.0%) have expected count less than 5. The minimum expected count is 1.88.

A12. BIFIDLAD - AGE

Crosstab

			AGEGRP					Total
			31-40	41-50	51-60	61-70	71-80	
BIFIDLAD	Present	Count	2	1	3	1		7
		% within AGEGRP	18.2%	3.4%	6.3%	2.0%		4.7%
	Not present	Count	9	28	45	50	10	142
		% within AGEGRP	81.8%	96.6%	93.8%	98.0%	100.0%	95.3%
Total		Count	11	29	48	51	10	149
		% within AGEGRP	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	6.173[a]	4	.187
Likelihood Ratio	5.060	4	.281
Linear-by-Linear Association	3.356	1	.067
N of Valid Cases	149		

a. 5 cells (50.0%) have expected count less than 5. The minimum expected count is .47.

A13. BIFIDLAD – ETHNCITY

Crosstab

			ETHNCITY			
			Black	Indian	White	Total
BIFIDLAD	Present	Count	1	4	2	7
		% within ETHNCITY	14.3%	4.3%	4.3%	4.7%
	Not present	Count	6	90	45	141
		% within ETHNCITY	85.7%	95.7%	95.7%	95.3%
Total		Count	7	94	47	148
		% within ETHNCITY	100.0%	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	1.489[a]	2	.475
Likelihood Ratio	1.015	2	.602
Linear-by-Linear Association	.406	1	.524
N of Valid Cases	148		

a. 3 cells (50.0%) have expected count less than 5. The minimum expected count is .33.

APPENDIX B

B1. ONEWAY ANOVA - LCA LENGTH - ETHNICITY

Descriptives

LCA_CM

	N	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean		Minimum	Maximum
					Lower Bound	Upper Bound		
Black	61	7.20	5.364	.687	5.82	8.57	0	23
Indian	2	.53	.750	.530	-6.20	7.26	0	1
White	20	2.69	3.845	.860	.89	4.48	0	14
Total	83	5.95	5.381	.591	4.77	7.12	0	23

ANOVA

LCA_CM

	Sum of Squares	df	Mean Square	F	p
Between Groups	366.945	2	183.473	7.311	.001
Within Groups	2007.633	80	25.095		
Total	2374.578	82			

B2. T-TEST

Descriptives

LCA_CM

	N	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean		Minimum	Maximum
					Lower Bound	Upper Bound		
Female	25	4.35	5.200	1.040	2.20	6.50	0	23
Male	52	6.75	5.426	.752	5.24	8.26	0	22
Total	77	5.97	5.438	.620	4.74	7.20	0	23

Independent Samples Test

		t-test for Equality of Means		
		t	df	p
LCA_CM	Equal variances assumed	-1.840	75	.070

B3. CORRELATIONS

Correlations

		LCA_CM	AGE	HEIGHT
LCA_CM	Pearson Correlation	1	-.297**	.044
	Sig. (2-tailed)	.	.009	.703
	N	83	77	77
AGE	Pearson Correlation	-.297**	1	-.115
	Sig. (2-tailed)	.009	.	.320
	N	77	77	77
HEIGHT	Pearson Correlation	.044	-.115	1
	Sig. (2-tailed)	.703	.320	.
	N	77	77	77

**. Correlation is significant at the 0.01 level (2-tailed).

B4. MULTIPLE REGRESSION

Model Summary

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate
1	.410[a]	.168	.122	5.095

a. Predictors: (Constant), HEIGHT, ETHN2C Ethnic group, SEX, AGE

ANOVA[b]

Model		Sum of Squares	df	Mean Square	F	p
1	Regression	378.609	4	94.652	3.646	.009[a]
	Residual	1869.179	72	25.961		
	Total	2247.788	76			

a. Predictors: (Constant), HEIGHT, ETHN2C Ethnic group, SEX, AGE

b. Dependent Variable: LCA_CM

Coefficients[a]

Model		Unstandardized Coefficients		Standardized Coefficients	t	p
		B	Std. Error	Beta		
1	(Constant)	11.598	12.702		.913	.364
	SEX	1.618	1.419	.140	1.140	.258
	AGE	1.656E-03	.042	.006	.039	.969
	ETHN2C Ethnic group	-2.260	.931	-.366	-2.427	.018
	HEIGHT	-2.930	7.833	-.043	-.374	.709

a. Dependent Variable: LCA_CM

B5. CROSSTABS DOMINANCE - SEX

Crosstab

			SEX			
			Female	Male	Not indicated	Total
DOMINAN1 Dominance	Co-dominant	Count	2	7	1	10
		% within SEX	8.0%	13.5%	16.7%	12.0%
	Left	Count	1	4		5
		% within SEX	4.0%	7.7%		6.0%
	Right	Count	22	41	5	68
		% within SEX	88.0%	78.8%	83.3%	81.9%
Total		Count	25	52	6	83
		% within SEX	100.0%	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	p
Pearson Chi-Square	1.478[a]	4	.830
Likelihood Ratio	1.873	4	.759
Linear-by-Linear Association	.609	1	.435
N of Valid Cases	83		

a. 6 cells (66.7%) have expected count less than 5. The minimum expected count is .36.

B6. DOMINANCE – ETHNICITY

Crosstab

			ETHN2C Ethnic group			
			Black	Indian	White	Total
DOMINAN1 Dominance	Co-dominant	Count	7		3	10
		% within ETHN2C Ethnic group	11.5%		15.0%	12.0%
	Left	Count	4		1	5
		% within ETHN2C Ethnic group	6.6%		5.0%	6.0%
	Right	Count	50	2	16	68
		% within ETHN2C Ethnic group	82.0%	100.0%	80.0%	81.9%
Total		Count	61	2	20	83
		% within ETHN2C Ethnic group	100.0%	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	p
Pearson Chi-Square	.675[a]	4	.954
Likelihood Ratio	1.022	4	.906
Linear-by-Linear Association	.066	1	.798
N of Valid Cases	83		

a. 6 cells (66.7%) have expected count less than 5. The minimum expected count is .12.

B7. MEAN VALUES

Report

DOMINAN1 Dominance		AGE	HEIGHT
Co-dominant	Mean	52.00	1.6967
	N	9	9
	Std. Deviation	20.137	.05500
	Minimum	23	1.63
	Maximum	81	1.77
Left	Mean	44.00	1.6800
	N	5	5
	Std. Deviation	22.858	.08456
	Minimum	27	1.58
	Maximum	84	1.80
Right	Mean	45.73	1.6781
	N	63	63
	Std. Deviation	20.162	.08353
	Minimum	16	1.40
	Maximum	96	1.86
Total	Mean	46.35	1.6804
	N	77	77
	Std. Deviation	20.156	.08014
	Minimum	16	1.40
	Maximum	96	1.86

B8. MULTIPLE REGRESSION

Model Summary

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate
1	.178[a]	.032	-.022	.678

a. Predictors: (Constant), HEIGHT, ETHN2C Ethnic group, SEX, AGE

ANOVA[b]

Model		Sum of Squares	df	Mean Square	F	p
1	Regression	1.077	4	.269	.587	.673[a]
	Residual	33.052	72	.459		
	Total	34.130	76			

a. Predictors: (Constant), HEIGHT, ETHN2C Ethnic group, SEX, AGE

b. Dependent Variable: DOMINAN1 Dominance

Coefficients[a]

Model		Unstandardized Coefficients		Standardized Coefficients	t	p
		B	Std. Error	Beta		
1	(Constant)	3.743	1.689		2.216	.030
	SEX	-.204	.189	-.143	-1.081	.283
	AGE	-6.21E-03	.006	-.187	-1.098	.276
	ETHN2C Ethnic group	4.678E-02	.124	.062	.378	.707
	HEIGHT	-.289	1.042	-.035	-.277	.782

a. Dependent Variable: DOMINAN1 Dominance

B9. CROSS TABS SEPARATE ORIGIN OF LAD & LCX - SEX

Crosstab

			SEX			
			Female	Male	Not indicated	Total
LAD_LCX Separate origin of LAD and LCX	Present	Count	4	8		12
		% within SEX	16.0%	15.4%		11.8%
	Not present	Count	21	44	25	90
		% within SEX	84.0%	84.6%	100.0%	88.2%
Total		Count	25	52	25	102
		% within SEX	100.0%	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	4.422[a]	2	.110
Likelihood Ratio	7.258	2	.027
Linear-by-Linear Association	3.052	1	.081
N of Valid Cases	102		

a. 2 cells (33.3%) have expected count less than 5. The minimum expected count is 2.94.

B10. SEPARATE ORIGIN OF LAD & LCX – ETHNICITY

Crosstab

			ETHN2C Ethnic group			
			Black	Indian	White	Total
LAD_LCX Separate origin of LAD and LCX	Present	Count	4	1	7	12
		% within ETHN2C Ethnic group	6.6%	50.0%	35.0%	14.5%
	Not present	Count	57	1	13	71
		% within ETHN2C Ethnic group	93.4%	50.0%	65.0%	85.5%
Total		Count	61	2	20	83
		% within ETHN2C Ethnic group	100.0%	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	11.945[a]	2	.003
Likelihood Ratio	10.390	2	.006
Linear-by-Linear Association	10.481	1	.001
N of Valid Cases	83		

a. 3 cells (50.0%) have expected count less than 5. The minimum expected count is .29.

B11. RAMUS MARGINALIS – SEX

Crosstab

			SEX			
			Female	Male	Not indicated	Total
RM_TRIF Ramus Marginalis	Present	Count	2	7	2	11
		% within SEX	8.0%	13.5%	8.0%	10.8%
	Not present	Count	23	45	23	91
		% within SEX	92.0%	86.5%	92.0%	89.2%
Total		Count	25	52	25	102
		% within SEX	100.0%	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	.790[a]	2	.674
Likelihood Ratio	.800	2	.670
Linear-by-Linear Association	.000	1	1.000
N of Valid Cases	102		

a. 2 cells (33.3%) have expected count less than 5. The minimum expected count is 2.70.

B12. RAMUS MARGINALIS – ETHNICITY

Crosstab

			ETHN2C Ethnic group			
			Black	Indian	White	Total
RM_TRIF Ramus Marginalis	Present	Count	11			11
		% within ETHN2C Ethnic group	18.0%			13.3%
	Not present	Count	50	2	20	72
		% within ETHN2C Ethnic group	82.0%	100.0%	100.0%	86.7%
Total		Count	61	2	20	83
		% within ETHN2C Ethnic group	100.0%	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	4.573[a]	2	.102
Likelihood Ratio	7.363	2	.025
Linear-by-Linear Association	4.383	1	.036
N of Valid Cases	83		

a. 3 cells (50.0%) have expected count less than 5. The minimum expected count is .27.

B13. HIGH ORIGIN OF CONUS – SEX

Crosstab

			SEX			
			Female	Male	Not indicated	Total
CONUS_HO High origin of conus artery	Present	Count	4	11	1	16
		% within SEX	16.0%	21.2%	4.0%	15.7%
	Not present	Count	21	41	24	86
		% within SEX	84.0%	78.8%	96.0%	84.3%
Total		Count	25	52	25	102
		% within SEX	100.0%	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	3.759[a]	2	.153
Likelihood Ratio	4.580	2	.101
Linear-by-Linear Association	1.348	1	.246
N of Valid Cases	102		

a. 2 cells (33.3%) have expected count less than 5. The minimum expected count is 3.92.

B14. HIGH ORIGIN OF CONUS – ETHNICITY

Crosstab

			ETHN2C Ethnic group			
			Black	Indian	White	Total
CONUS_HO High origin of conus artery	Present	Count	12	2	2	16
		% within ETHN2C Ethnic group	19.7%	100.0%	10.0%	19.3%
	Not present	Count	49		18	67
		% within ETHN2C Ethnic group	80.3%		90.0%	80.7%
Total		Count	61	2	20	83
		% within ETHN2C Ethnic group	100.0%	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	9.487[a]	2	.009
Likelihood Ratio	7.882	2	.019
Linear-by-Linear Association	.459	1	.498
N of Valid Cases	83		

a. 3 cells (50.0%) have expected count less than 5. The minimum expected count is .39.

B15. SEPARATE ORIGIN OF CONUS – SEX

Crosstab

			SEX			
			Female	Male	Not indicated	Total
CONUS_SO Separate origin of conus artery	Present	Count	1	2		3
		% within SEX	4.0%	3.8%		2.9%
	Not present	Count	24	50	25	99
		% within SEX	96.0%	96.2%	100.0%	97.1%
Total		Count	25	52	25	102
		% within SEX	100.0%	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	1.005[a]	2	.605
Likelihood Ratio	1.717	2	.424
Linear-by-Linear Association	.694	1	.405
N of Valid Cases	102		

a. 3 cells (50.0%) have expected count less than 5. The minimum expected count is .74.

B16. SEPARATE ORIGIN OF CONUS – ETHNICITY

Crosstab

			ETHN2C Ethnic group			
			Black	Indian	White	Total
CONUS_SO Separate origin of conus artery	Present	Count	3			3
		% within ETHN2C Ethnic group	4.9%			3.6%
	Not present	Count	58	2	20	80
		% within ETHN2C Ethnic group	95.1%	100.0%	100.0%	96.4%
Total		Count	61	2	20	83
		% within ETHN2C Ethnic group	100.0%	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	1.123[a]	2	.570
Likelihood Ratio	1.888	2	.389
Linear-by-Linear Association	1.076	1	.300
N of Valid Cases	83		

a. 4 cells (66.7%) have expected count less than 5. The minimum expected count is .07.

B17. ANOMALOUS PATH OF LCX – SEX

Crosstab

			SEX			
			Female	Male	Not indicated	Total
LCX_PATH Anomalous path of LCX	Present	Count	6	15	2	23
		% within SEX	24.0%	28.8%	8.0%	22.5%
	Not present	Count	19	37	23	79
		% within SEX	76.0%	71.2%	92.0%	77.5%
Total		Count	25	52	25	102
		% within SEX	100.0%	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	4.241[a]	2	.120
Likelihood Ratio	4.917	2	.086
Linear-by-Linear Association	1.814	1	.178
N of Valid Cases	102		

a. 0 cells (.0%) have expected count less than 5. The minimum expected count is 5.64.

B18. ANOMALOUS PATH OF LCX – ETHNICITY

Crosstab

			ETHN2C Ethnic group			
			Black	Indian	White	Total
LCX_PATH Anomalous path of LCX	Present	Count	18	1	4	23
		% within ETHN2C Ethnic group	29.5%	50.0%	20.0%	27.7%
	Not present	Count	43	1	16	60
		% within ETHN2C Ethnic group	70.5%	50.0%	80.0%	72.3%
Total		Count	61	2	20	83
		% within ETHN2C Ethnic group	100.0%	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	1.188[a]	2	.552
Likelihood Ratio	1.175	2	.556
Linear-by-Linear Association	.565	1	.452
N of Valid Cases	83		

a. 2 cells (33.3%) have expected count less than 5. The minimum expected count is .55.

B19. BIFID LAD – SEX

Crosstab

			SEX			
			Female	Male	Not indicated	Total
LAD_BIFI LAD bifurcates	Present	Count	2	9	3	14
		% within SEX	8.0%	17.3%	12.0%	13.7%
	Not present	Count	23	43	22	88
		% within SEX	92.0%	82.7%	88.0%	86.3%
Total		Count	25	52	25	102
		% within SEX	100.0%	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	1.318[a]	2	.517
Likelihood Ratio	1.389	2	.499
Linear-by-Linear Association	.167	1	.683
N of Valid Cases	102		

a. 2 cells (33.3%) have expected count less than 5. The minimum expected count is 3.43.

B20. BIFID LAD – ETHNICITY

Crosstab

			ETHN2C Ethnic group			
			Black	Indian	White	Total
LAD_BIFI LAD bifurcates	Present	Count	11	2	1	14
		% within ETHN2C Ethnic group	18.0%	100.0%	5.0%	16.9%
	Not present	Count	50		19	69
		% within ETHN2C Ethnic group	82.0%		95.0%	83.1%
Total		Count	61	2	20	83
		% within ETHN2C Ethnic group	100.0%	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	11.925[a]	2	.003
Likelihood Ratio	9.816	2	.007
Linear-by-Linear Association	1.103	1	.294
N of Valid Cases	83		

a. 3 cells (50.0%) have expected count less than 5. The minimum expected count is .34.

B21. MEAN VALUES

AGE HEIGHT * LAD_LCX Separate origin of LAD and LCX

LAD_LCX Separate		AGE	HEIGHT
Present	Mean	58.17	1.6925
	N	12	12
	Std. Deviation	27.035	.10056
	Minimum	26	1.48
	Maximum	89	1.86
Not present	Mean	44.17	1.6782
	N	65	65
	Std. Deviation	18.050	.07652
	Minimum	16	1.40
	Maximum	96	1.83
Total	Mean	46.35	1.6804
	N	77	77
	Std. Deviation	20.156	.08014
	Minimum	16	1.40
	Maximum	96	1.86

AGE HEIGHT * RM_TRIF Ramus Marginalis

RM_TRIF Ramus		AGE	HEIGHT
Present	Mean	37.00	1.7067
	N	9	9
	Std. Deviation	13.964	.07365
	Minimum	20	1.60
	Maximum	59	1.83
Not present	Mean	47.59	1.6769
	N	68	68
	Std. Deviation	20.597	.08082
	Minimum	16	1.40
	Maximum	96	1.86
Total	Mean	46.35	1.6804
	N	77	77
	Std. Deviation	20.156	.08014
	Minimum	16	1.40
	Maximum	96	1.86

AGE HEIGHT * CONUS_HO High origin of conus artery

CONUS_HO High		AGE	HEIGHT
Present	Mean	41.00	1.7053
	N	15	15
	Std. Deviation	14.663	.07396
	Minimum	20	1.57
	Maximum	69	1.83
Not present	Mean	47.65	1.6744
	N	62	62
	Std. Deviation	21.168	.08097
	Minimum	16	1.40
	Maximum	96	1.86
Total	Mean	46.35	1.6804
	N	77	77
	Std. Deviation	20.156	.08014
	Minimum	16	1.40
	Maximum	96	1.86

AGE HEIGHT * CONUS_SO Separate origin of conus artery

CONUS_SO Separate		AGE	HEIGHT
Present	Mean	38.33	1.6100
	N	3	3
	Std. Deviation	18.583	.07937
	Minimum	23	1.55
	Maximum	59	1.70
Not present	Mean	46.68	1.6832
	N	74	74
	Std. Deviation	20.267	.07938
	Minimum	16	1.40
	Maximum	96	1.86
Total	Mean	46.35	1.6804
	N	77	77
	Std. Deviation	20.156	.08014
	Minimum	16	1.40
	Maximum	96	1.86

AGE HEIGHT * LCX_PATH Anomalous path of LCX

LCX_PATH Anomalous		AGE	HEIGHT
Present	Mean	42.52	1.6829
	N	21	21
	Std. Deviation	14.421	.07226
	Minimum	20	1.55
	Maximum	69	1.79
Not present	Mean	47.79	1.6795
	N	56	56
	Std. Deviation	21.865	.08350
	Minimum	16	1.40
	Maximum	96	1.86
Total	Mean	46.35	1.6804
	N	77	77
	Std. Deviation	20.156	.08014
	Minimum	16	1.40
	Maximum	96	1.86

AGE HEIGHT * LAD_BIFI LAD bifurcates

LAD_BIFI LAD bifurcates		AGE	HEIGHT
Present	Mean	37.82	1.6836
	N	11	11
	Std. Deviation	12.640	.06054
	Minimum	22	1.57
	Maximum	56	1.75
Not present	Mean	47.77	1.6798
	N	66	66
	Std. Deviation	20.882	.08332
	Minimum	16	1.40
	Maximum	96	1.86
Total	Mean	46.35	1.6804
	N	77	77
	Std. Deviation	20.156	.08014
	Minimum	16	1.40
	Maximum	96	1.86

APPENDIX C

CONFIGURATION DESCRIPTION FOR TORTUOSITY INDEX PROGRAM

```
C
C The input data should be in the file IN.dat
C arranged as follows (the C on the left is not in it):
C  15
C  X     Y1    Y2    Y3    Y4    Y5    Y6
C 0.0    1.1   2.3   4.5   6.1   7.7   8.2
C 0.1    1.1   2.3   4.5   6.1   7.7   8.2
C 0.2    1.1   2.3   4.5   6.1   7.7   8.2
C 0.3    1.1   2.3   4.5   6.1   7.7   8.2
C 0.4    1.1   2.3   4.5   6.1   7.7   8.2
C 0.5    1.1   2.3   4.5   6.1   7.7   8.2
C 0.6    1.1   2.3   4.5   6.1   7.7   8.2
C 0.7    1.1   2.3   4.5   6.1   7.7   8.2
C 0.8    1.1   2.3   4.5   6.1   7.7   8.2
C 0.9    1.1   2.3   4.5   6.1   7.7   8.2
C 1.0    1.1   2.3   4.5   6.1   7.7   8.2
C 1.1    1.1   2.3   4.5   6.1   7.7   8.2
C 1.2    1.1   2.3   4.5   6.1   7.7   8.2
C 1.3    1.1   2.3   4.5   6.1   7.7   8.2
C 1.5    1.4   2.8   4.5   6.1   5.3   6.1
C
C This can be create with an editor (MS EDIT will do)
C or with a word processor, but save it as "text" file!
C The first number '15' is the number of points for
C each curve. Te program allows for a maximum of 100
C X-values.
C The results will appear in the file OUT.dat
C This can be viewed with a word processor and printed
C etc
C I have not got round to creating interesting graphics
C for you, but the shell is here, though not doing
C anything for now.

C
      WINAPP
C
      PROGRAM  NIRU
      INCLUDE <WINDOWS.INS>
      PARAMETER (NCURVES=24,NDIM=100)
C     NPOINTS <= NDIM !
      INTEGER*4 I,N,NT,NPOINTS
      CHARACTER KEY
      REAL*8 S,S1,ND,D
      REAL X(NDIM),Y(NDIM,NCURVES)
      REAL SLENGTH(NCURVES)
      CHARACTER*4 SCREEN,CH(NCURVES+1)
cC    INTEGER*2 XMAX,YMAX
      CHARACTER*20 TITLES(NCURVES)
      DATA TITLES/'curve 1','curve 2','curve 3',
```

```
     .          'curve 4','curve 5','curve 6',
     .          'curve 7','curve 8','curve 9',
     .          'curve 10','curve 11','curve 12',
     .          'curve 13','curve 14','curve 15',
     .          'curve 16','curve 17','curve 18',
     .          'curve 19','curve 20','curve 21',
     .          'curve 22','curve 23','CURVE24'/

      SCREEN = 'VGA'
      NT=1
      OPEN(UNIT = 5, FILE = 'C:\NIRU\IN2.dat')
      OPEN(UNIT = 6, FILE = 'C:\NIRU\OUT.dat')
      WRITE(*,5)
    5  FORMAT(1X,'You should have already put all your'/
     .         'input data in IN.dat. The results wiil'/
     .         'appear in OUT.dat. Continue (y or n)?'/)
      CALL GET_KEY@(KEY)
cc      READ *,KEY
      IF ((KEY .NE. 'y') .AND. (KEY .NE. 'Y')) THEN
        WRITE(*,6)
    6    FORMAT(1X, 'Close window and edit IN.dat')
        STOP
      ENDIF
C Read number of points for each curve
      READ(5,*)NPOINTS
C DUMMY READ TO NEXT POSITION
CC      READ(5,*)(CH(I),I=1,NCURVES+1)
C Clear arrays
      DO 50 I = 1,NPOINTS
      X(I)=0.0
      DO 50 J=1,NCURVES
      Y(I,J)=0.0
   50 CONTINUE
C Input the data from file IN.dat:
      DO 100 I=1,NPOINTS
      READ(5,90)X(I),(Y(I,J),J=1,NCURVES)
   90 FORMAT(F8.2,24F8.2)
C       Y(I,J) = I/100.0
C   5  FORMAT(F10.3)
  100 CONTINUE
C Output the data to file OUT.dat as a check:
      WRITE(6,101)NPOINTS
  101 FORMAT(1X,'NPOINTS = ',I5)
      DO 200 I=1,NPOINTS
      WRITE(6,90)X(I),(Y(I,J),J=1,NCURVES)
  200 CONTINUE
C Processing:
C Index1: total length/shortest path
      DO 250 J=1,NCURVES
      SLENGTH(J)= 0.0
  250 CONTINUE

      DO 500 J=1,NCURVES
      DO 300 I=1,NPOINTS-1
      SLENGTH(J)= SLENGTH(J)
```

```
     .  +SQRT((X(I+1)-X(I))**2
     .  + (Y(I+1,J)-Y(I,J))**2)
 300  CONTINUE
      SLENGTH(J) = SLENGTH(J)/
     .             SQRT((X(NPOINTS)-X(1))**2
     .    + (Y(NPOINTS,J)- Y(1,J))**2)
 500  CONTINUE

C Output results to file:
C     DO 620 J=1,NCURVES
      WRITE(6,610)SLENGTH
 610  FORMAT(1X,' NORMALISED SINUITY INDICES ARE:'
     .        /8X,24F8.3)
C 620  CONTINUE

      CALL GRAPH0(X,Y,NDIM,NPOINTS,NCURVES,SCREEN,TITLES,NT)
      CALL UPDATE_WINDOW@(0)
CC     CALL GRAPH(X,Y,NPOINTS,NCURVES,SCREEN,TITLES,NT)
CC     CALL UPDATE_WINDOW@
C     CALL TEXT_MODE@

      CLOSE(5)
      CLOSE(6)
 999  CONTINUE
      STOP
      END
C ---------------------------------------------------------------------
C
C     ********************************************************
      SUBROUTINE GRAPH0(X,Y,NDIM,NPOINTS,NGRAPHS,SCREEN,TITLES,NT)
C     called first-time only (VGA 640 x 480 pixels)
C     ************************************************
C     *                                      *
C     *  OUTPUT ONE OR MORE GRAPHS TO THE SCREEN       *
C     *  SCALING ACCORDINGLY                           *
C     *  THIS ROUTINE IS FOR ILLUSTRATIVE PURPOSES ONLY  *
C     *  IT ASSUMES THE X-DATA IS SORTED               *
C     *                                      *
C     ************************************************
C
      CHARACTER*(*) SCREEN
      CHARACTER*(*) TITLES(NGRAPHS)
      CHARACTER*8 LABEL
      CHARACTER*8 LAB2,CNUM
      INTEGER*4 NT
      REAL X(NDIM),Y(NDIM,NGRAPHS),XMIN,XMAX,YMIN,YMAX
      INTEGER*2 COLOUR(6)
      INTEGER*2 BLACK,BLUE,INVERSE_BLUE,GREEN,INVERSE_GREEN,

+CYAN,INVERSE_CYAN,RED,INVERSE_RED,MAGENTA,INVERSE_MAGENTA,

+BROWN,INVERSE_BROWN,YELLOW,INVERSE_YELLOW,WHITE,INVERSE_WH
ITE,
     +INTENSE,BLINKING
      PARAMETER(BLACK=0)
```

```
      PARAMETER(BLUE=1,INVERSE_BLUE=16)
      PARAMETER(GREEN=2,INVERSE_GREEN=32)
      PARAMETER(CYAN=3,INVERSE_CYAN=48)
      PARAMETER(RED=4,INVERSE_RED=64)
      PARAMETER(MAGENTA=5,INVERSE_MAGENTA=80)
      PARAMETER(BROWN=6,INVERSE_BROWN=96)
      PARAMETER(YELLOW=14,INVERSE_YELLOW=8+96)
      PARAMETER(WHITE=7,INVERSE_WHITE=7*16)
      PARAMETER(INTENSE=8)
      PARAMETER(BLINKING=128)
      DATA COLOUR/RED,YELLOW,GREEN,CYAN,BROWN,WHITE/
C SET SCREEN MODE:
      CALL VGA@
C EXTRACT VALUE OF NT AS CHARACTER STRING:
      LAB2 = CNUM(NT)
      LABEL = 'NT='
      CALL APPEND_STRING@(LABEL,LAB2)
C
C    DETERMINE THE DATA RANGE
C
      XMAX=X(1)
      YMAX=Y(1,1)
      XMIN=XMAX
      YMIN=YMAX
      DO 1 I=1,NPOINTS
        XMAX=MAX(XMAX,X(I))
        XMIN=MIN(XMIN,X(I))
        DO 1 J=1,NGRAPHS
          YMAX=MAX(YMAX,Y(I,J))
          YMIN=MIN(YMIN,Y(I,J))
 1      CONTINUE
C FIXED Y-SCALE (CC if not required):
      YMAX = 3.0
      YMIN = -1.0
C .....
C
C     DECIDE WHERE TO PUT THE ORIGIN
C
      IF(XMIN.LT.0.0)THEN
        XMAX=MAX(ABS(XMAX),ABS(XMIN))
        XMIN=-XMAX
        IX0=640/2
      ELSE
        XMIN=0
        IX0=0
      ENDIF
      IF(YMIN.LT.0.0)THEN
        YMAX=MAX(ABS(YMAX),ABS(YMIN))
        YMIN=-YMAX
      ELSE
        YMIN=0
      ENDIF
      XSCALE=640/(XMAX-XMIN)
      IF(SCREEN.EQ.'VGA')THEN
        YSCALE=480/(YMAX-YMIN)
```

```
      NYPOINTS=480
    ELSE
      YSCALE=350/(YMAX-YMIN)
      NYPOINTS=350
    ENDIF
    IF(YMAX.EQ.0)THEN
      IY0=0
    ELSE
      IY0=NYPOINTS/2
    ENDIF
C
C    DRAW THE AXES
C
    CALL DRAW_LINE@(IX0,0,IX0,NYPOINTS-1,WHITE)
    CALL DRAW_LINE@(0,IY0,639,IY0,WHITE)
C
C    AND TICKS
C
    IEPS=10
    DO 2 I=1,10
      CALL DRAW_LINE@(IX0+IEPS,I*NYPOINTS/10-1,IX0,I*NYPOINTS/10-1,WH
   +ITE)
2     CALL DRAW_LINE@(I*80-1,IY0+IEPS,I*80-1,IY0,WHITE)
C
C    DRAW THE GRAPHS
C
    DO 4 I=2,NPOINTS
      IX1=(X(I-1)-XMIN)*XSCALE+0.5
      IX2=(X(I)-XMIN)*XSCALE+0.5
      DO 3 J=1,NGRAPHS
        IY1=(Y(I-1,J)-YMIN)*YSCALE+0.5
        IY1=NYPOINTS-1-IY1
        IY2=(Y(I,J)-YMIN)*YSCALE+0.5
        IY2=NYPOINTS-1-IY2
        CALL DRAW_LINE@(IX1,IY1,IX2,IY2,COLOUR(J))
3       CONTINUE
4     CONTINUE
    IY=NYPOINTS*2/3
    IX=500
    DO 5 J=1,NGRAPHS
      CALL DRAW_TEXT@(TITLES(J),IX,IY,COLOUR(J))
5   IY=IY+20
    IX = IX-20
    CALL DRAW_TEXT@(LABEL,IX,IY,COLOUR(2))
    RETURN
    END
C
C    ******************************************************
    SUBROUTINE GRAPH(X,Y,NPOINTS,NGRAPHS,SCREEN,TITLES,NT)
C
C    ***************************************************
C    *                                 *
C    *  OUTPUT ONE OR MORE GRAPHS TO THE SCREEN      *
C    *  SCALING ACCORDINGLY                  *
C    *  THIS ROUTINE IS FOR ILLUSTRATIVE PURPOSES ONLY  *
```

```
C     * IT ASSUMES THE X-DATA IS SORTED              *
C     *                                              *
C     ************************************************
C
C fOLLOWING 4 needed to speed up graphics by wiping out
C only the old curves in Y0:
C must dimension Y0 to NPOINTS x NGRAPHS maximally
      PARAMETER(NPNTS=257)
      PARAMETER(NGRPHS=6)
      PARAMETER(NPSNGS=NPNTS*NGRPHS)
      REAL Y0(NPNTS,NGRPHS)
C
      CHARACTER*(*) SCREEN
      CHARACTER*(*) TITLES(NGRAPHS)
      CHARACTER*8 LABEL,LABEL0
      CHARACTER*8 LAB2,LAB20,CNUM
      INTEGER I,J
      INTEGER*4 NT
      REAL X(NPOINTS),Y(NPOINTS,NGRAPHS),
     .     XMIN,XMAX,YMIN,YMAX
      INTEGER*2 COLOUR(6)
      INTEGER*2 BLACK,BLUE,INVERSE_BLUE,GREEN,INVERSE_GREEN,

+CYAN,INVERSE_CYAN,RED,INVERSE_RED,MAGENTA,INVERSE_MAGENTA,

+BROWN,INVERSE_BROWN,YELLOW,INVERSE_YELLOW,WHITE,INVERSE_WH
ITE,
     +INTENSE,BLINKING
      PARAMETER(BLACK=0)
      PARAMETER(BLUE=1,INVERSE_BLUE=16)
      PARAMETER(GREEN=2,INVERSE_GREEN=32)
      PARAMETER(CYAN=3,INVERSE_CYAN=48)
      PARAMETER(RED=4,INVERSE_RED=64)
      PARAMETER(MAGENTA=5,INVERSE_MAGENTA=80)
      PARAMETER(BROWN=6,INVERSE_BROWN=96)
      PARAMETER(YELLOW=14,INVERSE_YELLOW=8+96)
      PARAMETER(WHITE=7,INVERSE_WHITE=7*16)
      PARAMETER(INTENSE=8)
      PARAMETER(BLINKING=128)
      DATA COLOUR/RED,YELLOW,GREEN,CYAN,BROWN,WHITE/
C Initialization for wipeouts:
      DATA LABEL0/'      '/
      DATA ((Y0(I,J),I=1,NPNTS),J=1,NGRPHS)/NPSNGS*1.0/
C SET SCREEN MODE:
CC      CALL VGA@           !only for DBOS/DOS
cc      CALL CLEAR_SCREEN@    !only if no wipeouts wanted
C EXTRACT VALUE OF NT AS CHARACTER STRING:
      LAB2 = CNUM(NT)
      LAB2 = CNUM(NT-10)
      LABEL = 'NT='
C     LABEL0 = 'NT='
      CALL APPEND_STRING@(LABEL,LAB2)
      CALL APPEND_STRING@(LABEL0,LAB2)
C
C     DETERMINE THE DATA RANGE
```

```
C
      XMAX=X(1)
      YMAX=Y(1,1)
      XMIN=XMAX
      YMIN=YMAX
      DO 1 I=1,NPOINTS
        XMAX=MAX(XMAX,X(I))
        XMIN=MIN(XMIN,X(I))
        DO 1 J=1,NGRAPHS
          YMAX=MAX(YMAX,Y(I,J))
          YMIN=MIN(YMIN,Y(I,J))
 1      CONTINUE
C FIXED Y-SCALE (CC if not required):
      YMAX = 3.0
      YMIN = -1.0
C .................
C
C     DECIDE WHERE TO PUT THE ORIGIN
C
      IF(XMIN.LT.0.0)THEN
        XMAX=MAX(ABS(XMAX),ABS(XMIN))
        XMIN=-XMAX
        IX0=640/2
      ELSE
        XMIN=0
        IX0=0
      ENDIF
      IF(YMIN.LT.0.0)THEN
        YMAX=MAX(ABS(YMAX),ABS(YMIN))
        YMIN=-YMAX
      ELSE
        YMIN=0
      ENDIF
      XSCALE=640/(XMAX-XMIN)
      IF(SCREEN.EQ.'VGA')THEN
        YSCALE=480/(YMAX-YMIN)
        NYPOINTS=480
      ELSE
        YSCALE=350/(YMAX-YMIN)
        NYPOINTS=350
      ENDIF
      IF(YMAX.EQ.0)THEN
        IY0=0
      ELSE
        IY0=NYPOINTS/2
      ENDIF
C
C     DRAW THE AXES
C
      CALL DRAW_LINE@(IX0,0,IX0,NYPOINTS-1,WHITE)
      CALL DRAW_LINE@(0,IY0,639,IY0,WHITE)
C       CALL DRAW_LINE@(0,0,639,0,WHITE)
C
C     AND TICKS
C
```

```
      IEPS=10
      DO 2 I=1,10
        CALL DRAW_LINE@(IX0+IEPS,I*NYPOINTS/10-1,IX0,I*NYPOINTS/10-1,WH
     +ITE)
2       CALL DRAW_LINE@(I*80-1,IY0+IEPS,I*80-1,IY0,WHITE)
C
C     DRAW THE GRAPHS
C
C Draw over old points i.e. wipeout previous curves
      DO 40 I=2,NPOINTS
        IX1=(X(I-1)-XMIN)*XSCALE+0.5
        IX2=(X(I)-XMIN)*XSCALE+0.5
        DO 30 J=1,NGRAPHS
          IY1=(Y0(I-1,J)-YMIN)*YSCALE+0.5
          IY1=NYPOINTS-1-IY1
          IY2=(Y0(I,J)-YMIN)*YSCALE+0.5
          IY2=NYPOINTS-1-IY2
          CALL DRAW_LINE@(IX1,IY1,IX2,IY2,BLACK)
30       CONTINUE
40      CONTINUE
C Draw new curves
      DO 4 I=2,NPOINTS
        IX1=(X(I-1)-XMIN)*XSCALE+0.5
        IX2=(X(I)-XMIN)*XSCALE+0.5
        DO 3 J=1,NGRAPHS
          IY1=(Y(I-1,J)-YMIN)*YSCALE+0.5
          IY1=NYPOINTS-1-IY1
          IY2=(Y(I,J)-YMIN)*YSCALE+0.5
          IY2=NYPOINTS-1-IY2
          CALL DRAW_LINE@(IX1,IY1,IX2,IY2,COLOUR(J))
3        CONTINUE
4       CONTINUE
      IY=NYPOINTS*2/3
      IX=500
      DO 5 J=1,NGRAPHS
        CALL DRAW_TEXT@(TITLES(J),IX,IY,COLOUR(J))
5     IY=IY+20
      IX = IX-20
      CALL DRAW_TEXT@(LABEL0,IX,IY,BLACK)
      CALL DRAW_TEXT@(LABEL,IX,IY,COLOUR(2))
C STORE OLD VALUES FOR NEXT WIPEOUT:
      LABEL0=LABEL
      DO 11 I=1,NPOINTS
        DO 11 J=1,NGRAPHS
          Y0(I,J)=Y(I,J)
11    CONTINUE
      RETURN
      END
```

APPENDIX D

TABLE D1: WF - no previous history of CAD

ANATOMICAL PROCEDURE	COMMENTARY
LOCATING ACCESS VESSELS	The RSA and RCCA were dissected and transected to allow access into the vessel lumen. The RSA was cannulated and the aortic lumen and coronary ostia were flushed using water. The endoscope was passed into the RCA and the coronary ostia were located. Difficulty was experienced in locating and approaching the left ostium. This was due to an awkward location a considerable distance away from the STJ.
CANNULATION & FLUOROSCOPY	The RCO was cannulated using a 4mm Fogarty catheter. The balloon was inflated to secure the orifice. 10ml PbO-microfil solution was injected into the lumen of the catheter. Procedure was done under fluoroscopy. The passage of the radio-opaque solution was observed on the screen. Images were recorded on AP and LAT projections. The C-arm was rotated around the specimen to produce a 3-D moving image, which was recorded on video. Fine branches extending from the RCA, ascending in the midline and laterally, outside the cardiac shadow were observed. The LCA was not cannulated due to damage of the catheter. It was therefore not injected or studied.
DISSECTION TECHNIQUE	The anterior thoracic wall was approached by reflecting the skin and the pectoral muscles. The ribs were dislocated at the costo-chondral junctions and the sterno-costal section was carefully harvested. There were no visibly injected vessels in the anterior mediastinum.
	Inspection of the exposed pleura showed no signs of injected vessels. The ribs were then cut away to expose a larger field of access into the mediastinum.

GROSS INSPECTION	There were no signs of injection material in the fibrous pericardium. An incision was made into the anterior aspect of the fibrous pericardium to expose the parietal and visceral pericardial layers. There was no sign of injection material in these layers. A number of injected vessels were observed on the ascending aorta, confirming the branches observed on radiographic inspection. The branches were carefully traced to their areas of origin.
DETAILED OBSERVATION & MORPHOMETRY	The RCA gave off a conus branch [ExD = 2.0mm]. Conus artery tortuous with 3 branches of noticeable size at its proximal end. 2 branches extended downwards and posteriorly around the main RCA to supply the right ventricular myocardium and epicardial fat in the vicinity. The 3rd ascending branch supplied part of the anterior surface of the infundibulum and the epicardial fat before bifurcating at the level of the root of the RCA. The left branch continued to give off fine twigs to the infundibulum and the other continued to ascend on the anterior surface of the aorta up to the arch. This branch [L=6.3cm; ExD=0.8mm] gave off 7direct branches of equal size [ExD=0.2mm] that ramified over the anterior surface of the aorta. The RCA gave off a corresponding branch 2cm from its origin. The branch [L=7.9cm; ExD=0.6mm] extended up to the origin of the BCT. There were 10 direct branches of equal size [ExD=0.2mm]. The right atrial branch arising from the RCA gave branches that extended to the base of the SVC. All the branches were seen to anastomose with the vasa vasora of the aorta and SVC, respectively. The branches from the conus artery anastomosed with those from the RCA over the anterior aspect of the ascending aorta.

TABLE D2: WM - PREVIOUS HISTORY OF CAD, MULTIPLE BYPASS GRAFTS

ANATOMICAL PROCEDURE	COMMENTARY
LOCATING ACCESS VESSELS	The RSA and RCCA were dissected and transected to allow access into the vessel lumen. The RSA was cannulated and the aortic lumen and coronary ostia were flushed using water. The endoscope was passed into the RCCA and the coronary ostia were located. LC ostium location above STJ, superior to a ridge.
CANNULATION & ANGIOGRAPHIC OBSERVATION	The RCO was cannulated using a 4mm Fogarty catheter. The balloon was inflated to secure the orifice. 10ml PbO-microfil solution was injected into the lumen of the catheter. Procedure was done under fluoroscopy. The passage of the radio-opaque solution was observed on the screen. Images were recorded on AP and LAT projections. The C-arm was rotated around the specimen to produce a 3-D moving image, which was recorded on video. LCO was cannulated with a 4mm Fogarty, injection showed poorly perfused left side, grafted vessels were visualised.
DISSECTION TECHNIQUE	The anterior thoracic wall was approached by reflecting the skin and the pectoral muscles. The ribs were dislocated at the costo-chondral junctions and the sterno-costal section was carefully harvested. There were no visibly injected vessels in the anterior mediastinum.
GROSS INSPECTION	Inspection of the exposed pleura showed no signs of injected vessels. The ribs were then cut away to expose a larger field of access into the mediastinum. There were no signs of injection material in the fibrous pericardium. An incision was made into the anterior aspect of the fibrous pericardium to expose the parietal and visceral pericardial layers. There was no sign of injection material in these layers. However, the pericardial area was extensively vascualrised with tortuous branches. The left lung was severely adhesed to the left side of the pericardium. The left and right ventricular myocardium was atrophic and extensively covered by fatty tissue. The fatty tissue was not well vascularised.
DETAILED OBSERVATION & MORPHOMETRY	The specimen was poorly injected and due to previous extensive intra-thoracic exploration, the results obtained were not anatomically viable by normal standards. There was no observation of naturally enhanced ECC connections.

TABLE D3: WM - PREVIOUS HISTORY OF CAD, MULTIPLE BYPASS GRAFTS

ANATOMICAL PROCEDURE	COMMENTARY
LOCATING ACCESS VESSELS	The RSA and RCCA were dissected and transected to allow access into the vessel lumen. The RSA was cannulated and the aortic lumen and coronary ostia were flushed using water. The endoscope was passed into the RCCA and the coronary ostia were located. Difficulty was experienced in locating and approaching the left ostium. This was due to an awkward location a considerable distance away from the STJ.
CANNULATION & FLUOROSCOPY	The RCO was cannulated using a 4mm Fogarty catheter. The balloon was inflated to secure the orifice. 10ml PbO-microfil solution was injected into the lumen of the catheter. Procedure was done under fluoroscopy. The passage of the radio-opaque solution was observed on the screen. Images were recorded on AP and LAT projections. The C-arm was rotated around the specimen to produce a 3-D moving image, which was recorded on video. The LCO was similarly cannulated. Fluoroscopy demonstrated ascending branches from LCA and RCA as well as lateral branches from both vessels, outside the cardiac shadow. A large transverse branch showed filling from the RCA and anastomosis with corresponding branch from LCA territory.
DISSECTION TECHNIQUE	The anterior thoracic wall was approached by reflecting the skin and the pectoral muscles. The ribs were dislocated at the costo-chondral junctions and the sterno-costal section was carefully harvested. There were no visibly injected vessels in the anterior mediastinum.
GROSS INSPECTION	The anterior fibrous pericardium was highly vascularised, however, there was no sign of infiltration of material. The epicardial fat was extensively vascularised. ECC's were observed on the anterior and posterior surfaces of the ascending aorta. Fine branches were also observed on the infundibulum, right and left pulmonary arteries, both pairs of veins, SVC, IVC with extension through the diaphragm.
DETAILED OBSERVATION &	The RCA gave off a conus branch [ExD = 0.8mm] at the origin of the RCA main trunk in conjunction. The conus branch supplied branches [ExD=0.2-0.4mm] to the surrounding fatty tissue and formed an anastomosis with the corresponding

MORPHOMETRY	the surrounding fatty tissue and formed an anastomosis with the corresponding left branch. A noticeable anastomosis was observed on the anterior and posterior surfaces of the ascending aorta. The anterior branch arose from the RCA at the junction between the RCA and conus artery. The vessel measured l=84mm and ExD=0.4mm. It gave off 14 branches. The posterior aspect of the aorta is supplied by the LCX branches and measured ExD=0.4mm, L=28mm. The vessel gives of 5 branches [ExD=0.2mm]. An anastomosis if formed between right and left sides as well as with the vasa vasora of the aorta. Vessels around the infundibular region give rise to branches that encircle the left and right pulmonary arteries. The LPA branch measured ExD=0.6mm. A main branch extends from it and continues to the hilum of the lung L=22mm, ExD=0.2mm. The LPA branch measured ExD=0.4mm. 3 noticeable branches stemmed from it. Branches extending from the posterior right atrial branches extend to the superior and inferior right pulmonary veins [RPV] The superior RPV has a branch encircling it ExD=0.8mm and gives of a branch L=20.5mm that bifurcates. The upper branch supplies the superior RPV and ExD=0.6mm[tapering to 0.4mm], L=16mm. The lower branch extends to the inferior RPV L=23mm, ExD=0.6mm[tapering to 0.4mm]. These branches send off smaller vessels to ramify towards the pleura and hila of the lungs. Branches extending from the posterior left atrial branches extend to the superior and inferior left pulmonary veins [LPV] The superior LPV has a branch encircling it ExD=0.6mm and gives of a branch L=18mm that bifurcates. The upper branch supplies the superior LPV and ExD=0.4mm L=12mm. The lower branch extends to the inferior LPV L=14mm, ExD=0.4mm. These branches send off smaller vessels to ramify towards the pleura and hila of the lungs. The right atrial branches from RCA gives off an ascending branch to the vasa vasora of the SVC, L=37mm, ExD=0.2mm. 5 fine branches extend on the surface of the SVC. The posterior atrial branch gives off a descending branch ExD=0.2mm, L=33mm that passes on the IVC and extends into the diaphragmatic opening fro the IVC. The main branch gives off a horizontal branch half way down its length, which divides into 3 finer branches on the anterior surface of the IVC. A descending branch from the anterior atrial branch also passes through the IVC opening in the

	diaphragm. At the superior aspect of the diaphragmatic orifice, there are 4 branches that surround the IVC as it enters the orifice. They measured ExD=0.2mm each.

TABLE D4: WF - NO PREVIOUS HISTORY OF CAD, NO SIGNS OF THORACIC SURGERY.

ANATOMICAL PROCEDURE	COMMENTARY
LOCATING ACCESS VESSELS	The RSA and RCCA were dissected and transected to allow access into the vessel lumen. The RSA was cannulated and the aortic lumen and coronary ostia were flushed using water. The endoscope was passed into the RCCA and the coronary ostia were located. Difficulty was experienced in locating both ostia. Upon visualisation, the ostia appeared narrow and the left ostium , almost slit-like.
CANNULATION & FLUOROSCOPY	The cannulation was unsuccessful. Although the ostia were approached, it would not allow for the insertion of the catheter.
DISSECTION TECHNIQUE	The specimen was dissected without intra-aortic injection. The standard dissection procedure was used and careful attention was paid to connecting vessels. This was done under magnification.
GROSS INSPECTION	Although the connections were not as clearly demonstrable as occurs with injection, the cardiac area was highly vascularised. Connections showed up due to the presence of blood in the lumen. The fat vessels were abundant and the anastomoses towards the hila of the lungs was pronounced.
DETAILED OBSERVATION & MORPHOMETRY	Further dissections showed ostial occlusion of the RCA - hence the inability to insert the catheter. The conus branch was large and measured 0.8mm diameter and branches to the sub-epicardial fat pads and pulmonary trunk were clearly visible. However, they were technically difficult to measure. Therefore no recordings were taken. The LCA was partially patent at the ostial portion. However, the LAD and LCX were sclerosed diffusely and brittle to the touch. Based on observation alone, ECC's were observed on the IVC, SVC and PT. Branches were seen extending to the LPA and LPV's. There was an abundance of

	ECC's on the ascending aorta, extending as far as the origin of the great vessels. However, no accurate data could be recorded. It can be concluded that this case represents a scenario of the patient showing no symptomatic signs of severe CAD but presenting with diffuse disease at necropsy.

TABLE D5: WM - NO PREVIOUS HISTORY OF CAD, NO SIGNS OF THORACIC SURGERY.

ANATOMICAL PROCEDURE	COMMENTARY
LOCATING ACCESS VESSELS	The RSA and RCCA were dissected and transected to allow access into the vessel lumen. The RSA was cannulated and the aortic lumen and coronary ostia were flushed using water. The endoscope was passed into the RCCA and the coronary ostia were located. The ostia were easily visualised.
CANNULATION & FLUOROSCOPY	The cannulation procedure was modified, by replacing the original guide wire with thin guitar wire that extended passed the lumen opening at the Fogarty tip. A bend was added to the proximal end of the wire to allow for greater control and guidance into the ostia. The wire was slightly bent at the distal end close to the balloon area to allow for an easier approach to the ostia. The RCO was cannulated using a 5mm Fogarty catheter. The balloon was inflated to secure the orifice. 10ml PbO-microfil solution was injected into the lumen of the catheter. Procedure was done under fluoroscopy. The passage of the radio-opaque solution was observed on the screen. Images were recorded on AP and LAT projections. The C-arm was rotated around the specimen to produce a 3-D moving image, which was recorded on video. The LCO was similarly cannulated. Fluoroscopy demonstrated ECC's to the aortic and pulmonary regions.
DISSECTION TECHNIQUE	The anterior thoracic wall was approached by reflecting the skin and the pectoral muscles. The ribs were dislocated at the costo-chondral junctions and the sterno-costal section was carefully harvested. There were no visibly injected vessels in the anterior mediastinum.
GROSS INSPECTION	The anterior fibrous pericardium was highly vascularised, however, there was no sign of infiltration of material. The pericardium showed extensive vascularisation with connections to the pleural vessels.

	The epicardial fat was extensively vascularised. Especially noticeable around the apex and lower right atrial area. Although fine vessels were seen on the anterior surface of the ascending aorta, the absence of injection material suggests that they do not have coronary connections. The conus branch contributed an extensive supply to the anterior ventricular epicardial fat.
DETAILED OBSERVATION & MORPHOMETRY	The conus artery [0.8mm exd] gave numerous branches to the fat. The atrial branches were prominent [0.6mm exd]. The LCA gave a branch that arose at a point 11mm from the origin. It extended to the PT anterior surface and further to the LPA. The main branch measured 0.6mm exd & 86mmL. The 2 secondary branches measure 35 and 40mmL respectively and 0.4mm exd. The LCX gave off a branch 56mm from origin that extended to the LPV. The main branch was 46mmL and 0.6mm exd. 2 secondary branches measured 45 and 50mmL and 0.2mm exd. Fine branches were observed from the LAD supplying the fat around it. The RAA measured 0.6mm and only 1 branch was seen to extend to the SVC. There was no clear sign of ECC's to the RPA or RPV's, although the area was vascualrised to some degree.

TABLE D6: WM - NO PREVIOUS HISTORY OF CAD, NO SIGNS OF THORACIC SURGERY

ANATOMICAL PROCEDURE	COMMENTARY
LOCATING ACCESS VESSELS	The RSA and RCCA were dissected and transected to allow access into the vessel lumen.
CANNULATION & FLUOROSCOPY	The RCO was visualized. However, there was evidence of extensive ostial occlusion. A stellate shaped plaque covered most of the opening. Cannulation was therefore not possible. The left ostium was not easily visualized endoscopically. This was due to the extensive distortion of the aorta. The internal walls of the aorta were severely sclerosed. Cannulation of both ostia was unsuccessful.
DISSECTION TECHNIQUE	The anterior thoracic wall was approached using a minimally invasive method. The sterno-clavicular joints were dislocated and the anterior section of sternum was removed, leaving the ITA's on the surface of the pericardium. A wider access was gained using a surgical spreader. The area around the root of the aorta was carefully dissected and the proximal portions of the RCA and LCA were isolated. An incision was made into the aorta and the endoscope passed once again to try and locate the left ostium. This was possible by getting rid of the occlusive visual effect of the distorted aorta. The vessels were cannulated and injected retrogradely using red microfil. The injection was partially successful, but not satisfactory for detailed data recording.
GROSS INSPECTION	The anterior fibrous pericardium was highly vascularised, and showed extensive vascularisation with connections to the pleural vessels. The epicardial fat was extensively vascularised. ECC's were large enough to dissect even without injection filling. The PT was extensively vascularised and the connections extended toward the PA's and PV's on both sides. ECC's were seen on the IVC, SVC and AA. Ostial occlusion of the RCA was confirmed. A particularly large vessel was seen arising from the post occluded site on the RCA and it extended upwards ramifying over the AA.

	Secondary branches extended to the PT and connected with pericardial vessels. A large branch can be seen arising from the RAA and descending toward the IVC. The vessel courses the sulcus terminalis.
DETAILED OBSERVATION & MORPHOMETRY	No report

TABLE D7: WF - NO PREVIOUS HISTORY OF CAD, NO SIGNS OF THORACIC SURGERY.

ANATOMICAL PROCEDURE	COMMENTARY
LOCATING ACCESS VESSELS	The RSA and RCCA were dissected and transected to allow access into the vessel lumen. The RSA and BCT were enlarged and RSA with tortuous course.
CANNULATION & FLUOROSCOPY	The RCO and LCO were easily visualized. There was no sign of ostial occlusion or extensive vessel disease. Successful cannulation was achieved, however, the injection was unsuccessful, due to deflation of the balloons.
DISSECTION TECHNIQUE	The costo-chondral junctions were disarticulated and the anterior sternum section reflected. The ITA's were dissected away from the ribs and left with origins intact. Wider access achieved by removing a greater extent of the ribs. The clavicles were disarticulated and removed along with the 1st ribs. A longitudinal slit was made on the anterior fibrous pericardium, without disturbing the pericardial vessels.
GROSS INSPECTION	The anterior fibrous pericardium was highly vascularised. The epicardial fat was not clearly vascularised. The right lung was adherent to the right side of the heart and ascending aorta as well as to the diaphragm. Inspection of the great vessels showed many fine vascular patterns surrounding the adventitia. The right surface of the ascending aorta was extensively vascular with pleural connections. Vessels were observed along the RPV's, LPV's, SVC.
DETAILED OBSERVATION & MORPHOMETRY	The RCA was located. There was a high degree of sclerosis present. Two branches were seen to arise from the left border. The 1st and smaller, supplied the fat over the infundibular area. The 2nd and larger, representing the conus artery, supplied ventricular branches to the infundibulum. The conus artery was bridged by a strap of myocardium. A right branch arose 4mm from the origin of the RCA. The stem immediately bifurcated to give rise to an ascending branch [B1] that adhered to the ascending aorta and a branch [B2] that travelled to the right and posteriorly, on the surface of the aorta.

	B1 measured 206mm in length and 0.6mm ext/d. The branch continued towards the aortic arch and anastomosed with branches from the fat as well as branches from the left side, coursing over the PT. There were 4 secondary branches [0.2mm]. The branch then terminated by anastomosing with the left pericardiocophrenic artery that arose 70mm from the LITA. B2 measured 30mm length and 0.2-0.6mm ext/d. It then branched into an extensive network of 12 secondary branches [0.2mm] on the right and posterior aspect of the ascending aorta. The LCA showed signs of diffuse disease. The LAD and LCX were patent. A branch arose 6mm from point of origin of LCA on the right side. Coursed downward and then upward along the adventitia of the PT. The branch continued toward the ligamentum arteriosus and dipped behind, followed a right course and terminated at the anastomotic area of the right B1. The Branch measure 139mm length and 0.6mm ext/d. There were 2 secondary branches [0.2-0.6mm] and fine network of vessels in the PT adventitia [immeasurable size].

TABLE D8: WF - NO PREVIOUS HISTORY OF CAD, NO SIGNS OF THORACIC SURGERY

ANATOMICAL PROCEDURE	COMMENTARY
LOCATING ACCESS VESSELS	The RSA and RCCA were dissected and transected to allow access into the vessel lumen. The LCCA was exposed.
CANNULATION & FLUOROSCOPY	The RCO and LCO were easily visualized. There was no sign of ostial occlusion or extensive vessel disease. Successful cannulation and injection was achieved. Red Microfil was used as injection material. The left system filled easily. The right system filled at a slower pace and a highly distended and tortuous right marginal, posterior descending artery was observed.
DISSECTION TECHNIQUE	The ITA's were dissected and exposed at their origins. The costo-chondral junctions were disarticulated and the anterior sternum section reflected. The ITA's were dissected away from the ribs and left with origins intact. Wider access achieved by removing a greater extent of the ribs. The clavicles were disarticulated and removed along with the 1st ribs. A longitudinal slit was made on the anterior fibrous pericardium, without disturbing the pericardial vessels.
GROSS INSPECTION	The anterior fibrous pericardium was highly vascularised. The vessels extended around the pleura and over the lungs. The epicardial fat was vascularised to a lesser extent. The vessels around the hila of the lungs were pronounced and anastomoses between the pericardial vessels could be observed. Inspection of the great vessels showed many fine vascular patterns surrounding the adventitia. The right surface of the ascending aorta was extensively vascular and the vessels extended up to the right subclavian and common carotid. Vessels were observed along the RPV's, LPV's, SVC, IVC.

DETAILED OBSERVATION & MORPHOMETRY

There was an average amount of epicardial fat, with no sign of extensive vascularisation. A prominent anastomosis was observed between the LAD and L marginal branches via the anterior ventricular branches. The main RCA and LCA were relatively free of atherosclerosis, although markedly distended, particularly the PD portion of the RCA.

AORTA

- The conus artery was identified originating from a common stem with the RCA.
- A network of fine branches [5 main, 0.2mm exd] spread out around the root of the anterior surface of the ascending aorta. 12mm from the origin of the RCA, a branch arose from its posterior aspect. The branch measured 0.6mm exd and ascended on the anterior right surface of the aorta for 37mm of length. At this point it bifurcated into 2 equal sized branches [0.4-0.6mm]. B1 continued to the left for 44mm and anastomosed with the vessels on the adventitia in the region. B2 extended to the right for 59mm before anastomosing with an unidentified branch [*thyroid*?] coming from the left side.

  The ascending aorta was extensively vascularised all around, mainly via contribution from the RCA branches. A prominent anastomotic circle was observed on the right surface of the ascending aorta, up to the aortic arch.

SVC

- The atrial branch arose 23mm from the RCA origin, measured 1.2mm exd. It continued between the aorta and RA appendage towards the LA appendage. At the base of the SVC, it gave off a major branch 1.0 mm exd that ascended on the SVC. The branch continued to ascend with a tapering diameter to 0.6 mm. There were 6 lateral branches of 0.2mm exd each. The vessel length measured 40mm and extended up to the union of the brachiocephalic veins. The lateral secondary branches anastomosed with lateral branches from the atrial appendage artery, to form a network of vessels over the anterior surface of the SVC.

LPV

- The main atrial branch continued to the left while giving off branches

	that encircled the LPV. 3 main circular vessels, of 0.4 mm exd each. These vessels anastomosed with vessels from the posterior pericardium and continued to the hilum of the lung. The main branch terminates by supplying the LA appendage. Anastomoses were also observed between branches around the left principal bronchus [0.6mmd]. **IVC** • As the RCA turns around the right inferior margin to the posterior interventricular groove, a descending branch is given off to the IVC [0.4 mm]. It continues beneath the diaphragmatic opening, fanning out into fine branches - there are 6 secondary branches. A branch arises from the LCxA that descends and then curves upward around the base of the IVC [0.6mm D, 84mm L] 4 secondary branches arise from the inner aspect of the LCX branch and anastomose with branches from the RC branch as well as the vasa vasorum of the IVC. **PT** • A network of fine vessels was seen on the PT - originating from LCA. In addition, tiny beads of Microfil was found within the pulmonary arteries, suggesting that the injection material had passed through the vascular channels of the pulmonary system, possibly via the bronchial connections. **BRONCHIAL** • A main bronchial trunk [2mmd] arose from the aorta, it split to give a branch to each side. The branch to the right contained injection material. Filling was traced to the main atrial branch [1.0mm] around the RPV. On further inspection of the anterior ventricular anastomoses between LAD and L marginal, an anastomotic branch was traced from its anastomosis with a branch of the L marginal to a point of anastomosis at the origin of the LCA. At this area, a cruciate pattern of anastomoses was observed. The lower left branch from the L marginal joined a lower right branch from the fat over the PT to form a union at an area to the left of the LCA origin, between the ascending aorta and PT. The upper right branch joined the upper left branch and continued to the right, beneath the ascending aorta. The branch then traveled underneath the aortic arch and then ascended along the trachea from the left principal bronchus, diagonally across the anterior surface of the trachea and then along the right margin of the trachea. It measured 24.4cm in length and 1.8mm diameter throughout its course. As it travels across the trachea, it

	anastomoses with branches from the tissue surrounding the trachea. It anastomoses with a branch arising from the right tracheal region as well as the right pericardiocophrenic artery. At a point 1 cm from the carina, an anastomosis between a right bronchial branch was observed. The anomalous vessel continued to terminate within the tissue of the posterior lobe of the thyroid gland, closely related to an anomalous inferior thyroid artery. The inferior thyroid artery arose from the first part of the subclavian artery in isolation as the first ascending branch, bilaterally. On the right, the vertebral, ITA and thyro-cervical trunk of 2 branches was identified. On the left, the ITA and thyro-cervical trunk originated from a common trunk. On the right side of the trachea, the anomalous branch contributed to an extensive anastomosis between the pericardiocophrenic, bronchial, tracheal, pulmonary and aortic vessels.

TABLE D9: WF - NO PREVIOUS HISTORY OF CAD, NO SIGNS OF THORACIC SURGERY. RIGHT MASTECTOMY

ANATOMICAL PROCEDURE	COMMENTARY
LOCATING ACCESS VESSELS	The RSA and RCCA were dissected and transected to allow access into the vessel lumen. The LCA was exposed.
CANNULATION & FLOUROSCOPY	The RCO was notably large and patent, LCO appeared more slit-like. There was no sign of ostial occlusion or extensive vessel disease. Successful cannulation and injection was achieved. Red microfil was used as injection material. The left system filled poorly. The right system filled easily, the RCA was large and tortuous. An unusual filling of a structure appeared to the right of the RCA system, in the vicinity of the SVC. RCA system appeared to be dominant angiographically.
DISSECTION TECHNIQUE	The ITA's were dissected and exposed at their origins. The costo-chondral junctions were disarticulated and the anterior sternal section reflected. The ITA's were dissected away from the ribs and left with origins intact. Wider access achieved by removing a greater extent of the ribs. The clavicles were disarticulated and removed along with the 1st ribs. A longitudinal slit was made on the anterior fibrous pericardium, without disturbing the pericardial vessels.
GROSS INSPECTION	The anterior fibrous pericardium was highly vascularised. The vessels extended around the pleura and over the lungs. The vascularization of the epicardial fat was of an average amount. An extensive anastomotic pattern was observed between the vessels on the anterior fibrous pericardium. Inspection of the great vessels showed many fine vascular patterns surrounding the adventitia. The right surface of the ascending aorta was extensively vascular and the vessels extended up to the right common carotid artery. Right lung adhesion to right fibrous pericardium - vessels extending to pleura. Vessels were observed along the RPV's, LPV's, SVC, IVC, AA.
	The epicardial fat vessels were demonstrable. The RCA was tortuous and its

DETAILED OBSERVATION & MORPHOMETRY	vasa vasora was clearly visible and formed and extensive anastomosis around the vessel. Injection material was found within the SVC, IVC and the left and right brachiocephalic veins. The cardiac veins also contained injection material. Evidence of the microfil having passed through a capillary system was found. **AORTA** • Conus artery arose 5mm from origin of the RCA. Opposite the conus, a branch arose directly from the RCA ascended on the anterior surface of the ascending aorta [91mmL, 0.4mmD - 7:2° branches, 0.2mmD]. The 2° branches arose 3 from the right and 4 from the left of the main branch. • The vasa vasora of was seen to anastomose with the fine vessels. A prominent anastomosis was found on the right posterior aspect of the ascending aorta. The right atrial branch contributed to the connections. • The anastomosis completely surrounded the aorta and the vessels extended up to 1cm above the origin of the right common carotid. Laterally, the branches anastomosed with vessels from the vasa vasorum of the PT. **SVC** • A large amount of Microfil was contained within the SVC. • 2 fine vessels were observed extending from the R atrial branch to anastomose with the vasa vasora on the anterior surface. • A branch originating from the LCX, 7mm from its origin at the LCA bifurcation was seen to extend across the left atrial appendage pass on the anterior surface of the left PV to terminate at the superior aspect of the right atrial appendage at the base of the SVC. The vessel extended 2-3 fine branches to the SVC, although it gave off no branches in its course [64mm L / 1.0mmD tapering to 0.6mm] **LPV** • From atrial artery, branch extending toward hilum [39mmL / 0.6mmD and tapering] **IVC** • A network of fine vessels was observed around the cardiac portion of the IVC. 2 vessels were seen extending beyond the pericardium and beneath the diaphragm [0.2mm D] • A main branch arose form the LCX [52mmL / 0.2mmD] with

	approximately 8 2° branches and anastomosed with descending branches from the base of the right atrium. These branches were traced to the RCA. • This anastomotic network was prominent in the region of the posterior inter ventricular groove where the LCX and RCA usually anastomose. • Microfil was found within the IVC lumen. **PT** • Not prominently vascularised. Vessels to vasa vasora arose from the LCX and contributions from the aorta via its vasa vasora branches were observed. **TRACHEAL** • Filling of vessels around the trachea was observed [0.6mmD]. These extended from the anterior surface of the descending aorta and were traced to the fine vessels in the tissue over the pulmonary arteries as well as those of the serous pericardium. **POSTERIOR PERICARDIAL** • The posterior fibrous pericardial vessels were seen to form anastomoses with the fat vessels found around the roots of the aorta and PT.

APPENDIX E

APPENDIX E1: ANGIOGRAM VARIATIONS FOR TOTAL SAMPLE

	AGE	SEX	ETHTHNICITY	LAD/LCX	RM	CONUC	DOMINANCE	LCX PATH	BIFID LAD	
1	32	M	W				R			
2	60	M	B				R			
3	66	M	I				R			
4	58	F	I		#		R			
5	40	M	B				R		#	
6	36	F	I				R			
7	52	M	W	#			R			
8	58	F	W				R		#	
9	74	F	I				R			
10	73	F	I		#		R			
11	59	M	I		#		R			
12	69	M	I		#		R			
13	41	M	W				R			
14	51	M	I				R			
15	51	M	I				R			
16	38	M	I		#		R		#	
17	67	F	W				L			
18	62	M	W				R			
19	62	M	W				R			
20	39	M	W				L			
21	54	M	W				R			
22	45	F	I				R			
23	38	M	I		#		R			
24	56	F	W		#		R			

25	58	M	W				R			
26	84	F	W				R			
27	61	F	B				R			
28	36	M	I				R			
29	35	M	I				R			
30	63	M	I				R			
31	52	M	W				R			
32	41	M	W				R			
33	42	F	I				R			
34	67	M	I				R			
35	65	M	I				R			
36	44	M	I				R			
37	46	F	W				R			
38	50	M	I				R			
39	52	F	W				R			
40	56	M	W				R			
41	63	F	I				R			
42	69	M	I				R			
43	43	M	I				R			
44	50	M	W				R			
45	54	M	I				L			
46	39	M	I				L			
47	66	F	I				C			
48	67	M	I				C		#	
49	56	M	I				C		#	
50	58	F	I				L		#	
51	62	M	W				R			
52	50	M	I				R			
53	46	F	I				R			
54	60	M	W				R			
55	78	F	W				R			
56	50	M	W				R			
57	50	M	W				L			
58	77	F	W				R			
59	64	M	I				R			

60	72	M	W				R			
61	69	M	W				R			
62	63	F	I				R			
63	60	M	I				R			
64	56	M	B				R			
65	77	M	W				R			
66	63	M	I				R			
67	54	M	I				R			
68	67	M	W				R			
69	55	M	I				R			
70	52	M	W				R			
71	63	F	W				R			
72	62	M	I				R			
73	64	M	I				R			
74	58	M	C				R			
75	64	M	W				R			
76	55	M	I				L			
77	67	M	I				R			
78	62	F	I				R			
79	50	M	I				L			
80	65	M	I				C			
81	44	M	I				R		#	
82	46	M	I				R			
83	66	F	B				R			
84	61	M	W				R			
85	56	M	I				R			
86	57	M	I				L			
87	44	M	W				L			
88	50	M	W				L			
89	66	M	I				R			
90	63	F	I				R			
91	47	F	B				R			
92	61	M	I				L			
93	51	M	I				L			
94	69	M	I				R			

95	49	M	I				R			
96	68	M	W				R			
97	59	M	I				R			
98	60	M	B				R			
99	56	M	I				R			
100	67	M	I				R			
101	66	F	I				R			
102	53	M	I				R			
103	48	M	I				R			
104	47	M	I				R			
105	62	F	I				R			
106	56	F	W				R			
107	46	M	I				R			
108	64	M	I				R			
109	69	F	I				R			
110	76	F	I				R			
111	65	F	W				R			
112	48	M	W				R			
113	61	M	I				R			
114	67	F	I				R			
115	52	F	W				R			
116	32	M	I				R			
117	47	M	I				R			
118	67	M	I				R			
119	61	F	I				R			
120	57	M	I				R			
121	50	M	I				R			
122	57	M	I				R			
123	43	M	I				R			
124	58	M	I				L			
125	47	M	I				R			
126	40	M	I				R			
127	60	M	W				R			
128	56	M	I				R			
129	70	M	I				R			

130	69	M	I				R			
131	62	M	W				R			
132	51	F	W				R			
133	68	M	W				R			
134	65	F	W				R			
135	60	F	I				R			
136	74	M	I				L			
137	56	F	W				L			
138	58	F	W				R			
139	66	M	W				R			
140	52	M	I				R		#	
141	51	F	I				R			
142	55	M	I				R			
143	50	M	I				R			
144	75	M	I				R			
145	59	M	I				R			
146	61	M	I				C			
147	60	M	I				R			
148	62	M	I				R			
149	56	M	I				R			
150	67	M	I				R			
151	68	M	I				R			

APPENDIX E2: CADAVERIC VARIATIONS FOR TOTAL SAMPLE

	AGE	SEX	ETHNIC	HT	LAD/LCX	RM	CONUS/ho	CONUS/so	DOMINANCE	BIFID LAD	LCX PATH
1	34	M	B	1.70					R		
2						#	#		R	#	
3	24	M	B	1.60					R	#	
4	22	M	B	1.70		#	#		R	#	
5	30	M	B	1.70					R		
6	37	M	B	1.75		#			R		
7	17	M	B	1.70					R		
8						#			C		
9	40	M	B	1.73	#				C		#
10	26	M	I	1.57	#		#		R	#	
11	54	F	I	1.74			#		R	#	#
12	80	F	W	1.40					R		
13	84	F	W	1.63	#				L		
14	89	F	W	1.48	#				R		
15	70	F	W	1.68					R		
16	70	M	W	1.65					R		
17	86	F	W	1.68					R		
18	88	F	W	1.86	#				R		
19	80	M	W	1.68					R		
20	51	F	B	1.71	#				R		
21	55	F	B	1.80					R		
22	26	M	B	1.76	#				R		
23	23	F	B	1.62					R		
24	76	M	W	1.75	#				R		
25	35	F	B	1.68					R		
26	81	M	W	1.63	#				C		
27	96	M	W	1.70					R		
28	80	M	W	1.75	#				R		
29	80	M	W	1.77					C		
30	70	F	B	1.63					R		

31									R		
32	50	M	B	167					C		
33	32	M	B	1.83		W	#		R		
34	35	M	B	1.67					R		
35	35	M	B	1.71					R		
36	41	M	B	1.65			#		R		#
37	57	F	B	1.69		#			R		
38	27	M	B	1.67		#			L		
39	29	M	B	1.64					R		#
40	20	M	B	1.79			#		R		#
41	69	F	W	1.63			#		C		#
42	38	F	B	1.77			#		C		
43	33	M	B	1.58				#	L		
44	20	M	B	1.67		#	#		R		
45	24	M	B	1.75					R		#
46	36	M	B	1.77					R		
47	32	F	B	157					R		#
48	23	M	B	170				#	C		#
49	28	M	B	160					R		
50	53	M	B	168			#		R		#
51	42	M	B	163			#		R		#
52	25	M	B	175			#		R	#	#
53	45	M	B	155					R		#
54									R		#
55	38	M	B	173					R		
56									R	#	
57	48	F	B	1.56					R		
58	16	F	B	1.57					R		
59	36	M	B	1.80		#			L		
60	38	M	B	1.65					R	#	
61									R	#	#
62	52	F	B	1.57					R		
63	55	M	B	1.70					R		#
64	30	M	W	1.74	#				R		
65	48	M	B	1.70			#		R	#	#

66	59	F	B	1.55				#	R		
67	40	M	B	1.72					L		
68	45	F	W	1.68					R		
69	35	M	B	1.74					R	#	
70	50	M	B	1.70					R	#	
71	27	M	B	1.70	#				R		
72	56	M	W	1.78		#	#		R		#
73	43	M	B	1.65	#				C		
74	23	M	B	1.70					R		#
75	38	M	B	1.73					R	#	
76	44	F	W	1.55					R		#
77	56	F	W	1.64					R	#	#
78	59	F	B	1.60	#	#			R		
79	30	M	B	1.75					R		
80	60	F	B	1.68			#		R		#
81	54	M	B	1.78					R		#
82	47	M	W	1.68					C		
83	44	M	B	1.72					R		

APPENDIX E3: MYOCARDIAL BRIDGES IN TOTAL SAMPLE

	AGE	SEX	ETHTHNICITY	HT	P/LAD	M/LAD	D/LAD	LCX	PDA	RCA
1	34	M	B	1.70		2.9				
2	24	M	B	1.60			0.71			
3	22	M	B	1.70		0.9				
4	30	M	B	1.70						
5	37	M	B	1.75		1.9				
6	17	M	B	1.70	2.55	0.9				
7	26	M	B	1.57	4.55					
8	48	F	I	1.56		14.5				
9	16	F	B	1.57		25.5				
10	36	M	B	1.80		IM	IM			
11	38	M	B	1.65		19.5				
12	55	M	B	1.70		28.5			10	
13	30	M	B	1.74		IM				
14	48	M	B	1.70	7					
15	59	F	W	1.55		IM				
16	42	M	B	1.72		IM				
17	27	M	B	1.70				4L		
18	36	M	B	1.77				2L		4L
19	24	M	B	1.75	14.5					
20	32	F	B	1.57		IM				
21	23	M	B	1.69	15	19.5	11.0			
22	28	M	B	1.60		24.0				
23	27	M	B	1.67	16	16.0				
24	36	M	B	1.57	16	16.0				
25	24	M	B	1.79	10	52.5				
26	32	M	B	1.67		20.0	23.5			
27	23	M	B	1.65		17.5				
28	28	M	B	1.71		24.0				
29	27	M	B	1.67		3 L		2L		
30	29	M	B	1.83	11				IM	

31	20	M	B	1.67		36.1				
32	20	M	B	1.78		13.7				
33	41	M	B	1.65		25.2				
34	35	F	B	1.70		8.70	3.1			
35	35	M	W	1.60		10.0				
36	32	F	B	1.75		28.0				
37	50	M	B	1.68		9.0				
38	56	M	B	1.68		32.0				
39	43	M	B	1.72		43.0				
40	23	M	W	1.60		13.0				
41	59	M	B	1.69		12.0				
42	30	M	B	1.71		46.0				
43	60	M	B	1.68		25.0				IM
44	47	M	B	1.65		20.0				
45	44	M	B	1.70		13.0				

APPENDIX E4: LENGTH MEASUREMENTS OF THE LCA

	AGE	SEX	ETHNICITY	HT	LCA/cm
1	34	M	B	1.70	0.54
2					1.17
3	24	M	B	1.60	0.27
4	22	M	B	1.70	0.84
5	30	M	B	1.70	1.18
6	37	M	B	1.75	0.80
7	17	M	B	1.70	0.68
8					0.99
9	40	M	B	1.73	-
10	26	M	I	1.57	-
11	54	F	I	1.74	1.06
12	80	F	W	1.40	1.40
13	84	F	W	1.63	-
14	89	F	W	1.48	-
15	70	F	W	1.68	1.70
16	70	M	W	1.65	1.60
17	86	F	W	1.68	1.00
18	88	F	W	1.86	-
19	80	M	W	1.68	0.60
20	51	F	B	1.71	-
21	55	F	B	1.80	1.30
22	26	M	B	1.76	-
23	23	F	B	1.62	1.70
24	76	M	W	1.75	-
25	35	F	B	1.68	1.20
26	81	M	W	1.63	-
27	96	M	W	1.70	0.50
28	80	M	W	1.75	-
29	80	M	W	1.77	2.40
30	70	F	B	1.63	0.40

31					1.45
32	50	M	B	167	1.30
33	32	M	B	1.83	0.90
34	35	M	B	1.67	1.00
35	35	M	B	1.71	0.40
36	41	M	B	1.65	1.50
37	57	F	B	1.69	0.75
38	27	M	B	1.67	1.10
39	29	M	B	1.64	0.70
40	20	M	B	1.79	0.90
41	69	F	W	1.63	0.40
42	38	F	B	1.77	0.60
43	33	M	B	1.58	1.45
44	20	M	B	1.67	0.80
45	24	M	B	1.75	0.90
46	36	M	B	1.77	2.20
47	32	F	B	157	-
48	23	M	B	170	1.40
49	28	M	B	160	1.00
50	53	M	B	168	1.10
51	42	M	B	163	0.90
52	25	M	B	175	0.75
53	45	M	B	155	0.75
54					0.40
55	38	M	B	173	1.00
56					0.75
57	48	F	B	1.56	0.40
58	16	F	B	1.57	1.05
59	36	M	B	1.80	1.35
60	38	M	B	1.65	0.85
61					0.60
62	52	F	B	1.57	2.25
63	55	M	B	1.70	0.80
64	30	M	W	1.74	-
65	48	M	B	1.70	1.55

66	59	F	B	1.55	0.90
67	40	M	B	1.72	1.00
68	45	F	W	1.68	1.05
69	35	M	B	1.74	0.55
70	50	M	B	1.70	1.00
71	27	M	B	1.70	-
72	56	M	W	1.78	1.40
73	43	M	B	1.65	0.70
74	23	M	B	1.70	0.85
75	38	M	B	1.73	0.70
76	44	F	W	1.55	0.40
77	56	F	W	1.64	0.60
78	59	F	B	1.60	1.00
79	30	M	B	1.75	1.20
80	60	F	B	1.68	0.50
81	54	M	B	1.78	1.00
82	47	M	W	1.68	0.60
83	44	M	B	1.72	0.55

APPENDIX E5: HISTOPATHOLOGICAL ANALYSIS

	LAD I			LAD II			LAD III		
	1+	2+	3+	1+	2+	3+	1+	2+	3+
1		#							
2			#				#		
3									
4									
5	#								
6	#			#					
7	#						#		
8	#			#					
9	#								
10									
11							#		
12									
13									
14	#							#	
15									

16									
17									
18				#			#		
19	#								
20									
21	#								
22	#								
23	#								
24									
25									
26									
27									
28									
29									

APPENDIX F

Division of Plastic and
Reconstructive Surgery

Department of Surgery
School of Medicine
University of Louisville
Louisville, Kentucky 40292
Office: (502) 852 6880
Fax: (502) 852 8915

UNIVERSITY of LOUISVILLE

May 5, 2003

Nirusha Lachman B Med Sc, M Med Sc

Dear Ms Lachman,

I am writing to confirm that the individuals whose bodies you used for your Ph.D. studies at the University of Louisville had given appropriate consent for this work.

All the bodies used in the Fresh Tissue Dissection Laboratory have been bequeathed to the University of Louisville Body Bequeathal Program. In bequeathing their bodies, individuals do not specify the use that is to be made of their body. They give a more general permission, signing a statement that they give their body to meet the needs of any of the University's officially approved research and educational programs.

You were accepted by the Department of Surgery and the Department of Anatomical Sciences as an external post-doctoral student. Thus your work became an officially approved research program under the terms of the Body Bequeathal Program, and your use of the bodies fully met the expressed wishes of the deceased.

Sincerely,

Robert D. Acland, M.D., F.R.C.S.,
Professor, Department of Surgery

APPENDIX G

MANUFACTURER'S INDEX FOR MICROFIL[R]

CLEAR MICROFIL SILICONE RUBBER INJECTION COMPOUND IS MADE BY FLOW TECH INCORPORATED.

THE PROCUCT IS LISTED AS **# MV-132**

MANUFACTURER'S INDEX FOR FOGARTY[R] CATHETERS

FOGARTY[R] THROUGH LUMEN EMOLECTOMY CATHETERS ARE MADE BY BAXTER HEALTH CARE CORPORATION.

THE PRODUCT IS LISTED AS **# 12TLW404F**

RESEARCH PROFILE

RESEARCH PROFILE

PROFESSIONAL RESEARCH AFFILIATIONS

- Active member of the American Association of Clinical Anatomists
- Active member of the Anatomical Association of Southern Africa

RESEARCH AWARDS

- Received Invitation Fellowship to the University of Louisville, Kentucky, USA, as Fellow of Clinical Anatomy, 1999-2000.
- Received Oppenheimer Travel grant, in support of the above research venture, 1999.
- Received Bob Symington Award from the Anatomical Association of Southern Africa for Excellence in Research, 1998.
- Received grant from American Association of Anatomists in support of Education Research, 2001
- Received University of Durban Westville, Faculty of Health Researcher of the Year, 2001 award.
- Received Technikon Natal award for best oral presentation at Research Day
- Received Technikon Natal award for best poster presentation at Research Day

FULL LENGTH PAPER PUBLICATIONS

- **Lachman N,** Satyapal KS. MORPHOMETRY OF THE INTERNAL THORACIC ARTERIES. Surgical and Radiologic Anatomy [Europe], 20:1, 1998

- **Lachman N,** Satyapal KS. DESCRIPTION AND INCIDENCE OF VARIANT TERMINAL BRANCH OF THE INTERNAL THORACIC ARTERY AND CLINICAL SIGNIFICANCE. Surgical and Radiologic Anatomy [Europe], 21 : 351-354, 1999

- Vanker EA, **Lachman N,** Satyapal KS and Kleinloog R. CLINICAL SIGNIFICANCE OF INTER-ETHNIC DIFFERENCES IN ITA MORPHOMETRY. Cardiovascular Journal of Southern Africa, vol 11:02, April-May, 2000

- **Lachman N**, Satyapal KS, Kalideen JM and Moodley TR. LITHOPEDION: A CASE STUDY. Clinical Anatomy [American], vol 13 : February 2001

- Omar YO, **Lachman N** and Satyapal KS. UNUSUAL ORIGIN OF INTERNAL THORACIC ARTERY: A CASE REPORT. Surgical and Radiologic Anatomy [Europe], 23:127-129, 2001

- **Lachman N**, Acland RD and Rosse C. ANATOMICAL EVIDENCE FOR THE ABSENCE OF A MORPHOLOGICALLY DISTINCT CRANIAL ROOT OF THE ACCESSORY NERVE IN MAN. Clinical Anatomy [American], 2002, Volume 15, No 1, 2002, pp.4-10. *[Cover Article with Editorial Commentary and Acknowledgement]*

- **Lachman N,** Acland RD, Vanker EA, Austin EH, Arronson BA and Satyapal KS. A MORPHOMETRIC STUDY OF THE EXTRA-CORONARY COLLATERALS – PRELIMINARY FINDINGS. Journal of Anatomy [British], 2001, *reviewed and recommended for publication*

REFEREED ABSTRACT PUBLICATIONS

- **Lachman N**, RD Acland, EA Vanker, BA Arronson, EH AUSTIN and KS SATYAPAL A MORPHOMETRIC STUDY OF THE EXTRA-CORONARY COLLATERALS, Clinical Anatomy, Vol 14, no1, pp76 - Publication of the Proceedings of the Joint Meeting of the American and British Associations of Clinical Anatomists, July 19-22, 2000, St John's College, Cambridge, UK.

- **Lachman N**, RD Acland and C Rosse. THE CRANIAL ROOT OF THE ACCESSORY NERVE: AN ANATOMIC MYTH REVISITED. Clinical Anatomy, Vol 14, no1, pp76 - Publication of the Proceedings of the Joint Meeting of the American and British Associations of Clinical Anatomists, July 19-22, 2000, St John's College, Cambridge, UK.

- **Lachman N**, Satyapal KS. AN UNUSUAL VARIANT BRANCH OF THE INTERNAL THORACIC ARTERY. Proceedings of the Anatomical Society of Southern Africa : April 1995 and J Anat. 1996, 188-504.

- **Lachman N**, Satyapal KS. MORPHOMETRIC ANALYSIS OF THE INTERNAL THORACIC ARTERIES. Proceedings of the Anatomical Society of Southern Africa : April 1996 and J Anat. 1997.

- **Lachman N**, Satyapal KS. CLINICAL SIGNIFICANCE OF THE MORPHOMETRY OF THE INTERNAL THORACIC ARTERIES. Proceedings of the European Association of Clinical Anatomists, September 1997.

- **Lachman N**, Satyapal KS. RACIAL DIFFERENCES OF INTERNAL THORACIC ARTERY MORPHOMETRY : SIGNIFICANCE IN CORONARY ARTERY BYPASS GRAFTING. Proceedings of the 15th Annual Scientific Session of the Anatomical Association of Clinical Anatomists, June, 1998.

CONFERENCE PRESENTATIONS: INTERNATIONAL

- ***Lachman etal.***, *Department of Human Biology, Technikon Natal.* CLINICAL SIGNIFICANCE OF THE INTERNAL THORACIC ARTERIES. **4th Congress of the European Association of Clinical Anatomists, Lille, France, September, 1997.** *[podium]*

- ***Lachman etal.***, *Department of Human Biology, Technikon Natal* DESCRIPTION AND INCIDENCE OF VARIANT TERMINAL BRANCH OF THE INTERNAL THORACIC ARTERIES AND CLINICAL SIGNIFICANCE. **4th Congress of the European Association of Clinical Anatomists, Lille, France, September, 1997.** *[poster]*

- ***Lachman etal.***, *Department of Human Biology, Technikon Natal* RACIAL DIFFERENCES OF INTERNAL THORACIC ARTERY MORPHOMETRY : SIGNIFICANCE IN CORONARY ARTERY BYPASS GRAFTING. **15th Annual Scientific Session of the American Association of Clinical Anatomists, Lexington, Kentucky, June 1998.** *[podium]*

- ***Lachman etal.***, *Department of Human Biology, Technikon Natal* MORPHOMETRIC STUDY OF THE EXTRA-CORONARY COLLATERALS – PRELIMINARY FINDINGS. **Tripartite Meeting of the Anatomical Society of Great Britain and Ireland, Cambridge, UK, July, 2000.** *[podium]*

- ***Lachman etal.***, *Department of Human Biology, Technikon Natal* THE ELEVENTH CRANIAL NERVE – AN ANATOMICAL MYTH REVISITED. **Joint Meeting of the American and British Associations of Clinical Anatomists, Cambridge, UK, July 2000.** *[podium]*

- ***Lachman etal.***, *Department of Human Biology, Technikon Natal* THORACIC OUTLET SYNDROME: ARTERIAL COMPRESSION. **Tripartite Meeting of the Anatomical Society of Great Britain and Ireland, Cambridge, UK, July, 2000.** *[poster]*

- ***Lachman etal.***, *Department of Human Biology, Technikon Natal* THE INTRAMURAL LEFT ANTERIOR DESCENDING CORONARY ARTERY. **International Symposium for Morphological Sciences, July, 2001, South Africa.** *[poster]*

- ***Lachman etal.***, *Department of Human Biology, Technikon Natal* THE USE OF LEAD-OXIDE FOR DEMONSTRATION OF EXTRA-CARDIAC COLLATERAL VESSELS IN POST-MORTEM ANGIOGRAPHY. **International Symposium for Morphological Sciences, July, 2001, South Africa.** *[poster]*

- ***Lachman etal.***, *Department of Human Biology, Technikon Natal* CAST PREPARATION OF ABDOMINAL VISCERA USING POLYESTER CYSTIC CASTING RESIN. **International Symposium for Morphological Sciences, July, 2001, South Africa.** *[poster]*

- *Lachman et al., Department of Human Biology, Technikon Natal* : ANGIOGRAPHIC MANIFESTATION OF INTRAMURAL CORONARY ARTERIES. **Indian Association of Anatomists, Manipal, India, 2001** *[podium]*

- *Lachman et al., Department of Human Biology, Durban Institute of Technology:* DIAGNOSTIC INDICATOR FOR THE PRESENCE OF THE INTRA-MURAL LAD. **American Association of Clinical Anatomists, Gainesville, Florida, USA, 2002** [podium]

CONFERENCE PRESENTATIONS: NATIONAL

- ***Lachman etal.**, Department of Human Biology, Technikon Natal.* MORPHOMETRIC ANALYSIS OF THE INTERNAL THORACIC ARTERIES. 26th Congress of the Anatomical Association of Southern Africa, university of Witwatersrand, 1997.

- ***Lachman etal.**, Department of Human Biology, Technikon Natal.* MORPHOMETRY OF THE INTERNAL THORACIC ARTERIES. 27th Congress of the Anatomical Association of Southern Africa, University of Cape Town, 1997. *This presentation was awarded the Bob Symington Award for research excellence.*

- ***Lachman etal.**, Department of Human Biology, Technikon Natal.* RACIAL DIFFERENCES OF INTERNAL THORACIC ARTERY MORPHOMETRY : SIGNIFICANCE IN CORONARY ARTERY BYPASS GRAFTING. University of Maputo, Mozambique, 1998.

- ***Lachman etal.**, Department of Human Biology, Technikon Natal.* LITHOPEDION : A CASE STUDY. 29th Congress of the Anatomical Association of Southern Africa, Eastern Transvaal, April, 1999.

- ***Lachman etal.**, Department of Human Biology, Technikon Natal.* SEX DIFFERENCES IN ITA MORPHOMETRY. 29th Congress of the Anatomical Association of Southern Africa, Eastern Transvaal, April, 1999.

- ***Lachman etal.**, Department of Human Biology, Technikon Natal* MORPHOMETRIC STUDY OF THE EXTRA-CORONARY COLLATERALS – PRELIMINARY FINDINGS. 30th Congress of the Anatomical Association of Southern Africa, Pretoria, April, 2000.

- ***Lachman etal.**, Department of Human Biology, Technikon Natal* THORACIC OUTLET SYNDROME: ARTERIAL COMPRESSION. 30th Congress of the Anatomical Association of Southern Africa, Pretoria, April, 2000.

- ***Lachman etal.**, Department of Human Biology, Technikon Natal.* CLINICAL SIGNIFICANCE OF ECC'S – PRELIMINARY FINDINGS. Surgical Society of Southern Africa, July, 2001

- ***Lachman etal.**, Department of Human Biology, Durban Institute of Technology.* THE CLINICAL SIGNIFICANCE OF CORONARY ARTERY VARIATIONS. Anatomical Society of Southern Africa, Durban, SA, April 2002.

- ***Lachman etal.**, Department of Human Biology, Durban Institute of Technology.* SHOULD CRANIAL NERVES BE RECLASSIFIED? Anatomical Society of Southern Africa, Durban, SA, April 2002.

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- ***Lachman etal.**, Department of Human Biology, Durban Institute of Technology.* LUNG PLASTINATION. Anatomical Society of Southern Africa, Durban, SA, April 2002.

- ***Lachman etal.**, Department of Human Biology, Durban Institute of Technology.* BREAST NECROSIS FOLLOWING INTERNAL THORACIC ARTERY HARVEST. Anatomical Society of Southern Africa, Durban, SA, April 2002.