THORACIC

Is there a biomechanical cause for spontaneous pneumothorax?

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Abstract

OBJECTIVES: Primary spontaneous pneumothorax has long been explained as being without apparent cause. This paper deals with the effect of chest wall shape and explains how this may lead to the pathogenesis of primary spontaneous pneumothorax.

METHODS: Rib cage measurements were taken from chest radiographs in 12 male pneumothorax patients and 12 age-matched controls. Another group of 15 consecutive male thoracic computerised tomography (CT) were investigated using paramedian coronal and sagittal CT reconstructions to assess apical lung shape. A finite element analysis (FEA) model of a lung apex was constructed, including indentations for the first rib guided by CT scan data, to assess pleural stress. This model was tested using different anteroposterior diameter ratios, producing a range of thoracic indexes.

RESULTS: The pneumothorax patients had a taller chest ($P = 0.03$), wider transversely ($P = 0.009$) and flatter ($P = 0.03$) when compared with controls, resulting in a low thoracic index. Prominent rib indentations were found anteriorly and posteriorly on the lung surface, especially on the first rib on CT. FEA of the lung revealed significantly higher stress (×5–×10) in the apex than in the rest of the lung. This was accentuated (×4) in low thoracic index chests, resulting in 20-fold higher stress levels in their apex.

CONCLUSIONS: The FEA model demonstrates a 20-fold increase in pleural stress in the apex of chests with low thoracic index typical of spontaneous pneumothorax patients. Mild changes in thoracic index, as occurring in females or with aging, reduce pleural stress. Spontaneous pneumothorax occurring in young male adults may have a biomechanical cause.

Keywords: Pneumothorax, spontaneous • Pneumothorax, pathogenesis • Biomechanics

INTRODUCTION

Primary spontaneous pneumothorax is a disease with no apparent cause, occurring in the absence of underlying lung disease [1]. It is a significant global health issue [2]. The incidence is 18–28/100 000 cases per annum for men and 1.2–6/100 000 for women [3]. Not long ago these patients were thought to have a 'heritable defect' in their structure [4]. It has long been recognized [5] that the typical patient with primary spontaneous pneumothorax is a tall, thin male [6, 7], and although body height is perhaps controversial [8], these patients have a diminished anteroposterior diameter and low thoracic index (ratio of anteroposterior and lateral chest diameters) [9]. Video-assisted thoracic surgery (VATS) has shown subpleural blebs in almost all patients (76–100%), with blebs commonly present in patients reaching surgery [10]. Bullae are often seen on computerised tomography (CT) scanning [11]. The pathogenesis remains unknown [1].

This paper examines the present understanding of the aetiology and pathogenesis of primary spontaneous pneumothorax, examines apical lung anatomy and models the lung using finite element analysis (FEA) in order to better understand the stresses on the pleura.

METHODOLOGY

Rib cage measurements were taken from posteroanterior and lateral chest radiographs in 12 male patients presenting with spontaneous pneumothorax and from 12 age-matched controls admitted with blunt chest trauma (see Fig. 1). Patients older than 30 years and females were excluded. The rib cage measurements were based on a previously reported method for thoracic dimensions [12].

Apical lung shape was investigated using paramedian coronal and sagittal reconstructions of a CT thorax series of 15 consecutive
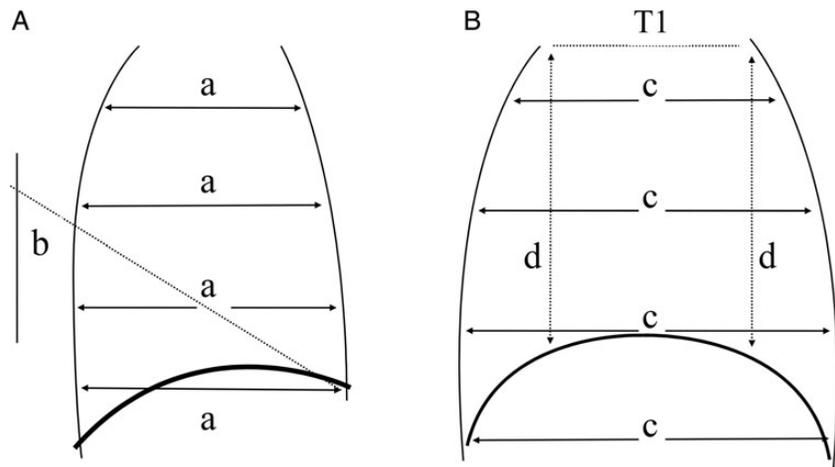
Figure 1: Diagrams of measurements taken on chest X-ray made on (**A**) lateral projection and (**B**) posteroanterior (PA) projection. Measurements include (*a*) depth of chest anteroposteriorly at various chest levels (*b*) angulation of rib (*c*) lateral width of chest at various levels (*d*) height of rib cavity from apex to dome of diaphragm. After Bellemare *et al.* [12].
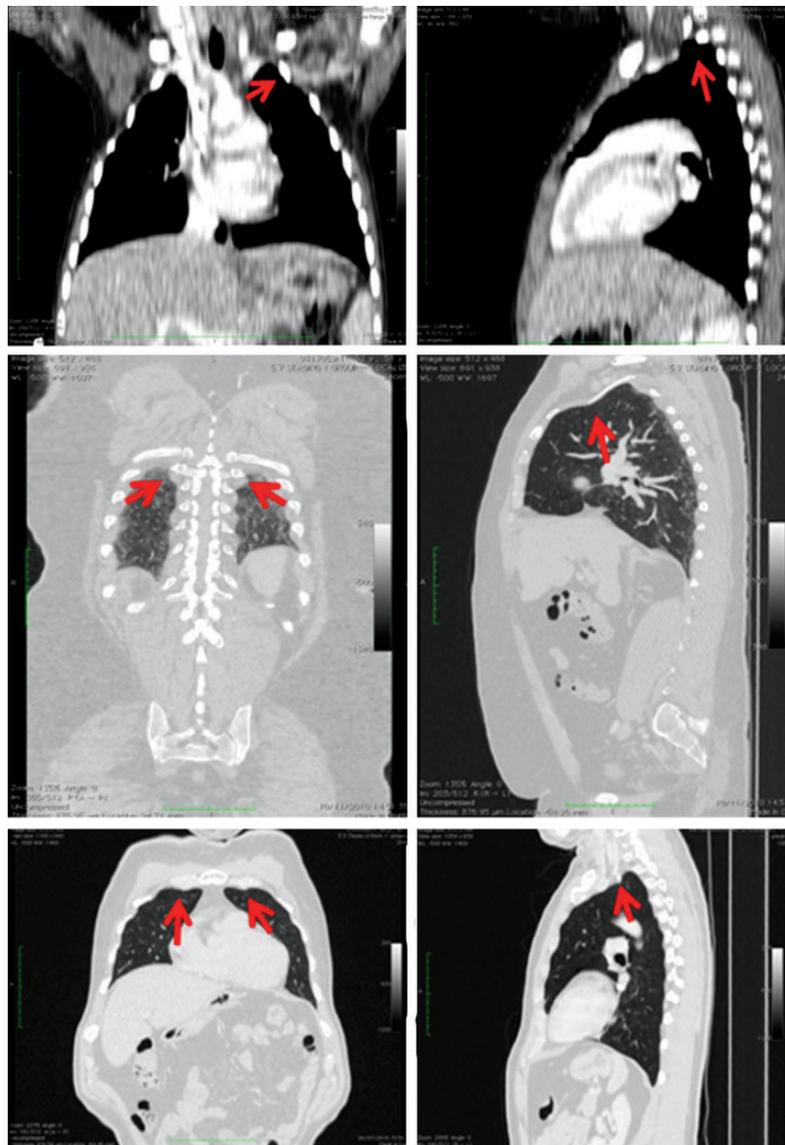
Figure 2: Coronal and parasagittal CT reconstructions from different individuals showing indentations on the lung produced by the upper ribs, especially the first ribs. The arrows show the grooving caused by the first rib.
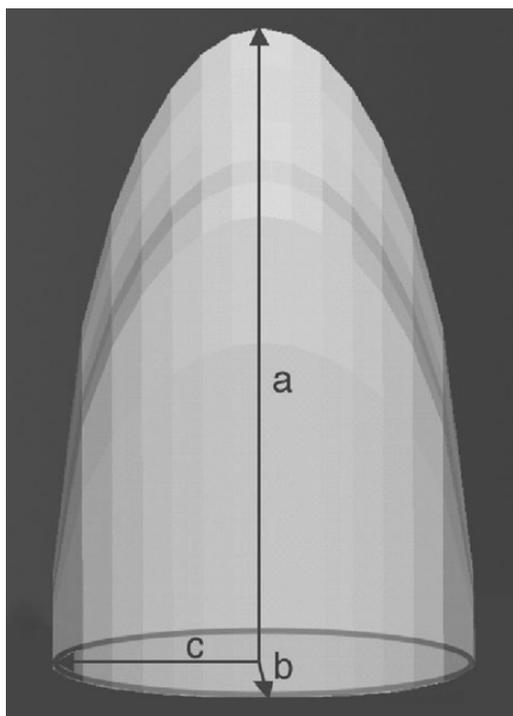
Figure 3: Side view of lung model, where the height of the lung *a* was 24 cm, the transverse radius of the lung *b* was 8 cm and the anteroposterior radius of the lung *c* was variably modelled depending on the posterior to lateral lung diameter ratios.

Table 1: The results of chest measurements of the pneumothorax and control groups using Bellemare's methodology [12]

Parameter	Controls	Pneumothorax	P-value
n	12	12	
Age	24.1	25.0	0.20 ns
Depth of chest at level T3/4	131.5	127.3	0.24 ns
Depth of chest at level T5/6	171.6	164.7	0.21 ns
Depth of chest at level T7/8	193.0	182.8	0.17 ns
Depth of chest at level T9/10	199.5	177.5	0.03
Angle of sixth rib with the vertical	63.2°	53.8°	0.02
Width of chest at level T3/4	214.1	244.2	0.009
Width of chest at level T5/6	253.2	268.8	0.04
Height of right chest	211.7	236.2	0.03

adult Caucasian males to assess the presence of indentations on the lung caused by the first rib (Fig. 2).

A finite element model of a pressure vessel having an indentation at the apex (as in the case of the lungs) was constructed to investigate the effect of changing the anteroposterior to lateral diameter ratios, and thus better visualize how the stress levels vary in the lung. This model was based mathematically on a six-degree polynomial, where the height of the lung *a* was 24 cm, the transverse radius of the lung *b* was 8 cm and the anteroposterior radius of the lung *c* was variably modelled depending on the posterior-to-lateral diameter ratios (Fig. 3). The model was then built and analysed in Ansys v11 (ANSYS, Inc., Philadelphia, PA, USA) finite element simulation package using linear modelling. The model was meshed using the element SOLID 187, where it was assumed that the composing material of the pressure vessel was elastic with a Poisson's ratio of 0.3. A series of analyses were performed while changing the ratio of the anteroposterior and lateral diameters mimicking different body shapes ranging from round to the flattened, low thoracic index shape that is associated with spontaneous pneumothorax by changing the variable *c* (Fig. 3) to obtain thoracic indices (ratio of anteroposterior to lateral chest diameters) of 5:8, 6:8, 7:8 and 8:8 (round). This was done to assess whether differences in anteroposterior diameters would influence stress distribution patterns in the pleura. The base of the model was constrained superoinferiorly but allowed to inflate freely in the transverse plane. The other parts of the model could inflate freely. All models were subjected to a pressure of 40 kPa [13].

T-test statistics were performed using Excel (Microsoft Corp, Richmond, VA, USA). The null hypothesis was considered valid at a *P*-value of <0.05.

RESULTS

The male pneumothorax patients had a taller chest (*P* = 0.03), transversely wider at T3/4 and T5/6 levels (*P* = 0.009 and *P* = 0.04) and flatter at T9/10 level (*P* = 0.03), as compared with a group of controls matched for average age (Table 1).

The CT data showed that the apex was not a simple ellipsoid, as is commonly illustrated, but had grooves marking indentations by the upper ribs, especially the first and second ribs. There was clearly a flattened teat appearance to the apex with shouldering caused by the grooves. The prominence of the upper ribs anteriorly and posteriorly varied on an individual basis. This grooving was present in all CTs examined in differing degrees. These indentations are demonstrated in Figs 2 and 3.

A series of four FEA simulations (Fig. 4) of the lung revealed significantly higher stress (×5–×10) in the apex than in the rest of the lung. This was accentuated (×4) in low thoracic index chests, resulting in ×20 higher stress levels in the apex of low thoracic index chests. The magnitude of maximal stress in all models is shown in Fig. 5.

DISCUSSION

Primary spontaneous pneumothorax was first recognized as different from the then prevalent disease of tuberculous pneumothorax by Kjaergard in 1931, who recognized the presence of apical emphysematous areas with or without additional scarring. Later Devillers suggested that the pathophysiology was secondary to subpleural bullae [14]. The fact that population height has increased [15] and patients with primary spontaneous pneumothorax are often tall thin males [1] may explain why the incidence is increasing [16].

Proposed aetiologies have included the 'low position of the diaphragm . . . (said to) concentrate the tension on the lung apices favouring both vesicle formation and rupture' [17] and congenital apical cysts with abnormally thin pleura with little elastic supporting tissue. Other suggested pathophysiological mechanisms included 'increased gravitational pull on the apex' [18], localized hypoperfusion and ischaemia causing 'wear and tear on such blebs with rupture and spontaneous pneumothorax' [19] and 'stress on the
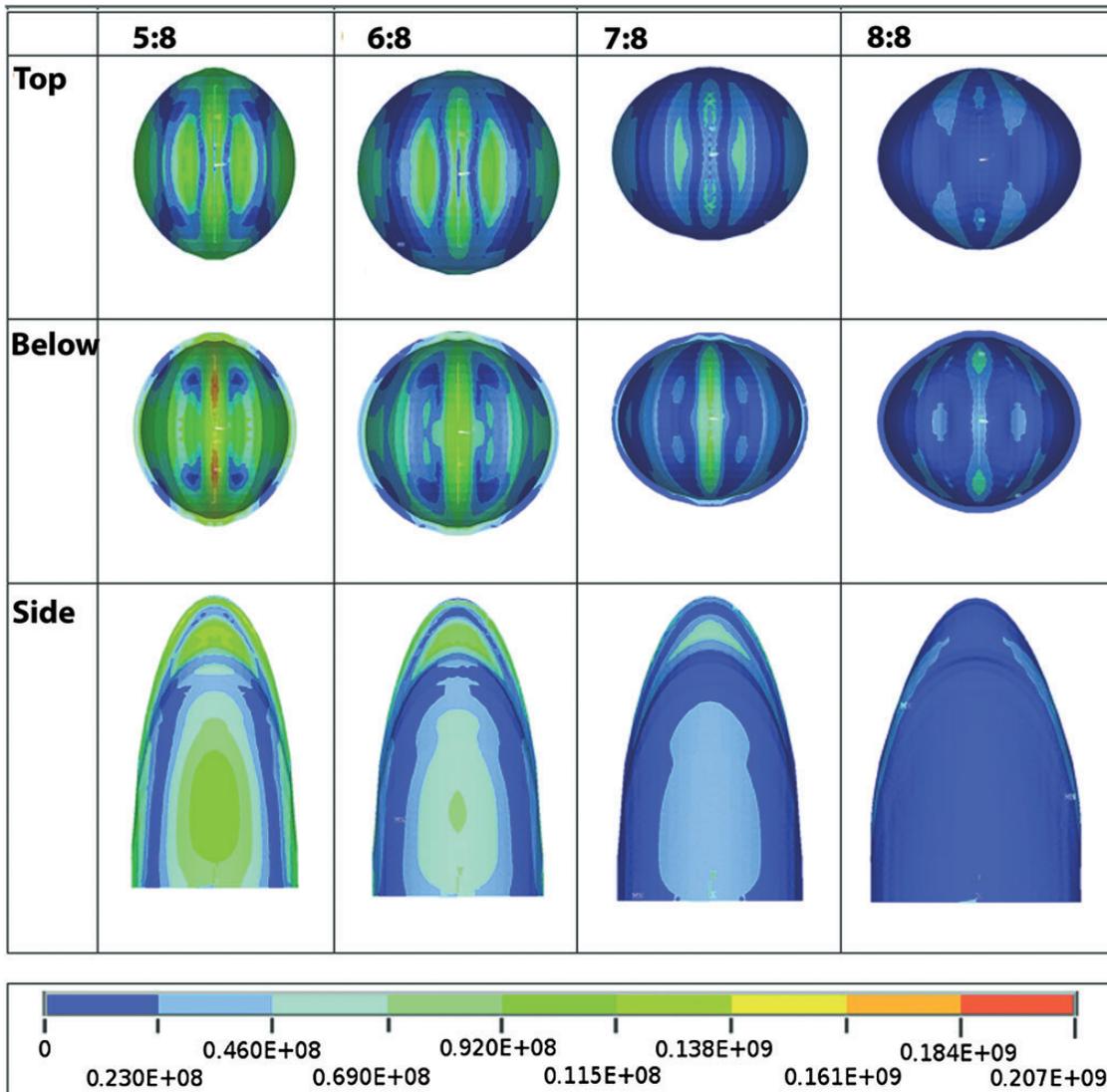
Figure 4: Stresses within the pleura of the lung on maximal inflation with differing chest wall anteroposterior to lateral diameter ratios. Pleural stress is maximal at the apex and at the site of the first rib furrows. Pleural stress is maximal with a slender base ratio of 0.6 (5:8).
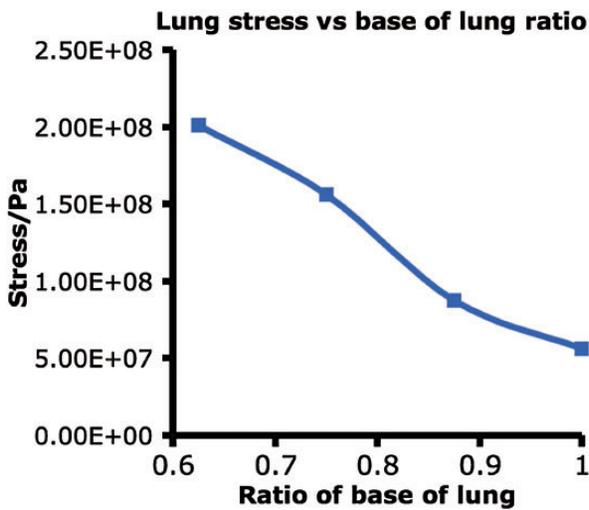

Figure 5: Maximum apical stress on maximal coughing of 40 kPa is reached with slender base ratio of 0.6 (5:8). Apical stress decreases as the base becomes rounder.

lung apex, causing subsequent expansion and rupture of a bulla' [20]. The greatest mechanical stress was found to be present at the apex due to a mix of 'gravity-induced stress and surface pressure' [21]. Other possible mechanisms quoted were due to 'repeated trauma, caused by friction of the lung against the chest wall' and also due to the sharp edge of the first and second ribs cutting into lung tissue and weakening the bullae [22].

Most spontaneous pneumothoraces have apical blebs or bullae [7, 11]. Bullae seem to leak air at certain pressure due to pleural porosity [23]. Sometimes scar tissue indicating a previous bulla is found. Rarely they are present in the apex of the lower lobe.

The wall stress in pressure vessels is proportional to the radius of curvature (Law of Laplace). The distending pressure on coughing is equal in all parts of the lung (Pascal's Law). Since pleural pressure is more negative at the apex than at the base of the lung, alveoli at the lung apex are exposed to an even greater distending pressure differential than at the base of the lung, especially in tall individuals [20].

Based on the CT data and the low thoracic index present in spontaneous pneumothorax patients [9], an anteroposteriorly
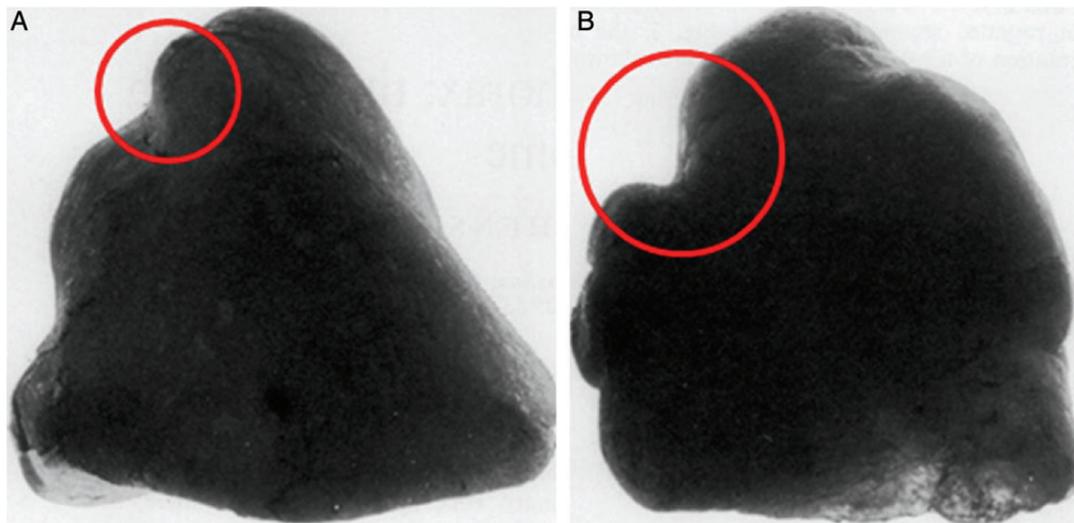
Figure 6: Moulds of the apex of the lung for two different specimens showing prominent indentations (**A**) anteriorly and (**B**) posteriorly due to prominent first and second ribs that would feel sharp to a surgeon. Reproduced from reference [22] with permission from BMJ Publishing Group Ltd.
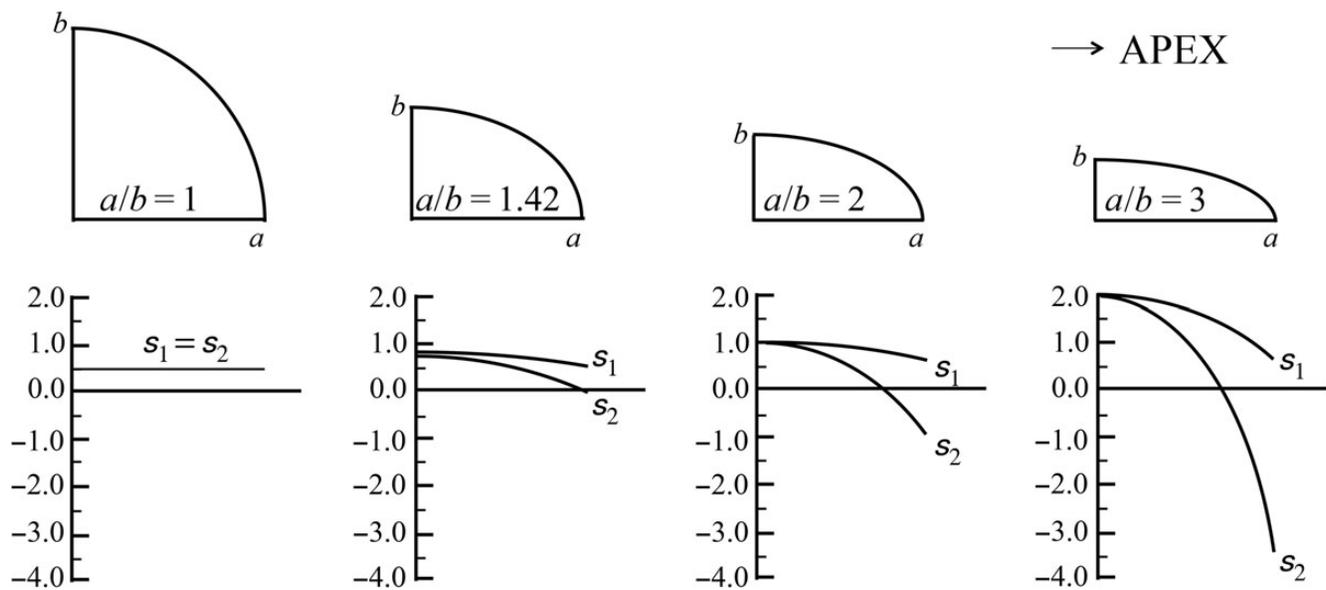
Figure 7: Ratios of stress in spheres and in ellipsoids, with varying ratios of major-to-minor (*a/b*) axis. After Fryer DM, Harvey JF. High Pressure Vessels. New York, NY: Chapman and Hall, 1997, 27–28.

flattened teat shaped apex was selected as the shape for stress analysis using finite element modelling and analysis. FEA modelling is an ideal solution for calculating wall stress in complex pressure vessels, as it involves applying a series of nodes in a mesh-like structure to model the original in three dimensions, thus reducing the complexity of the problem from an infinite into a finite number of problems. Each node on the mesh has boundary conditions and loads applied to it, simulating the original loads and resultant stress and strain gradients. The anteroposteriorly flattened teat shape is clearly seen in Fig. 6 in moulds of the apical portion of the lung, with the first two ribs marked as grooved indentations.

Geometrically the apex of the lung generally has a prolate or elongated spheroidal shape similar to that of a rugby ball or bullet. When the ratio of the height to the base of the apex protruding beyond the first rib exceeds 1.42 (Fig. 7), the circumferential stress component (marked σ_2 in Fig. 7) becomes negative. This means that there is instability at the shoulder area of the prolate spheroid or tearing as the pressure vessel pulls apart. The negative curvature of the rib grooves would also result in the reversal of the normal surface loads (circumferential stresses), leading to tearing in the shoulder areas due to the reversal from positive-to-negative curvature. A low thoracic index body shape causes a further 4-fold increase in apical stress. This would cause fatigue and failure in a pressure vessel. A tall, unsupported apex is therefore prone to increased stress; this geometrical shape may also explain the occurrence of pneumothorax in the apex of the lower lobe.

The results show that the narrowest model was associated with highest stress in the apex and shoulder areas of the apex when compared with the rounder models. This would imply that tall thin patients are prone to excess stress located at apices and the grooved surrounding area during coughing. This excess stress is associated with the surface force reversal, and instability and tearing occurs. Bullae at the apex of these patients would be stretched. There would be thinning of the pleura with porosity [24], possibly with repair by scarring. The process could lead to rupture and primary spontaneous pneumothorax in some patients.

Females have a smaller chest than males, with small diminutions in both anteroposterior and lateral rib cage diameter [12]. This smaller size results in less pleural forces being generated on coughing. This may be sufficient to prevent pneumothoraces. The same mechanism also follows in children, and only when adult body size in adolescence is reached does the risk of pneumothorax appear.

The chest wall changes with age [25]; in particular, the thoracic index increases as the ribs droop with age, dropping outwards laterally with age. There is a generalized rounding of the thoracic cage associated with aging and obesity, and chest anteroposterior diameter increases with both age and increasing BMI [12], while the lateral diameter remains static. This generalized rounding with age would result in a decrease in apical stress, as demonstrated in our 8:8 ratio model, explaining how patients outgrow pneumothorax and how therefore primary spontaneous pneumothorax is a disease affecting mostly young people with low thoracic index chest walls, clarifying the higher incidence in young service personnel [16].

After pneumothorax surgery, such as apicectomy and apical parietal pleurectomy, the pleural forces remain unchanged and could result in recurrence of pneumothorax; however, an effective pleurodesis would result in a pleura supported by adhesions to the chest wall, thus relieving pleural stresses. A possible explanation for prolonged air leak after pneumothorax surgery may be the time taken for effective pleural adhesions to form. Other technical issues may include residual leaking bullae and incomplete staple lines. Pleural stresses may also be the explanation for progression of emphysema after bullectomy, since the lung moulds itself to the pleural cavity, thus leading to severe apical stress, which can theoretically be limited by concurrent pleurodesis.

This work is the first paper that relates spontaneous pneumothorax, chest wall shape and pleural stress. Although a mechanism for disease progression dependent on pleural stress has been identified, it is clear that additional work is required for a fuller understanding of this mechanism. It is hoped that this study will stimulate further investigations in this field.

CONCLUSION

Primary spontaneous pneumothorax appears to be to a low thoracic index moulding the lung into a shape that causes an environment of high apical stress that leads to pleural buckling and fatigue, with resulting pleural tissue porosity and bulla formation. Mild thoracic index differences, such as those occurring in women

or with aging, can reduce stress levels. This may explain the predominant presentation in young male adults.

Conflict of interest: none declared.

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