

Arterial hypertension – the timeline of a concept

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Received: February 1 2022, Accepted: February 22, 2022

Abstract

Nowadays, it is well acknowledged that arterial hypertension is the leading global risk factor for cardiovascular diseases and the largest contributor to cardiovascular mortality in many parts of the world, including Europe. However, we rarely think about the evolution of its diagnosis, its changing definition, or the gradual understanding of its significance. In this paper, the milestones of the arterial hypertension concept will be presented, highlighting the decisive steps that led to the actual methods of blood pressure measurement in a clinical setting in the first part. The second part will reveal the contradictory evolution of the concept of hypertension until its current definition in relation to cardiovascular risk.

Keywords: arterial hypertension, blood pressure measurement, cardiovascular risk factor.

Introduction

It is well acknowledged that arterial hypertension (HTN) is the leading global risk factor for cardiovascular diseases (CVD) [1] but also the most influential contributor to cardiovascular mortality worldwide, including in Europe [2]. However, the evolution of its diagnosis methods, changing definition, or the gradual understanding of its significance are rarely considered. In this paper, the mile-

stones of the arterial hypertension concept will be presented.

Milestones in blood pressure measurement

The first documented measurement of blood pressure (BP) comes from the reverend Stephen Hales who inserted a pipe attached to tubing into the left crural artery of a dying horse [3]. Several techniques and devices developed in the 19th century to measure blood pressure [4, 5] led to Scipione Riva-Rocci's invention of a mercury sphygmomanometer that could be used in clinical practice. This device has initially measured only the systolic blood pressure through palpation [6]. Korotkoff listened with a stethoscope to the sounds emitted by

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a compressed artery during the deflation of the cuff and published in 1905 his hypothesis that the first knocking sound of this suite corresponds to the systolic BP and the disappearance of these sounds indicates the diastolic BP [7]. His hypothesis was later confirmed in animal models and humans through intraarterial catheterization [8]. In a short time, an aneroid device was adapted by Henri Vaquez and Charles Laubry and commercialized by Spengler starting in 1911 [9]. Due to mercury-related health concerns, the mercury manometer has essentially been replaced by aneroid or electronic devices toward the end of the 20th century [10]. Ambulatory blood pressure monitoring development started in 1966 when Sokolov *et al.* published their research based on a semiautomatic device [11, 12].

For us, it is an unimaginable medical practice not to evaluate the blood pressure during patients' work-up. However, among the misconceptions related to BP, some went against its measurement, fearing that "we pauperize our senses and weaken clinical acuity" by using sphygmomanometers (Table 1) [13].

Identification of higher blood pressure as a pathogenic condition and risk factor for cardiovascular disease

Richard Bright remarked on the association between kidney damage, left ventricular hypertrophy, and thickened blood vessels [14]. Subsequently, George Johnson suggested that renal vascular remodeling may be an adaptation to high BP [15]. Kirkes in 1855 and Traube in 1856 went further and advanced the hypothesis that increased BP could be the link between left ventricular hypertrophy and kidney damage [16]. All these observations led to the assumption that high blood pressure is an effect of kidney disease. However, when Samuel Wilks observed that left ventricular hypertrophy might occur in the absence of kidney disease, it became clear that high BP is not necessarily the consequence of an injured kidney and that increased BP could induce per se severe complications to the brain or heart. This hypothesis was proved by a young but brilliant physician of Guy's Hospital in the United Kingdom, Frederick Akbar Mahomed, also the first who attempted the clinical measurement of BP [17]. The earliest statistical evidence about the adverse effects of HTN and its impact on survival came from the insurance industry in the United States at the beginning of the 20th century. In 1911, the medical director of the Northwestern Mutual Life Insurance Company wrote: "The sphygmomanometer is indispensable in life insurance examinations, and the time is not far distant when all progressive life insurance companies will require its use in all exam-

inations of applicants for life insurance" [18]. The 1925 report revealed that mortality increases rapidly with increased blood pressure above the average value [19]. In 1939, it was noticed that for entry ages ≥ 40 , systolic blood pressure was a more important predictor of death than diastolic pressure, while for entry ages of < 30 years, the influence of diastolic pressure was more significant than systolic pressure [20]. A very severe clinical form of HTN was named "malignant" by a group of physicians from Mayo Clinic in 1928 [21].

Despite all the evidence that was accumulating, many physicians continued to believe that high blood pressure values are necessary for adequate organ perfusion, especially in the elderly. In 1912, Sir William Osler was firmly against the correction of high BP: "The extra pressure is a necessity – as purely a mechanical affair as in any great irrigation system with old, encrusted mains weekly channels. Get it out of your heads, if possible, that the high pressure is the primary feature, and particularly the feature to treat." [22]. In 1931, John Hay – Professor of Medicine at Liverpool University, asserted: "There is some truth in saying that the greatest danger to a man with high blood pressure lies in its discovery because then some fool is certain to try and reduce it" [23]. In line with this approach, US cardiologist Paul Dudley White wrote: "Hypertension may be an important compensatory mechanism which should not be tampered with, even if we were certain that we could control it" [24]. However, in 1949, Charles Friedberg, in his textbook "Diseases of the Heart" recommended not to treat mild benign hypertension, defined as values higher than 210/100 mmHg [25].

In the context of these debates during the third and fourth decade of the 20th century, the famous case of the American President Franklin D Roosevelt brought to the attention of the medical world and the public the ravages that hypertension can do if it remains untreated. In 1933, when he took office as the 32nd President of the United States of America, he was known for his battle with poliomyelitis but, without doubt, had no history of cardiovascular disease. In 1937, a medical report stated that the physical condition of the President was very good although his BP was 177/100 mmHg.

On the day of the Pearl Harbour attack, the BP was 190/105 mmHg; when landing in Normandy has started, BP was 226/118 mmHg and by the time of the Yalta Conference in February 1945, BP was 260/150 mmHg. In fewer than 30 days, his BP exceeded 300/190 mmHg, and he died of a presumed cerebral hemorrhage at 63 years old. Moreover, his physician declared that his neurological condition "came out of the sky" [26]. In the beginning, attempts to treat him included massages and phenobarbital, and when he began to develop heart failure, he was put on a low salt diet and digitalis [25].

Following this tragic event, the US Congress approved the National Heart Act in 1948 and created

Table 1. Milestones in blood pressure measurement.

1738	Stephan Hales
	<ul style="list-style-type: none"> • First documented measurement of blood pressure was performed in a horse [3]
1828	Jean-Leonard Poiseuille
	<ul style="list-style-type: none"> • Development of the U-tube Mercury manometer that helped standardize BP to the units of measure still used today – mmHg [4]
1881	Samuel Siegfried Karl Ritter
	<ul style="list-style-type: none"> • First sphygmomanometer (in Greek <i>sphygmo</i> means pulse and <i>manometer</i> means pressure monitor) [5]
1896	Scipione Riva-Rocci
	<ul style="list-style-type: none"> • Invention of the modern BP mercury sphygmomanometer consisting of an inflatable cuff (produced in the original version by Dunlop) connected to a column of mercury [6]
1905	Nikolai Korotkoff
	<ul style="list-style-type: none"> • The “sound of Korotkoff” allows the measurement of both systolic and diastolic BP [7, 8]
1911	First aneroid device
	<ul style="list-style-type: none"> • Invented by Henri Vaquez and Charles Laubry and commercialized by Spengler [9]
1966	Ambulatory Blood Pressure Monitoring
	<ul style="list-style-type: none"> • The development of ambulatory blood pressure monitoring starts in 1966 when Maurice Sokolov <i>et al.</i> published their research based on a semiautomatic device [11, 12]
1974	First fully automated oscillometric sphygmomanometer
	<ul style="list-style-type: none"> • Released by Panasonic [13]

the premises for the Framingham Study, which is an ongoing population-based, observational cohort study that was initiated to prospectively investigate the epidemiology and risk factors for cardiovascular disease [27]. The Framingham Study established hypertension as a major cardiovascular risk factor; the evidence that HTN increases coronary heart disease incidence was found in 1957, and the prognostic significance of electrocardiogram – left ventricular hypertrophy – in hypertensives was revealed in 1961, and HTN was established as the main risk factor for stroke in 1965. In 1971, it was shown that systolic BP is superior to diastolic pressure in predicting cardiovascular disease. Beyond all that, the Framingham Study demonstrated that HTN occurs usually in association with other atherogenic risk factors. Clustering with metabolically linked factors has been shown to be related mainly to insulin resistance promoted by weight gain and abdominal obesity [28].

Nevertheless, the Framingham Study also clarified the greater impact of systolic than diastolic pressure on cardiovascular risk proving that in persons whose diastolic pressures did not exceed 95 mmHg, cardiovascular disease risk increased progressively with the systolic pressure at all ages in both men and women. Each standard deviation augmentation in systolic BP raised by 40–50% the propen-

sity to cardiovascular disease, whereas for diastolic pressure, the increment was 30–35% (Table 2) [29]. Much evidence was needed on the importance of systolic *versus* diastolic pressure to rule out a simple presumption. It is not known precisely where this statement came from, but it can also be found in the 1927 edition of the classic Cecil’s medical textbook: “The blood pressure is found to be distinctly raised; systolic pressures of 250 mmHg are quite common. More significant than the systolic pressure is the increase in diastolic pressure increases, for it has been shown that as the diastolic pressure increases, the arteries lose their normal elasticity and efficiency, and greater work is consequently thrown upon the heart. It is therefore important to bear in mind, in determining the prognosis, that those individuals whose diastolic blood pressures remain relatively low, despite high systolic pressures, are decidedly less apt to suffer cardiac decompensation and cerebral apoplexy” [30]. Following the Framingham Study, many other studies confirmed that systolic BP exerts a greater influence than diastolic pressure [31].

Perception of HTN was beginning to change to a large extent. By 1960, Pickering was emphasizing that HTN is a “quantitative” and not a “qualitative” trait, meaning that there is a continuous relationship between arterial pressure and mortality over the whole range of arterial pressure: “Arterial

Table 2. Pathogenic effects of high blood pressure.

1836	Richard Bright
	<ul style="list-style-type: none"> • First report of the association between kidney damage, left ventricular hypertrophy and thickened blood vessels [15]
1850	George Johnson
	<ul style="list-style-type: none"> • Renal vascular remodeling might be an adaptation to high blood pressure [16]
1855–1856	William Senhouse Kirke and Ludwig Traube
	<ul style="list-style-type: none"> • Increased blood pressure could be the link between left ventricular hypertrophy and kidney damage [17]
1874	Frederick Akbar Mahomed
	<ul style="list-style-type: none"> • First attempt of blood pressure measurement in a clinical setting • Identification of left ventricular hypertrophy occurrence in the absence of kidney disease [18]
1906	Some life insurance companies from the US start measuring systolic BP
	<ul style="list-style-type: none"> • Important data have been collected over the time regarding the relation of arterial hypertension with mortality [19, 20]
1928	Keith, Wagener, and Kernohan
	<ul style="list-style-type: none"> • Enunciation of the “malignant hypertension” syndrome [21]
1945–1948	Franklin D Roosevelt
	<ul style="list-style-type: none"> • Death of the 32nd President of the USA due to a presumed cerebral hemorrhage at the age of 63 years after almost 15 years of severe and untreated hypertension [26]. • President Truman signs National Heart Act in 1948 [27].
1948	Beginning of Framingham Heart Study
	<ul style="list-style-type: none"> • The Framingham Study established hypertension as a major cardiovascular risk factor [27–29]

pressure is a quantity, and its adverse effects are related numerically to it. The dividing line (between normal blood pressure and hypertension) is nothing more than an artifact.” [32]. This assertion explains the variable definitions of HTN in the last decades of time. The current definition for HTN of the European Society of Cardiology/European Society of Hypertension relies, in essence, on the Multiple Risk Factor Intervention Trial (MRFIT) study results and consider that hypertension is the level of blood pressure at which the cardiovascular risk is doubling, meaning 140 mmHg for systolic BP and 90 mmHg for diastolic BP [33–35]. However, in the US, the delineation between normal and pathologic has been decreased to 130/80 mmHg since 2018 [36], and this decision was primarily motivated by the SPRINT trial, which reported that among patients at high risk for cardiovascular events but without diabetes, targeting a systolic blood pressure of less than 120 mm Hg, as compared with less than 140 mm Hg, resulted in lower rates of fatal and nonfatal major cardiovascular events and death from any cause, although significantly higher rates of some adverse events were observed in the intensive-treatment group [37]. In the future, we can expect that even lower values will define HTN since recent data from the Multi-Ethnic Study of Atherosclerosis have indicated that beginning at a systolic BP level as low as 90 mm Hg, there appears to be a

stepwise increase in the presence of coronary artery calcium and the risk of incident atherosclerotic vascular disease with increasing systolic BP levels [38].

Bearing in mind the current criteria for defining hypertension and its recognition as a significant cardiovascular risk factor, the discovery of mechanisms and specific therapy for high blood pressure over the last century will be presented in a future issue of this journal.

Conflict of Interest

The author confirms that there are no conflicts of interest.

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