OXFORD

CORE CONCEPTS IN CARDIAC SURGERY

Edited by

David Taggart and **Yasir Abu-Omar**

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OXFORD
UNIVERSITY PRESS

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Great Clarendon Street, Oxford, OX2 6DP,
United Kingdom

Oxford University Press is a department of the University of Oxford.
It furthers the University's objective of excellence in research, scholarship,
and education by publishing worldwide. Oxford is a registered trade mark of
Oxford University Press in the UK and in certain other countries

First Edition published in 2018

Impression: 1

Published in the United States of America by Oxford University Press
198 Madison Avenue, New York, NY 10016, United States of America

British Library Cataloguing in Publication Data

Data available

Library of Congress Control Number: 2018941854

ISBN 978–0–19–873546–5

Printed and bound by
CPI Group (UK) Ltd, Croydon, CR0 4YY

Contents

List of Abbreviations

ACAB	atraumatic coronary artery bypass
ACE	angiotensin-converting-enzyme
ACT	activated clotting time
AF	atrial fibrillation
AHA	American Heart Association
AI	aortic insufficiency
AR	aortic regurgitation
AS	aortic valve stenosis
AV	aortic valve
AVJ	aorto-ventricular junction
AVN	atrioventricular node
AVR	aortic valve replacement
BAV	bicuspid aortic valve
BH-TECAB	beating heart total endoscopic coronary bypass grafting
BIMA	bilateral internal mammary artery
BITA	bilateral internal thoracic artery
BMI	body mass index
BOS	bronchiolitis obliterans syndrome
CABG	coronary artery bypass graft
CAD	coronary artery disease
CF	continuous flow
CP	chordal procedure
CPB	cardiopulmonary bypass
CPR	cardiopulmonary resuscitation
CT	computed tomography
DAPT	dual antiplatelet therapy
DCD	donation after cardiac death
DDAVP	desmopressin acetate
DES	drug-eluting stent
DHCA	deep hypothermic circulatory arrest
DLCO	diffusing capacity of the lungs for carbon monoxide
DT	destination therapy
EACTS	European Association for Cardio-Thoracic Surgery
ECGI	electrocardiographic imaging
ECMO	extracorporeal membrane oxygenation
EDHF	endothelium-derived hyperpolarizing factor
ERP	effective refractory period
FAA	functional aortic annulus
FDA	Food and Drug Administration
FFR	fractional flow reserve
GEA	gastro-epiploic artery
HF	heart failure
HIFU	high-intensity focused ultrasound
IABP	intra-aortic balloon pump
ICU	intensive care unit
IEA	inferior epigastric artery
IEOA	indexed effective orifice area
IMA	internal mammary artery
IRAD	International Registry of Acute Aortic Dissection
ISDN	isosorbide dinitrate
ITA	internal thoracic artery
IVC	inferior vena cava
LA	left atrium
LAA	left atrial appendage
LAD	left anterior descending artery
LCC	left coronary cusp
LHB	left-sided heart bypass
LIMA	left internal mammary artery
LITA	left internal thoracic artery
LRA	leaflet resection with an annuloplasty
LV	left ventricular
LVAD	left ventricular assist devices
LVEDP	left ventricular end diastolic pressure
LVEF	left ventricular ejection fraction
MACCE	major adverse cardiac and cerebrovascular events
MCS	mechanical circulatory support
MI	myocardial infarction
MIDCAB	minimally invasive direct coronary bypass

MIMVS	minimally invasive mitral valve surgery
MLD	minimal lumen diameter
MR	mitral regurgitation
MRA	magnetic resonance angiography
MV	mitral valve
MVST	multivessel small thoracotomy
NC	non-coronary
NCC	non-compaction cardiomyopathy
NIRS	near-infrared spectroscopy
NYHA	New York Heart Association
ONCAB	on-pump coronary artery bypass
OPCAB	off-pump coronary artery bypass
OR	operative room
PA	pulmonary artery
PAU	penetrating atherosclerotic ulcer
PCI	percutaneous coronary intervention
PDA	patent ductus arteriosus
PGD	primary graft dysfunction
PLA	posterolateral artery
PPM	patient–prosthesis mismatch
PTCA	percutaneous transluminal coronary angioplasty
PTFE	polytetrafluoroethylene
PTLD	post-transplantation lymphoproliferative disease
PV	pulmonary vein
PVI	pulmonary vein isolation
PVR	pulmonary vascular resistance
QoL	quality of life
RA	radial artery
RAA	right atrial appendage
RCA	right coronary artery
RCC	right coronary cusp
RCT	randomized controlled trial
RFA	radiofrequency ablation
RGEA	right gastroepiploic artery
RIMA	right internal mammary artery
RITA	right internal thoracic artery
RV	right ventricular
RVAD	right ventricular assist device
SAM	systolic anterior motion
SAN	sinoatrial node
SAVR	surgical aortic valve replacement
SCA	sudden cardiac arrest
STJ	sinotubular junction
STS	Society of Thoracic Surgeons
SV	stroke volume
SVC	superior vena cava
SVG	saphenous vein graft
SVST	single-vessel small thoracotomy direct-vision bypass grafting
TAAA	thoracoabdominal aortic aneurysm
TAVI	transcatheter aortic valve implantation
TECAB	totally endoscopic coronary artery bypass
TEE	transesophageal echocardiography
VAD	ventricular assist device
VAJ	ventriculo-aortic junction
VAS	ventricular assist system

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Chapter 1

CABG conduits and graft configuration

David Glineur

Coronary artery bypass graft (CABG) conduits

Left internal thoracic artery (LITA)

Anatomy

The internal thoracic artery (ITA) supplies the anterior chest wall; it arises from the subclavian artery near its origin and travels downward inside the chest wall, approximately one centimeter from the edges of the sternum. It runs posterior to the internal intercostal muscles, but anterior to the transverse muscles. The ITA divides into the musculophrenic artery and the superior epigastric artery around the sixth intercostal space.

Histology

The major characteristic of the LITA is the presence of more elastic laminae compared to gastro-epiploic artery (GEA), inferior epigastric artery (IEA), or radial artery (RA), which contain more smooth muscle cells in their walls and are therefore less elastic.[1]

Intima Both connective tissue and smooth muscle are present in the intima, the border of which is delineated by the internal elastic membrane. The internal elastic membrane may not be conspicuous due to the abundance of elastic material in the tunica media.

Media This is the thickest of the three layers. Smooth muscle cells are arranged in a spiral around the long axis of the vessel, and secrete elastin in the form of lamellae, which are fenestrated to facilitate diffusion. These lamellae, and the large size of the media, are the most striking histological features of elastic arteries. In addition to elastin, the smooth muscle cells of the media secrete reticular and fine collagen fibers and proteoglycans. No fibroblasts are present.

Adventitia This is a relatively thin connective tissue layer. Fibroblasts are the predominant cell type, and many macrophages are also present. Collagen fibers predominate and elastic fibers (not lamellae) are also present. The collagen in the adventitia prevents elastic arteries from stretching beyond their physiological limits during systole. Blood vessels supplying the adventitia and outer media, known as the vasa vasorum, are also present.

Endothelium function

Endothelium acts as an antithrombotic barrier as well as a modulator of vascular tone and growth. For these reasons, it is believed to be the milestone of the graft long-term patency. In response to a variety of agonists, endothelial cells generate three major autacoids that regulate vascular relaxation and other endothelium-dependent vascular functions[2]: nitric oxide (NO), prostacyclin (PGI2), and endothelium-derived hyperpolarizing factor (EDHF).

Lüscher and colleagues[3] studied endothelium-dependent relaxation in internal mammary arteries, internal mammary veins, and saphenous veins. Vascular rings with and without endothelium were suspended in organ chambers, and isometric tension was recorded. Acetylcholine, thrombin, and adenosine diphosphate evoked potent endothelium-dependent relaxation in the mammary artery but weak responses in the saphenous vein. In the mammary artery, relaxation was greatest in response to acetylcholine, followed by thrombin and adenosine diphosphate. In the saphenous and mammary veins, relaxation was less than 25%. Relaxation was unaffected by indomethacin but was inhibited by methylene blue and hemoglobin, suggesting that endothelium-derived relaxing factor was the mediator. Endothelium-independent relaxation in response to sodium nitroprusside was similar in arteries and veins. Lüscher concluded that endothelium-dependent relaxation was greater in the mammary artery than in the saphenous vein.

The specificity of the ITA explains why it is less damaged by arteriosclerosis compared to other arteries, a phenomenon that has been studied with an ultrasonic system.[4] This study revealed that the intima-media complex of the ITA is protected from the influence of arteriosclerosis, in comparison with the morphological changes found in the intima-media thickness of the common carotid artery. This demonstrated protective mechanism underlines the widespread use of the ITA as a CABG conduit.

In addition, β-adrenoceptor agonists do not induce a significant relaxation of the ITA, and the use of β-adrenoceptor antagonists do not lead to IMA vasospasm.[5]

Patency

Long-term LITA to the left anterior descending artery (LAD) patency is usually greater than 90% (range 83–98% in historical studies)[6–27] (Table 1.1). Factors known to potentially influence patency include the degree of preoperative proximal coronary stenosis, the time from CABG in non-LAD arteries, sex, date of surgery, target other than LAD, and smoking status.

Degree of coronary stenosis ITA graft patency decreased as proximal coronary stenosis decreased.[28] These findings are consistent with the physiology of arterial grafts. ITAs are able to autoregulate size and blood flow in response to demand. As proximal coronary stenosis decreases, competitive flow increases, and demand for ITA graft flow falls. This cascade of events results in ITA constriction and, over time, increased risk of atrophy and occlusion.[29–32]

Kawasuji and colleagues[33] performed angiography one month after CABG in 100 patients with ITA to LAD grafts; all grafts were patent, but 15% (2/13) of those performed to coronary arteries with 50% or less stenosis were severely constricted. Seki and colleagues[30] observed either severe constriction or occlusion in 9.5% (14/147) of ITA grafts

Table 1.1 Summary of different studies on LITA patency

First author	Year	Studied/ Operated	Percent studied	Interval	Graft patency (%)
Green[6]	1972	70/165	42	2 wk–3 y	97
Kay[7]	1974	91/628	14	19.5 mo	98
Barner[8]	1976	139/307	45	20 days	95
		139/307	45	13 mo	90
Tector[9]	1976	43/275	15	9–24 mo	95
Geha[10]	1979	175/208	82	2 wk	99
		?/208	49	6 mo–5 y	97
Tyras[11]	1980	527/765	69	1 mo	95
		?/765	65	1 y	93
		?/765	63	5 y	90
Lytle[12]	1980	46/100	46	20 mo	91
Tector[13]	1981	88/298	29	60–108 mo	94.40
Singh[14]	1983	34/	NA	3–12 y	94
Grondin[15]	1984	37/40	92	1 mo	97
		32/40	80	1 y	88
		20/40	50	10 y	84
Okies[16]	1984	259/4183	6	5 y, 10 y	83, 70
Lytle[17]	1985	140/?	NA	5 y	97
Loop[18]	1986	855/2306	37	8.7 y	96
Zeff[19]	1988	37/39	92	8.9 y	95
Ivert[20]	1988	91/99	92	2 wk	94
		84/99	85	1 y	90
		66/99	67	5y	89
Goldman[21]	1990	237/670	23	1 y	93
Fiore[22]	1990	182/200	91	13 y	82
Galbut[23]	1990	53/947	6	2 mo–15 y	92
Boylan[24]	1994	57/100	57	<10 y, >10 y	93, 90
Goldman[25]	1994	167/1,031	25	3 y	90
FitzGibbon[26]	1996	456/476	96	6 mo	95
		123/476	26	5 y	80
Gills[27]	1997	25/25	100	4–6 h	96

studied 16 days to 62 months after CABG. Two (14%) of these failed ITA grafts bypassed LADs with more than 50% stenosis, whereas 12 (86%) bypassed LADs with 50% or less proximal stenosis.

Target ITA patency is the most durable of grafts performed to the LAD, possibly because of the ease of anterior coronary arteries grafting, but also because the amount of myocardium supplied by the LAD is greater than that supplied by other coronary arteries, resulting in a larger blood flow demand. ITA grafts with greater blood flow demand are less likely to fail.[34] In contrast, Glineur *et al.*[35] could not find any significantly difference at 6 months between the right internal thoracic artery (RITA) directed to the lateral wall of the heart versus the LITA to the LAD territory (Table 1.2).

Gender Because of their smaller size, women have smaller coronary arteries than men. Technical difficulties associated with grafting small arteries are one possible cause of the higher operative risk observed in women, and may also be responsible for lower graft patency.[28]

Risk factors Smoking is strongly associated with progression of coronary artery disease (CAD), and patients who continue to smoke after CABG have a higher risk of return of angina, myocardial infarction, and coronary reintervention.[28] In addition, multivariable analysis revealed that a history of smoking decreased ITA graft diameter.[6] These effects on both coronary arteries and ITA grafts probably account for the lower graft patency we observed in smokers.[36]

Right internal thoracic artery (RITA)

Anatomy, histology, endothelium function

There are no significant differences between the left ITA and right ITA in terms of anatomy, histology, and endothelial function.[37] There were also no statistical differences between LITA and RITA concerning mean intimal diameter (1.52 ± 0.24 *vs.* 1.58 ± 0.28 mm, P <0.06), medial diameter (2.21 ± 0.27 *vs.* 2.52 ± 0.28 mm, P <0.15), or wall thickness (0.39 ± 0.12 *vs.* 0.41 ± 0.16 mm, P <0.47). The intimal diameters diminished significantly from the origins (1.69 ± 0.34 and 1.86 ± 0.41 mm, respectively) to the terminations (1.25 ± 0.26 and 1.14 ± 0.25 mm, respectively) of both vessels.

In order to determine whether endothelial function differs between left and right ITA segments in a Y-graft configuration, Glineur *et al.* studied 11 patients 3 years after surgery.[38] The endothelium-dependent vasodilator substance P was selectively infused (1.4 up to 22.4 pmol/min in doubling dose increments) in the ostium of ITA Y-grafts. A maximal endothelium-independent vasodilatory response was then obtained by intragraft infusion of 2 mg isosorbide dinitrate (ISDN).

A similar dose-dependent vasodilatory response to substance P was observed in the left and in the right ITA (Figure 1.1). No difference in maximal endothelium-dependent response to substance P (7.4 ± 4.3% in left ITA and 8.1 ± 5.3% in right ITA) or in maximal endothelium-independent response to ISDN (12.2 ± 4.4% in left ITA and 10.6 ± 8.1% in right ITA) was observed. The endothelium-dependent and the endothelium-independent