Blood Pressure and Mortality

A quantitative review and a Bayesian network linking systolic and diastolic pressure to cardiovascular death

Summary

Blood pressure is one of the most thoroughly measured predictors of death in all of medicine, and the relationship is unusually clean. In large prospective data, the risk of dying from vascular disease climbs smoothly and steeply with usual blood pressure, with no safe ledge below which risk stops falling, at least down to about 115 over 75 (Lewington et al., 2002). Randomized trials then show that pushing pressure lower actually prevents events and deaths, which turns the observed association into a causal one (SPRINT Research Group, 2015, 2021). This note reviews what is known, separates the distinct roles of systolic and diastolic pressure, and builds a small Bayesian network that takes age, sex, systolic, and diastolic pressure and returns a probability of cardiovascular death over ten years. The network is calibrated from national survey data for the blood-pressure side and from the large meta-analyses for the mortality side, and it is written so that the two pressures carry different information rather than echoing each other.

The dose-response: usual pressure and vascular death

The anchor is the Prospective Studies Collaboration, a pooled analysis of about one million adults in 61 studies with roughly 56,000 vascular deaths and no known vascular disease at entry. Within every decade of age, the risk of vascular death rose in a straight line on a log scale as usual blood pressure rose, with no threshold detected down to 115 over 75. Each 20 mmHg of higher usual systolic pressure, or about 10 mmHg of higher diastolic pressure, went with roughly a doubling of the death rate from stroke and from heart disease at ages 40 to 69 (Lewington et al., 2002).

Two features of that finding matter for any model. First, the proportional effect weakens with age. The relative risk attached to a given pressure difference at ages 80 to 89 is about half what it is at ages 40 to 49, but because old age carries so much more baseline risk, the absolute number of extra deaths per unit of pressure is larger in the old, not smaller (Lewington et al., 2002). Second, the systolic and diastolic numbers are not two independent risks to be added. They move together and are alternative readings of the same underlying pressure, which is why a 20 mmHg systolic step and a 10 mmHg diastolic step carry about the same weight. The single best summary in that analysis was the average of the two, the mid-blood-pressure, with systolic alone slightly better than diastolic alone and the pulse pressure, the gap between them, the weakest of the simple choices (Lewington et al., 2002).

Systolic, diastolic, and pulse pressure: which number matters when

That last point has an important exception that depends on age, and it is the reason a serious model should keep systolic and diastolic separate rather than collapsing them. In younger adults, diastolic pressure is the better predictor of coronary risk. Somewhere around the fifties the

importance crosses over, and after about age 60 systolic pressure and the pulse pressure dominate while diastolic pressure becomes, if anything, inversely related to coronary risk (Franklin et al., 2001). The mechanism is arterial stiffening. As large arteries stiffen with age they no longer cushion the pulse, so systolic pressure rises while diastolic pressure falls, widening the pulse pressure. A wide pulse pressure is therefore a marker of stiff arteries, and it independently predicts coronary heart disease in older adults (Franklin et al., 1999).

This is why isolated systolic hypertension, a high systolic with a normal or low diastolic, is the characteristic pattern of later life and is genuinely dangerous rather than reassuring. Treating it reduces strokes, as the SHEP trial established (SHEP Cooperative Research Group, 1991). The practical upshot is that the same diastolic value can mean opposite things at different ages, so a model that only sees the average of the two pressures will miss the stiffness signal that low diastolic carries in the old.

The diastolic J-curve

The smooth downhill relationship has a known caveat at the low diastolic end. In older patients, and especially those with existing coronary disease or under aggressive treatment, very low diastolic pressure, below roughly 60 to 70 mmHg, is associated with higher coronary mortality rather than lower, which traces out a J-shaped curve (Franklin et al., 2001). Part of the explanation is physiology, since the coronary arteries fill during diastole, so a very low diastolic pressure can starve the heart muscle of its own blood supply. Part of it is reverse causality, since people who are already sick, frail, or in heart failure tend to have low diastolic pressure for those reasons rather than because low pressure harmed them. The J-curve is much weaker or absent for stroke, which fits the perfusion explanation, since cerebral flow depends more on systolic pressure. The lesson is not that high pressure is safe, but that in the old the diastolic number can fall for unhealthy reasons, and the model below treats the lowest diastolic band at high systolic as a wide pulse pressure rather than as a low-risk state.

From association to cause: the trials

Observational dose-response curves cannot by themselves prove that lowering pressure helps, because the people with lower pressure differ in many ways. The randomized evidence closes that gap. SPRINT assigned 9,361 adults at high cardiovascular risk, without diabetes, to a systolic target below 120 or below 140. The lower target cut the rate of major cardiovascular events by about a quarter and all-cause mortality by about a quarter to a third, a benefit large enough that the trial was stopped early (SPRINT Research Group, 2015). The longer final report confirmed both the cardiovascular and the mortality benefit (SPRINT Research Group, 2021). The intensive target also produced more of certain harms, including low blood pressure, fainting, and acute kidney changes, so the trial is a statement about averages in a high-risk group rather than a universal target. In causal terms, SPRINT is the interventional counterpart to the observational curve. It is the difference between seeing that lower pressure goes with longer life and acting to lower pressure and watching life lengthen.

A Bayesian network for blood pressure and mortality

The pieces above can be assembled into a small Bayesian network. The point of doing so is not prediction for its own sake, but to have one transparent object that holds the joint behavior of

age, sex, both pressures, and death, and that answers conditional questions by the rules of probability rather than by intuition.

Structure

The network has five variables. Age and sex are root causes. They influence both pressures, and systolic pressure also influences diastolic pressure, which captures the fact that the two readings are correlated and that their relationship shifts with age. All four upstream variables then feed the outcome, a binary node for death from cardiovascular disease within ten years.

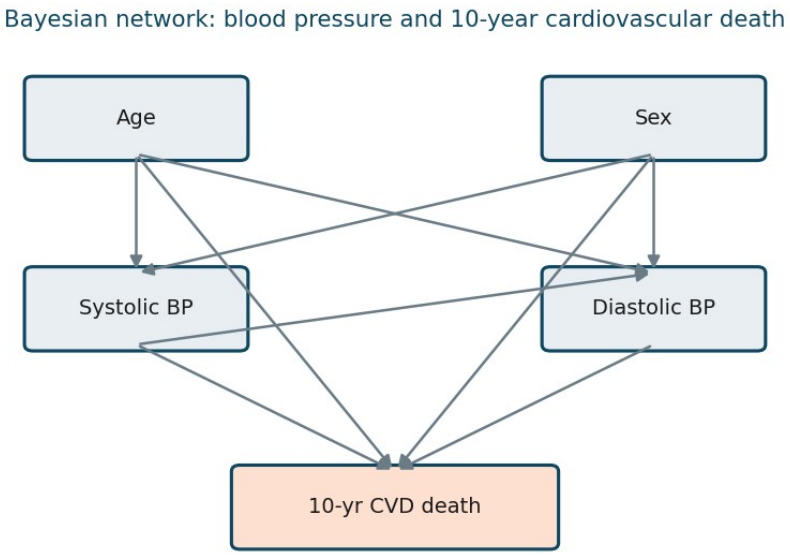

Figure 1. The network. Age and sex drive both pressures, systolic informs diastolic, and all four feed the ten-year cardiovascular-death node. The arrows are modeling choices about dependence, not claims that every link is purely causal.

How the conditional tables were built

The blood-pressure side is estimated from real measurements. The conditional distributions of systolic given age and sex, and of diastolic given age, sex, and systolic, come from the NHANES 2009 to 2012 examination microdata, weighted to the US population, for adults aged 40 to 79. So the network knows, from data, that pressure rises with age and that diastolic tends to plateau or fall relative to systolic in older people.

The mortality side is built from the meta-analyses rather than fitted to one cohort, and it is deliberately written so both pressures count. The hazard is the product of two terms. A level term raises risk with the mid-blood-pressure, doubling for roughly every 15 mmHg of mid-pressure above the 115 over 75 reference, with the doubling factor easing off with age in line with the observed attenuation of relative risk (Lewington et al., 2002). A pulse-pressure term adds risk as the gap between systolic and diastolic widens, with an effect that grows with age, reflecting arterial stiffness (Franklin et al., 1999). Because one term tracks the level of pressure and the other tracks its spread, systolic and diastolic readings carry distinct information rather than collapsing to a single average. The baseline ten-year risk at the reference pressure is set by age and sex; those baseline numbers are illustrative, chosen to reproduce the published relative risks and a realistic age and sex gradient rather than to match a single study exactly.

What the model says

Across the modeled population of adults aged 40 to 79, the network returns an overall ten-year cardiovascular-death probability of about 3.5 percent. The interesting part is the conditional structure. For a man aged 60 to 69, the risk runs from about 3 percent at an optimal pressure under 120 over 80 to about 27 percent at 160 or above over 100 or above, nearly a ninefold spread driven by pressure alone.

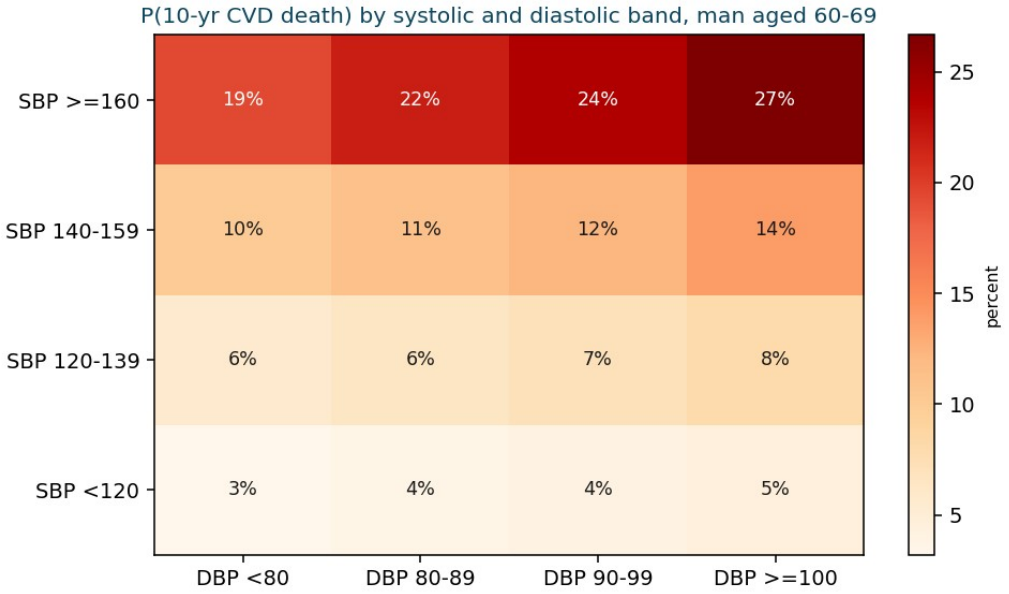

Figure 2. Modeled probability of ten-year cardiovascular death for a man aged 60 to 69, across systolic bands (rows) and diastolic bands (columns). Risk climbs steeply with systolic pressure and more gently with diastolic. The high-systolic, low-diastolic corner stays elevated because a wide pulse pressure carries its own risk. Absolute values are illustrative; the pattern follows the cited evidence.

The age gradient is just as important as the pressure gradient. The same systolic step adds far more absolute risk in an older person, which is the model's version of the finding that the absolute toll of pressure is greatest in old age even as the relative effect fades.

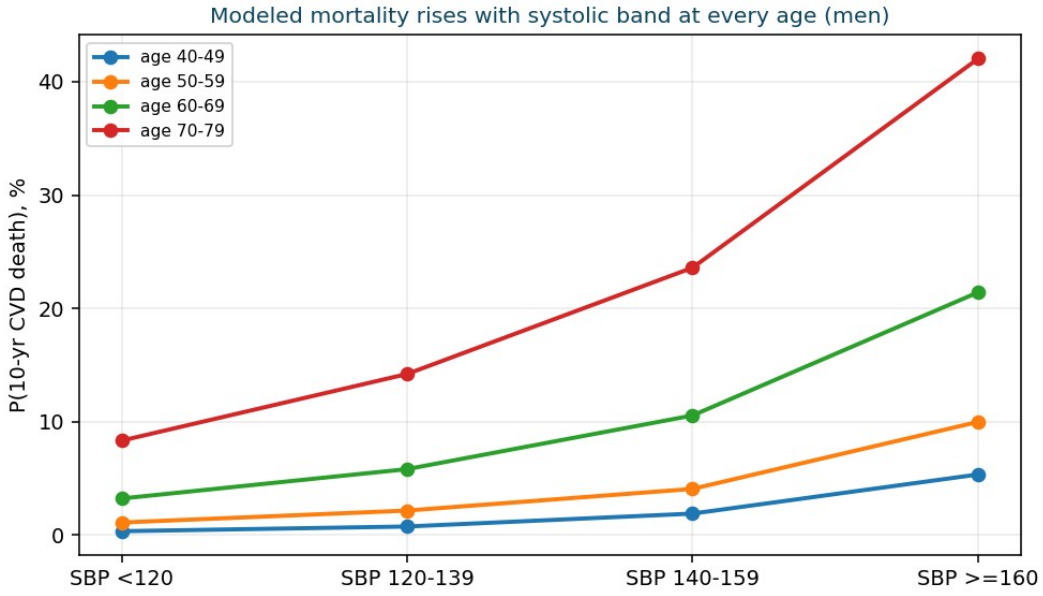

Figure 3. Modeled ten-year cardiovascular-death probability for men, by systolic band, at four ages, marginalizing over the data-derived diastolic distribution. Every line rises with systolic pressure, and the lines fan apart with age.

The network also answers individual conditional questions. A man aged 60 to 69 with a systolic of 140 to 159 and a diastolic of 90 to 99 carries a modeled ten-year cardiovascular-death probability of about 12 percent. The same man at a systolic under 120 would sit near 4 percent. That contrast is exactly the quantity a treatment decision turns on, and SPRINT is the evidence that moving along this axis by treatment, not just observing people who differ, captures most of that benefit. The pulse-pressure term shows its effect at the extremes. For a man aged 70 to 79 with a systolic of 160 or more, dropping the diastolic below 80 does not lower his modeled risk, because the resulting wide pulse pressure offsets the lower average pressure, which is the network's echo of the diastolic J-curve.

What the model is not

The network is a teaching and reasoning tool, not a clinical calculator. The relative-risk structure is anchored in the large studies, but the absolute probabilities depend on baseline assumptions and should be read as illustrative. The variables are coarse bands rather than continuous pressures. The mortality node is built from population averages, so it does not represent an individual's other risk factors, and it does not fully capture the steep harm at very low diastolic pressure seen in frail treated patients, since that pattern is driven partly by reverse causality that a forward model of this kind cannot express. Natural extensions are a treatment node, which would let the network answer interventional do-style questions directly rather than by comparing conditioned states, an explicit pulse-pressure or arterial-stiffness node, and the usual companions of blood pressure such as cholesterol, smoking, and diabetes.

References

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