



What is the optimal cut-off threshold in blood pressure measurement? Who invented 140/90 mmHg?

¿Cuál es el umbral de corte óptimo en la medición de la presión arterial? ¿Quién inventó el 140/90 mmHg?

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Nobody nowadays dares to doubt that high blood pressure is a cardiovascular risk factor and inexorably progressive and fatal. However, let's cite two experts on cardiovascular medicine in the 30th:

1. «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» – JH Hay, 1931.¹
2. «Hypertension may be an important compensatory mechanism which should not be tampered with, even where it is certain that we could control it» –Paul Dudley White, 1937.²

Thus, we have undoubtedly made remarkable progress in the last 85 years. After the death of USA President Franklin Delano Roosevelt, the evidence documented on his medical record that blood pressure progressively increased in the previous five years of life brought great concern. According to the thinking of that time, Ross McIntire, his family physician and a specialist in otorhinolaryngology, did not prescribe antihypertensive treatment. By 1944, pressures of more than 180/105 mmHg were documented, and he presented clear signs of heart failure. His doctor was treating him for bronchitis and sinusitis.³

Although 120/80 mmHg is often considered the standard upper threshold

blood pressure for adults, its precise measure is an issue. Actual devices developed for blood pressure measurement come from the old mercury sphygmomanometers that allowed «standardized» measurement. Despite the high inter-observer variability, this vital sign has been accepted over decades as an extraordinary health marker of great importance. Thus, health staff and predominantly physicians were the only ones authorized to measure blood pressure for a long time. In 1905, Nikolai Korotkoff, a Russian military surgeon, wrote in a very brief report to the Imperial Military Medical Academy his auscultatory technique for obtaining systolic and diastolic blood pressure. This technique only requires a sphygmomanometer (blood pressure cuff) and stethoscope for listening to Korotkoff's sound.⁴ The first sharp tapping sound defines the systolic pressure, and its disappearance defines the diastolic pressure.

Hypertension is a major risk factor for ischemic and hemorrhagic stroke, myocardial infarction, heart failure, chronic kidney disease, peripheral vascular disease (PVD), cognitive impairment, and premature death. So, what is the appropriate cut-off point for diagnosing a hypertension patient? Remember that blood pressure measurements give us systolic and diastolic values, respectively. The first reflects the blood impulse propagated in the arteries by the left ventricular contraction and the resistance of these arteries. On the other hand,

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diastole reflects the rebound distensibility of the arteries on the blood flow that attempts to return and is stopped by the closure of the aortic valve. However, many observational studies have found that both systolic and diastolic blood pressure exhibit a graded and independent relationship with mortality and morbidity (ESH-ESC 2013). Untreated hypertension can be associated with a gradual increase in blood pressure, potentially culminating in a state of resistance to treatment caused by associated vascular and renal damage.

The classic observational epidemiological research, elegantly analyzed by Macmahon and Peto, triggered a vital reflection. As diastolic and systolic pressures increase above 75 mm and 115 mmHg, respectively, the risk of a severe cardiovascular event (ischemic heart disease or cerebral vascular event) also increases significantly over the next 5 to 10 years.

Mathematical weightings and fittings had to be made with multiple rectilinear models to associate blood pressure levels with cardiovascular risk directly. So, one thing is clear: it is not a rectilinear phenomenon; no matter how many adjustments are made, biological phenomena always show some degree of internal variation. Generally, it is curvilinear, i.e., there is a first stage where the risk is 1.0 to 1.5 times greater with pressures of 115 to 135 mmHg in the systolic and 75 to 85 mmHg in the diastolic. Subsequently, generally, as of pressures of 140/90 mmHg for each 20 mmHg increase in systolic pressure and for each ten mmHg in diastolic pressure, the risk doubles. In the 70's- and 80's-decade, mathematical models of linear regression were introduced as predictors of events. However, it must be emphasized that many environmental, patient, and physician factors influence blood pressure measurement. For example, cuff size is crucial, and pressure values vary from 5 to 10 mmHg to the real one.

The recommended cuff sizes are: For an arm circumference of 22 to 26 cm, the cuff should be «small adult» size: 12 × 22 cm. For an arm circumference of 27 to 34 cm, the cuff should be «adult» size: 16 × 30 cm. For an arm circumference of 35 to 44 cm, the cuff should be «large adult» size: 16 × 36 cm. For an arm circumference of 45 to 52 cm, the cuff should

be 16 × 42 cm. Now, being honest, how many physicians or health care staff who measure blood pressure have these standard cuffs?

HYPERTENSION IS NOT JUST A MATTER OF MMHG

However, reducing to «numbers» (mmHg) the cardiovascular risk in the patient living with arterial hypertension is to be unclear about the problem in a comprehensive manner. Blood pressure is a significant marker, but of course, a 40-year-old patient with 145/93 mmHg with no other cardiovascular risk factors will not be the same as another one with the same blood pressure level, of the same age and gender but with diabetes, obesity, and dyslipidemia. The latter will have a very high risk. Thus, we must understand the gradual elevation of blood pressure as a dynamic and progressive pathobiological and pathophysiological phenomenon. Intra- and intercellular signaling cascades are activated. This vasoreactivity also activates anti-inflammatory and vasorelaxant compensatory mechanisms. However, if the insult is perpetuated, the equilibrium towards biochemical and cellular mechanisms originates mechanisms of structural changes secondary to cellular proliferation, giving rise to arterial stiffness, atherosclerosis, and arteriolosclerosis.

WHO INTRODUCED 140/90 MMHG, AND HOW CAN WE ACHIEVE PREDICTIVE INDEPENDENCE OF A VARIABLE?

Multivariate linear regression models were introduced in the 70s and 80s as a robust strategy to elaborate mathematical predictive and association models where randomness would participate as little as possible. How do we compare several variables with each other? To rescue those that maintain their «independence» to be significantly associated with the target variable under study was the guideline that gave way to what was called «multivariate analysis».⁵ The high risk of this method is the possibility of spurious variables leaking out and ruining the final interpretation. Removing confounding variables, such as collinearity, interference,

synergy, or antagonism, required statistical skills and full scientific knowledge of the area.

Thus, blood pressure levels went from observational studies to formal cohort research with robust mathematical analysis and modeling. The «sovereignty of blood pressure» was maintained as an «independent» continuous risk variable predictive of major cardiovascular events. However, later on, to make this knowledge practical (in its clinical use), it was necessary to search for cut-off points to turn continuous into categorical. Thus, the so-called ROC curves were used, where the «model» for defining cut-off points was found to have the best sensitivity and specificity (Figure 1).

But stop and think about it: will there be a difference between 138/88, 140/90, or 143/93 mmHg on the risk continuum? If your answer is no, the objective of this editorial will have been achieved. Exactly! We can't get to reductionism in medical thinking. The number of violations and assumptions that are made when trying to make the inaccuracy of a biological phenomenon, which per se, is variable, oscillatory, and dependent on several biological, biochemical, and environmental

circumstances, is to want to «tear away» from the physician the most valuable thing he has, his clinical judgment. There is no mathematical model that outperforms it, even artificial intelligence.

GOOD CLINICAL JUDGMENT

What happened once we accepted that 140/90 mmHg is a mathematical construct to define risk population classification? Once the value was agreed upon, many studies on arterial hypertension were conducted, both clinical and basic. It was possible to integrate follow-up cohorts to evaluate the impact of pharmacological and non-pharmacological strategies in short-, medium- and long-term follow-up.

The impact was such that 140/90 mmHg was popularized worldwide and emerged as a goal or treatment objective in all the guidelines and directives in the world. The efficacy and safety of all antihypertensive drugs are based on their ability to reduce blood pressure to < 140/90 mmHg. The dispute to find the best drug is «confined» to differences as small as 3-5 mmHg.

And that's not all. Since many drugs achieved the goal of reducing to < 140/90 mmHg, competition arose in other areas such as half-life, adverse effects, pleiotropic effects, costs, and adherence. Thus, the physician returns to the essence of his *raison d'être* and stops seeing the patient as a number to realize that there are multiple factors involved in cardiovascular risk in the real world. Syndrome X, or metabolic syndrome, appeared in the 80s to try to connect different risk factors that are frequently associated. Today, attempts are being made to popularize the cardio-reno-metabolic syndrome, which is not a syndrome at all but a new construct to draw attention to a pathophysiological situation shared by obesity, diabetes, hypertension, and dyslipidemia.

For this reason, in the practice of medicine, clinical judgment is insuperable. The same patient with 145/95 mmHg can be «low» risk or very high risk, depending on the context.

So, it means that we should ignore 140/90 mmHg. Nothing is false other than that; the importance is to take it for what it is, «a frame

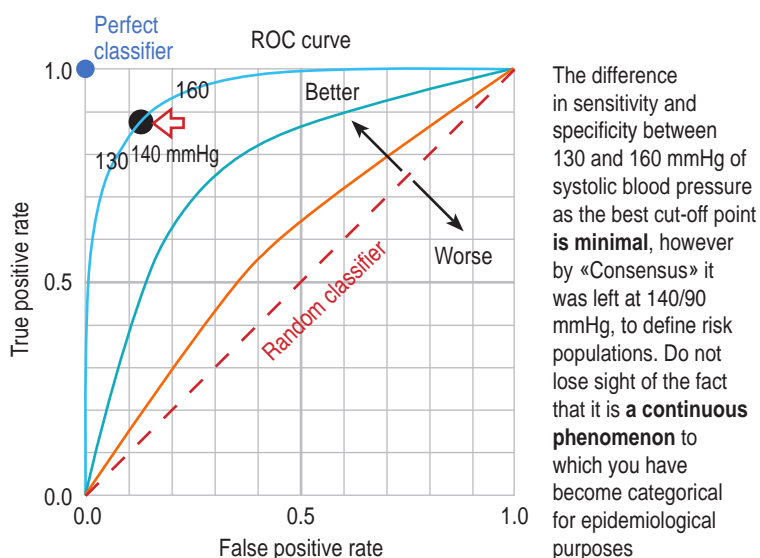


Figure 1: Receiver operating characteristic curves are the graphic representation that allows visualization of the best value as a classificatory cut-off point for a continuous phenomenon that one wishes to make categorical. What is sought is the point with the best sensitivity and predictive specificity. ROC = Receiver Operating Characteristic.

of reference», and the context of the patient should draw your attention. Thus, step 1, the figure per se, should draw my attention because it is already a point to consider in the pathological blood pressure elevation continuum. Step 2: make sure that the measurement is accurate and that it was made under optimal conditions. Step 3: make sure it is not a white coat phenomenon.

For this reason, taking and recording blood pressure outside the doctor's office is essential. A good log of home measurements or an ABPM will be of considerable diagnostic help. Step 4: define your patient's context to the possibility of other related risk factors. Step 5: Stratify your patient's risk by determining whether they are low, intermediate, high, or very high risk depending on the number of associated factors, presence or absence of target organ damage, structural damage, or history of a significant cardiovascular event, as well as blood pressure level. It will even help you decide which type of combination drug therapy is best for your patient.

WHAT IS THE GOAL < 140/90 MMHG OR LESS THAN 130/80 MMHG?

Last but not least, a plan of therapeutic goals must be drawn up. Again, do not get bogged down in an exact amount. The accumulated evidence indicates that it should be maintained at < 140/90 mmHg to reduce cardiovascular risk. However, ideally, it is to achieve < 130/80 mmHg without reaching values below 110/70 mmHg. Attaining this range of reduction has been associated with greater protection from

cardiovascular risk. However, to achieve these blood pressure ranges without taking into account the goals of the other associated risk factors is to consign the patient to continue to be at cardiovascular risk. Therefore, spare no effort to achieve comprehensive control of your patient.

Another grave mistake is not focusing on long-term strategies. It will be in vain for the patient to maintain optimal pressures for six months if they then abandon the treatment. It is common for the patient to change or modify it for «not feeling anything», whether they take it or not. The primary failures to change the natural history of arterial hypertension in Mexico and the world are due to underdiagnosis, inappropriate treatment, non-adherence, medical inertia, and lack of physician-patient communication. A work plan should be established in which the patient is an active and co-responsible co-participant.

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