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بسم الله الرحمن الرحيم

شبكة المعلومات الجامعية

التوثيق الالكتروني والميكروفيلم

شبكة المعلومات الجامعية

جامعة عين شمس

التوثيق الالكتروني والميكروفيلم

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بالرسالة صفحات لم ترد بالاصل

IMPACT OF RECRUITABLE COLLATERAL BLOOD FLOW ON CORONARY INTERVENTION.

Thesis
Submitted to the faculty of Medicine
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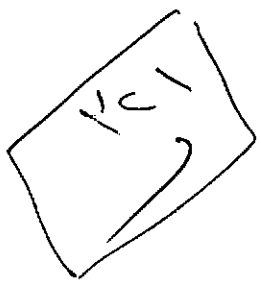

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INTRODUCTION

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It is well known in clinical practice that a coronary artery may become gradually or even suddenly occluded without causing myocardial infarction or even left ventricular dysfunction. Therefore in a number of patients collateral flow must be considerable or at least sufficient to meet the metabolic demands of the myocardium at rest [1,2].

Among the numerous risk factors for restenosis after successful percutaneous transluminal coronary angioplasty (PTCA); sever stenosis before angioplasty [3,4]. The presence of high grade stenosis before angioplasty of the infarct related coronary artery is associated with the presence of collateral circulation to the area perfused by the completely obstructed artery [5].

Although the importance of the collateral circulation of the heart has been recognised for decades, no methods are currently available for quantitative assessment of coronary collateral blood flow in conscious humans [6,7].

Despite the rapid developments of techniques to measure coronary artery blood flow in the clinical catheterization laboratory, the contribution of collateral blood flow to the total myocardial perfusion can not be measured separately, and the collateral blood flow is therefore assessed only qualitatively, indirectly or simply neglected [8,9,10].

Experimental studies have shown that collateral perfusion in the distribution of a totally occluded artery can vary widely and can be as high as 60-80% of normal maximal perfusion [11,12].

Recent experimental studies have indicated that recruitable blood flow at coronary artery occlusion can be expressed as a fraction of normal maximal myocardial blood flow by simultaneous recordings

of mean arterial , coronary wedge and central venous pressures respectively [11,12].

It was hypothesized that if fractional collateral flow exceeds 25% then collateral flow will be sufficient to prevent ischaemia at rest even during coronary occlusion.

REVIEW OF LITERATURE

THE CORONARY TREE

The arterial system:

The inlet of the coronary arterial system consists of two major vessels, which are the first to branch from the aorta. In the dog, the right ventricle is perfused by the right coronary artery while the left coronary artery perfuses the interventricular septum and the free wall of the left ventricle. Yet, in humans, the inferior part of the septum and the left ventricular free wall are perfused by the right coronary artery in approximately 80% of cases (right dominance). The arterial tree consists of large arteries ranging from several mm to 400 μm and branching like a tree into small arteries and arterioles (with a diameter < 400 μm). The arterioles are the smallest arterial conduits surrounded by smooth muscle cells. Although the distinction between large and smaller arteries is somewhat arbitrary, it corresponds to physiological and clinical difference [1,3].

Large arteries include the left main stem, the right coronary artery, the left anterior descending artery, the left circumflex artery, and their diagonal, marginal, and septal branches. The latter penetrate straight into the septum and are completely surrounded by myocardial tissue. All other large arteries run over the epicardial surface of the heart. Therefore, these large arteries will be referred to as epicardial arteries. Epicardial arteries follow a regular, dichotomous branching system. The edges of the vascular lumen of these vessels are sharply delineated at coronary angiography.

These epicardial vessels can be the site of atherosclerotic narrowing which is the main cause of exercise-induced myocardial ischaemia and may lead to thrombotic occlusion and myocardial infarction. The large coronary arteries are the only part of the coronary circulation amenable to mechanical revascularization [13].

Normal epicardial coronary arteries do not create any significant resistance to blood flow. Even at high flow rates induced by intravenous infusion of adenosine, only a negligible pressure difference exists between central aorta and the most distal part of angiographically smooth epicardial coronary arteries in humans [14]. The epicardial coronary arteries, therefore, are also called conduit or conductance vessels. The absence of a pressure drop along a normal epicardial coronary artery during maximal vasodilatation is an essential prerequisite for the concept of pressure-derived fractional flow reserve [15].

The arteries of less than 400 μm will be referred to in this review of literatures as resistive vessels. At coronary angiography they are not clearly delineated but appear as a myocardial blush of contrast medium. Resistive vessels are able to vasodilate under physiological and pharmacological stress. This modulation of resistance is paramount for matching myocardial blood flow to variable energy requirements. The ability of coronary resistive vessels to vasodilate may be impaired before the development of angiographically visible atherosclerosis [16, 17,18]. Often, the vasodilatory reserve is also abnormal in angiographically normal arteries in patients with remote coronary artery disease [19].

The classical two-compartments model of the coronary circulation (conduit and resistive vessels) does not account for the heterogeneity in the distribution of coronary resistance with respect to regulation of coronary blood flow [20]. Based on sequential differences in the response to shear stress and adenosine, resistive vessels in the human coronary circulation have been divided into two groups:

The proximal compartment consists of the pre-arteriolar vessels with a diameter ranging from 100-400 μm. Their tone is controlled by coronary flow, distending pressure and myogenic tone, and modulated by the autonomic nervous system and endothelial function [13].

The distal compartment consists of arterioles with a diameter of less than 100 μm. They are influenced primarily by the perfusion pressure at the origin of the vessel and by myocardial metabolism [13].

Capillaries:

The capillary bed does not have a branching structure but consists of a network of interconnected vessels of similar diameter. The myocytes are organized within the capillary network and are connected to the capillary wall by collagen struts [21]. The diameter of the capillaries is approximately 5 μm [22,23,24] However, the capillary bed is distensible and will be influenced by its inner pressure and by the contractile state of the adjacent myocytes [24]. The capillary density reported in the human heart approximates 3500/mm^2 and appears to be lower in the subendocardium than in the subepicardium [24,25,26,27]. The distance

between two capillaries averages 17 μm. The normal diameter of a myocyte is 18 μm and increases up to 30 μm with hypertrophy. Each myocyte is lined by at least one capillary. This close anatomic structure suggests an intense functional link [13].

Collaterals

Collaterals are vessels structured as a connecting network between different coronary arteries. Their function is to bypass obstructions in arterial branches and thus, to protect myocardial tissue that otherwise would become ischemic. Unlike a generally accepted impression that collaterals were the privilege of only a happy few, it is now well recognized that rudimentary connecting vessels are always present at birth. They are probably remenants of the embryonic arterial network and may develop progressively under the influence of various stimuli [13].

EMBRYOLOGY OF COLLATERAL CIRCULATION

The growth and maturation of ventricular myocyte (cardiomyocyte) are rapid processes that appear to be under partial control by locally generated growth factors [23].

Growth and development of the mammalian ventricle during the fetal and neonatal periods is to a large extent dependent on underlying changes occurring in the cardiomyocyte population, cardiomyocyte growth in cell number in mammals is primarily limited to fetal periods of development (*proliferative growth*) [20,30].

Subsequent increase in ventricular mass during the neonatal period is primarily the result of increased cellular size (Hypertrophy) of a definite population of cardiomyocytes. Cardiomyocytes occupy the heart tissue yet compose only 20-40% of the final total cellular population of the ventricle [31]. In addition the ventricle of the neonate undergoes rapid tissue remodeling that is associated with capillary angiogenesis and the formation of an extensive extracellular matrix (*ECM*) [30,32,33].

During this transition from fetal to neonatal development, cardiomyocytes exist while the cell cycle complete additional rounds of DNA synthesis associated with the process of binucleation and polyploidation, and begin their long term maturational process of cellular hypertrophy [34,35].

Capillary Angiogensis:

In concern with the terminal proliferative events in cardiomyocyte population, the length and number of ventricular