Established in 1871

Swiss Medical Weekly

Formerly: Schweizerische Medizinische Wochenschrift

An open access, online journal • www.smw.ch

Original article | Published 03 August 2021 | doi:10.4414/SMW.2021.w30041
Cite this as: Swiss Med Wkly. 2021;151:w30041

Electrocardiographic alterations by pneumothorax: a case-control study with review of the literature

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Summary

BACKGROUND: Numerous ECG alterations due to pneumothorax have been reported. The objective of the study was to establish the presence of ECG changes associated with pneumothorax in the literature, and in a cohort of patients with proven pneumothorax compared with age- and sex-matched healthy controls.

METHODS: A systematic review for ECG alterations associated with pneumothorax was performed. We then reviewed our hospital database for patients with pneumothorax and identified all patients with an ECG available at this time. The retrieved ECG alterations in the systematic review were identified in our pneumothorax patients and compared with a healthy sex- and age-matched control group. Accordingly, we calculated sensitivity and specificity for all alterations.

RESULTS: Seventeen ECG alterations were found and defined from the systematic review. Our pneumothorax cohort consisted of 82 pneumothorax patients and 82 control patients. Specificity was mostly more than 90%, but sensitivities were low. Phasic R voltage (pneumothorax group 25.6% vs control group 1.2%), T-wave inversion (31.7% vs 2.4%), prolonged QTc (11.0% vs 2.4%), right axis deviation (14.6% vs 3.6%) and QRS voltage ratio in aVF/I >2 (41.5% vs 22.0%) were significantly more frequent in pneumothorax patients compared with controls.

CONCLUSION: The sensitivity of published ECG signs in predicting pneumothorax in our cohort was low, which means that ECG findings are an unsuitable tool for pneumothorax screening. However, presence of these ECG signs might raise a suspicion of pneumothorax in patients presenting with dyspnoea, or unclear chest discomfort.

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Introduction

Pneumothorax is defined as collapse of the lung due to a collection of air in the pleural cavity between the parietal pleura and the lung. It may be spontaneous or traumatic. Pathological findings as to the cause on conventional chest radiography are absent in primary spontaneous pneumothorax, although small blebs and bullae are often found on computed tomography (CT). Underlying pathological findings in the lungs are found in secondary spontaneous pneumothorax. Traumatic pneumothorax is caused by lesions of the thoracic wall, or lesions of the lung or the pleura via the airways. The incidence of spontaneous pneumothorax ranges from 14.1–22.7/100,000 with a male to female ratio of 3.3:1 [1, 2]. Traumatic pneumothorax is found in about 20% of all chest injuries [3].

The diagnosis is confirmed by imaging. The electrocardiogram (ECG) as a diagnostic tool is not mentioned in the diagnostic algorithms for pneumothorax in any guidelines [4]. However, it is mentioned in “chest pain” guidelines [5] and a door-to-ECG time of 10 minutes is recommended by the American Heart Association [6].

Numerous ECG alterations due to pneumothorax have been reported over the last century. One of the first case series on ECG alterations was published in 1928 and showed changes of the QRS axis with nonspecific alterations in repolarisation [7]. Subsequently, many other alterations were reported, mostly in case reports. Defining and recognising these signs in patients entering the emergency department (ED) with chest pain or dyspnoea could lead to immediate imaging, potentially avoiding delayed treatment of the pneumothorax.

The objective of this study was to collect all ECG alterations in pneumothorax patients and perform a systematic literature review to establish the diagnostic accuracy of these alterations in a large case-control series of patients, retrospectively assessed from our hospital database.

Materials and methods

A literature search of the electronic PubMed/MEDLINE and EMBASE databases was carried out from inception to 18 September 2019 to identify literature concerning ECG

alterations and pneumothorax. A search algorithm was established using a combination of the following terms: pneumothorax AND (ECG OR electrocardiogram OR electrocardiographic) (see the appendix). The local ethics committee approved the study (EKOS 16/093).

Patient selection

The electronic medical records and ECG database at our institution were retrospectively searched for patients leaving the emergency department (ED) with a final diagnosis of pneumothorax (cases) between 01 January 2011 and 31 December 2015. All patients treated with chest tube insertion and an ICD-code of spontaneous tension pneumothorax (J93.0), other spontaneous pneumothorax (J93.1), other pneumothorax and air leak (J93.8), pneumothorax unspecified (J93.9) or traumatic pneumothorax (S27.0) were included if they had a 12-lead ECG recorded at ED admission. Exclusion criteria were: patients without placement of a chest tube, patients with an imaging modality not allowing quantification of pneumothorax size (ultrasound diagnosis, chest X-ray in the supine position, or a chest CT scan with large slices), patients with known or acute heart or lung diseases, phrenic nerve palsy, chest deformities, prior lung resection, left bundle branch block, severe arrhythmias and pacemakers. Control patients were retrieved as follows: for each pneumothorax patient, we searched on the ECG database of the hospital for a sex- and age-matched patient in the same year. The age match started from 1 January of the year of birth of the pneumothorax patient, continuing sequentially to subsequent days until a patient with an ECG in the same year was found. This patient was finally screened using the same exclusion criteria as for the pneumothorax cohort. We defined a single control patient for each pneumothorax patient only – if a control patient was already included by the matching process, the next one identified following the same rules was chosen. In addition, we searched for follow-up ECGs in all pneumothorax patients after recovery of the pneumothorax. All ECGs were taken immediately after ED admission before chest tube insertion.

ECG interpretation

ECGs of all case and control patients were systematically examined by one emergency physician (BM) and an experienced electrophysiologist (PA) unaware of the clinical diagnosis of the patient. Every ECG was assessed for the presence of the ECG alterations retrieved by the literature search. From this review we defined 17 ECG signs to be analysed (see supplementary table A3 in the appendix). In the case of different interpretations of an ECG, the final diagnosis was made by consensus between both physicians.

Pneumothorax size assessment

The diagnosis of pneumothoraxwas based on chest X-ray or chest CT. The size of the pneumothorax on the chest X-ray was calculated using the Collin's formula [8]. The size of pneumothorax on the chest CT scan was calculated by radiologists unaware of the ECG findings using volumetry per manual segmentation of the slices. The size was calculated as a percentage of the hemithorax volume.

Statistics

Sensitivity and specificity for pneumothorax were calculated for each ECG sign. Subgroup analyses were made for the location, gender, aetiology (trauma vs non-trauma) and size of the pneumothorax. A separate subgroup consisting of pneumothorax patients with follow-up ECGs was analysed to describe the evolution of ECG changes. The McNemar test was used to establish statistically significant differences between proportions in the paired data, and the chi-square test was used for unpaired data. Continuous paired variables were compared using the Wilcoxon signed-rank test or paired t-test as appropriate. Continuous unpaired data were compared using Student's t-test or the Mann-Whitney test as appropriate. To compare the prevalence of the ECG signs in the subgroups, the Z score was used. We defined a level of significance of 0.05. Point biserial correlation was used to assess any correlation between the presence/absence of ECG signs and pneumothorax size. Cohen's kappa was used to test interobserver agreement of the ECG signs. Continuous data are presented as mean ± standard deviation or median with interquartile range as appropriate. Categorical data are presented as percentages. The IBM SPSS Version 25.0 software package was used for the statistical calculation.

Results

Population characteristics

We identified 836 patients with a final diagnosis of pneumothorax, of whom 82 included for the analysis. Ultimately, 82 age- and sex-matched controls were retrieved. The patient selection pathway is presented in figure 1.

Baseline characteristics of the pneumothoraxpatients and controls are presented in table 1. There was no case of tension pneumothoraxor documented cardiac contusion/lesion in traumatic pneumothorax patients included into the study.

Prevalence of ECG signs

The prevalence (sensitivity) and specificity according to the different ECG signs are given in table 2.

The most frequent ECG signs seen in pneumothorax patients were S reduction in V2 or V3 and T-wave inversion, which was most often visible in lead aVL (19 of 26 cases, 73.1%). The prevalence of these ECG signs was similar in the control patients. Seven of the 17 analysed ECG signs had a specificity of >98%, although sensitivity was low for these signs. Prevalence differed significantly between the cases and controls for phasic QRS voltage, T-wave inversion, right axis deviation and aVF/I QRS voltage ratio >2 (table 2). Four good examples of ECG signs associated with pneumothorax with resolution in a follow-up ECG are shown in figure 2.

Subgroup analyses

P-wave inversion in lead I was more frequent in right-sided pneumothorax (25.0 vs 5.6%, $p = 0.024$), whereas a baseline shift with P pulmonale (27.8 vs 9.1%, $p = 0.023$), the S <1.2 mV in V2 (72.2 vs 43.2%, $p = 0.016$), and the S <0.9 mV in V3 (61.1 vs 36.9%, $p = 0.030$) were more frequent with left-sided pneumothorax. Prevalence did not differ

significantly between left- and right-sided pneumothorax for any of the other signs.

Incomplete right bundle branch block was significantly more frequent in males (18.9 vs 0%, p = 0.022), whereas P pulmonale was more frequent in females (20.8 vs 12.1%, p <0.001).

There were several differences between trauma vs non-trauma patients. Phasic voltage ECG (p = 0.01), P pulmonale (p = 0.028), P inversion in I (p = 0.035), baseline shift with P pulmonale (p = 0.003), T-wave inversion (p = 0.005), aVF/I >2 (p <0.001), and ST elevation (p = 0.008) were more frequent with spontaneous pneumothorax, whereas left axis shift (p = 0.028) and long QTc (p = 0.423) were more frequent in traumatic pneumothorax. Mean size of the pneumothorax in the non-trauma group was 56.7% vs 22.8% in the trauma group (p <0.001).

The prevalence of a positive ECG sign correlated statistically significantly with pneumothorax size for phasic QRS voltage, aVF/I QRS voltage ratio >2, T wave inversion, P pulmonale, baseline shift with P pulmonale, and P inversion in lead I.

Twenty-one pneumothorax patients had a follow-up ECG a median of 87 days (IQR 19.5–571.5) after the initial ECG. The prevalence, evolution of an initial positive EGC sign and the specificity in this subgroup is presented in figure 3. In patients with a left-sided pneumothorax, there was a significant increase in R-wave amplitude in left-sided ECG leads after resolution of the pneumothorax (p = 0.0025 for V5 and p = 0.036 for V6), whereas the increase in R-wave amplitude in these leads was not statistically significant in patients with right-sided pneumothorax (p = 0.94 for V5 and p = 0.26 for V6).

Discussion

Different ECG alterations in acute pneumothorax have been published over past decades. QRS phasic voltage, T-wave inversion, QRS right axis deviation, aVF/I QRS voltage ratio >2 and QTc prolongation were the ECG signs that were significantly more frequent in our pneumothorax patients than in controls.

Only one third of our pneumothorax patients showed T-wave inversion on the ECG. However, this finding showed the strongest prevalence with pneumothorax. In contrast to

Figure 1: Flowchart of patient selection for the pneumothorax (PT) group. COPD = chronic obstructive pulmonary disease.

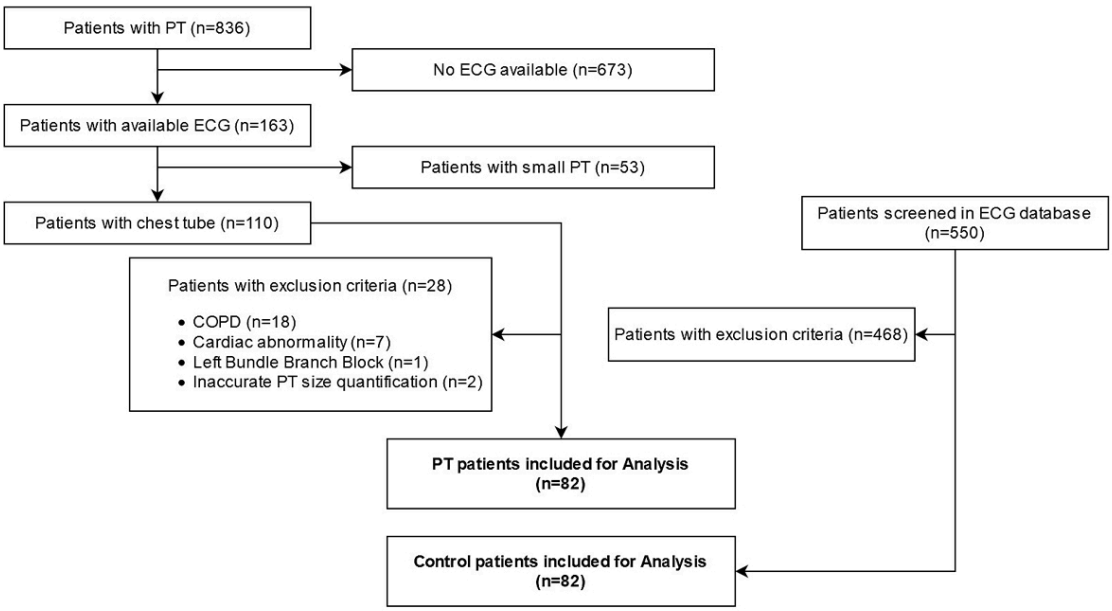


Table 1:
Baseline characteristics of the study population.

	Cases (n = 82)	Controls (n = 82)
Age (years), median (IQR)	53 (29.25–69.75)	53 (29.25–69.75)
Sex, n (%)		
– Male	58 (69.9)	58 69.9)
– Female	24 (29.3)	24 (29.3)
Site of pneumothorax, n (%)		
– Left	36 (43.9)	–
– Right	46 (56.1)	–
Clinical characteristics of PT, n (%)		
– Spontaneous	35 (42.7)	–
– Traumatic	47 (57.3)	–
Size, %	37 ± 32	–

IQR = interquartile range; PT = pneumothorax

our findings, in a study published by Krenke et al. [9], only 12.2% of pneumothorax patients showed T-wave inversions, 6.1% of patients in V2 to V6 (mostly V2), 3.6% in lead III, and only one patient (2.5%) in lead aVL. This is in contrast to a study by Kurisu et al., who analysed 10 patients with left-sided pneumothorax [10]. None of this group had T-wave inversion in the precordial leads. Unfortunately Kurisu did not analyse T-wave inversions in the limb leads. One of the ECG alterations most specific for pneumothorax in our study was phasic QRS voltage (or electrical alternans), which is associated with pericardial effusion and atrioventricular junctional tachycardias with an accessory pathways also [11]. Unfortunately, there is not a clear definition of "alternans" in the literature. Kurisu defined it as a variation of the R wave amplitude of at least 5 mm in at least one lead, whereas Huang defined it as phasic voltage variations of the R wave of over 2 mm in lead II [12]. Other authors described only the pattern without a metrical definition. In our cohort, we decided to use a variation in the R wave amplitude of at least 3 mm in at least one lead, as the best compromise between sensitivity and specificity.

The prevalence of the QRS voltage ratio avF/I >2 in our study was only 41.5%, as opposed to the 100% found by Kurisu. The reason for this difference is unclear.

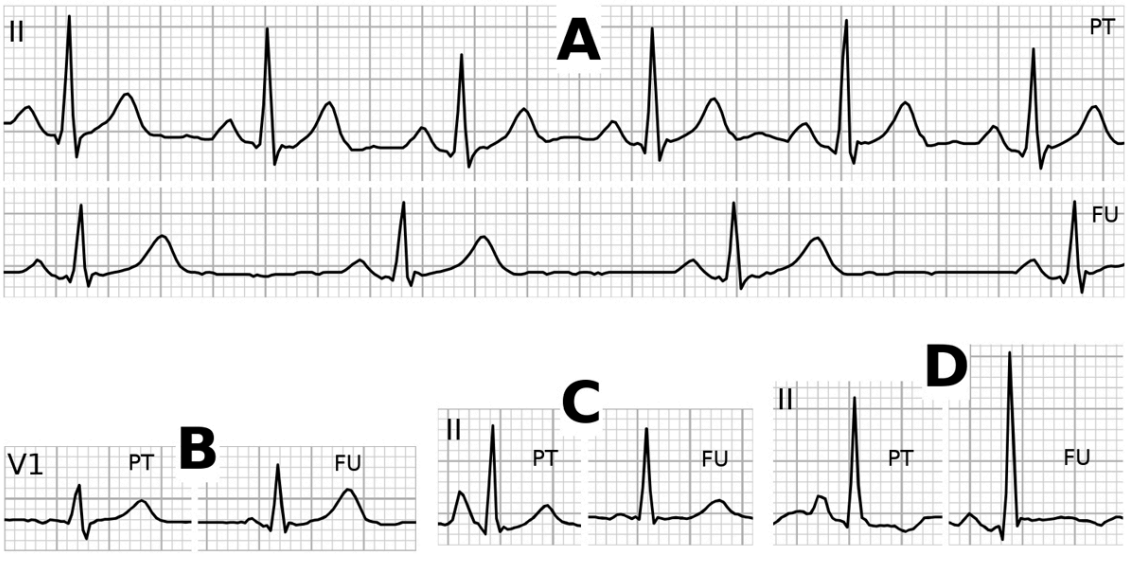
In a series described by Huang, the combination of three signs (reduction in S-wave in V2 and V3, and phasic QRS voltage) was found only in a group with large pneumothoraxes (light index >20%). Krenke et al. also showed that the number of ECG signs predicting pneumothorax was associated with the size of the pneumothorax. The number

Table 2:
Sensitivity and specificity for the presence of pneumothorax for different ECG signs and point biserial correlation with pneumothorax and size.

ECG sign	Prevalence in cases / sensitivity (%, n = 82)	Prevalence in controls (%, n = 82)	Specificity (%, n = 82)	p-value for difference in proportions	Point biserial correlation on PT size (Rpb)	Interobserver agreement (Cohen's kappa)
Phasic QRS voltage	25.6	1.2	98.8	<0.001	+0.3, p = 0.01	1.0
Right axis deviation	14.6	3.6	96.3	0.039	0, p = 0.57	1.0
Left axis deviation	7.3	4.9	95.1	0.683	−0.1, p = 0.24	1.0
Incomplete RBBB	13.4	11.0	89.0	0.803	−0.1, p = 0.43	0.8
RBBB	4.9	0	98.8	0.371	0, p = 0.80	1.0
P pulmonale	14.6	0	100	0.001	+0.3, p <0.01	0.9
P-wave inversion in lead I	15.8	0	100	<0.001	+0.5, p <0.01	1.0
Baseline shift with P pulmonale	17.1	0	100	<0.001	+0.4, p <0.01	0.9
T-wave inversion	31.7	2.4	97.6	<0.001	+0.4, p <0.01	0.9
Low QRS voltage	8.5	4.9	95.1	0.547	0, p = 0.97	0.8
QRS voltage ratio aVF/I >2	41.5	22.0	79.3	0.015	+0.2, p = 0.03	1.0
ST segment elevation	18.3	14.6	85.4	0.628	+0.2, p = 0.16	0.9
ST segment depression	1.2	0	100	1.000	−0.1, p = 0.25	1.0
S <1.2 mV in V2	57.3	62.2	37.8	0.596	−0.1, p = 0.54	1.0
S <0.9 mV in V3	46.3	53.7	46.3	0.511	0, p = 0.74	1.0
Prolonged QTc	11.0	2.4	97.6	0.046	−0.1, p = 0.28	1.0
Baseline shift with ST elevation ("spiked helmet sign")	0	0	100	Not available	Not available	1.0

CI = confidence interval; ECG = electrocardiogram; PT = pneumothorax; RBBB= right bundle branch block (Brugada pattern was searched for and not found in any of the cases).
The p-value for difference in proportions is given by McNemar (odds-ratio). Interobserver agreement is given with Cohen's kappa.

Figure 2: Examples of positive ECG signs by pneumothorax (PT) with resolution at follow-up (FU) in the same patient. A. Lead II with phasic QRS voltage changes. B. Lead V1 with P-wave inversion. C. Lead II with P pulmonale. D. Lead II with baseline shift before the P-wave onset.



of positive ECG signs and pneumothorax size were not directly compared in all of these studies.

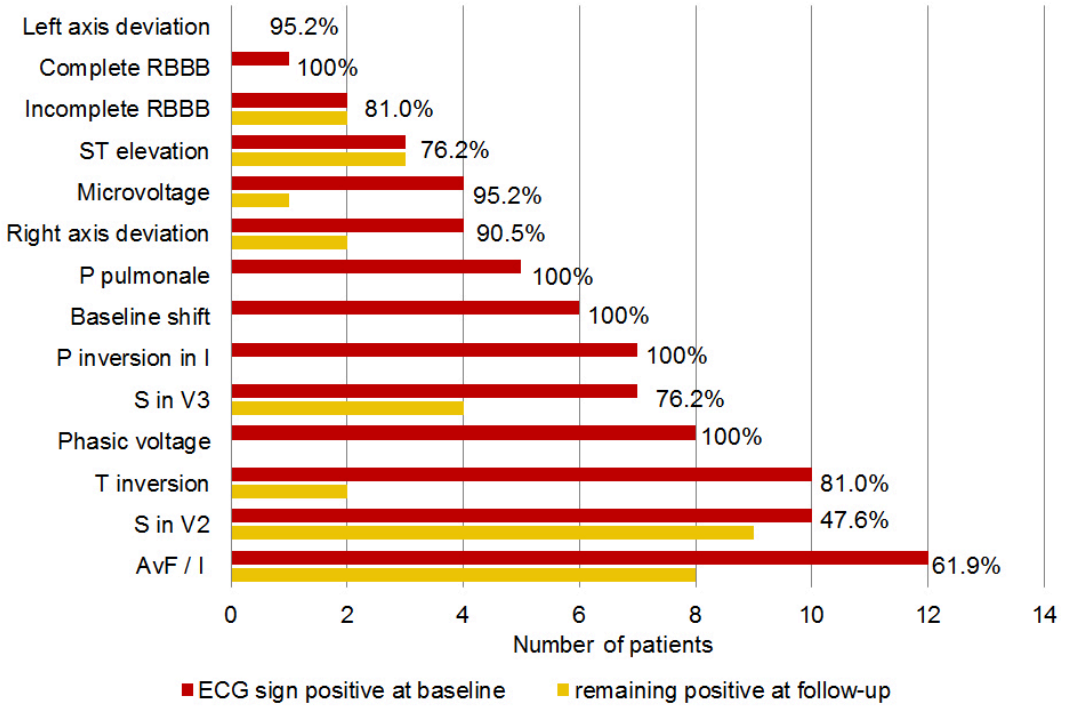
QTc prolongation was reported by Athanasopoulos et al. [13], who described QTc prolongation in 11 pneumothorax patients, although without findings for the prevalence of this sign.

The mechanisms of ECG alterations are discussed in many studies and seem to have multiple explanations. Cardiac displacement (downward and posterior) and (clockwise) rotation around the long axis seem to be responsible for many ECG alterations [12]. In addition, Feldmann [14] and Littmann [15] described T-wave inversion and QRS depression in pneumothorax patients in the supine position, demonstrating a "geometrical" relation between a pneumothorax and ECG alterations. They postulated an "insulation effect" of retrosternal free air, which might contribute to these changes. It is believed that phasic QRS voltage alteration is caused by the same displacement (in particular downward), emphasising the respirophasic pendular motion of heart [16]. Additionally, Huang et al. [12] hypothesised a lack of the parenchymal lung support in pneumothorax, which lets the heart "swing" within the respiratory cycle. Furthermore, right axis deviation is associated with right ventricular strain due to the collapsed lung, resulting in increased pulmonary resistance [17, 18]. Consequently reduced venous return and hypotension causing tachycardia and ischaemia might be another potential mechanism of ST segment alterations [19, 20].

Athanasopoulos et al. [13] hypothesised that autonomic imbalance could play a role in QTc prolongation and that epicardial stretch might result in the "spiked helmet sign" [21, 22]. In addition, takotsubo cardiomyopathy due to acute pneumothorax has been described in the literature, and increased sympathetic activation is discussed as an underlying pathophysiological concept, which again might cause ECG changes (e.g., tachycardia, QTc prolongation and ST segment alterations) seen in pneumothorax patients[23]. The mechanisms leading to T-wave inversion in patients with a pneumothorax are unknown. However, it is tempting to speculate as to whether a pneumothorax might cause ischaemia of the inner layers of the myocardium due to intrathoracic pressure shifts or catecholaminergic stress. The fact that so-called "false positive" elevation of cardiac troponins has been observed in pneumothorax patients [24] and that T-wave changes are more frequent in more severe pneumothorax might support this hypothesis.

Our study has several limitations. Because of the retrospective design, we were not able to test the published ECG alterations in our individual pneumothorax patients before and after treatment of pneumothorax, because most of the patients had no follow-up ECG. Analysis of pneumothorax in patients without lung and/or heart pathologies resulted in patients with traumatic or primary spontaneous pneumothorax only. However, it is possible that some patients had unknown heart/lung pathologies. This might reduce the power of ECG alterations for pneumothorax diagnosis only, and might somewhat reduce their specificity. Many patients with pneumothorax had to be excluded because no ECG has been recorded before pneumothorax treatment for a variety of reasons, but most possibly because an ECG has not been established as a tool to search for pneumothorax routinely. The study period goes back to 2015 based on an old database search. Because of the results presented, we decided to not update the study period, because we are sure that this would not have changed our results substantially. The choice of selecting only patients

Figure 3: Prevalence (number of patients) and evolution of positive ECG signs in 21 patients with pneumothorax and a follow-up ECG. The number at the end of the bars denotes the specificity for the ECG sign in this subgroup of 21 patients.



with chest tube insertion was also made because this was initially defined as an indirect measure of a clinically relevant pneumothorax size. Since cardiac troponins were not measured routinely in all pneumothorax patients, we cannot exclude that ECG alterations in traumatic pneumothorax could in part be due to heart contusion too. The small collective may have also contributed to statistical bias.

Conclusion

Taken together, we showed that the specificity for many of the listed ECG abnormalities is high, whereas the sensitivity is low. Thus, ECG signs are not suitable as a screening tool for pneumothorax. However, the presence of positive ECG signs discussed above might raise a suspicion of pneumothorax in patients presenting with dyspnoea or unclear chest discomfort in the ED, leading to early imaging and, in the case of confirmation, early treatment of pneumothorax.

Financial disclosure

This research received no external funding.

Potential competing interests

The authors declare that there is no conflict of interest regarding the publication of this paper.

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Appendix

Literature review to identify literature concerning ECG alterations and pneumothorax

Methods

This literature review conforms to the statement on Preferred Reporting Items for Systematic reviews and Meta-Analyses [1].

Search strategy

A literature search of the electronic PubMed/MEDLINE and EMBASE database was carried out from inception to September 18, 2019. A search algorithm was established using a combination of the following terms: pneumothorax AND (ECG OR electrocardiogram OR electrocardiographic).

Eligibility criteria

We included in this systematic review studies that filled the following inclusion criteria: (a) only English-language publications; (b) original article published in peer-reviewed journal; (c) studies and case reports with patients presenting with a pneumothorax and ECG changes. Exclusion criteria were: (a) articles not within the field of interest of this review; (b) review articles with only narrative descriptions (i.e., without patients).

Study selection

Titles and abstracts of the retrieved studies were reviewed by one researcher (BM), applying the inclusion and exclusion criteria mentioned above. The results were checked by all authors and approved. Articles were rejected if they were clearly ineligible. The full texts of the potentially eligible articles were reviewed as above to confirm or exclude their eligibility for inclusion. The results were presented for approval to all authors. Disagreements were resolved in a consensus meeting.

Data extraction

For each included study, one author (BM) manually extracted data relevant to the review aims using a customised form. Information regarding basic study data (authors, year of publication, country of origin, type of study), patient characteristics (number of patients, mean age, gender), methods (12-lead ECG, paper “strip” [1- or 2-lead ECG] or monitor) and outcomes (resolution of ECG changes after resolution/drainage of the pneumothorax) were retrieved. One author (BM) independently checked all extracted data.

Outcome measures

The primary outcome was the collection of all described ECG changes in patients with pneumothorax. The secondary outcome was the prevalence of each ECG sign considering all studies and differences on side and size of pneumothorax.

Quality assessment

All studies (including case reports) were critically appraised based on the presence of a 12-lead ECG associated with pneumothorax and after resolution of the pneumothorax (or before "therapeutic" pneumothorax). In studies without a follow-up ECG the control group was analysed.

Results

Literature search

The literature search from PubMed/MEDLINE, EMBASE and Cochrane CENTRAL databases yielded a total of 358 records. After reviewing language, titles and abstracts, 69 were selected as potentially eligible articles. The full text was retrieved for all. Following eligibility assessment, 3 articles with exclusion criteria were excluded from the systematic review. Finally, 66 studies [2–67] including 464 patients were identified as potentially relevant and were selected for the systematic review. These studies covered the period from inception to 18 September 2019. Search results and articles selections are displayed in a PRISMA flow chart (fig. A1).

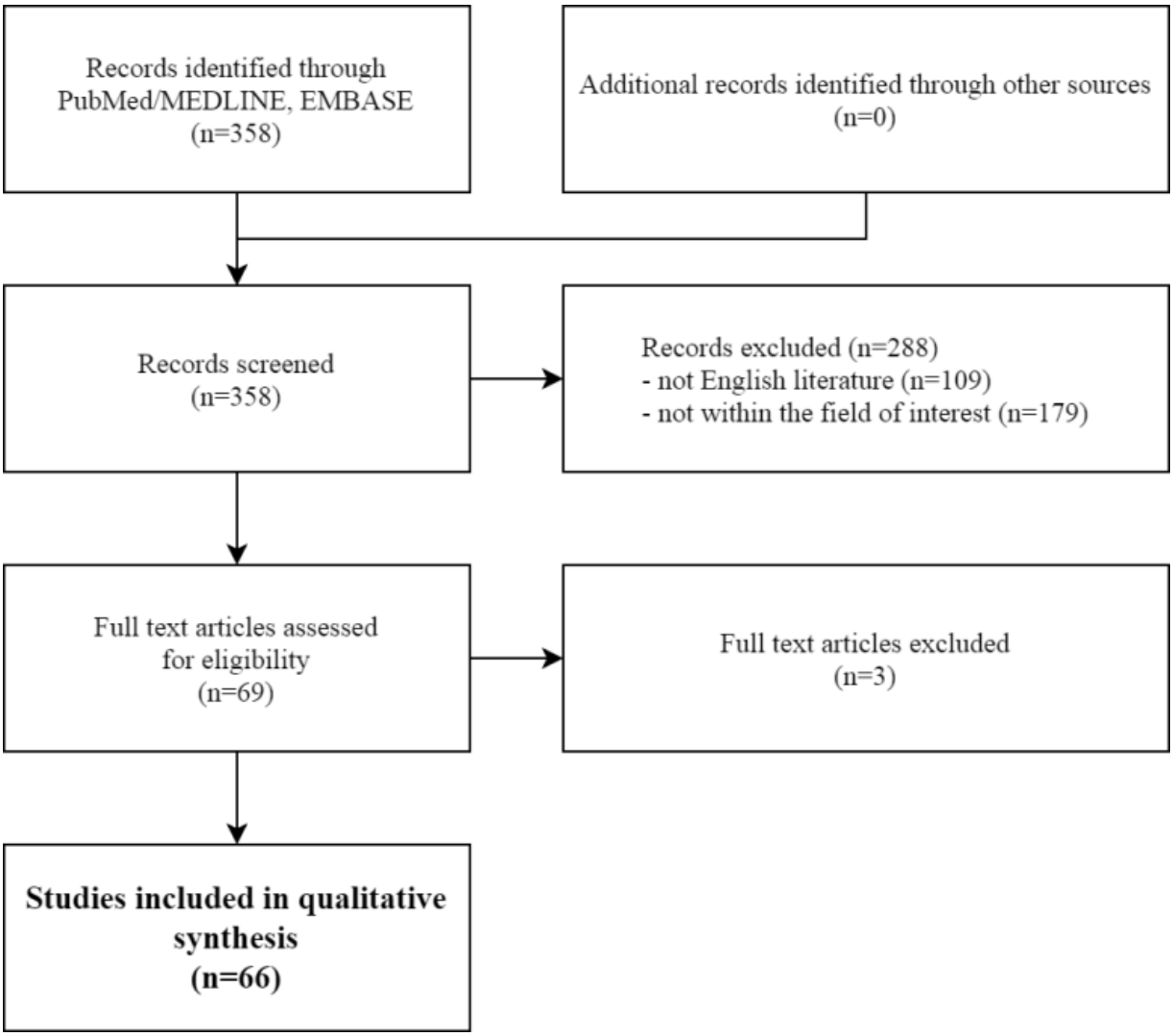

Figure A1. PRISMA flow chart of results and article selection.

Selected studies

The characteristics of selected studies are reported in table A1.

Table A1: Characteristics of selected studies.

Authors	Year	Country	Study design	No. of patients	% Male	Mean age ±SD (years)	% Left PT
Schmidt DC et al. [67]	2018	Denmark	Case Report	1	0	34	100
Ruhela M et al. [66]	2018	India	Case Report	1	100	37	100
Barcos JC et al. [65]	2017	Argentina	Case Report	1	0	68	100
Liu PY et al. [64]	2017	Taiwan	Retrospective, observational	60	100	22.5 ± 1.1	100
Lee W et al. [63]	2017	South Korea	Retrospective, observational	45	84.4	36.4 ± 23	55.5
Yeom SR et al. [62]	2017	South Korea	Case Report	1	100	35	100
Abdelghany M et al. [61]	2017	USA	Case Report	1	100	18	0
Robert J et al. [60]	2017	France	Case Report	1	100	84	0
Chada J et al. [59]	2017	USA	Case Report	1	100	64	0
Alzghoul B et al. [58]	2017	USA	Case Report	1	100	63	0
Kenzaka T et al. [57]	2016	USA	Case Report	1	100	43	100
Fei J et al. [56]	2015	Japan	Case Report	1	0	53	100
Tomcsányi J et al. [55]	2015	Hungary	Case Report	1	0	83	0
Tomiyama Y et al. [54]	2014	Japan	Case Report	1	0	0.6	0
Littmann L et al. [53]	2014	USA	Case Report	1	100	73	0
Huang SC et al. [52]	2014	Taiwan	Retrospective, observational	63	100	22.8 ± 9	100
Wieters JS et al. [51]	2014	USA	Case Report	1	100	68	0
Johnson P et al. [50]	2014	USA	Case Report	1	100	62	0
MacMahon M et al. [49]	2013	UK	Case Report	1	NR	1	100
Ali RZ et al. [48]	2013	USA	Case Report	1	100	56	0
Patané S et al. [47]	2013	Italy	Case Report	1	0	22	100
Anderson R et al. [46]	2013	Australia	Case Report	1	0	56	0
Senthilkumaran S et al. [45]	2011	India	Retrospective, observational	66	60*	38.7 ± 5.3*	58
Punn R et al. [44]	2011	USA	Case Report	1	0	14	100
Praserthdam W et al. [43]	2010	USA	Case Report	1	0	56	0
Saks MA et al. [42]	2010	USA	Case Report	1	100	46	0
Soltani P et al. [41]	2009	USA	Case Report	1	100	64	100
Mitsuma W et al. [40]	2009	Japan	Case Report	1	100	30	100
Oghlakian G et al. [39]	2009	USA	Case Report	1	100	54	0
Chan WH et al. [38]	2009	Taiwan	Case Report	1	10	86	100
Krenke R et al. [37]	2008	Poland	Prospective, observational	40	77.5	43.7 ± 19.1	55
Kurisu S et al. [36]	2008	Japan	Retrospective, observational	10	90	50 ± 26	100
Kurisu S et al. [35]	2008	Japan	Case Report	1	100	78	100
Bansal S et al. [34]	2007	UK	Case Report	1	100	22	100
Price JW et al. [33]	2006	Canada	Case Report	1	0	56	100
Ortega-Carnicer J et al. [32]	2002	Spain	Case Report	1	100	70	100
Lin JS et al. [31]	2001	Taiwan	Case Report	1	100	21	0
Strizik B et al. [30]	1999	USA	Case Report	1	0	82	100
Ti LK et al. [29]	1998	Singapore	Case Series	2	100	58 ± 3	100
Alikhan M et al. [28]	1998	USA	Case Series	5	0	54.6 ± 18.8	0
Goddard R et al. [27]	1997	USA	Case Report	1	100	18	0

Kozelj M et al. [26]	1997	Slovenia	Case Report	1	100	43	0
Paige GB et al. [25]	1996	USA	Case Report	1	100	6	0
Raev D et al. [24]	1996	Bulgaria	Case Report	1	100	66	100
Botz G et al. [23]	1992	USA	Case Report	1	100	55	100
Ruo W et al. [22]	1992	USA	Case Report	1	100	88	100
Keller N et al. [21]	1987	Denmark	Case Report	1	100	25	100
Werne CS et al. [20]	1985	USA	Case Report	1	100	50	100
Feldmann T et al. [19]	1984	USA	Case Report	1	100	53	0
Diamond JR et al. [18]	1982	USA	Case Report	1	0	36	100
Habibzadeh MA et al. [17]	1980	USA	Case Report	1	100	57	100
Hallengren B et al. [16]	1979	Sweden	Case Report	1	100	37	100
Slay RD et al. [15]	1979	USA	Case Report	1	100	52	0
Athanasopoulos C et al. [14]	1979	Greece	Case Series	11	63.6	28.5	54.5
Kuritzky P et al. [13]	1976	USA	Case Report	1	0	26	100
Kleine LW et al. [12]	1976	Belgium	Case Report	1	100	78	100
Walston A et al. [11]	1974	USA	Case Series	7	100	55.7 ± 13.3	100
Summers RS et al. [10]	1973	USA	Case Series	2	100	52 ± 15	50
Copeland RB et al. [9]	1970	USA	Case Series	2	100	52 ± 32	100
Fox JR et al. [8]	1956	USA	Case Report	1	100	21	100
Sreenivasan BR et al. [7]	1956	Singapore	Prospective, observational	20	NR	NR	50
Silverberg C et al. [6]	1950	USA	Prospective, observational	6	83.3	33.5 ± 98.8	100
Armen RN et al. [5]	1949	USA	Prospective, observational	45	100	28.5	55.5
Feldman D et al. [4]	1948	USA	Prospective, observational	23	39.1	30.1 ± 12.1	56.5
Littmann D et al. [3]	1946	USA	Case Series	2	NR	NR	100
Master A et al. [2]	1928	USA	Case Series	7	100†	24 ± 5†	28.6

ECG = electrocardiogram; PT = Pneumothorax; SD = standard deviation; USA = United States of America; UK = United Kingdom; NR = not reported.
Notes: *Data of the only 15 patients with ECG changes, † Data of only 2 patients.

The studies were conducted all over the world. The sample size of the included trials ranged from 1 to 66 patients. There was only one “modern” (2008) prospective observational trial [37]. The other four prospective observational studies were very old (1948–1956) [4–7] and the patients had pulmonary tuberculosis and were undergoing artificial pneumothorax. Thirteen studies [2, 3, 9–11, 14, 28, 29, 36, 45, 52, 63, 64] included collections of a few case reports or retrospective observational studies of patients presenting to the emergency department. Most of the studies were case reports (48). The mean age was 46 ± 21.7 years with 74.6% males and 62.7% patients with a left-sided pneumothorax.

Quality assessment

A 12-lead ECG was recorded in all prospective studies and all case series, as well as in 40 case reports. ECG changes in 8 case reports were seen on a monitor during resuscitation or anaesthesia. Each study included patients with known pneumothorax (or before execution of it), basically a case-control design. No study analysed patients with dyspnoea or chest pain. In the retrospective studies without follow-up ECG, a control group of age-matched disease-free subjects showed only amoderate association between observed changes and presence of pneumothorax.

ECG signs to be analysed

Table A2. Definition and inclusion or exclusion of the different ECG signs in the retrospective cohort.

ECG sign	Definition of the sign	Included
Phasic QRS voltage	Variation of the QRS amplitude of at least 0.3 mV (see fig. 2)	Yes
Right axis deviation	QRS axis in the frontal plane +90° to +180°	Yes
Left axis deviation	QRS axis in the frontal plane −30° to −90°	Yes
Incomplete RBBB	QRS duration of 110–120 ms with QRS morphology criteria for RBBB	Yes
RBBB	QRS duration greater than or equal to 120 ms AND rsr', rsR', or rSR' configruration in leads V1 or V2 AND S wave of greater duration than R wave or greater than 40 ms in leads 1 and V6 AND normal R peak time >50 ms in lead V1. Brugada pattern will be screened and signalised	Yes
P pulmonale	P wave amplitude >0.25 mV in the inferior leads (II, III and aVF) or P wave amplitude >0.15 mV in V1 and V2 (see fig. 2)	Yes
P wave inversion in lead I	Negative or isoeletric P wave in lead I (see fig. 2)	Yes
Baseline shift with P pulmonale	P pulmonale with upward shift of the baseline before the onset of the P wave (see fig. 2)	Yes
T wave inversion	T wave inversion in leads other than V1 and aVR	Yes
Low QRS voltage	QRS voltage <0.5 mV in the peripheral leads or <1 mV in the chest leads	Yes
QRS voltage ratio aVF/I >2	Ratio of R wave amplitude in lead aVF and R wave amplitude in lead I >2	Yes
ST-segment elevation	1 mm elevation of ST segment in at least one lead of 12	Yes
ST-segment depression	1 mm depression of ST-segment in V2, V3	Yes
V2S <12 mm	S wave in V2 less than 1.2 mV	Yes
V3S <9 mm	S wave in V3 less than 0.9 mV	Yes
Prolonged QTc	QTc (Bazett) >440 ms in men and >460 ms in women	Yes
Baseline shift with ST elevation ("spiked helmet sign")	ST segment elevation with upward shift of the baseline before the onset of the QRS complex	Yes
Reduced R wave amplitude	Increase in the R spike after resolution of PT	Excluded because requiring a follow-up ECG in the same subject. We did the analysis for V5 and V6 in the subgroup with a follow-up ECG
Increase R Wave amplitude V5–V6	Decrease in the R spike after resolution of PT in V5–V6	Excluded because requiring a follow-up ECG in the same subject. We did the analysis for V5 and V6 in the subgroup with a follow-up ECG.
Hyperacute T waves	Enlarged T waves	Excluded because no clear definition in the literature
LBBB	Supraventricular rhythm, QRS duration greater than 120 ms, lead V1 should have either a QS or a small R wave with a large S wave, lead V6 should have a notched R wave and no Q wave.	Excluded in the first analysis because analysis of the QRS complex and T waves would not have been possible as for the other signs
S1Q3T3 pattern	S wave in Lead I, Q wave and negative T wave in lead III	Excluded because no unambiguous definition in the literature
T wave in lead III >T wave in lead I	T wave in lead III (mV) >T wave in lead I (mV)	Excluded in the first analysis prior to the systematic review
AV block Mobitz 1	P waves with a constant rate (<100 bpm) with a periodic single nonconducted P wave associated with P waves before and after the nonconducted P wave with inconstant PR intervals	Excluded in the first analysis prior to the systematic review

ECG = electrocardiogram; PT = pneumothorax; RBBB = right bundle branch block; LBBB = left bundle branch block; AV = atrioventricular

ECG signs observed in patients with pneumothorax

Most frequently observed ECG signs in pneumothorax patients were phasic QRS voltage, low QRS voltage and reduced R wave amplitude, right-axis deviation, T wave inversion, ST-segment elevation and prolonged QTc. All 25 observed ECG alterations associated with pneumothorax are reported in table A3.

Table A3: ECG signs reported for patients with pneumothorax.

ECG sign	Reported in study	No. of patients with ECG sign
Phasic QRS voltage	[13], [16], [34–36], [56], [52], [60], [67]	23
Right axis deviation	[2], [4, 5], [8], [10, 11], [17], [22], [24], [28, 29], [31], [37], [41], [47], [51]	48
Left axis deviation	[29], [37]	3
Incomplete RBBB	[10], [37], [47]	5
RBBB	[59], [66]	2
LBBB	[50]	1
P pulmonale	[9, 10], [17], [31], [44]	6
P wave inversion in lead I	[51]	1
Baseline shift with P pulmonale	[30], [44]	2
T wave inversion	[4–6], [8], [11], [20], [28], [31], [33], [35], [37], [43], [50]	51
Low QRS voltage	[2], [8], [10], [12], [20, 21], [24], [28, 29], [32], [38], [54], [57]	20
QRS voltage ratio aVF/I >2	[35]	10
ST-segment elevation	[2], [15], [33], [43], [45, 46], [48], [58, 59], [61–63]	35
ST-segment depression	[2], [24], [45], [58], [63]	11
V2S <12 mm	[52]	27
V3S <9 mm	[52]	29
Prolonged QTc	[14], [42], [63]	20
Baseline shift with ST elevation (“spiked helmet sign”)	[53], [55]	2
S1Q3T3 pattern	[27], [31]	2
T wave in lead III >T wave in lead I	[4]	13
Reduced R wave amplitude	[3–7], [9–11], [17–19], [22, 23], [28, 29], [35–38], [40]	91
Increase R wave amplitude	[7], [49]	11
Hyperacute T waves	[42]	1
Brugada pattern	[65]	1
AV block Mobitz 1	[39]	1

ECG = electrocardiogram; PT = pneumothorax; RBBB = right bundle branch block; LBBB = left bundle branch block; AV = atrioventricular

Prevalence of ECG signs in patients with pneumothorax

We found overall 416 of the ECG signs described above in the 464 analysed patients (table A3). In the retrospective and prospective observational studies we found prevalences of these signs from 1.5% (T wave inversion by Krenke et al. 2018 [37]) to 100% (QRS voltage ratio aVF/I >2 by Kurisu et al. 2008 [36]). Pooled prevalence from more studies was only possible for right axis deviation [5, 36, 37, 63] (9.3% [13/140]), ST elevation [45, 63] (20.7% [23/111]), ST depression [45, 63] (5.4% [6/111]), phasic QRS voltage [36, 52] (27% [17/73]) and T wave inversion [4–6, 36, 37] (25.8% [32/124]).

ECG signs in relation to pneumothorax side and size

Analysis of side-related ECG findings associated with pneumothorax was possible for following signs, considering prospective and retrospective studies.

ST elevation and depression: Senthilkumaran et al. [45] analysed ST elevation and depression in 66 patients with pneumothorax. 58% were located on the left. There was no difference in prevalence between right- or left-sided pneumothorax.

Reduced/increased precordial QRS complex: Krenke et al. [37] described QRS complex reduction in left-sided pneumothorax and increase of R wave in V5-6 in right-sided pneumothorax. the same results were described by Sreenivasan et al. [7].

QTc: in the series of Athanasopoulos et al. [14] QTc prolongation was not related to the pneumothorax side.

Right axis deviation: seen with both sides, but mostly right-sided pneumothorax [2, 4, 5].

P-pulmonale, P wave inversion in lead I: seen with both sides, but mostly right-sided pneumothorax [4, 5, 10].

T wave inversions: seen with both sides, but mostly left-sided pneumothorax [4, 5].

An analysis of signs related to the size of pneumothorax was not possible given the existing data, but Huang et al. [52] found significance for up to 20% pneumothorax. Interestingly in some series [45, 63], the changes seen most were not related to the biggest pneumothorax, rather those between 30 and 60%.

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