

## CASE REPORT

# Music: a new cause of primary spontaneous pneumothorax

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Most cases of primary spontaneous pneumothorax are thought to be caused by air leaks at so-called “emphysema-like changes” or in areas of pleural porosity at the surface of the lung. Environmental pressure swings may cause air leaks as a result of transpulmonary pressure changes across areas of trapped gas in the distal lung. This is the first report of music as a specific form of air pressure change causing pneumothorax (five episodes in four patients). While rupture of the interface between the alveolar space and pleural cavity in these patients may be linked to the mechanical effects of acute transpulmonary pressure differences caused by exposure to sound energy in association with some form of distal air trapping, we speculate that repetitive pressure changes in the high energy-low frequency range of the sound exposures is more likely to be responsible. Exposure to loud music should be included as a precipitating factor in the history of patients with spontaneous pneumothorax.

Primary spontaneous pneumothorax is defined as the spontaneous occurrence of pneumothorax in patients without apparent underlying pulmonary disease. It typically occurs in young, tall, thin, smokers. Although no apparent underlying lung disease is present, most patients present with some abnormalities in the affected (and sometimes also at the contralateral) lung. Subpleural blebs or bullae (emphysema-like changes, ELCs) are seen in most of the patients, as shown by high resolution CT scanning<sup>1</sup> or during thoracoscopy or thoracotomy.<sup>2</sup> The actual site of air leakage, however, can be located at the ELCs which may be ruptured in some cases,<sup>3</sup> or elsewhere at the lung surface (“pleural porosity”).<sup>4</sup> Alternatively, air can enter after alveolar rupture with an air leak into the peribronchovascular interstitium (causing pneumomediastinum) and ultimately into the pleural space.<sup>5</sup> Whatever the exact site of the air leak, some kind of transpulmonary pressure difference between the alveolar space and pleural space—resulting in rupture of the alveolo-pleural barrier and penetration of air into the pleural space—has to be present.

We report four patients in whom five episodes of spontaneous pneumothorax occurred after exposure to loud music, a specific form of air pressure variation.

### CASE HISTORIES

#### Case 1

A 23 year old non-smoking man of Tunisian origin was transferred to our clinic for management of a first episode of primary spontaneous pneumothorax. Five days before his admission he had experienced a sudden right sided pleuritic chest pain and dyspnoea while attending a pop concert, standing quietly within a few metres of several large loud speakers. His medical history was negative except for a familial myopathy characterised by generalised Ehlers-Danlos like ligamentary hyperlaxity and muscle weakness.

He was treated with a 24 Fr chest tube drain but was transferred for thoracoscopic pleurodesis because of a persistent (72 hours) air leak. At thoracoscopy several apical blebs were visualised, one of which was ruptured. The ruptured bleb was coagulated and a thoracoscopic talcage with 3 g sterile, asbestos-free talc was performed. The postoperative recovery was uneventful and persistent pleurodesis was obtained. Three days later the patient was discharged. Two months later a small partial recurrence of a right sided pneumothorax occurred. Because there was only a 1 cm air rim around the right lower lobe, no active treatment was proposed. After 5 days the pneumothorax had resolved completely. There have been no recurrences during a 3 year follow up period.

#### Case 2

A 25 year old man of Moroccan origin was transferred to our clinic for management of a first episode of primary spontaneous pneumothorax. The day before admission he had experienced a sudden pleuritic left sided chest pain while visiting a dance hall. The pain occurred when he was standing quietly in the vicinity of a loud speaker. His past medical history included asthma, retinal loosening, and a perforated tympanum after otitis media. He was an active smoker (20–30 cigarettes per day for 7 years). He was treated with manual aspiration using a 16 G catheter.<sup>6</sup> After radiographic confirmation of re-expansion of the left lung, he was discharged the same day. There have been no recurrences in a 1 year period of follow up.

#### Case 3

A 23 year old white male smoker (10 pack years) was first seen with an episode of left sided primary spontaneous pneumothorax in November 1998. Because he mentioned that he had experienced two similar episodes of left sided pleuritic chest pain and dyspnoea in the past (which had resolved spontaneously), the diagnosis of recurrent pneumothorax was made and a thoracoscopic talcage was performed. Follow up was uneventful until February 2002 when he presented with a first episode of right sided spontaneous pneumothorax. A CT scan showed apical ELCs at both lung apices. A general work-up including measurement of  $\alpha_1$ -antitrypsin was negative. At the patient's request a right sided thoracoscopic talcage was performed. In July 2002 a very small right sided recurrence of pneumothorax occurred while on holiday in Spain. No active treatment was proposed since there was only a small rim of air around the lower lobe. While discussing the pathogenesis and precipitating causes of recurrent spontaneous pneumothorax during a follow up visit, we mentioned having seen two patients in whom exposure to loud music was associated with the occurrence of a pneumothorax. At that moment the patient also recalled that both his first left sided pneumothorax and second right sided pneumothorax had occurred while attending a heavy metal rock concert.

#### Case 4

A 19 year old white male smoker had suffered a first right sided pneumothorax when driving a 125 cc monocylinder motorbike. A chest radiograph showed a complete pneumothorax which was successfully treated by simple aspiration. Two years later he had bought a car in which he installed a 1000 Watt base box in the boot as he liked to listen to loud music in his car. While doing this he experienced a sudden pain in the right side of his chest followed by breathlessness. He immediately knew that his pneumothorax had recurred and this was subsequently confirmed radiographically. He is convinced that the very loud music in his car triggered this recurrent pneumothorax. He was advised to undergo thoracoscopic pleurodesis but elected to await events.

#### DISCUSSION

This is the first report of primary spontaneous pneumothorax occurring after exposure to very loud music. Although the occurrence of spontaneous pneumothorax in these young men may have been coincidental, the exact temporal relationship between the symptoms and the exposure to loud music suggests this may have been the cause.

Sound is a form of mechanical energy characterised by wave front propagation through a physical medium. Propagation of sound pressure waves through the respiratory system is a complex three-dimensional problem, but it is bound to result in pressure differences at the interface between media of different densities. (air, alveolar surface water and tissue). These pressure differences could tear the alveolar or ELC walls resulting in an air leak into the pleural space. We propose three types of mechanism that could be involved in this process.

Firstly, primary blast damage to gas containing organs such as ears or lungs can occur if the mechanical energy of sound is very high, as in blasts or explosions.<sup>7</sup> Very loud sounds at close range, as in our patients, could be considered as a miniature variant of "repetitive blasts", causing a lung blast injury (pneumothorax). Blast injury has generally been attributed to direct mechanical damage—that is, rapid compression/decompression forcing air against various usually delicate compartments such as the alveolar septa, causing them to rupture.<sup>8</sup> Besides its direct mechanical impact on the lung, blast injury has also been shown to induce biochemical changes such as antioxidant depletion and lipid peroxidation, both correlating with blast peak overpressure<sup>9</sup> which may last for hours after exposure. These changes may be involved in the development of the structural abnormalities that predispose to spontaneous pneumothorax. Exposing the lungs to one high or several low energy blasts may precipitate alveolar or ELC rupture. The cumulative effect of repetitive insults to the lung parenchyma or pleura due to these vibrations could lead to injury of the alveolar walls after exposure to high energy impulse noises or blasts.<sup>10</sup>

A second mechanism may be linked to the presence of a so-called check valve mechanism responsible for a time delay in pressure equilibrium, and for overdistension of distal lung regions. Patients with primary spontaneous pneumothorax may have an inflammatory "bronchiolitis" with distal air trapping.<sup>11</sup> Airway inflammation or mucus retention may cause a check valve phenomenon leading to intrapulmonary air pressure differences and possibly chronic anatomical changes (ELC or pleural porosity) and therefore pneumothorax. It is possible that loud music induces changes in atmospheric pressure that are less dramatic than those generated by blasts but which fail to be transmitted immediately to the region distal to this "check valve". The resulting transpulmonary pressure difference may be sufficient to cause rupture of alveolar or ELC walls. In case 1,

weakening of the alveolar surface may have been enhanced by the presence of the underlying Ehlers-Danlos-like connective tissue disorder which is often characterised by poor quality elastic tissue.

A third potential mechanism is related to a particular frequency band of the acoustic pressure wave spectrum. A series of publications has focused on the effects of exposure to low frequency (<500 Hz) high intensity (>90 dB) noise, a range of structural lung changes having been reported mainly in rats<sup>12</sup> but also in humans.<sup>13</sup> These data suggest that low frequency high intensity noise may lead to structural and functional changes in the airways, pleural mesothelium, and lung parenchyma. With frequencies from commercial loud speakers typically in the range of 30 Hz to 20 kHz, it is the lower frequency band of 30–150 Hz which is usually boosted in big music venues for enhanced effect. These low frequencies could indeed be particularly damaging to the lung parenchyma if they coincide with its natural frequency of around 128 Hz.<sup>14</sup> Furthermore, Mahagnah and Gavrieli<sup>15</sup> showed that, in normal humans, the lung acts as a low pass filter with flat transmitted energy up to 100 Hz or 300 Hz. In view of these experimental results, the most compelling observation in our four patients is that the lower frequency band, which tends to be boosted in the kind of music they listen to, corresponds with those frequencies, thereby ensuring maximal acoustic energy transfer to the lungs.

We conclude that exposure to loud music may be a cause of spontaneous pneumothorax. Possible mechanisms for this observation are suggested. While it may be linked to the mechanical effects of acute transpulmonary pressure differences caused by sudden exposure to sound energy in association with distal air trapping, we speculate that repetitive pressure changes in the high energy-low frequency sound range are more likely to be responsible. Exposure to loud sounds should therefore be included in the list of precipitating factors for primary spontaneous pneumothorax and, as illustrated by case 3, should be asked for specifically when taking the history of a patient presenting with pneumothorax.

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## LUNG ALERT .....

### BCG provides protection for a lifetime

▲ Aronson NE, Santosham M, Comstock GW, *et al*. Long-term efficacy of BCG vaccine in American Indians and Alaska natives: a 60-year follow-up study. *JAMA* 2004;**291**:2086–91

**T**his paper analyses the efficacy of BCG vaccination in 2792 American Indians and Alaska natives enrolled in a placebo controlled trial of BCG between 1935 and 1938. They were recruited aged 1 month to 20 years following a normal chest radiograph and absence of reaction to a strong dose of purified protein derivative of tuberculin. Follow up data were obtained from the Indian Health Service medical records, tuberculosis (TB) registries, death certificates, and participant interviews. TB cases were strictly defined as those with either culture identification of *Mycobacterium tuberculosis*, evidence of clinical disease with response to antituberculous treatment, or evidence of acid-fast bacilli/granulomata at autopsy. The BCG group (n = 1483) and placebo group (n = 1309) had case rates of 66 and 138 per 100 000 person years, respectively, giving a vaccine efficacy of 52% (95% CI 27 to 69). BCG appeared more efficacious against extrapulmonary than pulmonary TB (63% and 52%, respectively). Analysis by decade yielded efficacies of 54%, 62%, 62%, 12%, and 48%. This suggests a slight waning over time that was statistically significant in men but not in women (p = 0.02) although, because of the small number of TB cases, these results must be interpreted with caution.

The authors conclude that single dose BCG vaccination can give protection for up to 60 years. However, before extrapolating these findings to other populations, further research is needed to determine if use of different BCG strains or frequent exposure to *M tuberculosis* or environmental mycobacteria could influence the long term efficacy. Differences between the sexes also require investigation.

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