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Letter to Editor

Myriad Evidence and Presentations Spell out Upper Respiratory Infection Should be Responsible to the Essential Hypertension Especially the Systolic Phenotype that Is a Functional Compensation Counteracting Chronic Hypoxia Attack on Ventral Brain

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Letter to Editor

Hypertension is named a chronic Blood Pressure (BP) attack. With regarding to WHO documents in 2024, one over third prevalence among adults whose age covering 30–79 years [1]; and the serious is bring on a high incidence of vascular attacks. Unfortunately, till now, the cause has long been reckoned of idiopathic [2] and popularly believes due to the mechanism of BP regulation malfunction with wrong parameters withal the corresponding treatment is long-haul aiming to improve the vascular dynamic or involved. In present etiology, sympathetic outflow postulation is a polar for which note, in particular, almost 200 years ago people had already set their eyes on the sympathetic nervus for available to impact blood vessel elasticity [3] via which nervous system could reach out to regulate BP that, meanwhile presents a kind of global consistency through body. Now, it is well-known that with neuro-vascular reflex Autonomic Nervous System (ANS) is assigned to undertake this BP job.

Accordingly, people exploration on this BP attack may be translated to interrogate how a pathological factor induces disorder of sympathetic nervus; thus, hypertension actually becomes a subset of certain disease; however, whom we should recognize. For example, a plausible hypothesis sounds respiratory centre attack [4] yet the following question is what attack can damage such region that residing in medulla oblongata, and what pathway it adopts to commit its intruding? Similar questions likewise confront in peripheral components of ANS, such as the supposed transcranial sensors in dysfunction leads to error signal sent to central neurons that mislead sympathetic outflow to make cerebral hypoxia or ischemia [5]. For prove that, the first should be moving to make clear the sensor in histology or with what biochemistry characters.

An interesting note about the association between hypertension and neurodegeneration [6]. In which, neurodegeneration can be ascribed to hypertension for its pressure giving rise to bring on a persistent elasticity worsening in cerebral vasculature. But why not is the inversed case that ANS reaches out to upraise BP as a functional compensation in attempt to improve itself condition?

Well, with these arguments recapped the one our propose is not more than glucose metabolism anomalous in ANS whose neurons uptake sugar exceeds their capacity of translating to ATP by aerobic glycolysis; It can say, in our framework of pathogenesis, the uncanny is that other regions in cranium likely run regularly. Developing such pathology can be ascribed to Upper Respiratory Infection (URI) and its chronically foster; meanwhile that implicates this aberrant damage existing in everybody upon the earth, that tallies with its high prevalence. Herein, we suppose a chronically local hypoxia other than general note of global attack. Apparently, our postulation motivates why hypertension is so common and able to bring on a global impact via ANS neuropathology. On the other hand, the most people likely think of ridiculous for no sign indicates rhinovirus widespread invading people's brain. Even to covid in

which patients died, in whom, people still have not revealed any decisive bio-evidence, what is less; the sympathetic nervus attack existing.

Inside our framework proposed in [7] it reveals a physiological pathway may be abused to transfer overloading glucose into cranium bottom instead of pathogen's direct invasion that amounts to a process of chronically hyperglycaemia attack on ANS. It can say, without ANS exposure, pathogens still reach out to debilitate ANS; even more to if we generalize this model, the injury can be accrued from myriad factors to share so long as they can bring nasal mucosa with inflammation. For complexity of pathological sources, we thus know why the hypertension so refractory; on opposite, ANS that itself must reach out to solve this hyperglycaemia problem.

Apparently, as though the anaerobic glycolysis also is an important energic metabolism in nervous system [8] yet simultaneously yielding lactate an adverse impact is PH thus declines capable of worsening the homeostasis ANS lives on. Consequently, whereas oxygen saturation in serum impossibly of endless elevated, upraise BP so as to elevate the cerebral perfusing becomes the best option except sterilizing rhinovirus that often is the chief pathogen. This explains why the type of systolic BP is so common in low-land communities [9]; and answer why the isolated systolic phenotype BP more difficult in control than isolated diastolic if only in use of vascular protocol [10]. The high incidence among middle-aged people likewise implicates the consistency with our commonsense that number of age often is used to indicate people who begin with senescence in which course; hyperglycaemia growing in ANS starts chronically inhibiting body metabolism. This presents a strong consistency in all populations of animals. It explains that why coronavirus with the strongest virulence among well-known rhino pathogens, which epidemic brings with a high risk of hypertension [11].

For the case in out hand, a patient who is female with up to 80 years old of moderate overweight without diabetes, within 12 years hypertension of isolated systolic type for exceeding 180 mmHg has long been consulted as underlying condition. From February 2024, along with nasal disinfection against covid, out of our expectation, the systolic BP frequently below 140 mmHg that led to reducing maintaining dosage of calcium channel antagonist to half. The importance is her without any remarkable presentation of mental attack that may be credited to excessive-lower BP common seen in antihypertensives therapy the side effect. Without losing weight it sounds a spontaneous eradication; in particular, compliance of nasal disinfection presents with the positive correlation reflected on BP.

Discussion. We post the hypertension pathogenesis on the ANS condition. There is a logical principle if in presence of myriad causative factors impacting BP. For example, while peripheral sensors report wrong information, ANS still correct BP in dependence on the data from itself metabolism. That explains the denervation arterial baroreceptor does not effectively maintain the chronic hypertension [12] despite how the successful the procedure is at beginning [13]. This is why this sort of modelling always presents hard to reach what designers want.

The interesting is our model seems to exclude the isolated diastolic off framework. To the point, we have a case in hand whose diastolic BP exceeds 90 mmHg but rarely 100. Yet the exertional fatigue can bring on sys-diastolic hypertension. Therefore, Alprazolam (up to 20 mg, most often before bedtime) can improve.

There are two interesting episodes. The first is she coating her nose with honey and looked forwards to sugar nourishing nasal tissues via cutaneous uptake available. With two days for two 8 hours of coating-overnight, the third day, complained of headache and dais/systolic pressure provisionally exceeding 110/170 mmHg for 12 hours, a typic hypertensive presentation, we call it BP of Honney Attack (BPHA). This exposes the relationship between BP and overplus glucose in ANS. Second, while enhance rinsing at olfactory by the avail of mini electric-water-pomp that accelerates the hyperosmolar saline (> 1.5%, w/v) discharging at a few of weeks, the diastolic BP were drawn down into borderline range thereafter, this improvement made her acquired to gradually get rid of Alprazolam control within one month.

Well, the isolated diastolic BP attack sounds an association with olfactory condition; whereas, olfactory is likely one of myriad causes, too; for which, we have less evident or observation to make a sense; moreover, olfactory where people's study scarcely dabbles; except most often connects it with gastrointestinal tract, still less literatures there lies for reference. In sum, we agree that the essential hypertension is a physiological compensation serving for ANS to relieve stress from metabolic disorder though; yet this conception doesn't encourage people completely discard vascular administration, the all-or-nothing behaviour; conversely, it is vital in clinic for immediately to improve BP against involved complications onset. On the other hand, chronic hypertension may be an integrated result from myriad attacks. Alternatively, the most interesting is BPHA that can advertise much potential research to expedite their profound explorations in hypertension.

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Thank my darling; Ruan, Xin; whose unintended coating nose with honey gave a novel hypertension model, safe and measurable.

Conflict of Interest

None.

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