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REFERENCES


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 dilative 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.

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REFERENCES


Optimal Depth of Tracheal Intubation in Severe Scoliosis

To the Editor:

Severe scoliosis can pose significant problems during airway management. The difficulty in identifying the correctness of the tracheal tube position in patients with severe scoliosis and resultant short stature has been described.

A 35-year-old man was admitted with loss of consciousness following a fall during a seizure episode. His Glasgow Coma Scale score at admission was E3M6V4. Brain CT imaging showed a large extradural hematoma. Clinical examination revealed severe scoliosis of the thoracic spine and short stature (124 cm). For emergent evacuation of hematoma under anesthesia, the tracheal tube was fixed at 22 cm and position was confirmed by visual passage of the tube cuff 2 cm below the glottis, the presence of bilateral lung ventilation on auscultation, normal airway pressure, and oxygen saturation. Chest radiographs showed severe scoliosis with the appearance of a horizontal upper thoracic spine (Fig 1A, vertical white arrows), vertical ribs in the right hemithorax (horizontal white arrows), tube position above the carina (Fig 1A, black arrow), and short stature (Fig 1B).

Correct placement of the tracheal tube is vital for safe anesthetic/critical care. The optimal depth of tube placement depends