From Sleep-Disordered Breathing to Increased Left Ventricular Wall Stress in Heart Failure

To the Editor:

Sleep-disordered breathing (SDB) frequently occurs in patients with congestive heart failure (CHF). Both entities seem to be inherently associated, but mechanisms causally linking SDB with CHF still remain uncertain. Thus, in an issue of CHEST (March 2012), Carr et al. reviewed acute cardiopulmonary failure resulting from SDB.

Central sleep apnea is associated with increased circulation time in CHF due to a reduced stroke volume and is closely linked to the sympathetic tone. We showed that the severity of SDB is associated with increased end-diastolic and end-systolic left ventricular wall stress, but not with pump function. Thus, the question arises of whether cardiac function is indeed the key determinant.

Left ventricular wall stress is predominantly influenced by ventricular volume, myocardial mass, and the transmural pressure gradient. Ventricular wall stress is frequently increased in dilatative CHF, since cardiac hypertrophy is not appropriate to compensate for ventricular dilatation. Increased ventricular wall stress was associated with an altered autonomic tone. In obstructive sleep apnea, upper airways obstruction increases the negative intrathoracic pressure, which transmits increased distending forces on the left ventricle and thereby increases wall stress. Increased left ventricular wall stress was identified as the missing link between CHF and SDB and should not be underrated. These considerations also provide a rationale for the benefits of positive airway pressure ventilation therapy in CHF.

It has been suggested that repetitive nocturnal hypoxias interfere with an adequate ventricular hypertrophy in SDB, which enhances further wall stress and ventricular dilatation with worse prognosis. Increased left ventricular wall stress is associated with decreased heart rate variability with unfavorable consequences. Increased wall stress leads to the opening of stretch-activated cation channels, which increases the risk of sudden cardiac death. Wall stress is also associated with myocardial oxygen consumption, that is, increased wall stress can worsen wall motion abnormalities, further deteriorating cardiac function. We recommend, therefore, that ventricular wall stress should be considered as a diagnostic criterion in SDB and CHF. Normalization of high wall stress remains, however, an as-yet unmet therapeutic target.
on the distance from the mouth to the glottis and the tracheal length. A patient’s height generally influences the proper depth of intubation. In dwarfs, fixation of a tube at standard depth results in endobronchial intubation. In patients with short stature, a shorter depth of tracheal intubation is, therefore, expected. However, in patients with severe thoracic scoliosis and resultant short stature, both the tracheal length and oroglottic distance (neck height) are likely to be normal. Therefore, anticipating a shorter orocarinal distance and fixing the tube inappropriately at a lesser depth can lead to accidental extubation. Our experience suggests that short stature due to dwarfism should be differentiated from short stature due to severe scoliosis during tracheal intubation. Fiber-optic or fluoroscopic confirmation of the correct tube position is ideal and should be performed when feasible. However, when such examination is not possible, a meticulous clinical examination and chest radiograph confirmation can overcome the dilemma about the correct placement of tracheal tube.

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