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Collateral Circulation of the Heart

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Dedicated to Ladina, Luzi, Fia Rageth, Men Maria, my children and to Christina

Preface

The sober explanation for this book is a call by the Springer-Verlag, London, to edit a publication on 'The functional relevance of the collateral circulation' of the heart. Alternatively, it could be 'sold' as the result of my intention to reduce entropy of 18 years of scientific work on the topic of the coronary circulation, which was itself meant to diminish the amount of 'useless' energy. Such a process of reducing disarray in a system with the aim of grasping it better is related to simplification, which carries the risk of introducing error. This can be exemplified by the historic view of angina pectoris, which used to be simplified as being always fatal, thus obscuring for nearly two centuries the view of a 'self-healing' mechanism such as the collateral circulation of the heart. It would be naïve, to assume the present work to be free of erroneous oversimplification, because the very nature of scientific work is related to generating (simple) hypotheses with their subsequent falsification.

In that context and bluntly, my primary interest in the field of the collateral circulation was not initiated with a vision of eradicating the consequences of coronary artery disease (CAD) by promoting the growth of natural bypasses. The time for such sizeable ideas had passed in the 1970s with the start of the work by Wolfgang Schaper. My interest in the area related to maps, geometry charting landscape as a former cartographer and the linked process of minimizing error in doing so. At first sight, landscape is not organized and charting it realistically requires techniques of projecting it on a flat plane, while preserving distances, angles, object size relations. Apparently, biology in the sense of mathematical science is poorly organized. However, patterns of organization can be recognized, and this is even the case in ostensibly chaotic systems. In hindsight, my genuine interest in the field evolved from describing biological patterns with exact means, always including the calculation of the error made during mathematical modelling. Accordingly, one of my favourite occupation used to consist of modelling the coronary artery circulation, the model being a minimal cost function of energy expenditure for the transport of blood, whereby large 'tubes' dissipate little frictional energy but their construction and maintenance is costly because large, and vice versa. The 'maps' of coronary angiograms were crucial for delineating the local territory of 'irrigation', i.e. the so called ischaemic area at risk for infarction. The

oversimplification and, thus, error of the system consisted of demarcating different coronary regions, disregarding the possibility of links between them and the assumption that there are no intercoronary anastomoses.

The step between acknowledging inter-coronary anastomoses, links, natural bypasses, collaterals in the human coronary circulation and the structure of this book is small. The following questions, i.e. principle book chapters, arise instantaneously: are they relevant in the sense of life-saving for patients with CAD (Chapter 1); how can they be gauged (Chapter 2); how often are they present and how are they promoted *naturally* (Chapter 3); if present, how do they function physically (Chapter 4), and can they be promoted *artificially* (Chapter 5)? The epigrammatic answers to some of the above questions according to the actual state of (mis)calculation are: yes, they are relevant in every third patient with CAD and in every fourth without CAD; they can be measured invasively; they are able to dilate and constrict and do not function as rigid tubes; and yes, they can be promoted artificially. The demarcation between the main chapters is not absolute, i.e. there are 'anastomoses' among them for the purpose of allowing to read single chapters, and of reflecting the reality of multiple associations between the sub-topics. The potential 'error' of using the design of 'permeable' book chapters is the risk of redundancy, which on one hand, is an essential element of didactics. Conversely, it is anaesthetizing when applied in an overdose. Single- as compared to multi-authorship should reduce rather than amplify the risk of redundancy.

That human coronary collateral vessels are relevant *quo ad vitam* and that they are inducible artificially renders the subject of *collaterology*, which is aimed to be covered by this book, important from a medical standpoint of view. This is even more so considering the epidemiologic and economic burden of CAD and the fact that every sixth to fifth patient suffering from CAD cannot be treated sufficiently by conventional means. *Arteriogenesis*, the promotion of collateral artery growth employed in collaterology is on its way to be a treatment pillar of CAD, but the path is not as straightforward as thought before the first controlled clinical angiogenesis trials. With its winding pattern, it resembles the corkscrew shape of collateral vessels. This hallmark of vascular enlargement is caused by the fact that growth of the vessel is not restricted to one direction (cross-sectional calibre), but is ubiquitous, i.e. also lengthwise. In the absence of cardiac enlargement, an increase in vessel length translates into a meandering route. The latter evokes, again, the way of how scientific work advances with a generated hypothesis pointing into one direction, its (often occurring) falsification, which leads to a temporary retreat, meaning a change of the motion vector.

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Contents

Chapter 1
Relevance of the Human Coronary Collateral Circulation

1.1 Historical Aspects

The symptom of angina pectoris surfaced only in the late 18th century and became more prevalent even 150 years later. For a long time, the view prevailed that angina pectoris was almost always fatal. Conversely, developing tolerance to exercise-induced angina pectoris which could even 'cure' it was described by William Heberden. Structural channels connecting the right and left coronary arteries were first described by Richard Lower of Amsterdam in 1669. In 1757, the Swiss anatomist Albrecht von Haller also demonstrated anastomoses between coronary arteries. The first anatomic observations of anastomoses were possibly made in non-obstructed coronary arteries, because coronary artery disease (CAD) was much less prevalent than today. Using post-mortem imaging of the coronary circulation by a multitude of different techniques, a controversy on the existence of structural intercoronary anastomoses ensued, which was not settled in their favour before the first half of the 20th century in case of the presence of CAD and not before the early 1960s in case of the normal human coronary circulation by William Fulton. Since James Herrik's clinicopathologic observation in 1912 of the possibility of surviving sudden thrombotic coronary obstruction in the presence of intercoronary anastomoses, it has been recognized that the collateral circulation is a very important determinant of the rate and extent of myocardial cell death within an ischaemic zone. However, actual in vivo functional coronary collateral measurements during cardiac surgery and percutaneous coronary intervention (PCI) were first performed only in the 1970s and early 1980s respectively. The existence of in vivo functional collaterals in the absence of CAD was not been proven before 2003.

1.1.1 Introduction

In 1983, Proudfit pointed out that ischaemic heart disease could be justly called 'British disease' because of the origin of its pathophysiologic concept being based on William Heberden's description of angina pectoris in 1772, but more

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importantly on pathoanatomic observations and their accurate clinical interpretation by Edward Jenner (1749–1823; developer of the smallpox vaccine), Caleb Hillier Parry (1755–1822), Samuel Black (1764–1832) and Allan Burns (1781–1844).[1] This 'British team', working over 23 years, established the myocardial ischaemic theory of angina pectoris and clarified the origin of a very important disease in industrialized countries, but it was only 120 years later that leaders of the medical profession generally accepted the concept.[1] In parentheses, the question may be raised, why the symptom of angina pectoris was not described in detail before 1772. This could be due to previous absence or low prevalence of angina or to the lack of clinical insight to recognize it. It appears unlikely that clinical inability to detect angina could account for its late manifestation in the literature, and Michaels interpreted probably correctly that this symptom and, thus, also CAD 'surfaced' only in the late 18th century and became substantially more prevalent even 150 years later as a result of changing lifestyle[2] In the context of the pioneering work of the above 18th-century 'British quartet', it is speculative to state that CAD is not called 'British disease' because one aspect of CAD, i.e. the development of arterial detours around atherosclerotic obstructions or the growth of natural bypasses, was not built into the theory of CAD. Quite on the contrary, Parry's observations on angina pectoris and those of Jenner on the same condition focusing on John Hunter's case (see below) launched the view that this condition was almost always suddenly fatal.[3] Thus, while establishing a pivotal part of the concept of ischaemic heart disease, Jenner, Parry, Black and Burns unwillingly contributed at the same time to the expansion of the more than 300-year-controversy on the relevance of the human coronary collateral circulation since its first anatomic description by Richard Lower of Amsterdam in 1669.[4] This interpretation of the history of coronary collateral vessels cannot be applied to Heberden, because he reportedly did not make the link between angina pectoris and coronary obstruction, but attributed it in individual cases to an unknown cause or to mediastinal abscess or to 'contraction of the arch of the aorta or the arteries that go to the arm'.[1] Conversely, Heberden's contribution to the discovery of the coronary collateral circulation is rated very high as being the first describing the phenomenon of developing tolerance to exercise-induced angina pectoris, i.e. the occurrence of 'walking through' angina.[5] Hence, contemporaries and 'compatriots' in the second half of the 18th century simultaneously hold the view that angina pectoris was almost always deadly and could be well tolerated.

In this context, several questions related to the discovery and historical description of the human coronary collateral circulation emerge, which are subsequently detailed. What were the instruments for the detection of coronary anastomoses? How and when were the clinical features of CAD related to its collateral circulation? How and when was the collateral circulation in human *peripheral* artery disease discovered? What role did post-mortem detection of coronary anastomoses play as compared to in vivo assessment with regard to the debate on the relevance of collaterals? How was the term 'relevance'

interpreted, i.e. as *structural* presence of coronary anastomoses or as the myocardial salvaging *function* of structural collaterals? In which context was the term 'relevance' meant, in the presence of obstructive CAD or in normal hearts? How did early animal experiments on the collateral circulation contribute to respective findings in humans? Considering the multitude of these facets alone, it is not unexpected that controversy on the relevance of the human coronary collateral circulation had to evolve in the past.

1.1.2 First Observations of Collateral Vessels

Channels connecting the right and left coronary arteries were first described by Richard Lower of Amsterdam in 1669 (Table 1.1).[4] He detected the anastomoses by introducing fluid into one coronary artery post-mortem and observing its arrival in the other.[4,6] However, Prinzmetal et al.[7] suggested Thebesius to be the first who revealed by dissection the occurrence of anastomoses between both coronary arteries,[8] whereby the respective anatomic preparations had probably not been performed in normal hearts.[7] In 1757, the Swiss anatomist Albrecht von Haller (1708–1777; Fig. 1.1) also demonstrated anastomoses of the coronary arteries, whereby he described different origins and routes of coronary arterial detours, such as those going via the pulmonary artery root to the sulcus longitudinalis posterior of the right ventricle, those being located near the ventricular apex or close to the atria as well as extracardiac collaterals.[9] While the technique employed during these early observations of collateral vessels of the human heart was mostly that of mechanical dissection, it remains obscure whether normal or diseased hearts were examined. Considering the possibility that CAD was much less prevalent before the mid-18th century than thereafter,[2] the first anatomic observations of anastomoses were possibly made in non-obstructed coronary arteries.

Before the debate on the existence of structural collaterals started in the mid-19th century, William Heberden (1710–1801, Fig. 1.2) in 1772 systematically and meticulously described his clinical observations in 'nearly a hundred people' (three women and one boy, aged 12), who suffered from a disorder, '(T)he seat of it, and sense of strangling, and anxiety with which it is attended, may make it not improperly be called angina pectoris'.[5] Of particular importance in an entirely opposite sense for the further course of the history of the human coronary collateral circulation are two pieces of the text: (a) 'The termination of the angina pectoris is remarkable. For if no accident intervene, but the disease go on to its height, the patients all suddenly fall down, and perish almost immediately'. (b) 'With respect to the treatment of this complaint, I have little or nothing to advance: nor indeed is it to be expected we should have made much progress in the cure of a disease, which has hitherto hardly had a place, or a name in medical books. . . .I know one who set himself a task of sawing wood for half an hour every day, and was nearly cured. In one also, the disorder