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Case Report

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Can Bendopnea Occur In Healthy Individuals without Heart Failure?

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1. Case Report

During the last few years, the effects of forward thoracic bending on cardiovascular system have been specifically studied in patients with heart failure (HF). From a pathophysiological point of view, forward bending may cause the reduction of the thoracic volume, which in turn reduces the left ventricular end-diastolic volume and decreases preload. At the same time, the increase of intrathoracic pressure leads to the rise of cardiac afterload, and both the reduction of cardiac preload and the increase of afterload synergycally contribute to stroke volume decrease [1]. Moreover, forwarding flexion results in a progressive reduction of rib cage displacement, tidal volume, and minute ventilation and a progressive increase of abdominal and diaphragmatic contribution to tidal volume [1]. Forward flexion may also determine increase in sympathetic nerve activity [2]. Anatomically, the sympathetic nervous system ganglia are located in front of the thoracic vertebral joints. From a biomechanical perspective, the flexion of the spine stretches the sympathetic nerves, which in turn increases sympathetic nerve activity.

The anterior bending is responsible for the so-called bendopnea, a shortness of breath that occurs within 30 seconds of forwarding flexion. This new respiratory symptom has been firstly observed in patients with systolic [3] and decompensated [4]. HF. Notably, it has been described in a percentage of systolic HF patients ranging from 18% to 48.8% of total, a variability clearly dependent on the clinical context (acute vs. chronic HF) [5].

The increased intrathoracic or intrabdominal pressure induced by the anterior bending results in a positional elevation of right and left side filling pressures in HF patients [3]. The increased thoracic or abdominal pressure by anterior bending imposes greater pressure on the diseased heart [6].

Bendopnea typically occurs in patients with chronic heart failure with reduced ejection fraction (HFrEF), particularly if advanced or decompensated [7, 8].

It is associated with several clinical symptoms and congestive signs, such as dyspnea, orthopnea, paroxysmal nocturnal dyspnea, New York Heart Association (NYHA) functional class III or IV, high waist circumference and body mass index (BMI), abdominal fullness, elevated jugular venous pressure, pretibial edema, elevated serum levels of N-terminal pro-brain natriuretic peptide (NT-proBNP), worse cardiac index, increased pulmonary capillary wedge pressure, bi-atrial enlargement, increased systolic pulmonary artery pressure and increased use of loop diuretics [3, 5, 8-10].

A shorter-onset bendopnea is associated with a higher mortality rate [3, 8], particularly in those HF patients with advanced NYHA functional class (III–IV) and in those with HFrEF [8].

Some Researchers have demonstrated that bendopnea is not specific of HF, but may also be detected in various cohorts of non-HF patients. Indeed, it has been reported in 18 of 53 (33.9%) outpatient pulmonary arterial hypertension (PAH) patients [11], in 46 of 108 (42%) patients with severe aortic stenosis referred for surgical aortic valve replacement [12] and in 20 of 300 (6.7%) individuals attending to Primary Care or Internal Medicine consultation without HF [13]. Among individuals without HF, those with bendopnea had higher prevalence of obesity, chronic obstructive pulmonary disease, atrial fibrillation, chronic kidney disease, hypertension and diabetes mellitus, compared to those without bendopnea [13].

Recent evidence would suggest that the anterior bending might play an important etiopathogenetic role in the occurrence of bendopnea also in individuals who chronically suffer from abdominal and/or thoracic compressive phenomena on cardiac chambers, even in absence of any intrinsic myocardial dysfunction. With this regard, a number of recent studies have highlighted the important role exerted by thoracic and/or abdominal compressive phenomena on cardiac chambers in determining a subclinical deterioration of myocardial strain parameters, assessed by speckle tracking echocardiography (STE), in various cohorts of healthy individuals with no evidence of structural cardiomyopathy. These studies have noninvasively assessed the chest wall shape by using the modified Haller index (MHI), that allows the clinicians to quantify the degree of anterior chest wall deformity, without using ionizing radiation. It is easily measured by dividing the latero-lateral thoracic diameter (obtained by using a rigid ruler coupled to a level) by the antero-posterior (A-P) thoracic diameter (measured from the echocardiographic parasternal long-axis view, as the distance between the true apex of the imaging sector and the posterior wall of the descending aorta, visualized behind the left atrium) [14]. A MHI >2.5 is diagnostic of concave-shaped chest wall and/or pectus excavatum (PE), due to a narrow A-P thoracic diameter (generally <13.5 cm), whereas a MHI <2.5 identifies a normal chest shape conformation [15].

Despite normal indices of left ventricular systolic function on conventional transthoracic echocardiography, a subclinical myocardial dysfunction, defined as left ventricular (LV) global longitudinal strain (GLS) magnitude <20% on STE examination, has been demonstrated in healthy individuals with android obesity by two recent studies [16,17]. In these studies, a greater waist-to-hip ratio (WHR) and a narrower A-P thoracic diameter were the only independent predictors of subclinical impairment in LV-GLS in healthy individuals with android obesity. In other terms, the subclinical myocardial dysfunction observed in these individuals appeared to be primarily related to anthropometrics, such as WHR and A–P thoracic diameter, rather than intrinsic myocardial dysfunction. Accordingly, the bendopnea described in individuals with obesity might be related not only to intrinsically increased left ventricular filling pressures, but also to extrinsic abdominal and thoracic compressive phenomena on cardiac chambers.

Similarly to what detected in healthy individuals with android obesity, a subclinical attenuation of myocardial strain parameters has also been reported in healthy individuals with PE [18] and healthy pregnant women during the third trimester of pregnancy [19]. In these two categories of healthy individuals, the anterior chest wall deformity and the physiological rise in the diaphragm, respectively, showed a preponderant and independent role in affecting the myocardial deformation properties of both ventricles, in absence of any intrinsic myocardial dysfunction. The greater was the MHI value, the lower was the magnitude of biventricular strain parameters.

A narrow A-P thoracic diameter has also been correlated with a more severe restrictive respiratory pattern in patients with mild-to-moderate idiopathic pulmonary fibrosis [20]. Analogous situation may be observed in patients with severe thoracic kyphoscoliosis, a condition characterized by diminished chest wall compliance and impaired respiratory mechanics [21]. Finally, it is noteworthy that abdominal bloating and distension may impair respiratory mechanics in absence of any structural cardiomyopathy, similarly to android obesity [22].

We hypothesize that the forward bending might contribute to the bendopnea occurrence in healthy individuals with various degrees of anterior chest wall deformity due to a narrow A-P thoracic diameter and/or android obesity or abdominal distension, even in absence of intrinsic systolic and/or diastolic biventricular dysfunction. To date, as far as we know, the characteristics of bendopnea in these individuals has never been previously investigated. Accordingly, prospective observational studies are needed for evaluating the prevalence of bendopnea in healthy individuals with a narrow A-P thoracic diameter and/or android obesity.

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