Hello all,

I want to thank everyone for considering my petition calling on the NIH to end their silence regarding sub-optimal breathing and high blood pressure. The petition is still going strong so please sign on!

A bit of background on this... In 2006, two colleagues and I applied for an NIH research grant to study the correlation between breathing, heart rate variability, and blood pressure where a potential outcome, depending on findings, was that modification of the breathing pattern might be appropriate to mitigate elevated arterial pressure. The grant received review and was declined. One of the reasons it was declined was that, “we already know this” (that breathing frequency and depth is a key determinant of blood pressure). The disappointing thing is that if the NIH already knew this, they have been silent for 8 years now, at a time when the physical and financial health of the nation are in peril. The petition calls on the NIH to end their silence and add healthful breathing to their recommendations for managing hypertension, which already include healthy diet and exercise.

Regarding the petition, I’ve received a number of emails asking for clarification as to exactly how breathing affects blood pressure. I respond to those queries in this issue of Alternativz, titled simply, Inhale For Life. I’ll start with this assertion:

*If we inhale effectively, we are creating the conditions for normal blood pressure, and conversely, if we don’t we are creating the conditions for elevated blood pressure.*

In fact, if we observe arterial pressure, every time we inhale it drops (and every time we exhale it rises). But effective inhalation is key to reducing the pressure in the entire circulatory system in the long run. Here’s why...

Circulation consists of this loop: lungs -> left heart -> arterial tree -> capillaries -> venous tree -> right heart -> lungs. The autonomic nervous system concerns itself with maintaining blood flow in this loop, for without adequate flow cells perish. Sensors exist throughout major arteries that observe and gauge flow. When flow is low, the nervous system has the means of increasing it. It does so by increasing pressure. The simple formula for determining flow is:

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\text{Flow} = \frac{\text{Pressure}}{\text{Impedance}} \text{ (resistance to flow)}
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So if the sensors throughout the arterial tree signal that flow is low, the nervous system effects an increase in pressure to increase flow. Pressure is increased in two primary ways. The first is by increasing the stroke output and power of the heart beat, i.e. the heart pumps more blood and it pushes it into the aorta under higher pressure. Think of this as increasing the water pressure in your home so the upstairs shower has adequate water pressure.
The second is by reducing arterial capacity. Now think of a 1 inch garden hose full of water that suddenly shrinks in diameter. Pressure in the hose increases instantly and water squirts out the end under significant pressure. This is the kind of control that the nervous system has over the muscular walls of the arterial tree, i.e. it has the ability to narrow and widen arteries to govern pressure and flow.

Now, as a rule elevated blood pressure is a consequence of the nervous system facilitating one or both of these changes in the body for some reason. In the case of secondary hypertension (~5% of cases) it is principally due to organic obstruction of the arterial tree, the lungs, or the kidneys. I posit that essential or primary hypertension (~95% of cases) is also due to “obstruction” but this obstruction is not caused by failing organs. It is ultimately caused by relatively positive quiescent pressure in the thoracic cavity that prevents blood in the venous tree from returning to the chest. When this happens, venous blood flow slows and backs up. (In Chinese medicine this condition is referred to as “blood stagnation”.) When venous flow slows, arterial flow is forced to slow. When arterial flow slows, the nervous system calls for more pressure so as to maintain viable flow. When this happens, arterial pressure rises. (It’s interesting to note that when venous and arterial flows don’t match fluid begins to build up in the capillary circulation. This is a symptom of congestive heart failure.)

For most of us, primary hypertension is preventable by making sure that our inhalation is effective. This is because inhalation creates what amounts to a vacuum in the chest to which venous blood naturally returns via the right heart. Inhalation is a critical motive force that is necessary for effective circulation. Inhalation empties the venous tree (see Figure 1) making plenty of room for new blood flowing into the venous tree via the capillaries. When capillary circulation is robust, then the capillaries accommodate a higher volume of arterial blood, “circulation” continuing unimpeded.

Much of the blood that flows into the chest via the right heart during inhalation accumulates in the dense pulmonary capillary bed which can accommodate hundreds of milliliters, maximally about a wine bottle full of blood. When we exhale this blood is ejected via changing thoracic pressure which goes relatively positive, and by the highly compliant and elastic pulmonary capillaries. It makes its way through the left heart into the aorta where the pressure is once again elevated.

Thank you for your interest, Stephen Elliott