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ELECTROCARDIOGRAPHIC CHANGES IN PATIENTS WITH SPONTANEOUS PNEUMOTHORAX

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The aim of the study was to evaluate the prevalence of electrocardiography (ECG) abnormalities in subjects with spontaneous pneumothorax. Forty consecutive patients (mean age 43.7 ±19.1 years) with spontaneous pneumothorax participated in the study. There were 22 cases of left-sided and 18 cases of right-sided pneumothorax. The mean relative volume of pneumothorax was 51.4 ±24.7% according to the Light's index and 53.5 ±22.9% according to the Rhea method. Heart rate was significantly higher in patients with pneumothorax than after lung re-expansion (91 ±20 bpm *vs*. 72 ±16 bpm; P<0.001). Abnormal left axis deviation was found in 3 patients with left-sided and in 1 with right-sided pneumothorax, while abnormal right axis deviation was found in 2 patients with left-sided pneumothorax. Relevant QRS abnormalities (incomplete RBBB and T-wave inversion) were found in 4 patients (10%). QRS amplitude in V2-V6 leads was significantly decreased in left-sided pneumothorax, while the right-sided pneumothorax was associated with an increase in QRS amplitude in V5-V6. We conclude that ECG in subjects with pneumothorax often reveals significant abnormalities. The most significant abnormalities were seen in patients with massive right-sided pneumothorax.

Key words: *ECG, electrocardiography, pneumothorax*

INTRODUCTION

Few electrocardiography (ECG) textbooks describe changes in subjects with spontaneous pneumothorax. Very limited information is also provided in monographs and other texts dealing with pleural diseases. Most frequently, they merely mention the possibility of ECG changes in pneumothorax, and only few publications discuss these changes more comprehensively (1). This is most likely related to the assumption that the wide availability of chest X-ray makes differentiation between spontaneous pneumothorax and cardiac conditions not difficult. However, under certain circumstances, the similar symptomatology of spontaneous pneumothorax and cardiac conditions (chest pain, dyspnea) together with ECG abnormalities may lead to misdiagnosis and delay in the treatment of pneumothorax.

Attention to ECG changes in patients with pneumothorax was initially drawn in the first half of the 20th century. Numerous patients managed with artificial pneumothorax for pulmonary tuberculosis constituted a very-large research material (2). Later years brought reports describing ECG changes in pneumothorax with patterns similar to other serious respiratory and cardiovascular conditions, such as pulmonary embolism (3) and myocardial infarction (4, 5).

Only few summaries of ECG changes associated with pneumothorax have been published so far (2). Most of these studies were conducted in small groups of patients (6, 7). According to these studies, the most common ECG changes in pneumothorax include: right axis deviation, reduced QRS amplitude in precordial leads, and T wave inversion.

The aims of our study were: (1) to evaluate the prevalence of ECG changes in subjects with spontaneous pneumothorax, (2) to determine the nature of these changes, and (3) to assess the relationship between ECG changes and pneumothorax size, side, and the degree of hypoxemia as the potential underlying mechanism of the ECG changes in patients with pneumothorax.

MATERIAL AND METHODS

The study protocol was approved by an institutional Ethics Committee.

The study included 40 patients with spontaneous pneumothorax admitted to the Department of Internal Medicine, Pneumonology and Allergology, Medical University of Warsaw, Poland, between 2000 and 2005. The diagnosis of pneumothorax was based on clinical examination and chest X-ray. Only patients with indications for pleural drainage based on pneumothorax size and clinical symptoms were included in the study (8, 9). Subjects with tension pneumothorax and subjects who failed lung re-expansion within 5 days due to a massive air leak were excluded.

Estimated pneumothorax size was calculated in all eligible patients using Light's index (1, 10) and the Rhea method (10, 11). The calculations were performed independently by two authors (RK and TP). If the difference between the two authors did not exceed 5%, the mean of the two measurements was calculated, but for differences in excess of 5%, the final pneumothorax size was established by consensus.

At least two standard 12-lead electrocardiograms were obtained for each patient (after the diagnosis of pneumothorax, but prior to drain placement, and after a complete re-expansion of the lung has been achieved and confirmed radiologically). Electrocardiograms were obtained using E600G, Farum SA, Poland or Hewlett Packard M1772A electrocardiographs (paper feed: 50 mm/s, calibration: 1 mV = 10 mm). Four ECG parameters were evaluated: (1) heart rate, (2) morphology of P waves, QRS complexes and T waves, (3) axis deviation, (4) QRS amplitude changes in precordial leads.

ECG tracings obtained following pneumothorax evacuation and complete lung re-expansion were signed as baseline. Pneumothorax-induced changes were evaluated by comparing the baseline ECGs and pneumothorax ECGs (prior to chest drainage).

Axis deviation in the frontal plane was determined using triaxial system of bipolar extremity leads, according to Scheidt (12). Pneumothorax-related electric axis changes were defined as the difference between the axis position on baseline ECG and the axis position calculated from the pneumothorax ECG.

The following ECG parameters in precordial leads were evaluated: (1) QRS amplitudes in V1-V6, (2) height and depth of R and S waves in V1-V2 and V5-V6, and (3) position of the first lead (counting from V1 to V6) with R wave higher than the depth of the S wave. Normal QRS amplitude was defined as the mean amplitude in V1-V6 in the baseline ECG. The calculated value was compared with the mean QRS amplitude in V1-V6 calculated in the pneumothorax ECG. The impact of pneumothorax on the QRS amplitude change in precordial leads was calculated by subtracting the pneumothorax QRS amplitude from the baseline QRS amplitude (*e.g.*, $\Delta QRSV1 = QRSV1_{baseline} - QRSV1_{pneumothorax}$).

Arterial blood gases were sampled in 22 subjects following the diagnosis of pneumothorax, but before pleural drainage.

The statistical analysis was performed using a statistical software package (StatSoft, Inc. STATISTICA, version 8.0. www.statsoft.com.). All data were presented as means ±SD. The Shapiro-Wilk W test was used to assess the normal distribution for all variables. A t-test for unpaired samples was calculated to test for significance between the groups. For variables inconsistent with normal distribution, we used the corresponding non-parametric tests (Mann-Whitney U test or Wilcoxon signed-rank test for non-related and related samples, respectively). Spearman's rank correlation coefficient was measured to test for potential correlations between extent of pneumothorax and various ECG parameters. Results with P<0.05 were considered statistically significant.

RESULTS

The study group comprised of 40 subjects: 31 males (77.5%) and 9 females (22.5%) with the mean age of 43.7 ± 19.1 years (range: 18-86). There were 22 cases (55%) of left-sided pneumothorax (LSP) and 18 cases (45%) of right-sided pneumothorax (RSP). Twenty-four subjects had primary spontaneous pneumothorax and 16 had secondary spontaneous pneumothorax with several underlying conditions including chronic obstructive pulmonary disease (COPD) in 10 patients, post-tuberculosis in 2 cases, asthma in 1 patient, 1 case of lymphangioleiomyomatosis, 1 case of pneumonia, and 1 case of pulmonary metastases of breast cancer. The mean duration of pleural drainage was 7 ± 5 days (range: 1-26 days).

The mean pneumothorax size was 51.4 ±24.7% according to Light's index and 53.5 ±22.9% according to the Rhea method (11). Despite no significant differences in the mean pneumothorax size between primary and secondary pneumothorax groups, pre-drainage partial oxygen arterial pressure (PaO_2) was significantly lower in patients with secondary versus primary pneumothorax (66.2 ± 20.6 mmHg, n=15, *vs*. 82.5 ± 17.2 mmHg, n=7; P<0.05).

Heart rhythm and rhythm abnormalities

Sinus rhythm was found in all baseline and pneumothorax ECG tracings. Heart rate was significantly higher prior to than following lung re-expansion (91 ± 20 bpm *vs*. 72 ± 16 bpm, P<0.001). No heart rhythm abnormalities were noted on ECGs obtained during pneumothorax and no distinct correlations were found between heart rate and pneumothorax size, irrespective of the way it was calculated, or between heart rate and PaO_2 measured during pneumothorax.

Changes in the cardiac electrical axis

There was no significant difference in the position of the electrical cardiac axis in patients with left-sided *vs*. right-sided pneumothorax (53.2° *vs*. 62.6°, NS). The mean change in pneumothorax-related axis position in the frontal plane for the whole group was 25.6° ± 35.6°. Because 24 subjects had right axis deviation and 13 subjects had left axis deviation, the mean pneumothorax-related axis deviation was not significant (+1.7° ± 44.0°). In 3 patients, pneumothorax had no effect on the axis position. The mean change in cardiac axis was greater in left-sided *vs*. right-sided change (35.8 ± 54.6° *vs*. 22.3 ± 20.2°), but was not statistically significant (NS). We found a correlation between the absolute axis deviation and pneumothorax size calculated from Light's index (r=0.39, P<0.05).

In 22 subjects with left-sided pneumothorax, the absolute mean change in axis position was 31.9 ± 45.2°. Since pneumothorax resulted in right axis deviation in 12 of them and left axis deviation in 8 of them, the mean axis position changed only slightly (+1.8 ± 55.8°). In two patients, pneumothorax did not affect the axis position. Of the 4 patients with axis change in excess of 60°, right axis deviation was noted in three and left axis deviation in one patient. The greatest single change in axis position was revealed in a patient with pneumothorax-related left axis deviation (change from +85° to -120°, *i.e.*, by -205°). The mean left axis deviation in left-sided pneumothorax was smaller than right axis deviation (39.6 ± 67.8° *vs*. 29.5 ± 25.5°, NS).

In 18 patients with right-sided pneumothorax, the absolute mean axis change was 18.3 ± 17.7°. Since pneumothorax resulted in right axis deviation in 12 and left axis deviation in 5 subjects, the mean axis position changed by a mere +1.72°

Table 1. Details of cardiac axis deviation.

Side of pneumothorax	Pneumothorax-induced cardiac axis change	Number of cases (% of ipsilateral pneumothoraces)	Mean ±SD change in axis position (in angular degrees)
Left	Right	12 (54.5%)	39.6±67.8
Left	Left	8 (36.4%)	29.5±25.5
Left	No change	2 (9.1%)	0
Right	Right	12 (66.7%)	15 ±9.15
Right	Left	5 (27.8%)	29.8±28.7
Right	No change	1 (5.6%)	0

± 25.8°. In one patient with right-sided pneumothorax axis position did not change at all. Although right axis deviation was more common than left axis deviation, the greatest right axis deviation caused by right-sided pneumothorax did not exceed 30°. In two patients with left-sided pneumothorax axis deviation exceeded 30° (and 60° in one patient). The mean left axis deviation in right-sided pneumothorax was also greater than right axis deviation, but the differences were not statistically significant (29.8° ± 28.7° *vs*. 15.0° ± 9.15°, NS). The absolute axis deviation in right-sided pneumothorax correlated with pneumothorax size calculated from Light's index ($r=0.55$, $P<0.05$).

Detailed data on cardiac axis deviation in relation to the side of pneumothorax are shown in *Table 1*. There was no relationship between the frequencies of right and left axis deviation and the localization of pneumothorax, *i.e.*, whether it was right or left-sided. In all the subjects without pneumothorax, the axis fell within the acceptable limits for adults (between -30° and +100°). Pneumothorax caused abnormal left axis deviation (*i.e.*, less than -30°) in 4 patients and abnormal right axis deviation (*i.e.*, more than +100°) in 2 of them. The cause of abnormal axis deviation in the frontal place was left-sided pneumothorax in 5 patients and right-sided pneumothorax in only 1 patient.

Changes in morphology of P waves, T waves and QRS complexes

Significant ECG changes in T waves and QRS complexes were found in 4 patients (10%). *Table 2* summarises the features of these subjects and their ECG changes. ECG abnormalities were found in both patients with right and left-sided pneumothorax. Incomplete right bundle branch block was diagnosed in three

Table 2. Characteristics of the 4 patients with ECG changes.

	Side of pneumo-thorax	Sex M/F	Age (yr)	Axis position following re-expansion (angular degrees)	Axis position during pneumo-thorax (angular degrees)	Axis change (angular degrees)	PaO_2 on admission (mmHg)	ECG changes during pneumo-thorax	Pneumo-thorax size (Light's index; %)	Pneumo-thorax size (Rhea method; %)
1	R	F	26	65	90	+25	92.2	Negative T waves in III, aVF	79.2	81.5
2	R	M	50	0	-70	-70	46.4	Incomplete right bundle branch block	69.4	49.9
3	L	M	30	85	120	-205	NA	Incomplete right bundle branch block	74.8	85.5
4	R	M	40	72	90	+18	NA	Incomplete right bundle branch block	91.5	99.3

R - right-sided pneumothorax; L - left-sided pneumothorax; PaO_2 - arterial partial pressure of oxygen; NA - not available.

patients (one case of right-sided pneumothorax and two cases of left-sided pneumothorax) (*Fig. 1A* and *B*). One 26-year-old woman had T wave inversion in III and aVF leads (right-sided pneumothorax) (*Fig. 2A* and *B*). All the changes resolved following lung re-expansion.

When we compared the group of 4 patients with ECG abnormalities to the remaining 36 patients in whom no significant ECG changes were observed, we found significant differences in the size of pneumothorax. The mean pneumothorax size in subjects with ECG changes was 78.7% ± 9.4% and 78.8% ± 21.3% according to Light's index the Rhea method, respectively, whereas the respective values in the group of patients without ECG changes were 48.0% ± 23.9% and 50.4 ± 21.4% (P<0.05). Detailed features of the patients with and without ECG abnormalities are summarized in *Table 3*.

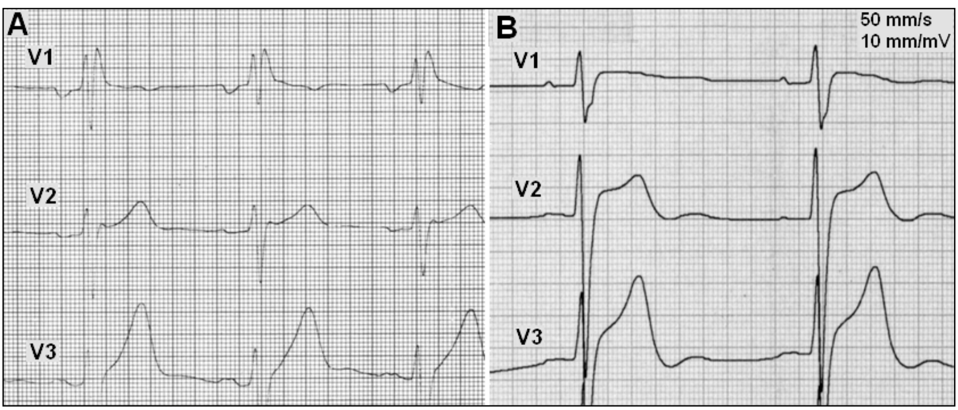

Fig. 1. A - Right bundle-branch block in a 30-year-old male with left-sided pneumothorax, ECG on admission; B - ECG after lung re-expansion.

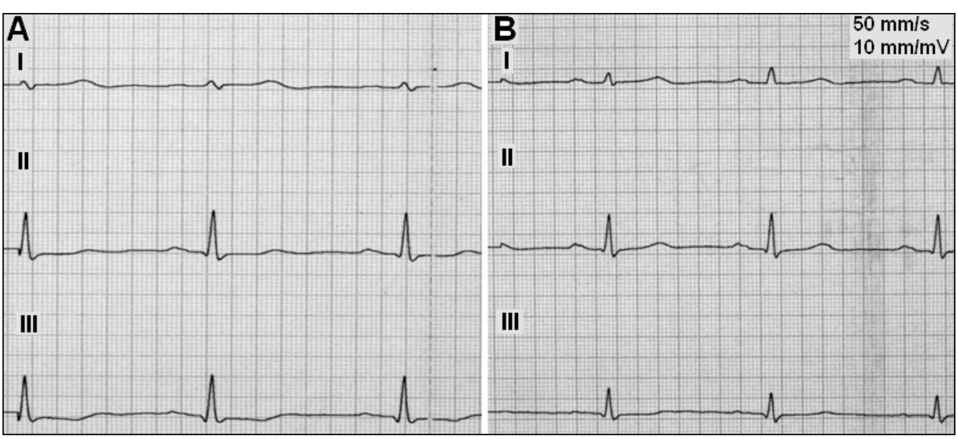

Fig. 2. A - Inverted T wave in lead III and a 26-year-old female with right-sided pneumothorax, ECG on admission; B - ECG after lung re-expansion.

Precordial QRS amplitude

The mean amplitude of the highest and lowest deflection of the QRS complex in V1-V6 leads in the pneumothorax ECG was significantly lower than that in the baseline ECG (1.2 ± 0.53 mV *vs*. 1.4 ± 0.47 mV, $P < 0.05$). The QRS complex amplitude in leads V1-V4 was significantly lower prior to than that following the pneumothorax evacuation. No such relationship was observed for V5 and V6 leads. The height of the R wave, the depth of the S wave in V1 and V2, and the height of the R wave in V5 and V6 were significantly lower in the pneumothorax ECG *vs*. baseline ECG. A reverse relationship was observed for the S waves in V5 and V6.

Table 3. A comparison of patients with and without pneumothorax-related ECG changes.

Parameter/variable	Patients without ECG abnormalities	Patients with ECG abnormalities	P value
Number of patients, n (%)	36	4	
Left-sided pneumothorax, n (%)	21 (58.3%)	1 (25%)	NS
Right-sided pneumothorax, n (%)	15 (41.7%)	3 (75%)	NS
Age in years (mean ±SD)	44.5 ±19.6	36.5 ±10.7	NS
Change in axis position caused by pneumothorax (°); (mean ±SD)	50.5 ±36.8	62.3 ±25.7	<0.05
PaO_2 before the placement of a drain (mmHg; mean ±SD)	71.6 ±20.4	69.3 ±32.4	NS
Pneumothorax size: Light's index (%; mean ±SD)	48.0 ±23.9	78.7 ±9.4	<0.05
Pneumothorax size: Rhea method (%; mean ±SD)	50.4 ±21.4	78.8 ±21.3	<0.05

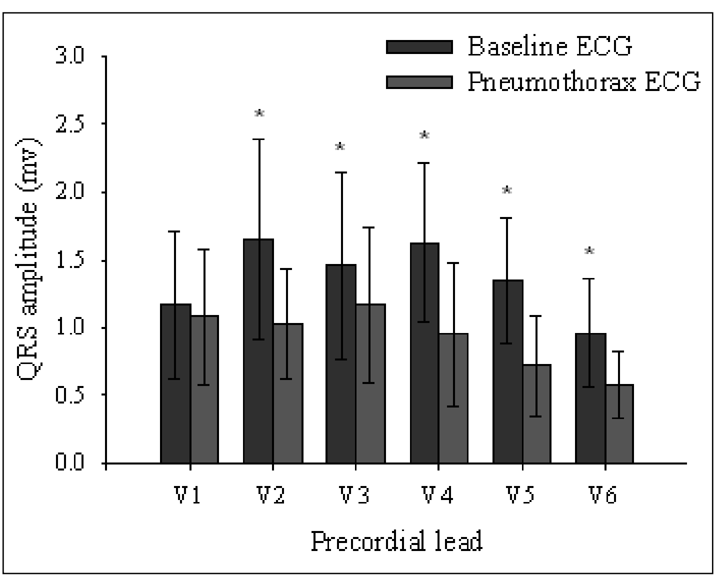

Fig. 3. QRS amplitude changes in precordial leads in patients with left-sided pneumothorax. *P<0.05, baseline ECG vs. pneumothorax ECG.

The mean amplitude of the highest and lowest deflection of the QRS complex in V1-V6 in left-sided pneumothorax was significantly lower during pneumothorax than that after lung re-expansion (0.9 ± 0.3 and 1.4 ± 0.5 mV; respectively, $P<0.05$). Left-sided pneumothorax resulted in a significant reduction of QRS amplitude in all leads except for V1 (*Fig. 3*). The effect of left-sided pneumothorax on the individual deflections in V2, V5, and V6 was reflected by a significant reduction of the height of the R wave in V5 and V6 and by a non-significant increase of the depth of the S wave in V5 and V6.

Right-sided pneumothorax did not change the mean QRS amplitude in precordial V1-V6 leads. Right-sided pneumothorax resulted in a significant reduction of QRS amplitude in V1 and V2, had no significant effect on QRS amplitude in V3 and V4, and significantly increased QRS amplitude in V5 and V6. The reduction of QRS amplitude in V1 and V2 was a net result of a reduced height of the R wave in V1 and V2 ($P<0.05$ for both changes) and a reduced depth of the S wave in V1 and V2 ($P<0.05$ and NS, respectively). The increased QRS amplitude in V5 and V6 was a net result of a significant increase in the depth of the S wave in V5 and V6 ($P<0.05$ for both changes) and a non-significant increase in the height of the R wave.

An analysis of the results for the entire study group and for the subgroups (left-sided and right-sided pneumothorax) failed to find a correlation between the precordial QRS amplitude change and the pneumothorax size.

DISCUSSION

Acute chest conditions have a fairly common symptomatology. Chest pain and dyspnea are the dominant manifestations in various dissimilar conditions, such as myocardial infarction, pulmonary embolism, and pneumothorax. ECG is one of the most important diagnostic tools used in the differential diagnosis of these conditions. In the majority of cases, ECG abnormalities suggest cardiac problems. However, there are a large number of non-cardiovascular conditions and diseases that may affect ECG, most notably: electrolyte imbalance, acute central nervous system conditions, hypo- and hyperthermia, hypo- and hyperthyroidism, drugs, and stimulants. Pneumothorax is rarely listed as a cause of ECG abnormalities. The possibility of such changes is most often mentioned in subjects with tension pneumothorax (13-16).

It is worth remembering that tuberculosis specialists were the first to draw attention to pneumothorax-related ECG changes. Artificial pneumothorax they used for the management of tuberculosis offered an opportunity to compare ECGs obtained prior to and after induction of pneumothorax. The considerable prevalence of tuberculosis and the widespread use of therapeutic pneumothorax made it possible to perform observations on relatively large groups of patients. Reports on the nature of ECG changes in patients with artificial pneumothorax were first published in the first half of the 20th century (2, 17). However, the ECGs obtained in that period were in a large part less informative, as they employed fewer leads (normally: 3 bipolar limb leads and 1 to 3 precordial leads), which considerably decreases the value of these observations (2). The first report on ECG changes in patients with pneumothorax drew attention to differences in electrocardiographic abnormalities in patients with artificial and spontaneous pneumothorax (17). Because of the above factors, the value of these early observations is quite limited.

We have found no studies in larger groups in the available literature that would allow us to draw conclusions on the incidence and patterns of ECG changes in spontaneous pneumothorax. Most commonly, the reports include isolated cases with ECG changes mimicking myocardial ischaemia (18), myocardial infarction (4, 5), pulmonary embolism (3), or anecdotal ECG changes, such as breathing-dependent QRS amplitude changes (19) or PR segment elevation (14).

Considering the circumstances, we aimed to analyze ECG changes in a large and relatively uniform group of subjects with spontaneous pneumothorax. Because we included all patients managed with pleural drainage for spontaneous pneumothorax (with the exception of lung re-expansion failures and subjects with persistent massive air leak) in whom technically adequate ECGs could be obtained, we were able to conduct a qualitative evaluation and assess the incidence of the changes. To the best of our knowledge no such data have been published so far.

Our results indicate that ECG changes in patients with spontaneous pneumothorax are a relatively common finding. If significant ECG changes were defined as pathological axis deviation (which we found in 6 patients) and the specific changes in the QRS and T wave morphology (in 4 patients), then the incidence of these changes in spontaneous pneumothorax would have to be estimated at 25% (10 out of 40 patients). Abnormal axis deviation is much more common in patients with the left than right-sided pneumothorax (5/22 or 22.7% vs. 1/16 or 6.25%). Changes in QRS (most commonly right bundle branch block) and T waves (inversion) were, on the other hand, more common in patients with the right than left-sided pneumothorax (3/16 or 18.75% *vs.* 1/22 or 4.5%). ECG changes could, therefore, be expected in subjects with both left and right-sided pneumothorax. An abnormal axis deviation is a predominating finding in the former group while QRS and T wave changes predominate in the latter.

The topic that generated considerable interest was the change in heart axis position as a result of the presence of air in the pleural cavity. Intuitively, pneumothorax should affect the axis position secondary to the change in the heart position. In all the seven cases of left-sided pneumothorax reported, Walston et al (20) confirmed axis deviation to the right (in the range from 11° to 235°). Previously, however, Armen and Frank (2), in an analysis of 43 patients, managed with pneumothorax due to tuberculosis, reported axis deviation to the right in the right and left-sided pneumothorax alike. These changes were present in about

40% of the cases (2). The analysis of cardiac axis changes in our material suggested that pneumothorax caused slight changes in the axis position in nearly all cases. While the presence of pneumothorax more commonly led to the right than left axis deviation (57.5% *vs*. 32.5%, NS), the mean deviation to the left was greater than that to the right (NS). Among patients with pneumothorax-induced abnormal axis deviation, deviation to the left was more common than deviation to the right (4 *vs*. 2 patients) and it was more commonly caused by left than right-sided pneumothorax (5 *vs*. 1 patients). In both cases, abnormal right axis deviation was caused by the left-sided pneumothorax. Abnormal left axis deviation was also much more commonly caused by the left-sided pneumothorax, although in one case it was associated with the right-sided one. Other authors have reported abnormal right axis deviation also in the right-sided pneumothorax, which was associated with acute right ventricular overload and enlargement (21). In summary, changes in the axis position are a common finding in pneumothorax. Abnormal axis deviation is found in about 15% of pneumothorax patients and is much more common in the left-sided pneumothorax. In subjects with the right-sided pneumothorax, the absolute axis deviation correlated with pneumothorax size calculated according to Light's index. No such relationship has been demonstrated for the left-sided pneumothorax.

T wave inversion in patients with pneumothorax is an important ECG abnormality and a relatively common finding in the left-sided pneumothorax. It might give rise to diagnostic uncertainties. Walston *et al.* (20) reported this abnormality in 3 out of 7 subjects with the left-sided pneumothorax (20). We found only one such case, which - in contrast to the previous studies - concerned a female patient with the right-sided pneumothorax. Walston *et al.* (20) explain the T wave changes by abnormal myocardial repolarisation possibly secondary to: abnormal myocardial perfusion, intrapleural pressure fluctuations, and/or pericardial cavity and increased resistance in the pulmonary circulation. It is very unlikely that the ECG abnormalities we found could have been caused by myocardial perfusion abnormalities, as they developed in a young, 26-year-old female with a PaO_2 of 92.2 mmHg at the time of the ECG.

We found no reports of a bundle branch block in patients with pneumothorax in the available literature. In our study we found 3 cases of incomplete right bundle branch block which developed in two subjects with the right-sided and one with left-sided pneumothorax. Two underlying mechanisms could be at play here: a compression of the right ventricle by the air accumulated in the right pleural cavity and increased pulmonary artery pressure with right ventricular strain leading to abnormalities in the heart conduction system.

The case reported by Janssens et al (22) proves that the presence of pneumothorax, especially tension pneumothorax, may precipitate myocardial ischaemia and the resulting ECG changes. The patients with ECG abnormalities we studied were slightly younger (NS) and had similar PaO_2 compared to patients without ECG changes. This may suggest that myocardial oxygenation abnormalities which may develop during pneumothorax may be at the root of these changes (hypoxemia, coronary flow abnormalities in the elderly).

We also noted the previously reported phasic voltage alterations (23) in the ECGs obtained in our pneumothorax patients. However, in contrast to the previous findings, we also confirmed this finding following evacuation of pneumothorax. We did not conduct a detailed analysis of the incidence and severity of these abnormalities or their relationship with the presence of pneumothorax.

An evaluation of precordial QRS amplitude revealed reduced mean amplitude sums of the highest and the lowest deflection of the QRS complex in V1-V6 in pneumothorax patients. The effect of left-sided pneumothorax on QRS amplitude in precordial leads differed from that of right-sided pneumothorax. The left-sided pneumothorax resulted in a significant reduction of QRS amplitude in all leads except for V1. A reduction in QRS amplitudes was also seen in all cases of left-sided pneumothorax reported by Walston et al (20). Reduced QRS amplitude in the left-sided pneumothorax has also been reported by other authors (4, 18, 24). The right-sided pneumothorax resulted in significant reductions of QRS amplitudes in V1 and V2 and significant increases of QRS amplitudes in V5 and V6. Only Alikhan and Biddison (7) observed reduced QRS amplitude in the right-sided pneumothorax, but failed to specify the leads in which these changes could be observed. The fact that QRS amplitude is reduced in patients with the left-sided pneumothorax only and that this change is mainly seen in the left ventricular lateral leads may suggest that these changes are caused by a displacement of the heart away from the chest wall, leading to the attenuation of electrical impulses. This hypothesis is supported by the observation that the right-sided pneumothorax leads to increased QRS amplitude in V5 and V6 (most likely due to decreased distance between the heart and the left lateral portion of the chest wall).

The underlying mechanisms of ECG changes in pneumothorax patients have not been fully elucidated. The multitude of changes and the lack of correlation between pneumothorax size and side suggest that more than one pathophysiologic mechanism may be involved. The following two factors seem most likely: rotation of the heart around its long axis and the presence of air between the chest wall (and therefore between the ECG electrodes) and the myocardium. Littmann (25) conducted an interesting study supporting the latter hypothesis. ECGs were obtained from two patients with spontaneous pneumothorax in supine and standing positions. In the supine position, reduced amplitudes of precordial R waves and T wave inversion were found. These changes were absent in the standing position. In both positions chest X-ray confirmed the difference in the volume of retrosternal space air. The author concluded that the accumulated air impaired electric impulse conduction between the myocardium and the electrodes giving rise to ECG changes. This mechanism seems to have been confirmed by Copeland and Omenn (26) and Feldman and January (27) who reported ECG changes mimicking myocardial infarction that disappeared when ECG was obtained in the standing position. Another potential mechanism explaining ECG changes involves an increase of in pulmonary artery pressure leading to right ventricular overload and hypoxemia leading to coronary blood flow abnormalities (22).

The issue of ECG changes co-existing with pneumothorax is not merely theoretical, as indicated in a report by Botz and Brock-Utne (24). Thanks to the knowledge on such changes these authors diagnosed left-sided pneumothorax intraoperatively, which allowed for prompt intervention and the avoidance of dangerous complications. The first sign of pneumothorax, in the absence of physical symptoms and in subjects in good overall condition, was reduced QRS amplitude (24).

In summary, pneumothorax-induced ECG changes are a relatively common finding. They may be expected in both left-sided and right-sided pneumothorax alike. Abnormal axis deviation is much more common in patients with the left-sided compared with right-sided pneumothorax, while changes in the QRS morphology (most commonly right bundle branch block) and T waves (inversion) appear more often in patients with the right-sided pneumothorax. The latter finding was only observed in patients with massive pneumothorax. The effects of left-sided vs. right-sided pneumothorax on precordial QRS amplitude changes are different. The change in QRS amplitude in these leads in pneumothorax patients is most likely secondary to alterations in the distance between the heart and the chest wall and to changes in electric impulse conduction pathways.

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