THE GREAT BLOOD PRESSURE SCAM

Given the hype around blood pressure, you would assume it had been clearly proven that lowering blood pressure improves survival. Those studies don't exist. Instead, a universal assumption exists that lowering blood pressure is good; a related assumption is that if a drug can lower blood pressure, it must be good and should be approved. This may help explain why there has been such a strong focus on the theoretical benefit of lowering blood pressure versus actually proving a benefit exists.

The West's fixation on blood pressure is due to it being much easier to measure than blood perfusion (healthy blood flow)**. When ever a drug exists that can treat a number or statistic, as the years go by, the acceptable number keeps on being narrowed, making more and more people eligible to take the drug. For example, once statins entered the market, the acceptable blood cholesterol levels kept on being lowered, and before long, almost everyone was told they would die from a heart attack unless they started a statin—despite the fact that statins have an almost non-existent mortality benefit and often cause severe side effects for roughly 20% of users.**

Broadly recommending these drugs appears unconscionable, but these unjustifiable guidelines are a product of clever pharmaceutical marketing and targeted bribery of public officials. In fact, many of the dogmas that underlie popular drugs are actually sales slogans created by a marketing company. A chemical imbalance from low serotonin has never been linked to depression (in fact, patients who commit suicide have elevated brain serotonin)**; acid reflux is due to too little acid in the stomach, though medical schools teach that it is due to too much acidity; and "sleeping" pills are sedatives that block the restorative phase of the sleep cycle. Each of these drugs is immensely harmful, but, due to how solidly these myths were established** (just like "safe and effective")**, large numbers of people continue to use them. Something similar happened in the field of blood pressure** (BP)**.**

One of the most pivotal studies began in 1973. With no studies offering proof that lowering a moderately elevated blood pressure reduced one's chance of dying, researchers in the UK undertook a massive, decade-long public study. The study recruited over 17,000 patients between ages 35 and 64 who had blood pressures below 200 (averaging 158/98 in men and 166/99 in women) **and randomly allocated them to take either a beta blocker, a thiazide diuretic or a placebo.**

The study showed that treating high blood pressure made no real difference in the likelihood of a coronary event such as a heart attack (the rate went from 5.5 to 5.2 per thousand patient-years)**, and likewise, almost no difference in the death rate** (253 versus 248 deaths—or a 0.06% reduced chance of dying)**. The study did show that lowering blood pressure made someone less likely to experience a stroke** (probably hemorrhagic)**, as there were 18 fatal and 42 non-fatal strokes in the treatment groups, versus 27 fatal and 82 non-fatal in the placebo group; this led to a small overall reduction in those strokes** (roughly 1 stroke delayed for around 900 years of treatment)**. On the other hand, roughly 25% of trial participants experienced side effects significant enough for them to withdraw from the study.**

After the trial, the term "heart disease" was abandoned in favor of "cardiovascular disease." This made it possible to create the perception that treating blood pressure stopped heart attacks, while simultaneously concealing the actual benefit—a small reduction in vascular strokes. The 2009 and 2020 Cochrane reviews both found that the negligible benefit from aggressive blood-pressure-lowering target was outweighed by the harms those drugs created.

Both reviews found a small increase in kidney disease (acute injuries or chronic kidney disease) **which makes sense because the kidneys are damaged by insufficient blood reaching them; kidney disease creates heart disease and raises blood pressure.**

When the blood pressure craze took off, there was a rush to bring blood-pressure-lowering drugs to market before actual proof of their benefit. That mindset continues to define this field. When your only tool is a hammer, everything starts looking like a nail. Experts on guideline panels are paid to create recommendations that result in more people taking the drugs.

As the years have gone by, the blood pressure thresholds keep getting lowered without supporting evidence, and more people are put on medication—roughly sixty million American adults. Originally, the focus was on treating DBP under the belief that the heart had to work harder if there was too much blood in the circulation; that this was believed for decades—but now is not—illustrates how arbitrary many medical dogmas are.

In many cases, the actual mechanism of a drug greatly differs from the purported one. Statins are sold on the basis of the claim that cholesterol causes heart disease and that they lower cholesterol. However, there is extremely little evidence that cholesterol causes heart disease, and more importantly, prior to statins hitting the market, there was no mortality benefit shown from other cholesterol-lowering medications.

Presently, four main types of antihypertensive drugs exist: *diuretics* (the oldest)**; *beta-blockers*; *calcium channel blockers*, and *ACE inhibitors* and related medications. Each drug works in a different manner, either by loosening the arterial walls, reducing the total blood in circulation or weakening the contraction of the heart for a combination of all three, with very different degrees of benefits seen from their use, despite them creating the same drop in blood pressure. This strongly argues that their effects are not due to lowering blood pressure, but rather to how each one specifically affects the body.**

Because less elevated blood pressures are more common than the higher ones, each time the treatment target is lowered by a small amount, it results in a large number of people getting started on the drugs. This is especially the case for the elderly, whose blood pressure rises to compensate for the decreased health of their arterial system; due to their calcified arteries, they have the least ability to tolerate insufficient blood pressure and simultaneously are the most likely to have elevated blood pressure.

Rather than 1 in 3 adults having high blood pressure (32%) **with the previous definition, the new guidelines will result in nearly half of the US adult population** (46%) **having high blood pressure, or hypertension. According to the revised guidelines, 79% of men and 85% of women over age 75 now have hypertension, and 71 and 78% of men and women in that age group, respectively, meet the threshold to start blood pressure medications.**

- **A 1998 review found that the known cardiovascular benefits of ACE inhibitors were not seen with calcium channel blockers, despite the latter having a more significant effect on blood pressure.**
- **A 2000 study of almost 3,600 diabetics found that a specific ACE inhibitor, despite minimally reducing blood pressure** (a reduction in SBP and 1.0 reduction in DBP)**, had a massive effect** (a 25% reduction) **on the risk of a heart attack, stroke or cardiovascular death.**
- **An eight-year-long double-blind study published in 2007 and funded by NIH, involving over 42,000 subjects, found that when two different types of blood pressure medications were used, there was no difference in their effect on blood pressure; however, their rate of preventing heart failure varied by 18 to 80%, depending on the drug, leading the investigators to conclude, "blood pressure reduction is an inadequate surrogate marker for health benefits in hypertension."**

CONVENTIONAL PERSPECTIVES

Once the outflow from the heart gets obstructed (for example, through aortic stenosis)**, the heart's left ventricle is known to become enlarged** (this is called *left ventricular hypertrophy* of LVH)**, yet, we typically ascribe the hypertension associated with LVH to being the *cause* of it, rather than a *result* of it. Similarly, if the blood flow through the lungs gets obstructed** (due to pulmonary hypertension)**, the right heart ventricle enlarges** (called *right ventricular hypertrophy* or RVH) **and eventually fails.**

In certain cases, this hypertension can be immediately reversed—for example, if it's being caused by chronic blood clots entering the lungs—at which point the heart immediately recovers. Another observation is that significant plaque within the artery that feeds the kidney raises blood pressure; if the kidney's blood flow is restored, the blood pressure immediately drops.

Because blood vessels are elastic structures filled with fluid, that fluid holds them under pressure. Blood pressure, in turn, is typically measured by determining how much external force is needed to exceed the artery's pressure is needed to exceed the arteries' pressure and compress it so that blood no longer flows through it. *Low* blood pressure (hypotension) **is a problem because it prevents blood from reaching the areas where it's needed; for example, *orthostatic hypotension* or *postural orthostatic tachycardia syndrome*** (POTS) **describe a common situation where people become lightheaded as they stand up, due to insufficient blood being pushed into the brain. In most cases, though, medicine chooses to focus on the consequences of high blood pressure.**

Within the existing model, one of the consequences of high blood pressure is that weakened blood vessels become more likely to break open and leak as higher blood pressure pushes against them. This is why emergency departments aggressively lower the blood pressure of patients who show up with symptoms of "hypertensive emergency" (such as a severe headache and a significantly elevated blood pressure)**. Likewise, whenever a critical blood vessel ruptures** (such as the aorta or in the brain)**, once the bleed has been confirmed, the first step in managing it is to lower the patient's blood pressure so less blood leaks out, after which they are sent to surgery.**

Other consequences of high blood pressure—according to the conventional model—include excessive pressure on the arteries (which strains and damages them, causing the lining of the vessels to become damaged and gradually develop atherosclerosis) **and damage to the internal organs** ("end organ damage")**, leading to premature failure and early death** (for example, from a heart attack or kidney failure)**.**

Because of these situations, high blood pressure is viewed as one of the greatest preventable causes of cardiovascular disease. Thus, a chief focus of all medical visits is ensuring that a patient achieves a sufficiently lowered blood pressure. Unfortunately, the chain of logic has quite a few gaps in it.

BLOOD PRESSURE VARIABILITY

Blood pressure is immensely variable. Pressures at the periphery (where BP is typically measured) **have been found to vary by around 14 points. The blood pressure should be measured 3 times, and the third reading should be taken as the true reading. The first two are going to be high from the body trying to overcome the resistance of the blood pressure cuff. This frequently leads to individuals being erroneously diagnosed with *hypertension* and given a prescription for blood-pressure-lowering medications, despite having normal blood pressures; those medications can then make them *hypotensive*. This phenomenon is so common** (constituting 15 to 30% of hypertension diagnoses) **that it is often referred to as "white-coat hypertension."**

Stress commonly elevates blood pressure; because visiting a doctor can be stressful, many patients have temporarily elevated blood pressures in the doctor's office.

The guidelines suggest the need for multiple measurements to confirm a hypertension diagnosis (for example, with home blood pressure monitoring)**, but unfortunately, this often does not happen in practice.**

There are other common sources of error when measuring blood pressure. One is using the wrong-sized cuff, and another is that patients frequently have significantly different blood pressures in each arm. This helps to explain why it is estimated that 25% of those diagnosed with hypertension *do not have it*.

Likewise, there is a surprisingly poor correlation between *peripheral* blood pressure and *central* blood pressure inside the aorta. One large study found a significant difference between the blood pressure within the aorta and the arm, and the central aortic pressure had a much stronger correlation to the likelihood of cardiovascular disease. Different classes of blood pressure medications have very different effects of *central* versus *peripheral* blood pressure.

WHAT AFFECTS BLOOD PRESSURE?

If a fluid at a set pressure tries to move through a tube, as the tube shrinks, the pressure it creates (for example, on the walls of the tube) **will *increase*, while, if the tube enlarges, the pressure it exerts will *decrease*. The body, in turn, continually controls where blood in the body goes by changing the heart rate and fully or partially constricting the arteries, allowing it to shunt blood to where it is most needed** (such as by dilating arteries in that area)**.**

Blood pressure is thus a product of two factors: the amount of blood in the arteries and the constriction or relaxation of the arteries containing it. *Arterial* BP is greater than *venous* BP and is what is measured externally; veins compress long before arteries do, and only arterial blood has a signature pulsatile wave created by the heartbeat. Because each beat of the heart pushes blood into the arteries and hence increases the pressure within the, two different blood pressure values exist—the baseline pressure (*diastolic* blood pressure DBP) **and the pressure when the heart contracts** (*systolic* blood pressure or SBP)**.**
The blood pressure values you see (such as 140/90) **represent the maximum and minimum *(the systolic over the diastolic)*. One reason why this stretching is important is because when the vessels contract back to their normal size once the systolic pressure fades, that recoil pushes blood further along into the circulation.**

WHAT CAUSES BLOOD PRESSURE?

Most cases of high blood pressure (90 to 95%) **are known as "essential" or "primary" hypertension, which is a fancy** (and rarely questioned) **way of saying "elevated blood pressure without a known cause." The belief that there is no known cause for most cases of elevated blood pressure has been widespread in allopathic medicine for decades. The remaining 5 – 10% of cases** ("secondary" hypertension)**, recognized causes include reduced blood flow to the kidneys** (which sets off a signal to raise the blood pressure because the kidney believes there isn't enough blood perfusion)**, sleep apnea, or having a rare tumor, which dumps large amounts of adrenaline into the blood, thereby constricting blood vessels and increasing the heart rate.**

A kidney (especially the left) **being in the wrong position—which is quite common—can functionally compress the renal artery. However, until there is an actual *stenosis*** (narrowing) **of the artery, this can be quite difficult to identify with conventional measurements. Additionally, poor sleep is immensely damaging to cardiovascular health, and those effects extend to blood pressure. In one study, a single night of partial sleep deprivation raised SBP by 6 and DBP by 3. Another study found that a night of sleep loss raised SBP by 4.5 and DBP by 2.6, and functional magnetic resonance imaging** (fMRI) **showed that sleep loss also impaired the brain's control of blood vessel function.**

Because the causes of most cases of hypertension aren't known to medicine, they focus on specific "risk factors" that are known to be associated with it, such as being over age sixty-five, having diabetes, insomnia,

obesity, not exercising, stress, being an alcoholic, having other people in your family who have high blood pressure, or "eating too much salt." The latter is the one we tend to hear most about, despite the fact that the most detailed review of this subject showed that drastic salt reduction typically results in less than a 1% reduction in blood pressure.

One of the most underappreciated causes of hypertension is exposure to microwave EMFs (cell phones, WiFi, Bluetooth, etc.) **and artificial lighting** (LED, fluorescent, halogen)**. These exposures trigger the *paraventricular nucleus* in the hypothalamus, which creates sympathetic dominance** (the fight-or-flight response)**, which raises blood pressure and blood sugar. Also, anxiety; frequently, effectively addressing anxiety can resolve a case of high blood pressure, which would otherwise receive pharmacologic treatment, often indefinitely.**

ATHEROSCLEROSIS AND BP

Many became suspicious of the existing blood pressure paradigm because they noticed that circulatory impairments would proceed or occur in tandem with elevating blood pressure, rather than happening long after an elevated blood pressure had had time to damage the arteries. Each came to a similar conclusion—the increased blood pressure must be a compensatory mechanism the body is using to counteract the fact that it is needed (which is recognized to occur with the kidneys and the brain)**.**

Calcification of the arteries (which stiffens them) **also increases blood pressure, as they can no longer expand as effectively and release the pressure within them. However, there are also three other reasons why this could occur.**

First, measuring blood pressure utilizes an external cuff to pressurize the arteries until the blood flow cuts off. If the artery has stiffened, that is much harder to do. When patients have severe atherosclerosis, their blood pressure is no longer measured in the arm's arteries, as those arteries won't compress from the cuff. At the same time, however, there is most likely a gradual stiffening that leads to the measured blood pressure being higher than it actually is.

Second, the health of the cardiovascular system depends to a large extent on the lining of the blood vessels being able to secrete nitric oxide, which, in turn, dilates the blood vessels, locally decreasing blood pressure and regionally increasing blood flow. Defective production or activity of nitric oxide (endothelial dysfunction) **typically precedes the development of atherosclerotic plaques and is much more severe by the time they've formed, as the surface of the endothelium has calcified at that point.**

For this reason, many believe the core issue is not calcification of the arteries or high blood pressure, but rather endothelial dysfunction, which in turn leads to abnormal growth of the endothelium and formation of harmful blood clots. Given that endothelial dysfunction happens to increased blood pressure, that again argues that the direction of causality for heart disease is incorrect.

Third, whenever the body is not getting enough blood, a sympathetic reflex will be triggered to increase the BP. For example, this is commonly seen in critically ill patients or those who have rapidly lost a significant amount of blood, at which point the blood vessels will tighten and the heart rate will increase.

BLOOD SLUDGING

Approximately a century ago, many Western researchers argued that blood cells clumping together—a condition that came to be known as "blood sludging"—was a root cause of many illnesses, particularly where a focal issue (such as burns or cancers) **created a systemic problem. This theory became quite influential, especially once it was possible to directly observe the phenomenon by using the appropriate microscope to look at the blood vessels in the eyes.**

Sadly, the concept of blood sludging has largely been forgotten and persists today only through live blood analysis, which is not only widely disparaged by allopathic medicine but is significantly more error-prone than the original method of looking into the eyes.

Once pockets of sludge are formed, the pressure build-up causes plasma (the non-cellular component of blood) **to begin leaking out into the tissues, leading to things like edema; the remaining blood thus becomes even more concentrated with these sludge pockets. Blood sludging disproportionately affects the smaller vessels** (because it takes significantly smaller sludges to obstruct them)**, the primary effect of blood sludging is injuries akin to microstrokes. However, because the larger blood vessels have their own small blood vessels** (the *vasa vasorum*, which sustain the arteries)**, the early generation of researchers frequently observed blood sludging to obstruct the vasa vasorum in an identical manner to what happens in smaller sludged vessels.**

Once the blood supply in a vessel's vasa vasorum is impaired, it causes the endothelial lining of the blood vessel to rapidly die and fall off, with the total damage in proportion to how much the blood supply is interrupted and how long that interruption persists. Once this happens, those lost cells can no longer release the critical nitric oxide; as a result, clots frequently form, and the blood vessel is no longer protected from damage (as endothelial cells create the primary protective layer of the blood vessels)**.**

Zeta potential is a theory of coagulation and dispersion measured as the electrical charge difference between a colloidal particle and its surroundings. (Colloidal particles are small particles, ranging in size from nanometers to microns, dispersed in fluid.) **A high zeta potential confers stability to colloidal systems, whereas conversely, colloids with low zeta potentials tend to coagulate. Vaccines—including the COVID shots and also the aluminum that many so-called "traditional" vaccines contain—are significant factors impairing zeta potential.**

Poor zeta potential is a primary cause of arterial damage (and the ability of vessels to then repair themselves) **and the lethal clots that damaged the endothelium can form. You can treat a variety of heart conditions by restoring the physiologic zeta potential. If the net charge of a colloidal system is made more negatively charged, particles will become more dispersed; if, on the other hand, the net charge becomes more positively-charged, particles will begin to clump together, forming successively larger colloidal agglomerations. Because the body depends on the continual circulation of fluids, this clumping can be extremely problematic. For example, once red blood cells clump together, microstrokes often follow.**

Each mobile agglomerate must be broken down into smaller sized particles as it flows along the arterial tree. The blood cells must eventually become completely discrete if they are to pass through the capillaries, whose small diameter permits the cells to pass only in single file. Considerable energy must be required to continuously break down these clumps ahead of each capillary—and then reform them past the capillary. Impaired zeta potential and deformed red blood cells correlate with poor health. Small wonder the workload of the myocardium is increased and blood pressure raised. This is one reason why grounding is so important for supplying the body with abundant electrons, which shift the zeta potential more negatively.

When researchers in 2019 compared the zeta potential of four groups—hypertension patients, myocardial infarction (MI) **patients, treated MI patients and healthy controls—they found significantly reduced zeta potentials in the first three groups compared to the controls. Likewise, when they looked at study participants' blood, they found significantly more clumping, blood cell deformation, and membrane fragility in cells from patients with cardiovascular disease.**

Several observations originally led researchers to hypothesize that high blood pressure might be the cause of heart disease.

- **First, high blood pressure has been observed to occur in association with heart disease.**
- **Second, significant vascular issues such as severe headaches, strokes and organ damage can occur when blood pressure is significantly elevated** (such as SBP above 200) **and improve once the blood pressure is rapidly lowered.**
- **Third, blood vessel damage never occurs in low-pressure areas of the body but does occur when blood vessels are suddenly transitioned to much higher pressures than normal, such as a vein being grafted onto the heart or the blood pressure suddenly being greatly elevated in the lungs.**

Unfortunately for the "high blood pressure causes heart disease" hypothesis, there's actually very little evidence that smaller elevations in blood pressure create issues. Much of the current dogma is based on the long-standing Framingham Heart Study, which "found" a linear relationship between blood pressure and the risk of death—meaning that continually lowering blood pressure decreases one's risk of dying. That has been the basis for continual recommendations for lower and lower blood pressures (though the National Institutes of Health n(NIH) also admits that an SBP below 90 is dangerous and unhealthy) **later found to overestimate one's risk of dying by 500%.**

Major problems with the paradigm include the following:

- **It creates a situation where no optimal blood pressure can exist. You absolutely cannot be healthy anymore—it's official. We have reached a situation whereby a SBP lower than 90 mmHg increases risk; and a blood pressure higher than 90 mmHg increases risk. So, you could say that anyone with a blood pressure of exactly 90 mmHg is healthy, so the land of health still exists as a microscopically thin sliver of habitable area. But for all intents and purposes, health has gone.**
- **It's exceedingly rare that things in biology follow a completely linear relationship.**
- **No one has ever done a study demonstrating that lowering blood pressure from 100 to 90 provides a benefit. Rather benefit is assumed from the linear model's prediction.**

Moreover, the original analysis of the Framingham study was flawed. Reanalysis of the data in 2000 instead found a relationship that was much more congruent with what's typically seen in nature. Specifically, rather than being a linear relationship, blood pressure had relatively little impact on mortality until a critical threshold was passed (going above 70 to 80% of the normal blood pressure for the person's age and gender.)**, at which point there was an exponential rise in their risk of death. A large number of people are diagnosed as having dangerous hypertension in the *linear* model, but not in the more accurate *threshold* model.**

The 2000 paper was mostly ignored. However, it eventually received this response from the National Heart, Lung, and Blood Institute (NHLBI)**, illustrating how resistant medical dogmas are to evidence that refutes them: "After careful review of this study, the NHLBI finds that id does not offer a basis for changing the current hypertension guidelines.**

Recent research with more modern technologies has led to the same conclusions. Rather than being linear, an age-dependent threshold exists that is not recognized by the guidelines. A SBP below 130 significantly increased the chance of death, and a DBP below 80 (a common consequence of blood pressure medications) **made patients 8 to 19% more likely to die and, once again, an SBP under 120 was associated with a higher risk of death.**

ANTI-HYPERTENSIVE MEDICATION HARMS

The typical allopathic management of blood pressure involves using a combination of drugs until they collectively achieve the desired blood pressure and simultaneously switching out drugs that cause more side effects than the patient can tolerate.

This is a problem because the drugs have very different effects on the body. On one hand, this is a good thing because it allows each of them to exert unique "benefits," but on the other hand, it means they each have unique side effects. Each should be considered on the basis of whether their individual effects are appropriate for the individual patient's situation, rather than whatever achieves the desired blood pressure—but as that would get in the way of drug sales, it never happens.

Typically, the most common side effect of blood pressure medications are complications of poor perfusion. BP medications increase the risk of fainting by 28% and are notorious for causing older individuals (who have calcified arteries and hence, difficulty getting blood to the brain) **to become lightheaded and then suffer potentially devastating falls. Three of the more troubling side effects are an 18% increased risk of acute kidney injuries** (affecting 1.5% of users)**; a 103% increased risk of *hyperkalemia*** (high potassium)**, which can be quite dangerous and affects 4.8% of users; and a 19% increase in the risk of lung cancer.**

To illustrate the problem with falls, a 2014 JAMA study of almost five thousand hypertensive adults over age seventy monitored three groups for three years: 14.1% received no antihypertensive medications, 54.6% were on moderate intensity medical therapy and 31.3% were on high-intensity medical therapy. Over the three-year period, 9% experienced falls, and 16.9% died. Those in the moderate-intensity treatment group were 40% more likely to have a fall that caused a serious injury, and 117% more likely to have a serious fall if they had a previous fall history. The high-intensity group was 131% more likely to have a serious fall (which begs the question of why they were still on those drugs)**. The authors did not report the rate of death between the groups but did find that calcium channel blockers had the highest rate of causing falls.**

In 2007, an important Israeli study found that discontinuing an average of 2.8 drugs per elderly patient reduced their one-year death rate from 45% to 21% and their hospitalization rate from 30% to 11.8%. The study did not provide specific data, but a significant degree of the benefit came from removing the antihypertensives that have become a core principle in geriatrics.

Emergency medicine recognizes that it is unwise to aggressively treat blood pressure in the emergency department (ED)**; the risk is that it will cause an ischemic stroke due to insufficient blood flow in the brain. It is worth quoting a review paper on this subject. Noting that the majority of IV medications given to achieve immediate BP reduction in the ED are done so inappropriately. There is no evidence-based thresholds at which asymptomatic but markedly-elevated BP in the ED benefits from immediate reduction. Rapid BP reduction can cause significant harm by impairing cerebral blood flow, and it has not been shown to improve clinical outcomes except in hypertensive emergencies.**

Many serious diseases result from low blood pressure, especially in the organs that are most sensitive to loss of blood flow. Low blood pressure is strongly linked to cognitive decline (the brain needs adequate blood to function)**. Likewise, as you lower blood pressure, the kidneys start to struggle as they, too, require sufficient blood flow to function. As we already saw, hypertension drugs increase the risk of an acute renal injury by 18%. In patients who had end-stage renal disease, those with blood pressures below 130 were 38.9% more likely to die compared to those with blood pressures between 10 and 149.**

When one's blood pressure is below 90, it is diagnosed as "hypotension." Critical care medicine views blood pressures below 90 as dangerous because organs do not get enough blood. The most common symptoms of hypotension are lightheadedness or dizziness. Other common symptoms include fainting (when it gets lower)**, blurry vision, confusion, nausea or vomiting, sleepiness, fatigue, and weakness. Given the high inaccuracy of blood pressure measurement and the fact that patients often are put on excessive hypertensive medications** (especially as they age and the body is less able to handle low blood pressures)**,**

these symptoms affect many blood pressure medication users to varying degrees. Stated another way, blood pressure medications can increase one's risk of developing hypotension.

The concerning data presented about side effects probably underestimates the actual rate of side effects because much of the data comes from industry clinical trials that deliberately find ways to downplay their drug's side effects. Independent patient surveys likely provide a far better perspective on the rate of symptomatic side effects. A Swedish survey, published in 2000, found that roughly one in five users experienced side effects.

Because of side effects, patients frequently stop taking antihypertensives. In a large study of three hundred seventy thousand patients under age sixty-five conducted between 2007 – 2014, almost one in four participants (23.5%) **stopped taking the drugs within roughly nine months of starting them, while 40.2% who continued taking them often skipped the medications. Large studies have found that patients are least likely to stop using ACE inhibitors** (and the related angiotensin II receptor blockers or ARBs) **and are most likely to stop using diuretics and especially, beta blockers. This is congruent with the rate of side-effects observed in practice.**

Given such high discontinuation rates, it is surprising how little awareness exists regarding blood pressure drugs' side effects, especially among medical doctors. In a 1982 study that compared how patients, their families and their doctors felt about the effects of these drugs. 48% of patients were taking beta blockers, 25% were on beta blockers plus diuretics, and 12% on diuretics only.

When asked whether quality of life had improved, stayed the same, or become worse on the blood-pressure-lowering drugs, 100% of doctors answered "improved," versus less than half; (48%) **of patients; meanwhile, 99% of relatives assessed their loved one's quality of life as worse, and 9% of patients agreed with that assessment. Most relatives characterized the worsening as moderate** (45%) **or severe** (30%)**, with commonly cited deterioration, including an undue preoccupation with sickness, irritability, and a decline in energy or general or sexual activity.**

SALT

Patients diagnosed with high blood pressure are frequently advised to undertake aggressive salt lowering. We do not support this approach because significant salt reduction has almost no effect on blood pressure. Furthermore, many people don't consider that hospital patients re routinely given large amounts of IV sodium chloride (9 grams of sodium in one bag of IV sodium chloride)**—receiving up to ten-times the daily recommended sodium chloride—but their blood pressure does not rise.**

Low sodium levels are strongly correlated with a risk of dying. Specifically, the salt consumption target we are told to follow increases one's risk of dying by 25%. A common reason for hospital admissions are symptoms resulting from low sodium levels; 15% to 20% of hospitalized patients have *low* sodium levels at admission. Many of the associations are due to the effects on the zeta potential of the aluminum, often contained in processed salt as a desiccant; this is something not found in natural salt products or in IV saline given at hospitals. Conversely, many of the benefits from hospital care are a result of IV fluids being routinely given as they somewhat restore the physiologic zeta potential.