

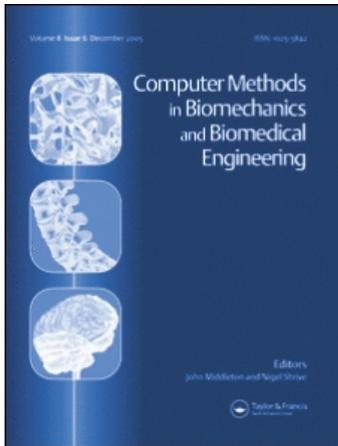
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A. Vaziri ^a; H. Nayeb-Hashemi ^a; B. Akhavan-Tafti ^b

^a Department of Mechanical, Industrial and Manufacturing Engineering, Northeastern University, Boston, MA, USA ^b School of Medicine, Stanford University, Stanford, CA, USA

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Computational model of rib movement and its application in studying the effects of age-related thoracic cage calcification on respiratory system

A. Vaziri^{a*}, H. Nayeb-Hashemi^a and B. Akhavan-Tafti^b

^aDepartment of Mechanical, Industrial and Manufacturing Engineering, Northeastern University, Boston, MA 02115, USA

^bSchool of Medicine, Stanford University, Stanford, CA, 94305, USA

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A 3D finite element model of rib cage movement is developed and used to study the role of age-related costal cartilage and sternocostal joint calcification, as well as respiratory muscle weakness on the 'bucket-handle' movement of human rib. The volume displacement of the rib cage is related to changes in its circumference using an empirical equation presented by Agostoni et al. (1965, *J Appl Physiol*, 20:1179–1186). A systematic study is carried out to quantify the role of costal cartilage, sternocostal joint calcification and muscle weakness on the volume displacement of the rib cage. The results provide insight into some of the mechanisms underlying age-related changes in the respiratory system.

Keywords: respiration mechanisms; rib cage volume displacement; costal cartilage calcification; respiratory muscles weakness; finite element method

1. Introduction

During the past several decades, the size of the elderly population has increased substantially and steadily across most of the western world (Lewis and Bottomley 1994; Diczfalussy 2001). This continued growth in elderly population requires understanding of *normal age-related changes* to enhance healthcare and develop more effective preventive measures and diagnostic and treatment procedures for elderly population. An example of such age-related changes is the change in the respiratory system from ageing. Respiration is a complex activity that involves contraction of respiratory muscles and movement of the rib cage to provide the required lung volume during inspiration. The rib cage displacement is based on an increase in its transverse and anteroposterior dimensions, together or separately (Figure 1(A); Luttgens et al. 1992). Analysis of the lung volume displacement during respiration entails studying body wall movement during respiration. Three mechanisms of the body wall movement during respiration are: rib cage expansion and contraction, anterior abdominal wall expansion or contraction and spine flexion–extension. Although spine movement can cause substantial displacement of the chest wall, it does not contribute significantly to the changes in lung volume (Smith and Mead 1986). The degree to which rib cage and abdominal wall movements contribute to the lung volume displacement during respiration depends on the body anatomy, body posture and breathing condition. The contribution of the rib cage movement to the volume displacement of the lungs was evaluated by Agostoni et al.

(1965), Grimby et al. (1968) and Loring and Mead (1982) for various human body postures. It should be noted that rib cage displacement not only contributes directly to the volume displacement of the lung, but also facilitates the movement of abdominal viscera and the primary act of the diaphragm [i.e. as the rib cage expands, it lowers abdominal pressure and thus permits a larger fraction of transdiaphragmatic pressure to go into lowering the pleural pressure (Mead et al. 1995)].

The reduction of lung flexibility in combination with respiratory muscle weakness and chest wall stiffening due to ageing diminishes pulmonary function. The age-related reduction of lung flexibility is linked to the general functional degradation of the protein elastin throughout the body and loss of elastic fibre attachments within the lungs (Smith 1997). On the other hand, the weakening of the respiratory muscles and the stiffening of the chest wall due to calcification of costal cartilages (costocalcinosis) and sternocostal joints are common age-related changes seen in 26% of population, which may result in a substantial decrease in volume displacement of the lungs during respiration. Weakness of the respiratory muscles due to ageing is associated with the general age-related loss of skeletal muscle fibres (Alnaqeeb et al. 1984; Fitts et al. 1984; Frontera et al. 1991; Carmeli et al. 2002) and it is generally hypothesised that the respiratory muscles display deficits similar to those observed in other skeletal muscles such as the limb and trunk muscles (muscle strength degrades by 30–40% between 30 and 80 years of age; Brooks and Faulkner 1995). On the other hand, thoracic

*Corresponding author. Email: vaziri@coe.neu.edu

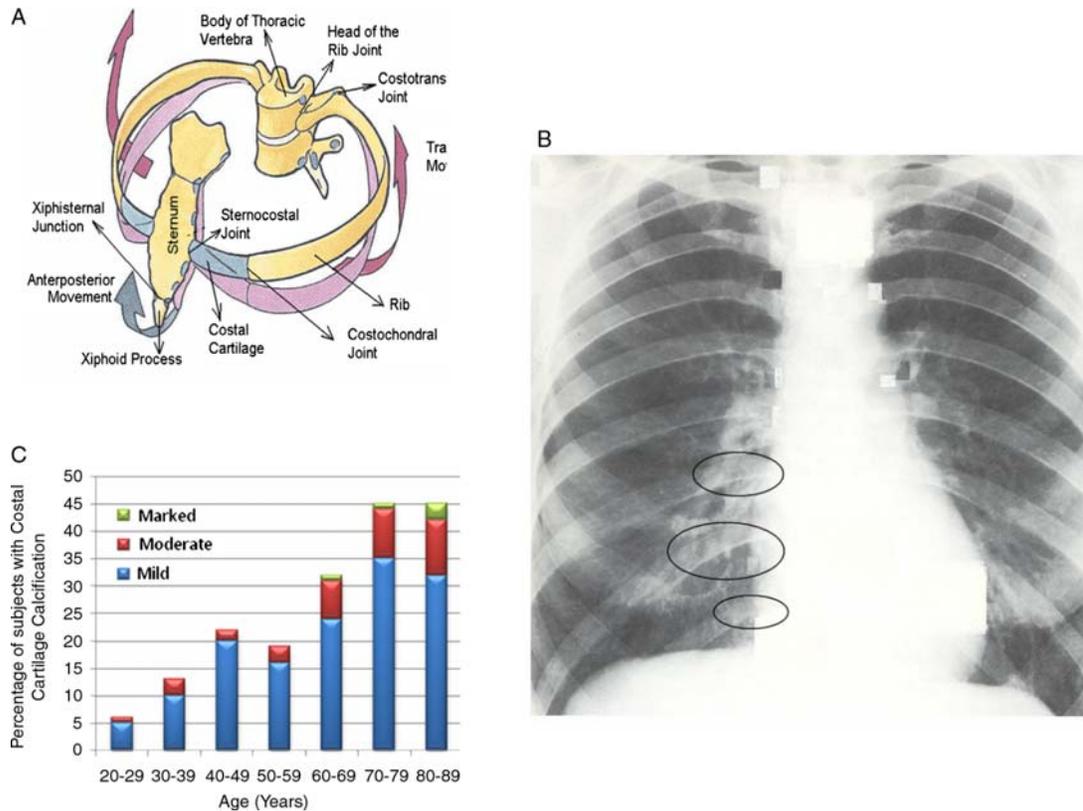


Figure 1. (A) Schematic diagram of the rib cage and its corresponding mechanisms of movement during inspiration. (B) X-ray picture of the rib cage, the ellipses indicate the sites of costal cartilage calcification. The image is adapted from Brooks and Faulkner (1995). (C) Correlation of costal cartilage calcification with age. The vertical axis shows the percentage of subjects suffering from costal cartilage calcification with different severity levels: mild (flecks of calcification at the costochondral junction), moderate (calcification extending into the cartilage) and marked (widespread calcification in at least six costal cartilages). The figure is plotted based on the results provided by Teale et al. (1989), which were obtained by reviewing 100 (50 men) chest radiographs from each of the seven age decades considered (third to ninth).

cage calcification usually initiates from costochondral junctions and extends toward the sternocostal junctions (Rao and Pai 1988; Ontell et al. 1997) as illustrated in Figure 1(B). Figure 1(C) shows the correlation between age and possibility of costal cartilage classification and its extent. The results are reproduced from the work of Teale et al. (1989), where the authors reviewed 700 chest radiographs to determine the site and extent of the calcification. The results show that the prevalence of costal cartilage calcification increases from 6% in the third decade of life to 45% in the ninth decade and, overall, it is more common in men. Similar results were reported by Edge et al. (1983).

In this study, we developed a full 3D model of rib movement using finite element method and employed it to relate the severity of costal cartilage calcification and respiratory muscle weakness to the rib cage volume displacement. This was achieved by studying the transverse and anteroposterior expansion of the rib cage during inspiration and relating them to the changes in the rib cage circumference and volume displacement using the

available empirical methods. The details of the biomechanical model of rib movement are described in Section 2. Section 3 provides a brief overview of the empirical equations used to relate the rib displacement to the rib cage volume displacement. Section 4 presents the results of our parametric study to quantify the effect of costal cartilage and sternocostal joint calcification, as well as respiratory muscles weakness on the volume displacement of the thoracic cage. Conclusions are made in Section 5.

2. Rib model development

2.1 Computational model of the human rib

A 3D computational model of the human fifth right rib is developed based on the typical dimensions of a 65 kg, 167 cm male rib as shown in Figure 2(A). The rib is taken to have a hollow elliptical cross section with the effective area of half of the total area. The costal cartilage is taken to be solid with an elliptical cross section. However, for investigating the effect of the costal cartilage calcification at the costochondral junction, three sets of models are

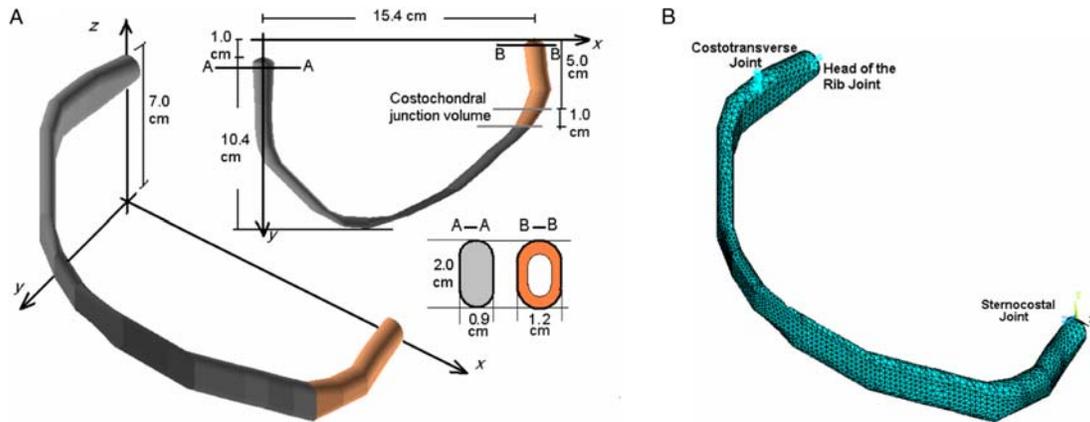


Figure 2. (A) A 3D computational model of the fifth right rib. The cross section of the model is also shown at A–A and B–B. (B) The finite element mesh of the model.

developed to represent the costal cartilage with mild, moderate and severe calcification:

- *Rib model with mild costal cartilage calcification (flecks of calcification at the costochondral junction, Teale et al. 1989).* In this model, the costal cartilage has two segments, 1 cm calcified costochondral joint segment followed by a healthy costal cartilage (Figure 2(B)).
- *Rib model with moderate calcification (calcification extending into the cartilage) with healthy sternocostal joint.* In this model, the entire costal cartilage is calcified.
- *Rib model with marked calcification with severely calcified synovial joint.* In this model, the entire costal cartilage is calcified and all degrees of freedom at the sternocostal joint are constrained.

It should be noted that based on our literature survey, there was no data available on the degree of sternocostal joint degradation by ageing. Here, we explored the cases of ‘healthy joint’ and ‘completely calcified joint’. The computational model of the fifth rib employed 20-node solid elements and consisted of 36,675 nodes distributed over 23,165 elements (Figure 2(B)). A mesh sensitivity study was conducted to ensure independence of the results from the computational mesh. The computations are performed using commercially available finite element modelling software ANSYS.

2.2 Material modelling

Both the human rib and costal cartilage were considered as linear elastic materials (Wang and Yang 1998). The rib had an elastic modulus $E = 11.51$ GPa and a Poisson ratio 0.3. The costal cartilage is a hyaline cartilage tissue, which acts as the flexible interface between the bones of the ribs and the sternal column. Most of the available data

on the human cartilage properties were obtained by studying the response of articular cartilage (Kempson et al. 1968; Mow et al. 1980; Weng et al. 1999; Vaziri et al. 2008). The costal cartilage is generally stiffer than the articular cartilage with material properties that depend on age and gender (Guo et al. 2007; Lau et al. 2008). In this study, the healthy costal cartilage was modelled as linear elastic with an elastic modulus 24.5 MPa and a Poisson ratio 0.4 (Yamada 1970). The linear elastic property assumption for the costal cartilage was taken for simplicity and by considering the time scale of rib movement and cartilage deformation during the ‘bucket-handle’ movement of the rib. This assumption is also consistent with the results provided by Ko and Takigawa (1953), which showed that the costal cartilage has an elastic limit about 50% of its ultimate strength. The results of our analyses revealed that the maximum stress at the cartilage is significantly lower than its elastic limit. It should be noted that several other studies have reported stiffness values in the range of 5–7 MPa for the costal cartilage, which is lower than the value considered in our study for the stiffness of the costal cartilage (Feng et al. 2001; Roy et al. 2004; Guo et al. 2007; Lau et al. 2008). The variability could be due to differences in measurement techniques, as well as the age and gender of the donors (Lau et al. 2008) and is not expected to change the key findings of the current study. The effect of calcification on the material properties of the costal cartilage depends on the severity and pattern of the calcification. Based on our literature survey, there is no data available to quantify the relationship between the severity of human costal cartilage calcification and the changes in its material properties. However, considering the available data on the stiffness of calcified articular cartilage of the knee joint (Mente and Lewis 1994; Vaziri et al. 2008), we assume that costocalcinosis increases the elastic modulus of the rib costal cartilage by a maximum

factor of 10. Here, the elastic modulus of the costal cartilage after calcification is denoted by \bar{E} . With this assumption, the maximum elastic modulus of the calcified cartilage is $\sim 2.1\%$ of the rib bone elastic modulus, which is in agreement with the relative stiffness of the knee calcified articular cartilage and bone (Mente and Lewis 1994).

2.3 Loading during inspiration

Correct modelling of rib articulations is critical for accurate analysis of the rib movement during respiration. The first to seventh ribs articulate with the lateral border of the sternum via their costal cartilages (sternocostal joints). Furthermore, a typical rib articulates with the vertebral column at two joints: at the joint of the head of the rib and at the costotransverse joint (Figure 1). The sternocostal joints of the second to seventh ribs are synovial joints, as well as the joints at the head of the ribs to vertebral column. The costotransverse joint of the fifth rib is typically a plane type synovial joint (Moore 1992). The synovial joint was modelled as a ball and socket joint as they give the rib the ability to rotate about its axis in the ‘bucket-handle’ movement of the rib. Chen (1978) used the same modelling assumption for studying the response of the chest under impact loading. The costotransverse joint was modelled as a roller, as the plane type of synovial joint permits gliding and sliding along the opposed surface of the joint, which is flat or almost flat (Moore 1992).

The finite element analyses were performed by considering large deformation and finite strain conditions under static loading. The static load applied to the rib was estimated by considering the effective force exerted by the respiratory muscles. The respiratory muscles are divided into two groups: muscles with primary function and muscles with secondary function of respiration. The contribution of each of the thoracic muscles to respiration is so complex that despite a few conducted studies, still the nature of forces and moments applied to the rib during an inspiration has not been completely understood (De Troyer et al. 1985; Luttgens et al. 1992; Loring 1992). Here without emphasising on the actual muscle force, the transverse movement of the rib was evaluated by applying a constant vertical distributed force along the top boundary line of the rib. This is based on the fact that intercostal muscles are the most effective muscles in forcing the rib cage to expand transversely. The magnitude of this constant distributed force was evaluated by trial and error in order to obtain a defined maximum vertical displacement in the ‘bucket-handle’ movement for a healthy rib. For studying the effect of costal cartilage and synovial joint calcification on the ‘bucket-handle’ movement of the rib, the same intercostals muscle force was exerted to the rib. The effect of the age-related muscle weakness was investigated by decreasing the value of the intercostal

muscles’ force systematically up to 40% (see Section 1). The applied effective normal force of weakened respiratory muscles, denoted by \bar{F} , is normalised by the effective normal force exerted to the rib by healthy respiratory muscles, F .

3. Relating rib movement to rib cage volume displacement

The computational model explained in Section 2, provides an estimate of the rib displacement during respiration which can be related to volume displacement of the thoracic cage using the empirical relationships discussed in Section 3. Based on the empirical equation presented by Agostoni et al. (1965), the volume displacement of the rib cage, denoted by ΔV , is related to the change of its circumference by

$$\Delta V = hK[(c_1^2 - c_0^2)/4\pi], \quad (1)$$

where h denotes the distance between the jugular notch and the sternoxiphisternal joint, c_1 and c_0 are the circumferences of the rib cage at the sternoxiphisternal joint at full inspiration and rest, respectively, and K is a geometrical parameter, which can be estimated from

$$K = \frac{b}{a} \left[1 + \frac{1 - (b/a)^2}{(da/a)/(db/b) + (b/a)^2} \right] \quad (2)$$

by assuming the cross section of the rib cage as an ellipse with minor and major radii denoted by a and b , respectively. In Equation (2), da and db are the changes in the minor and major radii of the rib from rest to full inspiration, respectively. To estimate the effects of costal cartilage calcification, sternocostal joint calcification and muscle weakness on the volume displacement of the rib cage, here we assumed that these changes do not change the overall rib movement kinematics and Equations (1) and (2) are applicable to both rib movement before and after calcification. The parameter η is defined as the ratio of rib cage volume displacement of the calcified and healthy rib cage in full respiration due to the ‘bucket-handle’ movement. To relate η to the results of our computational model, we assumed that the ratio of the change of radial expansion to lateral expansion, da/db , is independent of the costal cartilage condition. This allows a simple relationship for estimating η ,

$$\eta = (\bar{c}_1^2 - c_0^2)/(c_1^2 - c_0^2), \quad (3)$$

where c_1 and \bar{c}_1 denote the circumferences of the healthy and calcified rib cage in full inspiration at the sternoxiphisternal joint, respectively. Nonetheless, a more rigorous computational model, which encompasses the total rib cage, is necessary in order to evaluate the

validity of these assumptions. The value of c_1 and \bar{c}_1 in Equation (2) could be estimated from the results of the computational simulations of fifth rib movement by using the relationship between the circumference of the rib cage at the fifth rib and at the sternoxiphisternal joint. Agostoni et al. (1965) observed that the change of circumference of the rib cage at the sternoxiphisternal joint in full inspiration is approximately 50% greater than its change at the fifth rib.

4. Results

The effect of the severity of costal cartilage calcification on the 'bucket-handle' movement of the rib is explored by systematically varying the elastic modulus of the costal cartilage. In this set of calculations, first we assumed a maximum vertical displacement of 1 cm for the healthy rib cage in full inspiration denoted by δ . This value depends on body posture, sex and inspiration condition. For example, in the case of forced inspiration this value is significantly higher than regular inspiration. This value is reasonable for ordinary respiration. Then the forces applied by the respiratory muscles that lead to $\delta = 1$ cm were calculated from the detailed computational movement of a healthy rib described in Section 2, assuming uniform force distribution along the top side of the rib. Subsequently, this force was used in modelling the rib movement with calcified costal cartilage.

Since the results show that the rib movement has a very low sensitivity to the Poisson ratio of the costal cartilage, all the results presented here are for a constant costal cartilage Poisson ratio of 0.4 (the results are not presented for the sake of brevity). The results of our calculation on the effect of costal cartilage calcification

on the rib cage movement are summarised in Figure 3. Figure 3(A) shows the maximum vertical displacement of the rib with calcified cartilage, denoted by $\bar{\delta}$, normalised by δ . Figure 3(B) shows a complementary set of results where the vertical displacements are converted to thoracic cage volume displacement due to the 'bucket-handle' movement. The results indicate that mild costal cartilage calcification, as classified in Section 2, does not considerably influence the maximum vertical displacement of the rib and the volume displacement of the rib cage. In contrast, moderate calcification of the costal cartilages causes significant reduction in rib movement and thoracic cage volume displacement. For an increase in the elastic modulus by a factor of 10, $\bar{E}/E = 10$, the volume displacement of the rib cage is reduced by $\sim 44\%$. The sternocostal joint calcification further decreases the volume displacement of the rib cage due to the 'bucket-handle' movement ($\sim 17\%$ decrease for $\bar{E}/E = 10$).

The effect of respiratory muscles weakness on the rib movement and its volume displacement due to the 'bucket-handle' movement is quantified in Figure 4 for various severities of costal cartilage calcification. Forty percent reduction in the muscle force (i.e. $\bar{F}/F = 0.6$) leads to 39% and 17% reduction in the maximum vertical displacement of the rib for the case of $\bar{E}/E = 1$ and $\bar{E}/E = 10$, respectively. The same reduction in the muscle strength causes the volume displacement of the rib cage to reduce by $\sim 22\%$ and $\sim 9\%$ for the above mentioned degrees of calcification, respectively. In general, the effect of muscle weakness is more pronounced for a rib with less severe degree of costal cartilage calcification. The additional set of results shown in Figure 5 indicate that volume displacement of the

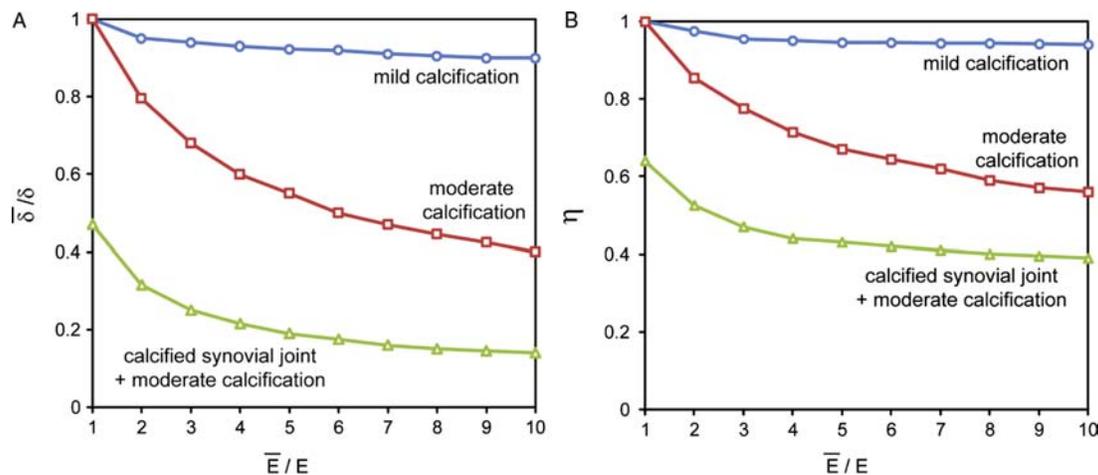


Figure 3. Effect of calcification severity of the costal cartilage and synovial joint on rib movement and volume displacement. The plots display the effect of increase in the elastic modulus of the costal cartilage due to calcification on: (A) the maximum vertical displacement of the rib due to bucket-handle movement and (B) the volume displacement of the rib cage. The results are presented for three different calcification levels.

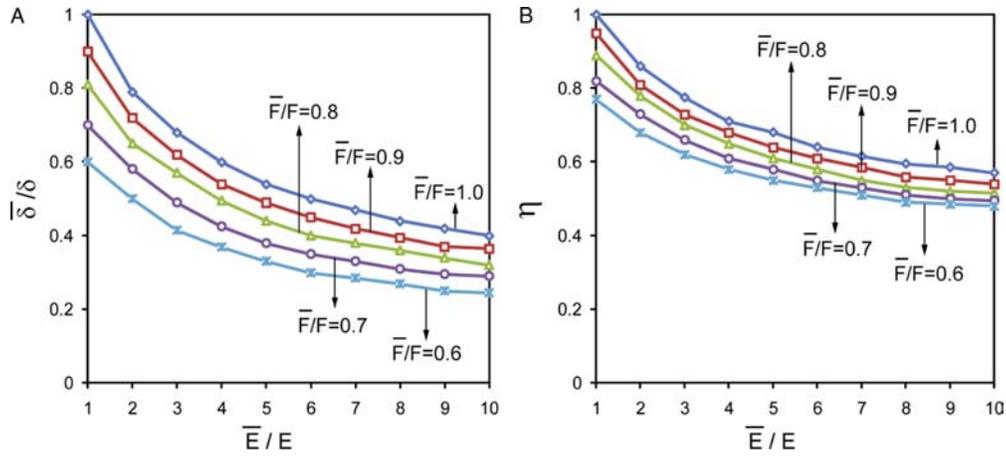


Figure 4. Effect of respiratory muscles weakness on rib movement and volume displacement. The plots display the effect of increase in the elastic modulus of the costal cartilage due to calcification on: (A) the maximum vertical displacement of the rib due to bucket-handle movement and (B) the volume displacement of the rib cage for various levels of respiratory muscles weakness.

thoracic cage induced by the ‘bucket-handle’ movement degrades approximately linearly with the reduction in respiratory muscle force for both healthy and calcified costal cartilage. The results again indicate the significant effect of the synovial joint calcification on the rib cage volume displacement during respiration.

5. Discussion and conclusions

In this study, we developed a 3D model of human rib and employed it to study the role of costal cartilage calcification, synovial joint calcification and respiratory

muscle weakness on the rib movement and volume displacement of the rib cage during respiration. Several limitations were inherent to the study design. We have used a simplified geometrical model of the rib cage as only one rib was used to study our hypothesis. We have described and modified the elastic restoring force of the rib according to the ventral articulation. It has been shown that the spine movement does not contribute to the lung volume displacement significantly, although it can cause substantial displacement of the chest wall (Smith and Mead 1986). Furthermore, we considered the bucket-handle rotation of the rib as the main rotation of the rib. This model requires external validation. In addition to the necessity for further theoretical investigation by modelling the total rib cage, the results of the present study motivate clinical investigations to validate the present results due to some uncertainties of the theoretical model. However, and despite these limitations, the preliminary study conducted here may serve as a functional model for studying the effects of age-related thoracic cage calcification on respiratory system and better explanation of certain clinical situations in the elderly population such as exertional dyspnea as further discussed below.

Dyspnea is frequently associated with conditions in which respiratory drive is increased or the respiratory system is subject to a mechanical load. These conditions are characterised by a sensation of air hunger or increased effort or work of breathing (Simon et al. 1990; Elliott et al. 1991). Manning and Schwartzstein (1995) and Killian (2002) provided a review of the pathophysiology of dyspnea and discussed the current trend in approaching this condition. Here, we briefly mention some of the proposed mechanisms of exertional dyspnea and their possible relation to age-related costal cartilage and rib

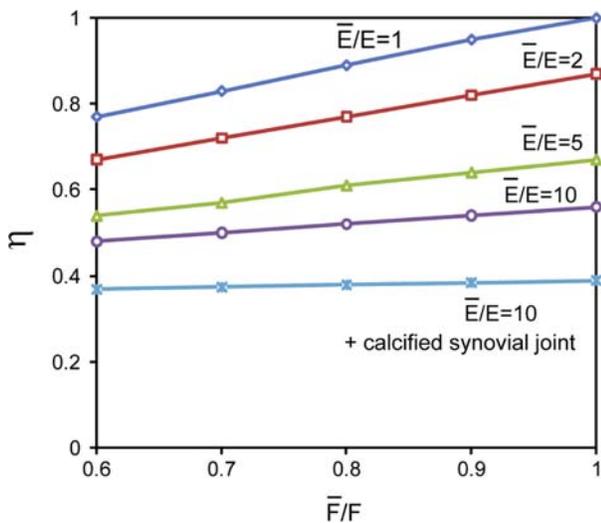


Figure 5. Volume displacement of the rib cage with moderate calcification versus the muscle force for different levels of increase in the elastic modulus of the costal cartilage due to calcification, as well as the rib with calcified synovial joint.

joint calcification, and weakness of the respiratory muscles, in view of the results presented here.

- *Sense of respiratory effort.* According to this theory, the sense of respiratory effort increases whenever the central motor command to the respiratory muscles must be increased; that is, when the muscle load is increased, or when the muscles are weakened. This theory clearly states the relationship between an increased muscle workload and dyspnea. Costal cartilage and joint calcification generate a greater resistance against rib cage expansion. This increased resistance, imposes an increased load on muscles and may partially account for a sense of respiratory effort.
- *Chest-wall receptors.* Based on this theory, the brain receives projections from a variety of receptors in the joints, tendons, and muscles of the chest wall that might influence the sensation of dyspnea. These afferent information from the chest wall, modify the intensity of dyspnea. Impulses in afferent pathways from proprioceptors in chest wall muscles and joints stimulate the inspiratory neurons. This effect probably helps increase ventilation during exercise. Costal cartilage and rib joint calcification restrict the movements of the chest wall and thus, reduce the stimulatory impulses to the brain.
- *Afferent mismatch.* According to this theory, dyspnea arises from disturbance in the force or tension generated by the respiratory muscles and the resulting change in muscle length and lung volume [Readers are referred to Schwartzstein et al. (1989, 1990) and Ganong (2001) for further discussion]. As already stated, costal cartilage and rib joint calcification generate a greater resistance against rib cage expansion resulting in a smaller change in muscle length subject to any constant force generated by respiratory muscles leading to an afferent mismatch.
- *Hypercapnia and hypoxia.* Hypercapnia is the retention of CO₂ in the body. It does occur in ventilation-perfusion inequality and when for any reason alveolar ventilation is inadequate in the various forms of pump failure. Hypoxia is O₂ deficiency at the tissue level. Hypoxic hypoxia is the most common form of hypoxia seen clinically [Readers are referred to Ganong (2001) for a more detailed discussion of mechanisms resulting Hypoxia and Hypercapnia]. An important factor in alveolar ventilation is the ability of lungs to expand properly. The results of the present study indicate that the costal cartilage and rib cage joint calcification result in significant reduction of the lung expansion during respiration. This leads to

poorer ventilation and thus may result in hypercapnia and hypoxic hypoxia.

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