The belief that “the higher your blood pressure, the higher your risk of death, and the lower your blood pressure, the lower the risk of death” is WRONG.
This FALSE belief goes back to 1968 when the Framingham study was published.
My impression is that this belief about blood pressure is thought to be one of the “sacred truths” in medicine.
But this belief is WRONG.
Systolic blood pressure and mortality

Sidney Port, Linda Demer, Robert Jennrich, Donald Walter, Alan Garfinkel

Summary

Background The current systolic blood-pressure threshold for hypertension treatment is 140 mm Hg for all adults. WHO and the International Society of Hypertension have proposed that normal pressure be lower than 130 mm Hg, with an optimum pressure of less than 120 mm Hg. These recommendations are based largely on the assumption that cardiovascular and overall mortality depend in a strictly increasing manner on systolic blood pressure. The Framingham study was instrumental in establishing this viewpoint. We reassessed data from that study to find out whether the relation is strictly increasing or whether there is a threshold in this relation.

Methods We used logistic splines to model the relation of systolic pressure to cardiovascular and all-cause mortality with age and sex as risk factors. We used age-specific and sex-specific rates. We tested for the independence of the slope parameters from pressure, using age-specific and sex-specific rates. We used logistic splines to model the relation of systolic pressure to cardiovascular and all-cause mortality with age and sex as risk factors.

Introduction

Current medical treatment of hypertension in adults aims to lower systolic blood pressure, for all ages and both sexes, to a resting value lower than 140 mm Hg. This threshold, which is partly arbitrary, is used to define hypertension. The sixth report of the Joint National Committee on the Detection, Evaluation, and Treatment of Hypertension (JNC/6) recommends that normal systolic pressure is less than 130 mm Hg and that an optimum pressure is less than 120 mm Hg.1 Such an aggressive approach to hypertension is based on the general belief that there is a strictly increasing relation between systolic blood pressure and cardiac risk. For example, JNC/6 states, “This relationship is strong, continuous, graded, consistent, and etiologically
**Background** The current systolic blood-pressure threshold for hypertension treatment is 140 mm Hg for all adults. WHO and the International Society of Hypertension have proposed that normal pressure be lower than 130 mm Hg, with an optimum pressure of less than 120 mm Hg. These recommendations are based largely on the assumption that cardiovascular and overall mortality depend in a strictly increasing manner on systolic blood pressure. The Framingham study was instrumental in establishing this viewpoint. We reassessed data from that study to find out whether the relation is strictly increasing or whether there is a threshold in this relation.
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Threshold for hypertension. A substantial proportion of the adults. There is an age-dependent and sex-dependent 140 mm Hg is a useful cut-off value for hypertension for all adults. WHO risk are, therefore, at no increased risk.

Interpretation The Framingham data contradict the concept that lower blood pressure implies lower cardiovascular and overall mortality depend in a strictly increasing manner on systolic blood pressure. The Framingham study was instrumental in establishing this viewpoint. We reassessed data from that study to find out whether the relation is strictly increasing or whether there is a threshold in this relation.
Background The current systolic blood-pressure threshold for hypertension treatment is 140 mm Hg for all adults. WHO and the International Society of Hypertension have adopted the current viewpoint. We reassessed the Framingham study was instrumental in establishing this belief is wrong. Whether the relation is strictly increasing or whether there is a threshold in this relation.

The current cutoff for hypertension for systolic blood pressure, that is the upper number, is 140 mm Hg for everybody.
Background The current systolic blood-pressure threshold for hypertension treatment is 140 mm Hg for all adults. WHO and the International Society of Hypertension have proposed that normal pressure be lower than 130 mm Hg, with an optimum pressure of less than 120 mm Hg. These recommendations are based largely on the assumption that a threshold in this relation.

The WHO and the International Society of Hypertension has proposed that normal blood pressure should be lower than 130 mm Hg, and ideally below 120 mm Hg.
This is based on the assumption that the higher your blood pressure, the higher your risk of death, and the lower your blood pressure, the lower your risk of death.
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But this assumption is WRONG.
This assumption originally came from the Framingham study that was published in 1968 and has been believed ever since.
However, the Framingham study used the WRONG statistical model which led to this FALSE belief.

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The Framingham study was instrumental in establishing this viewpoint. We reassessed data from that study to find out whether the relation is strictly increasing or whether there is an optimum pressure of less than 120 mm Hg. These new standards have generated some concerns.3

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against the predictions of the linear logistic model, we tested for the independence of the slope parameters from age and sex, and the reduced model with common slopes was used to produce a model different from the conventional linear logistic model. UCLA statistician Sid Port re-ran the statistics on the data from the original Framingham study.
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UCLA statistician Sid Port re-ran the statistics on the data from the original Framingham study.

When he did, he found that they used the wrong statistical model leading to this FALSE belief.
Interpretation The Framingham data contradict the concept that lower pressures imply lower risk and the idea that 140 mm Hg is a useful cut-off value for hypertension for all adults. There is an age-dependent and sex-dependent threshold for hypertension. A substantial proportion of the population who would currently be thought to be at increased risk of cardiovascular and overall mortality depend in a strictly increasing manner on systolic blood pressure. The optimum pressure of less than 120 mm Hg. These recommendations are based largely on the assumption that lower blood pressure implies lower risk. A substantial proportion of the national population has blood pressures higher than the 80th percentile. Since systolic blood pressure higher than the 80th percentile is sufficiently high to allow accurate estimates of the shape of the relation with systolic blood pressure. We chose the 80th percentile as the cut-off for high systolic blood pressure.

After re-anlyzing the statistics on the original Framingham data, Port found...

The original Framingham data DOES NOT support the belief that “lower [systolic] pressures imply lower risk”...
Interpretation The Framingham data contradict the concept that lower pressures imply lower risk and the idea that 140 mm Hg is a useful cut-off value for hypertension for all adults. There is an age-dependent and sex-dependent relation.

Port also found that the original Framingham data also does NOT support the belief that “140 mm Hg is a useful cut-off for hypertension all adults.”
Interpretation The Framingham data contradict the concept that lower pressures imply lower risk and the idea that 140 mm Hg is a useful cut-off value for hypertension for all adults. There is an age-dependent and sex-dependent risk of cardiovascular and all-cause death with systolic blood pressure. We used logistic splines to model the relation of whether the relation is strictly increasing or whether there is a threshold for hypertension. A substantial proportion of the population is affected by antihypertensive drug intervention.

In other words, this belief is WRONG.
Here we are in June 2009, and, as far as I know, nothing has been done to correct this false belief.
It would be very simple to prove or disprove what Sid Port discovered.
All you’d need to do is give the same data to several statisticians around the world and let them analyze the data independently.
This would prove or disprove what Sid Port is saying.
But, as far as I know, this has NOT been done.
Many people are being frightening into believing the have hypertension, and that they must take a drug to lower their blood pressure.
Several older people have told me...
...“I’m taking this blood pressure medicine which makes me feel lousy...”
“... but I know that I have to take it if I don’t want to die.”
But for a majority of people who have been told that they have hypertension, this is NOT true.
For most people with moderately elevated blood pressure, they are at NO GREATER RISK OF DEATH.
I believe the reason that this false belief continues to be perpetuated is because...
...drug companies don’t want doctors and patients to know the truth...
... because it would dramatically lower sales of blood pressure medicines.
The drug companies perpetuate this false belief...
... in order to scare you into thinking you’d better take their drugs or else you will die.
Many researchers have been paid by the drug companies, which influences what they say.
This is most unfortunate, but true.
I will post other highlights from this brilliant paper.
I believe that potassium bicarbonate is vastly superior to all prescription blood pressure medicines...
... for improving health.
I’ve been taking 1000 mg of potassium twice a day (2000 mg per day) in the form of potassium bicarbonate since 2000.
My blood pressure dropped from roughly 140/80 mm Hg to 124/73 mm Hg.
WARNING: Only take potassium under a doctor’s supervision. Too much potassium can kill you.
I

Summary

Theoretically, we humans should be better adapted physiologically to the diet our ancestors were exposed to during millions of years of hominid evolution than to the diet we have been eating since the agricultural revolution a mere 10,000 years ago, and since industrialization only 200 years ago. Among the many health problems resulting from this mismatch between our genetically determined nutritional requirements and our current diet, some might be a consequence in part of the deficiency of potassium alkali salts (K-base), which are amply present in the plant foods that our ancestors ate in abundance, and the exchange of those salts for sodium chloride (NaCl), which has been incorporated copiously into the contemporary diet, which at the same time is meager in K-base-rich plant foods. Deficiency of K-base in the diet increases the net systemic acid load imposed by the diet. We know that clinically-recognized chronic metabolic acidosis has deleterious effects on the body, including growth retardation in children, decreased muscle and bone mass in adults, and kidney stone formation, and that correction of acidosis can ameliorate those conditions. Is it possible that a lifetime of eating diets that deliver evolutionarily superphysiologic loads of acid to the body contribute to the decrease in bone and muscle mass, and growth hormone secretion, which occur normally with age? That is, are contemporary humans suffering from the consequences of chronic, diet-induced low-grade systemic metabolic acidosis?

Our group has shown that contemporary net acid-producing diets do indeed characteristically produce a low-grade systemic metabolic acidosis in otherwise healthy adult subjects, and that the degree of acidosis increases with age, in relation to the normally occurring age-related decline in renal functional capacity. We also found that neutralization of the diet net acid load with dietary supplements of potassium bicarbonate (KHCO₃) improved calcium and phosphorus balances, reduced bone resorption rates, improved nitrogen balance, and mitigated the normally occurring age-related decline in growth hormone secretion – all without restricting dietary NaCl. Moreover, we found that co-administration of an alkalinizing salt of potassium (potassium citrate) with NaCl prevented NaCl from increasing urinary calcium excretion and bone resorption, as occurred with NaCl administration alone.

Earlier studies estimated dietary acid load from the amount of animal protein in the diet, inasmuch as protein metabolism yields sulfurous acid as an end-product. In cross-cultural epidemiologic studies, Abelow [1] found that hip fracture incidence in older women correlated with animal protein intake, and they suggested a causal relation to the acid load from protein. Those studies did not consider the effect of potential sources of base in the diet. We considered that estimating the net acid load of the diet (i.e., acid minus base) would require considering also the intake of plant foods, many of which are rich sources of K-base, or more precisely base precursors, substances like organic anions that the body metabolizes to bicarbonate. In following up the findings of Abelow et al., we found that plant food intake tended to be protective against hip fracture, and that hip fracture incidence among countries correlated inversely with the ratio of plant-to-animal food intake. These findings were confirmed in a more homogeneous population of white elderly women residents of the U.S. These findings support affirming

Diet, evolution and aging

The pathophysiologic effects of the post-agricultural inversion of the potassium-to-sodium and base-to-chloride ratios in the human diet

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LONG-TERM POTASSIUM SUPPLEMENTATION LOWERS BLOOD PRESSURE IN ELDERLY HYPERTENSIVE SUBJECTS

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SUMMARY Following a randomised cross-over trial of the effect of a four-week 60 mmol/day potassium supplement versus placebo on blood pressure (BP), eight of the original 18 hypertensive subjects continued with a 48 mmol daily potassium supplement for four months. For these eight subjects 24-h potassium excretion during placebo, one month of 60 mmol and four months of 48 mmol daily potassium supplementation phases was 56 ± 23, 102 ± 28 and 90 ± 35 mmol/24 hours, respectively, and mean 24-h BP following each phase was 160 ± 16/89 ± 11, 147 ± 13/83 ± 12 and 145 ± 14/81 ± 9 mmHg respectively; a significant fall in mean 24-h SBP between four months of potassium supplement and placebo period of 15 ± 13 mmHg (95% CI: 4, 26 mmHg, p=0.02), although the fall in 24-h DBP was not significant (8 ± 11 mmHg, 95% CI: 0, 17 mmHg, p=0.08). Modest increases in dietary potassium intake could have significant effects on lowering BP in the large proportion of elderly subjects with hypertension. (Int J Clin Pract 1997; 51(4): 219-222)
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Why not try potassium (bicarbonate) first?