Brief Communication

Left ventricular hypertrophy linked with arterial hypertension through centralized aerobic-anaerobic energy balance compensation theory

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Abstract

We demonstrated intrinsic connections between left ventricular hypertrophy (LVH) and arterial hypertension (AHT) through the recently announced centralized aerobic-anaerobic energy balance compensation (CAAEBC) theory. CAAEBC has already demonstrated achievements in the treatment of AHT, diabetes myelitis (DM), and osteochondrosis. Such demonstration lifts the necessity to check the applicability of this theory to other non-communicable diseases (NCDs) and develop the proper way to model the main idea of CAAEBC.

Introduction

LVH is a condition in which an increase in left ventricular mass occurs secondary to an increase in wall thickness, an increase in left ventricular cavity enlargement, or both [1]. Most commonly, the left ventricular wall thickening occurs in response to pressure overload, and chamber dilatation occurs in response to volume overload. No doubt, it is associated with AHT [2-4].

For a while, the statistical correlation of AHT with brachiocephalic arterial blood [5-8] was mentioned as required theoretical consideration. And so far, just one pure theoretical analysis, where AHT was considered as a result of the obstructions of blood access to the brain, had been reported [9].

Recently we explained through the development of CAAEBC theory why the access to arterial blood flow to the rhomboid fossa is so critical to the body’s internal conditions regulation [10]. The visual explanation is exhibited in Figure 1. So far, we have demonstrated, that the proposed approach helps the body regain control of arterial blood pressure (BP) [11], HbA1c [12] and vertebral cartilage [13].

Should we check if LVH is on this list? To answer this question, first of all, it is necessary to check if LVH, caused by AHT, can be recovered. Indeed, the successful recovery was reported over two decades ago, but it becomes statistically noticeable only after half of the year of therapy [14]. To reach a plateau the observation should last at least three years. Therefore, it is worth trying. In this case, the initial scheme, where the first and last measurements of desired parameters we take approximately six weeks apart [10-13] should be corrected according to the time course of partial normalization of LVH in case of AHT recovery [14].
Discussion

LVH is, as mentioned in the Introduction, an abnormal increase in left ventricular mass. It is associated with coronary events, stroke, heart failure, peripheral arterial disease, and cardiovascular mortality in patients with AHT [4,15]. LVH is usually detected by electrocardiography, echocardiography, and magnetic resonance imaging. According to the American Society of Echocardiography and the European Association of Cardiovascular Imaging, LVH is defined as an increased left ventricular mass index (LVMI) greater than 95 g/m² in women and increased LVMI to greater than 115 g/m² in men [1]. It is divided into two groups – concentric and eccentric. A concentric LVH is an increased left ventricular mass index (LVMI) with a relative wall thickness ≥ 0.45, while eccentric LVH < 0.45 [4,15]. Increasing age in patients with AHT as well as diabetes mellitus in patients with AHT is associated with concentric LVH, whereas obesity, which is a volume overload state, and coronary artery disease in patients with AHT - with eccentric [16].

CAAEBC theory suggests that the restoration of the above-mentioned access with the subsequent strengthening of the cervical muscular corset will eventually lead to the normalization of the majority of internal body functions and, therefore, corresponding parameters. Therefore, we need to set the acquisition of LWMI before the therapy and six months after its completion.

It would be also convenient to step from medical record analysis to the experiment. The experimental data collection should be done on the appropriate animal model(s), the choice of which in contemporary conditions is associated with a wide variety of issues [17-25].

Conclusion

We demonstrate that the next steps to prove CAAEBC theory applicability to LVH should be

- Analysis of LVMI data taken before and six months after therapy in the Clinic
- Experiments on appropriate animal model.

Author contributions

Conceptualization, A.Y.S., A.A.V., B.A.G., A.Y.S.; writing—original draft preparation, A.Y.S., A.A.V.; writing—review and editing, A.Y.S., A.A.V.; visualization, A.A.V.; supervision, A.Y.S., and A.A.V.; project administration K.V.Z. All authors have read and agreed to the published version of the manuscript.

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