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How Vascular Hemodynamics Affects Blood Pressure

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To my wife Marna
I am so lucky to have her in my life

Foreword 1

I am delighted to introduce Paolo Salvi's volume on *Pulse Waves*, brilliantly summarizing the haemodynamics of hypertension and its implications for treatment. This is indeed a happy occasion for considering once more the variable approaches by which scientific knowledge advances, that is by the elaboration of complex models, to be subsequently simplified just to acknowledge that new factors and terms have to be entered to make the model match the realty.

The founding fathers of cardiovascular physiology were well aware of the complexity of the haemodynamic mechanisms underlying arterial blood pressure, and the models they developed tried to keep all known mechanisms under consideration. During the second half of the twentieth century, when blood pressure became an object of increasing clinical interest and a target for therapeutic intervention, the operational model being used was simplified and reduced to a pump working against a peripheral vascular resistance. Although certainly oversimplified, it cannot be denied this model helped achieving one of the greatest medical successes of the past century, that is the ability to effectively lower the most important risk factor for cardiovascular disease, high blood pressure.

With increasing attention being paid nowadays to hypertension in the elderly, it has been too easy to understand that the simplified model that has been so beneficial in developing current antihypertensive therapy has obvious limitations, and that in order to understand blood pressure increase with aging additional haemodynamic complexities must be added to the present model. Paolo Salvi, who has been and is in the forefront of new research in this area, deserves the highest appreciation for calling the doctors' attention to the reality of the complexities of haemodynamics, and for doing that in such a clear and efficacious way. Readers will find here a clear explanations of the role of large arteries in addition to those of the cardiac pump and vascular resistances, the role of pulse wave reflection, the possible differences between peripheral and central blood pressure, the new techniques and equipments allowing a more precise evaluation of haemodynamic patterns.

Scientific progress naturally generates not only knowledge but challenges. The major challenge in front of hypertension experts is demonstrating that the new models, the new equipments, the new measurements can effectively improve the

way we are diagnosing and treating hypertensive patients, and further improve their survival.

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Foreword 2

For a young clinical investigator working in the field of hypertension during the 1970s, the etiological aspects of hypertension disease, namely the renin-angiotensin-aldosterone system and the kidney, had to be considered as the dominant scientific problem. Most of the hypertensive specialists were first and foremost nephrologists and did not fully take into account that the prognosis of hypertension in humans could be significantly improved by the symptomatic (hemodialysis and transplantation) but not etiologic aspect of high blood pressure. Mostly, drug treatment of hypertension was emerging and the primary results of therapeutic trials were just becoming easier to evaluate.

An important aspect to consider in hypertension was that the disease was described as a mosaic of various pathophysiological alterations, involving numerous organs and/or functions, including the kidney, adrenal glands, renin, sympathetic nervous system, and arterioles. In fact, it seemed that there was no part of the body that was not implicated in the mechanisms of hypertension. However, the conduit arteries, which were the precise sites where the diagnosis of high blood pressure was established, were not considered of primary interest. Only the arterioles, a part of the vascular tree in which blood pressure was never measured, were important to investigate. On the other hand, the large arteries, i.e., the precise and specific site of the complications (rupture, thrombosis and aneurysms) of the disease, were thought to be important only as the location of atherosclerosis, a specific process very similar to, but in fact substantially different, from that of hypertension itself.

At this period, however, systemic hemodynamics was considered as a necessary basis for any understanding of the mechanisms of hypertension. This notion was widely accepted in both clinical and experimental situations, but in both fields, the hemodynamic description of hypertension referred only to linear models of circulation with simple measurements of steady flow. Only Poiseuille's and Laplace's laws were considered to be relevant to investigate the hemodynamic alterations. The evolution of the disease was presented as some kind of "struggle" between two exclusive components of the circulation: the heart and the arterioles. No other component of the circulation had to be extensively investigated. In particular, large arteries had no substantial role to be clarified within the mechanisms of

hypertension. Nevertheless, at that time (1970), the weight of evidence suggested that conduit arteries were clearly implicated in both the diagnosis of the disease and its complications. It was evident that the description of this important aspect of circulation in hypertensive subjects required a major modification of our conceptual approach to the hypertensive disease.

In the conventional approach, the vascular system is considered as a steady-flow network of conduit, which is characterized by mean blood pressure. However, this model focuses on the end product, the steady flow that is vital for perfusion of tissues and ignores the process that precedes and corresponds to the pulsatile nature of the arterial system and to the buffering function of large arteries and their Windkessel function. This process is the conversion of the intermittent, high-flow ejection by the left ventricle into the continuous peripheral flow and functions through the elastic properties of the aorta and large arteries already described in cardiovascular physiology. To take into account both steady and pulsatile hemodynamics phenomena, the pressure waveform should instead be conceived as a fluctuation, whose peak is systolic blood pressure and nadir the diastolic blood pressure, around a mean value, mean blood pressure. Furthermore, large arteries should be conceived not only on the basis of viscoelastic properties of the arterial wall but also on the basis of presence of wave reflections.

All these new properties of the cardiovascular system could not be detailed without new goals in the development of hypertension: not only the research in the causes of hypertension but also the reduction of cardiovascular risk. The association of hemodynamic development and of cardiovascular epidemiology has now transformed in men our concepts of the hypertensive cardiovascular disease. This story is detailed in the latest book by Paolo Salvi, Pulse Waves.

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Introduction

1

Arterial hypertension used to be considered a serious disease whose even fatal complications were an everyday occurrence. Nowadays, medical knowledge and scientific research have improved greatly, providing increasingly effective tools to assess pressure values. Moreover, health training and culture always emphasize that blood pressure must be kept under control.

This is the reason why we have started to consider blood pressure as part of a wider prevention context and no longer as an isolated pathological event. As a consequence, nowadays, hypertension is considered to be one of the main risk factors for cardiovascular diseases, particularly myocardial infarction and cerebral stroke.

In the future, there will be further evolution in the concept of hypertension, as it will be mainly considered as a symptom (Fig. 1.1). This concept will revolutionize the approach to hypertensive patients as it leads to exploration and understanding of the physiopathological events underlying increased blood pressure values. Faced with a patient who is suffering from high blood pressure, not only will we have to normalize the pressure values, but also we will try to understand, and possibly solve, the hemodynamic causes at the basis of the increased pressure

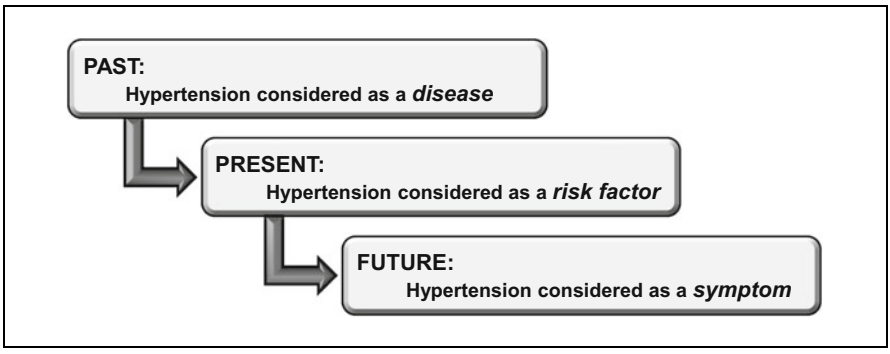

Fig. 1.1 Evolution of the concept of arterial hypertension

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values. In this way, the aim of therapy will be even to face the hemodynamic elements and resolve the main factors leading to hypertension.

Over the past 20 years, there has been a radical change in scientific knowledge, thanks to clinical research, and this has dramatically changed the approach to the hypertensive patient. Moreover, the outcomes of important clinical trials have recently pointed out some peculiar aspects of vascular hemodynamics, stressing the importance of the mechanical properties of the aorta and the large arterial vessels, of central blood pressure, of the amplification phenomenon, of incremental pressure, etc. To understand these elements, the basics of cardiovascular physiopathology are required, particularly vascular hemodynamics.

This book aims to be a clear and easy tool, providing the reader with the basic principles of vascular hemodynamic physiopathology, to offer a better approach to the hypertensive patient. Sometimes this teaching method needs to simplify things in a way that could seem even banal, but the purpose is always to give intelligible messages to everybody.

The author will try to take the reader by the hand, to describe the elements of blood pressure, starting from the definition of mean arterial pressure, going on with analysis of the basic principles of pulse pressure, and familiarizing the reader with a "dynamic" concept of blood pressure. Then, the forward and reflected components of the central pressure wave will be analyzed, providing the reader with self-study material to read the central blood pressure waveform and to understand the relationship between peripheral and central blood pressure.

Please point me at incomplete or unclear parts or even possible mistakes. Thank you in advance for your suggestions and comments. Any suggestion will be taken into consideration in the next editions, always to improve the quality of the book. Please send them to this e-mail: *salvi.pulsewaves@gmail.com.*

Reading aid

In this book, you will find some paragraphs, presented in a box and written in italics. These provide further in-depth study, sometimes rather complex. They can be skipped without affecting the comprehension of the book. However, they are suggested reading, as sometimes they deal with some oddities, and sometimes they help to understand some physical and mathematical parameters. They consider elements which may be of help in research and clinical applications. Anyway, reading of these parts is strongly recommended for practitioners who use methods aimed at studying viscoelastic properties of the large arteries in their clinical research and activity.

Mean Arterial Pressure

2

Let us consider the cardiovascular system as a simple hydraulic circuit, where there is a pump (heart) with a rhythmic activity (systole → diastole → systole …) that pushes a liquid (blood) into a tube (aorta), which divides over and over again (peripheral arteries → arterioles → capillaries) to be able to reach the farthest extremes (tissues).

This hydraulic circuit is very similar to a simple electric circuit, and we have to stress that electrical models are often used to verify cardiovascular hemodynamic phenomena.

According to Ohm's law (Fig. 2.1), the potential difference between the extreme points of an electric circuit ($\Delta V = V_1 - V_2$) is defined by multiplying the current (I) by the resistance of the circuit (R).

$$\Delta V = I \cdot R.$$

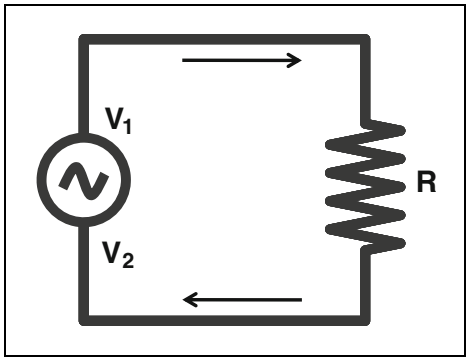

Fig. 2.1 Simple electric circuit

The cardiovascular system can be considered in a similar way (Fig. 2.2), and the law which defines blood pressure comes directly from Ohm's law:

- The difference in blood pressure values between the extreme points of the systemic circulation ($\Delta P = P_1 - P_2$) represents the potential difference between the extreme points of an electric circuit ($\Delta V = V_1 - V_2$);

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- The cardiac output (CO) represents the current (I);
- The systemic vascular resistance (SVR) represents the resistance of the circuit (R).

$$\Delta P = CO \cdot SVR.$$

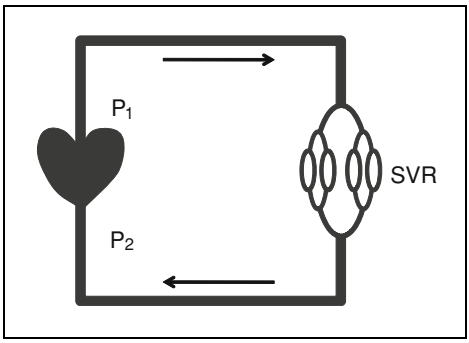

Fig. 2.2 Systemic circulation system model

As blood pressure back to the heart is very low, let us consider the pressure value as the value of blood pressure in ascending aorta (P), therefore the formula can be simplified by writing

$$P = CO \cdot SVR.$$

As cardiac output (CO) is given by multiplying stroke volume (SV) by heart rate (HR), the formula can be rewritten as

$$P = SV \cdot HR \cdot SVR$$

(blood pressure = stroke volume · heart rate · systemic vascular resistance).

However, we must point out that blood pressure values change during the cardiac cycle, so the term "P" defined by the formula above refers to mean arterial pressure (MAP). Therefore,

$$MAP = SV \cdot HR \cdot SVR.$$

According to this formula, blood pressure values depend on just three parameters: stroke volume, heart rate and systemic vascular resistance (Fig. 2.3). It is important to note that, for decades, both research and clinical application focused their attention on these three factors affecting mean arterial pressure.

There is no doubt that mean pressure is a very important parameter. Moreover, it has a quality which makes it even more interesting, namely its relative "stability" in the arterial tree. In other words, mean arterial pressure tends to remain unchanged in the arterial system, from ascending aorta to peripheral arteries (Fig. 2.4).