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The purpose of electrocardiography in acute and chronic diseases with right ventricular involvement

Zastosowanie elektrokardiografii w ostrych i przewlekłych chorobach z zajęciem prawej komory serca

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Abstract

Electrocardiography is a basic diagnostic tool and one that plays a significant role in differentiating many diseases, including those involving the right heart. The diagnosis of right ventricular (RV) pathology is difficult due to the complex structure and the wide spectrum of haemodynamic disorders resulting from its dysfunction. The use of a standard electrocardiogram (ECG) with right-sided leads can be a valuable complement of imaging tests, especially when these are not quickly available. Numerous electrocardiographic abnormalities are observed in the course of acute pulmonary embolism, chronic pulmonary hypertension, right ventricular myocardial infarction, or arrhythmogenic right ventricular cardiomyopathy, and some of these also have prognostic significance. Unfortunately, despite its simplicity and utility, ECG is insufficiently sensitive and specific to be the single tool in the recognition of RV pathologies. ECG is a common, inexpensive, non-invasive and easily accomplished complementary test, which can be useful in diagnostic algorithm of right heart diseases.

Key words: electrocardiography, pulmonary embolism, pulmonary hypertension, right ventricle infarction, arrhythmogenic right ventricular cardiomyopathy

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Introduction

Electrocardiography (ECG) has been an irreplaceable diagnostic tool for over 100 years in patients reporting chest pain, shortness of breath, syncope or palpitations. However, when analysing an ECG, do you also remember the possibilities of assessing the right ventricle? How often do you analyse ride-sided electrocardiogram? Since right ventricular (RV) diseases are not infrequent, better knowledge of specific electrocardiographic abnormalities in the course of these conditions could help the physician to avoid confusion and improve a patient's prognosis. It should be emphasised that an ECG examination is characterised by insufficient sensitivity and specificity to be used as the single diagnostic tool in cases of suspected right heart diseases. We discuss below the most critical conditions involving RV, and the most common electrocardiographic abnormalities that accompany them.

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Table 1. Common electrocardiographic abnormalities in course of acute pulmonary embolism (based on [2])

Standard ECG	Tachycardia > 100/min
	Atrial arrhythmias
	Right axis deviation (dextrogram)
	Features of right atrial overload/P *pulmonale*
	Incomplete or complete right bundle branch block (RBBB)
	S1Q3T3 pattern (McGinn-White sign) — S wave in lead I, Q wave in lead III with amplitude > 0.15 mV, negative T wave in lead III
	S1S2S3 pattern — S wave with amplitude > 0.15 mV in leads I–III
	QR sign in lead V1 — the Kucher sign
	Notched S wave in leads V1–V2
	Negative T wave in leads V1–V4 (less often V1–V6)
	ST segment depression in leads V4–V6 (less often V1–V6), less often in leads II, III, aVF
	ST segment elevation in leads III, aVR, V1
	Right axis deviation (dextrogyria)
	Low voltage in limb leads
Right ventricular leads V3R–V6R	Negative T wave in leads V3R–V6R

RBBB — right bundle branch block

Acute pulmonary embolism

The pathophysiology of ECG changes in the course of acute pulmonary embolism (APE) is complex. In addition to excessive pressure overload, damage and ischaemia of the myocardium, other more complex mechanisms are also suspected, including excessive local adrenergic stimulation [1]. So far, numerous ECG abnormalities caused by APE have been described, and studies are being continued in various centres around the world, including Poland. One of the first researchers to describe the S1Q3T3 pattern, typical of acute pulmonary heart in APE, was McGinn and White. Since then, electrocardiographic parameters of pulmonary embolism (PE) are better recognised/understood. The most common abnormalities in the course of APE include sinus tachycardia, the presence of negative T waves in leads V1–V4, depression or elevation of ST-T segment in certain lead groups, S1Q3T3 pattern, S1S2S3 pattern, right bundle branch block (RBBB), and other symptoms as set out in Table 1 (an example of an ECG of a patient with APE is shown in Figure 1). It is important to emphasise that electrocardiographic findings do not prejudge the diagnosis of the disease, and can in fact be completely correct even in the course of radiologically significant PE. ECG abnormalities are found in 80–90% of patients in intermediate and high-risk groups [2]. Interestingly, in the course of APE, the longest-lasting changes are negative T waves, while other symptoms usually disappear within two weeks of initiating proper treatment [3].

Significance in evaluation clinical course

Numerous studies have been conducted on the electrocardiographic diagnosis of PE. One of them is the study of Kukla et al. [4], who observed that ST segment depression in leads V4–V6 ($p = 0.02$), ST segment elevation in aVR lead ($p = 0.0007$), and ST segment elevation in lead V1 ($p = 0.0002$) occur significantly more often in high-risk APE than in low-risk APE [4]. Furthermore, researchers have shown that the presence of ST elevation in aVR lead significantly increases the number of complications during hospitalisation (38.3% vs. 12.5%; $p < 0.001$), and overall mortality, compared to people in whom this abnormality was not observed (16.5% vs. 6.9%; $p = 0.009$) [5]. Another study by the same group of researchers showed that the number of leads with negative T waves (odds ratio [OR] 1.46; $p = 0.001$), the presence of RBBB (OR 2.87; $p = 0.02$), and ST segment elevation in V1 (OR 3.99; $p = 0.00017$), and aVR leads (OR 2.49; $p = 0.011$) are an independent prognosis of cardiovascular complications during hospitalisation. This study also proved that the sum of the negative T-wave amplitudes, the number of leads with negative T waves, and the elevation of ST in lead V1, are independent predictors of in-hospital mortality [6]. On the other hand, the prognosis is the worst in patients with more than five leads with negative T waves [7].

The relationship between specific electrocardiographic features and the adverse clinical course of APE defined as progression to shock, the need for resuscitation, intubation, use of pressure amines, thrombolysis or thrombectomy, has also been shown in the recently published

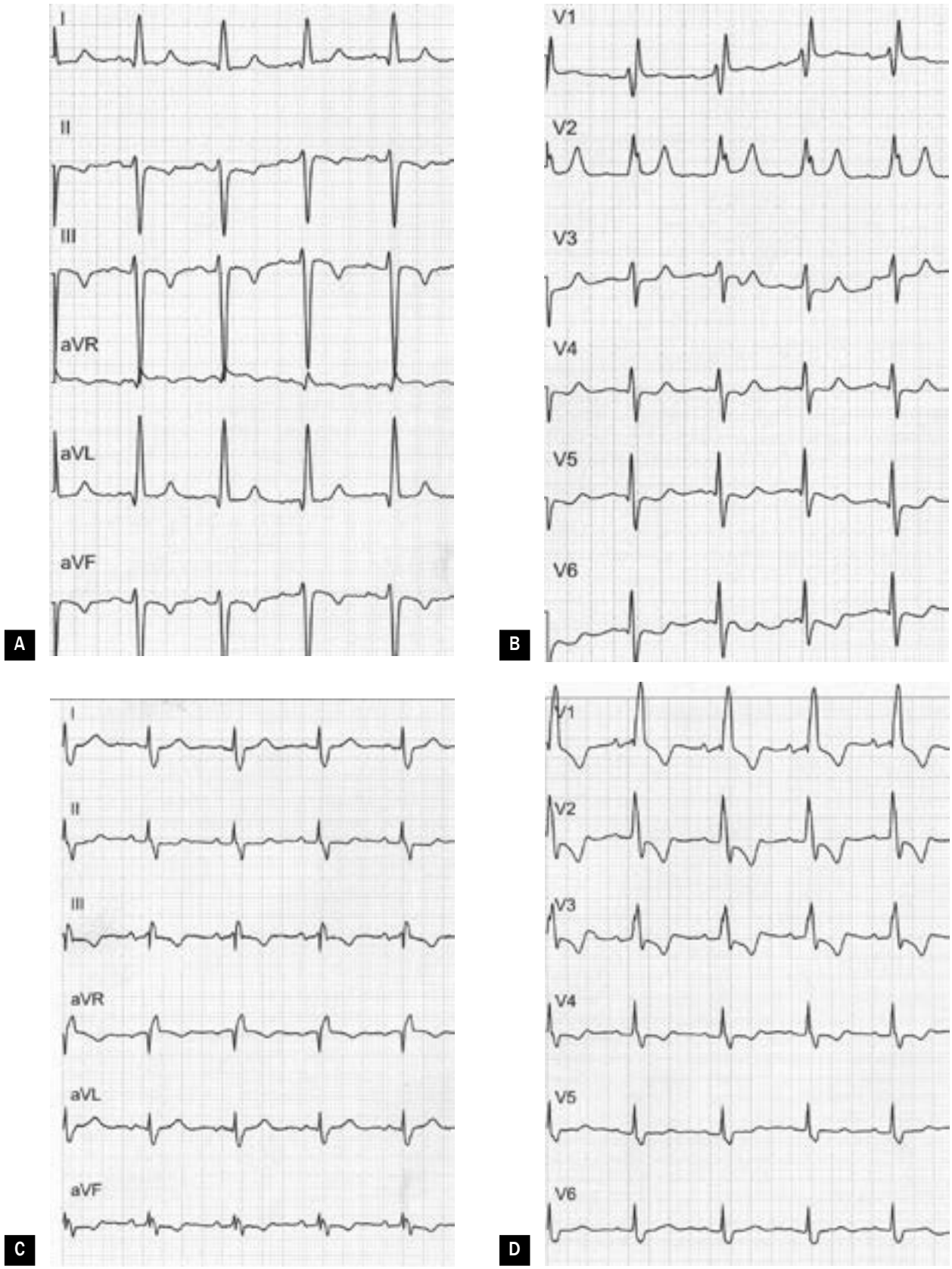

Figure 1A–D. Electrocardiographic examples of patient with acute pulmonary embolism (**A**, **B**) and chronic thromboembolic pulmonary hypertension with right ventricular hypertrophy (**C**, **D**); records of Piotr Bienias MD, PhD (25 mm /s, 10 mm/mV)

meta-analysis of Qaddour et al. [8] covering 39 studies. The total number of evaluated patients with APE was 9,198, and an independent relationship between the occurrence of an adverse clinical course was proven with regard to the following changes: tachycardia (OR 4.61; p < 0.001), atrial fibrillation on admission (OR 1.78; p < 0.001), complete RBBB (OR 2.47; p < 0.001), S1Q3T3 pattern (OR 3.89; p < 0.001), ST segment depression in V4–V6 leads (OR 2.71; p < 0.001), ST segment elevation in lead III (OR 3.06; p < 0.001), ST segment elevation in aVR lead (OR 3.29; p < 0.001), ST segment elevation in lead V1 (OR 5.14; p < 0.001), presence of negative T waves in leads V1–V4 and II, III, aVF (OR 2.45; p < 0.001), and qR sign in lead V1 (OR 4.65; p < 0.001). This meta-analysis also showed that most of the characteristics mentioned above are significantly associated with increased in-hospital mortality in the course of APE [8].

The importance of ST segment elevation in aVR in patients with APE must be underlined. Dynamic ST changes in this lead were considered nonspecific and without significant clinical value for many years, until it was demonstrated that they could be associated with stenosis of the left main coronary artery and/or multivessel disease (in the case of the co-existence of significant ST segment depression in ≥ other eight leads). In patients with APE, ST segment elevation in aVR indicates an increased risk of death and/or deterioration of the general condition with the development of shock, as demonstrated in 2013 by Zhong-Qun et al. [9] (in those patients ECG — ST segment depression in leads I, V4–V6 was registered).

When evaluating ECG in patients with APE, an accurate analysis of the morphology of the QRS complex is as important as analysis of repolarisation abnormalities. Already in the 1960s, Weber and Philips observed the presence of a Q wave in lead V1 in patients with APE and its relation with an adverse clinical course. In 2003, Kucher et al. again showed a relation between the occurrence of the qR/QR sign in lead V1 and an poor prognosis in the course of APE (since then this has been called the Kucher sign), and Casazza et al. [10] confirmed that the presence of the qR/QR syndrome in lead V1 is associated with in-hospital mortality in the group of patients with high risk of death (but the symptom was only observed in 15.9% of the patients).

Evaluating the probability of pulmonary embolism and right heart strain

Almost two decades ago, Daniel et al. [11] proposed a 21-point ECG scale, which could determine the probability of APE and the degree of pulmonary hypertension (PH) in its course. The Daniel ECG Scale includes sinus tachycardia, RBBB, incomplete RBBB, negative T waves in leads V1–V4, S waves in lead I, Q waves in lead III, negative T waves in lead III, and S1Q3T3 pattern (McGinn-White sign). The maximum number of points according to the Daniel scale is 21; the presence of at least 10 of these suggests PH over 50 mm Hg in an angiographic examination with a sensitivity of 23.5% and a specificity of 97.7% [11]. In a later analysis, Toosi et al. [12] showed that a score of 3 or more points indicates RV dysfunction (sensitivity 76%, specificity 82%), the risk of complications during hospitalisation (sensitivity 58%, specificity 60%), and an increased risk of death, while a score of below 3 points is associated with good short-term prognosis. The high negative predictive value of a result lower than 3 points, which allows to predict a positive clinical course with over 90% probability, was also confirmed by Kostrubiec et al. [13]. Despite the numerous studies conducted, the role of the Daniel ECG Scale has still not been clearly determined, and it is not included in routine clinical practice.

Another new electrocardiographic model enabling the prediction of the presence of RV overload in the course of APE was proposed in 2015 by Hariharan et al. [14]. Compared to the Daniel ECG Scale, the researchers presented a simplified 10-point TwiST index taking into account the occurrence of negative T waves in leads V1–V3 (5 points), S waves in lead I (2 points), and tachycardia (3 points). A TwiST score of 2 points or below excludes RV overload with 84% sensitivity [95% confidence interval (CI): 77–90%], while a TwiST score greater than or equal to 5 indicates RV overload with 93% sensitivity (95% CI: 88–97%) [14].

Fragmented QRS complex

Another QRS abnormality observed in the course of APE is QRS fragmentation (fQRS, fragmented QRS). It is suspected that fQRS is caused by ischaemia or the presence of a previous scar, which results in abnormal ventricular depolarisation. Fragmented QRS was first defined by Das et al. [15] in patients with coronary artery disease as the presence of at least one R' wave or notching of the S wave or R wave within narrow QRS complex (< 120 ms) of different morphologies not meeting the criteria for bundle branch block [15]. These criteria are used in ECG analysis of patients with APE, but they are not the only one. In a meta-analysis evaluating fQRS's prognostic value and including about 1,200 APE patients, an international team of researchers has demonstrated a clear relation between the presence of fQRS and the occurrence of cardiogenic shock (OR 4.71; p = 0.005), in-hospital death (OR 2.92; p < 0.001), as well as death during a two-year follow-up period (OR 4.42; p < 0.001) [16]. Therefore, fQRS seems to be a potentially useful prognostic marker for APE. Unfortunately, due to heterogeneous criteria of fQRS in the literature, and significant variability in the assessment of the presence of this parameter in ECG among expert centres, further analyses are still needed.

QT interval

Recently, exciting results of corrected QT interval in the course of APE have also been presented. In comparison to a healthy control group, it has been shown that in patients with APE, the average duration of the QTc interval in lead V1 was significantly longer (454.6 ± 44.3 *vs.* 417.5 ± 31.3 ms; $p < 0.001$), and the difference between QTc duration in lead V1 and lead V6 — greater (34.8 ± 30.5 *vs.* –12.5 ± 16.6 ms; $p < 0.001$). This Korean study also showed that QTc difference (V1–V6) greater than or equal to 20 ms makes it possible to identify individuals with APE with 82.2% sensitivity and 100% specificity [17]. Although the authors considered the parameter they identified to be an excellent diagnostic tool, it seems that before it is widely used, it must be validated by other centres and in different racial groups.

Differentiation from acute coronary syndrome

Abnormalities presented in ECG in the course of APE may raise diagnostic doubts and often require differentiation from other emergency conditions. The most important practical implication is that APE can clinically and electrocardiographically imitate myocardial infarction, both with and without ST segment elevation. Depending on the study, abnormal changes in the ST-T segment in APE patients, requiring differentiation from acute coronary syndrome (ACS), are reported in 40–70% of cases [3, 4, 18]. ST segment depression or the occurrence of negative T waves in the course of APE seems easy to explain. The presence of the ST segment elevations in leads V1–V3/V4 is probably caused by RV ischaemia, or may be the result of paradoxical embolism to the coronary arteries through a patent foramen ovale or atrial septal defect [19]. The aetiology of electrocardiographic changes imitating ACS with ST segment elevation in the inferior leads remains unclear [20]. Electrocardiographic differentiation of APE from ACS is still an important research goal. Among others, Kosuge et al. [20] noted that the coexistence of inverted T waves in leads V1 and III occurs in only 1% of patients with ACS, while in the course of APE it is found in 88% of cases ($p = 0.001$) [20].

Right-sided leads

Beyond standard ECG in patients suspected of APE, you can also use right-sided V3R–V6R leads in typical locations. Unfortunately, due to very limited test results, the final diagnostic value of right-sided ECG in patients with APE has not been fully established. However, the usefulness of right-sided ECG and its advantage over a standard ECG exam has been demonstrated in the initial differentiation of patients suspected of APE, which is confirmed by the results obtained by Turkish researchers. The most frequently reported abnormalities in V3R–V6R leads in patients with APE are negative T waves (64% sensitivity) and ST segment elevation (29% sensitivity) [21]. It should be emphasised that the final practical value of this registration requires verification in the course of further studies involving large groups of patients. It is also worth noting that only repolarisation disorders are subject to the analysis of V3R–V6R leads, because the voltages, and thus the potential deviations within the QRS complex, are too small.

The role of ECG in standards

Despite the undoubted utility of ECG in the diagnostic process of patients with APE, the current standards of the European Society of Cardiology (ESC) do not include standard ECG in the routine diagnostic scheme. Similarly, these standards do not indicate the role of ECG in short- and long-term risk stratification in patients with confirmed PE.

Pulmonary hypertension

The diagnostic value of ECG in the diagnosis of RV and PH hypertrophy is not significant, and it is widely known that it must be supplemented by echocardiography and, in certain situations, also by invasive measurement during right heart catheterisation. The commonly recognised features of RV hypertrophy in ECG examinations, presented in Table 2 [22], must lead to diagnostics in the direction of all forms of PH (an example of an ECG of a patient with APE is presented in Figure 1).

It seems that the size of RV hypertrophy is most closely correlated with the amplitude of R wave in lead V1; thus the higher the amplitude of R wave in lead V1, the higher the probability of RV and PH [23]. In one of the latest studies, Miura et al. [24] has proposed quick and simplified electrocardiographic screening for PH consisting of an accurate analysis of the S wave in lead V5 and the occurrence of negative T waves in the precordial leads. It has been proved that the depth of the S wave in V5 lead above 0.42 mV ($p < 0.001$), and the depth of the negative T wave in lead V4 above 0.28 mV ($p = 0.018$), are independent prognostic indicators of increased pulmonary arterial pressure by at least 25 mm Hg [24].

Importance in evaluating the clinical course

Multi-path analysis conducted by various teams has shown that ECG also has potential prognostic value in this group of patients. An example of such studies is the analysis of Sun et al. [25] who documented that widening the QRS complex of at least 120 ms in patients with idiopathic PH increases the risk of death 2.5-fold ($p < 0.024$). Similar results were obtained by Rich et al. [26], who also proved that in this group of patients QTc prolongation longer than or equal to 480 ms is associated with increased mortality

Table 2. Criteria of right ventricular hypertrophy in electrocardiogram (based on [22])

Diagnostic criteria
R wave in lead aVR ≥ 5 mm
R wave in lead V1 ≥ 7 mm
RSR wave in lead V1 — R' > 10 mm (QRS < 120 ms)
S wave in lead V5 > 10 mm
S wave in lead V6 > 3 mm
R wave in lead V1 + S wave in lead V5 or V6 > 10.5 mm
Other criteria
Dextrogram > + 110 degrees
R wave in lead V1 > S wave in lead V1
S wave in lead I and Q in lead III
Criteria of right atrial hypertrophy
Criterion for presence of right bundle branch block (RBBB)
R wave in lead V1 > 15 mm
Criterion for presence of left bundle branch block (LBBB)
No explicit criterion

[hazard ratio (HR) 3.09, 95% CI: 1, 14–8.38; p < 0.05]. Also, it was observed that end-diastolic volume (p <0.001) and RV mass (p < 0.05) both increase, and ejection fraction decreases (p < 0.05) along with the prolongation of the QTc interval [26]. Increased mortality in PH (especially arterial) has also been observed in people with V1 lead qR syndrome, which occurs in 18–43% of patients according to a specific study [27].

Thromboembolic pulmonary hypertension

It is estimated that 0.4–9.1% of people after an APE episode may develop chronic thromboembolic pulmonary hypertension (CTEPH) after several years [28]. This is the only type of PH that can potentially be completely cured, so it is vital to detect it early. Clinically, CTEPH is a diagnostic challenge, mainly due to the improvement after the APE episode, which may dull the patient's and the attending physician's vigilance (the so-called 'honeymoon period' usually lasting 6–24 months). The occurrence of right-sided heart failure symptoms or intensifying chest discomfort in patients with previous APE should be a spur to initiate diagnosis. Unfortunately, the symptoms mentioned above may occur when more than 40% of the arterial pulmonary placenta is obstructed. For this reason, research on the use of screening and non-invasive methods that would allow for early detection of this dangerous consequence of the EP is still ongoing. It is believed that the electrocardiographic features of right atrial and right ventricle overload may be present in almost 90% of patients with CTEPH [29]. In their pioneering work from 2004, Lewczuk et al. [30] described the most common abnormalities and their frequency in ECG registration in the CTEPH group, *i.e.* negative T waves in leads V1–V5 (in 43%), II, III and aVF (in 32%), P *pulmonale* (in 30%), and deviation of the electrical axis of the heart to the right (in 30% of patients). It should be stressed that the predictive value of the parameters enumerated above proved to be high in the case of CTEPH — both positive (> 80%) and negative (< 50%) [30].

In the above mentioned study by Miura et al. [24], of the role of ECG in predicting PH in a separate group of patients with CTEPH, the most important were the S-wave depth in lead V5 (p = 0.027) and the depth of the negative T-wave in lead V1 (p < 0.001) [24]. Recently, Klok et al. [31] attempted to develop a diagnostic model excluding CTEPH in patients after PE. After taking into account the following electrocardiographic parameters: the rSR'/RSr' syndrome, R/S wave ratio above 1 and R amplitude over 0.5 mV in lead V1 and electrical axis of the heart over 90°, they showed that the absence of the abnormalities mentioned above, combined with the correct concentration of N-terminal pro-B-type natriuretic peptide (NT-proBNP), allows to exclude the disease (> 90% predictive value). The presented model was also characterised by 94% sensitivity and 65% specificity in the diagnosis of CTEPH, and the presence of dextrogram plays a pivotal role in this algorithm [31].

Right ventricular infarction

Isolated right ventricular infarction (RVI) is rare. Most often, RVI accompanies an inferior wall (20–50%) or anterior wall of left ventricle infarction (10–15%) [32, 33]. A rarely-observed characteristic of RVI is a triad of symptoms, *i.e.* hypotension, jugular vein distention and clear lungs. Typically, an RVI diagnosis is confirmed by right-sided ECG V3R–V6R, and ST segment elevation at the J point greater than or equal to 0.5 mm (or in men < 30 years of age ≥ 1 mm). If RVI is suspected, only the ST segment elevation has diagnostic value, and its depression, or the occurrence of negative T waves, should be treated as an abnormal but nonspecific image. Thus, V3R–V6R lead assessment also does not allow to recognize RVI without ST segment elevation. It should be emphasised that the elevation of the ST segment by at least 1 mm in the V4R lead is the most sensitive (100%) and most specific (87%) electrocardiographic parameter in the diagnosis of RVI. Moreover, this is an independent predictor of complicated clinical course and in-hospital mortality [34].

RVI cases imitating anterior ischaemia of the left ventricle caused by occlusion of the right coronary artery in its proximal segment have also been described in the literature. In such cases, ST segment elevation is usually greater in lead V1 than in lead V2, and it decreases in subsequent leads (compared to myocardial infarction due to anterior descending branch occlusion). Concomitant diagnosis of

RVI in such cases is also indicated by the absence of pathological Q-wave in control ECG registration [35].

Right ventricular infarction and inferior wall infarction

In patients with RVI on a standard ECG, the most commonly observed abnormalities are ischaemic lesions in the lower wall area, which is caused by the high incidence of infarctions in these areas. The Polish guidelines on electrocardiographic diagnoses emphasise that in the case of typical features of the inferior myocardial infarction, a V3R––V6R lead reading should also be performed each time to exclude concomitant RVI or RV ischaemia. This implies specific pharmacological management (*i.e.* consideration of intravenous fluid therapy, avoidance of vasodilatation drugs) and also implies the need for prolonged monitoring, because patients with RVI display a significantly increased risk of developing various cardiac arrhythmias. Atrioventricular block, severe sinus bradycardia, and ventricular fibrillation are the most common arrhythmias in this patient group [36].

A large number of scientific studies on RVI or acute RV ischaemia come from the era before the age of the widespread use of cardiac troponins in diagnostics and before the era of invasive coronary revascularisation, which is an essential element of ACS therapy, and which results in better diagnosis of heart attacks and better clinical course. The consequences also include different dynamics and the evolution of myocardial infarction symptoms, including RVI in ECG registration, and another prognostic value of ECG changes that cannot be translated into current times.

Arrhytmogenic right ventricular cardiomyopathy

Arrhythmogenic right ventricular cardiomyopathy (ARVC) is a rare, genetically determined disease in which healthy myocardial tissue is replaced by fibrous and adipose tissue. The disease mainly affects RV, but it can also involve the left ventricle. Its incidence is 1:2,000–1:5,000 births and it is one of the most common causes of sudden cardiac death in young people and athletes. Diagnosing ARVC is not easy. Due to RV geometry, the assessment of its morphology and function in transthoracic echocardiography is limited. In turn, magnetic resonance imaging, which until recently was an indisputable diagnostic criterion, has a high risk of false-positive diagnoses. The currently used criteria for the diagnosis of ARVC developed by the appropriate working team take into account the co-occurrence of structural, functional, and histological changes in the heart, abnormalities in ECG recordings, and Holter monitoring, as well as family medical history [37]. Characteristic ECG changes play a key role in the diagnostic algorithm, although they are rare at the beginning of the disease — they occur only in about half of the patients. However, during more extended follow-up periods, various ECG abnormalities occur in virtually all patients.

Repolarisation abnormalities

The most commonly reported abnormalities are repolarisation disorders, while depolarisation abnormalities that are more specific to this disease are reported very rarely. Typical repolarisation disorders include negative T waves in leads V1–V3, and their presence in the absence of RBBB is considered a typical ARVC sign found in 59–83% of patients [38]. In addition, as shown in the study by Batchvarov et al. [39], special attention should be paid to the T wave in lead V1, which is more negative in patients with ARVC than in healthy people (0.21 ± 0.12 vs. 0.11 ± 0.06 mV; $p < 0.0001$). The researchers pointed out that so far, no detailed analysis of the T wave shape had been performed in the group of people suspected of having ARVC, and that, in their opinion, this assessment could play an important role in the initial diagnosis of cardiomyopathy [39].

Depolarisation abnormalities and epsilon waves

ECG depolarisation abnormalities found in ARVC include an image suggesting incomplete or complete R (not a real branch block, but an effect of intramural conduction disturbances), wide QRS complex in leads V1–V3 and III exceeding 110 ms, the widening of the S wave in leads V1––V3 greater than or equal to 55 ms, and an epsilon wave.

An epsilon wave is a low amplitude wave observed in right ventricular precordial leads, typically occurring between the end of the QRS complex and the beginning of the T wave. It was first described by Fontaine et al. in 1977 [40], and is considered as a characteristic symptom of ARVC, although it occurs in no more than 25% of cases. Noticing the epsilon wave in a standard ECG is not easy, because in the course of ARVC the QRS complex has various types of narrowing, especially in its end part and on the ascending arm of the S wave (so-called fragmentation of the QRS complex). It is said that the fragmentations occurring at the beginning, top, and end of the QRS complex should be considered as variants of the epsilon wave. It should be noted that sometimes even a few epsilon waves can be observed in severe cases of ARVC.

To increase the sensitivity of epsilon wave detection, it is recommended to perform ECG with a paper speed of 50 mm/s and double feature (*i.e.* 20 mm/mV). You can also use the so-called higher leads, *i.e.* the displacement of the V1/V2 electrodes to the III or II intercostal space. Also, by using the special lead modification proposed by Fontaine, the presence of the epsilon wave can be recorded in up to 75% of patients. The location of the electrodes according to Fontaine should be as follows: the electrode from the

left upper limb (yellow) should be placed at the height of the xiphoid process, the electrode from the right upper limb (red) at the level of the manubrium, and the electrode from the left lower limb (green) at the location of the precordial electrode V4 or V5.

The presence of the epsilon wave is not an independent risk factor for sudden death or arrhythmias, but it indicates a large extent of the disease process. It should also be noted that it can sometimes be observed in RVI, myocarditis, and sarcoidosis [41].

In recent years, ARVC researchers have also been interested in aVR lead. In the absence of typical [42] electrocardiographic features of ARVC as set out in Table 3, changes in this lead may facilitate a proper diagnosis. In the case of patients with cardiomyopathy in aVR lead, the following changes can often be observed: deep Q waves (≥ 0.3 mV) due to the presence of an electroanatomic scar, low R wave amplitude (≤ 0.2 mV) resulting from myocardial atrophy, and negative T waves (43). The above abnormalities are characterised by high sensitivity (97%), moderate specificity (81%), and, more importantly, high negative predictive value reaching 99%. The positive predictive value of the above parameters is low (30%). An epsilon wave is rarely observed in aVR lead (in approximately 5% of cases) [44]. It is often associated with heart failure (p = 0.04) and increased mortality (p = 0.03) [45].

Ventricular late potentials

It should be remembered that signal-averaged electrocardiography is still a high-value method in the diagnosis of ARVC — the presence of late potentials with specific characteristics in this group of patients has a high diagnostic value, but the availability of the examination is significantly limited [44].

Conclusion

A comprehensive assessment of the right ventricle of the heart is an exacting challenge which requires great meticulousness on the part of physicians. Electrocardiography remains a critical component of this diagnosis, in addition to in-depth medical history, physical examination, and imaging. An analysis of standard ECG along with right-sided ECG may be of crucial importance in making an accurate diagnosis as well as assessing prognosis.

We must say again that an ECG registration is characterised by insufficient sensitivity and specificity to be the one and only diagnostic tool in the case of diseases with a predominant RV involvement. Ongoing research, which seeks to broaden the knowledge of ECG changes in the course of diseases with RV involvement, gives hope for better use in future of this widely available diagnostic method.

Table 3. Typical electrocardiographic abnormalities in course of arrhythmogenic right ventricular cardiomyopathy (based on [42])

Repolarisation abnormalities
Inverted T wave in leads V1–V3 in patients > 14 years of age without RBBB
Inverted T wave in leads V1–V2 in patients > 14 years of age without RBBB or in leads V4–V6
Inverted T wave in leads V1–V4 in patients > 14 years of age in the presence of RBBB
Depolarisation abnormalities
Epsilon wave in leads V1–V3
Duration of QRS complexes in precordial leads V1–V3 and III (> 110 ms)
Widening of S wave in leads V1–V3 ≥ 55 ms in patients without RBBB (measurement taken from the nadir of S wave to the end of the QRS complex, including R waves in leads V1, V2 or V3)
Rhythm and conduction disorders
Complete or incomplete RBBB
Nonsustained or sustained ventricular tachycardia of left bundle-branch morphology with superior axis (negative or indeterminate QRS in leads II, III, and aVF and positive in lead aVL)
Nonsustained or sustained ventricular tachycardia with right ventricular outflow tract configuration or LBBB morphology with inferior axis (positive QRS in leads II, III, and aVF and negative in lead aVL)

RBBB — right bundle branch block; LBBB — left bundle branch block

Conflict of interest

The authors declare that there is no conflict of interest.

Streszczenie

Elektrokardiografia (EKG) odgrywa znaczącą rolę w różnicowaniu wielu chorób, w tym przebiegających z zajęciem prawego serca. Rozpoznanie patologii prawej komory (RV) bywa trudne ze względu na złożoną budowę oraz szerokie spektrum zaburzeń hemodynamicznych wynikających z jej dysfunkcji. Standardowy zapis EKG czynności serca uzupełniony o rejestrację odprowadzeń prawokomorowych może być w tym przypadku cennym uzupełnieniem badań obrazowych, zwłaszcza gdy te nie są szybko dostępne.

W przebiegu ostrej zatorowości płucnej, nadciśnienia płucnego, zawału prawej komory czy arytmogennej kardiomopatii prawokomorowej obserwuje się liczne nieprawidłowości elektrokardiograficzne pomocne w diagnostyce, a część z nich ma znaczenie prognostyczne. Niestety, mimo swojej prostoty i użyteczności EKG cechuje się niedostateczną czułością i swoistością, by mógł stanowić pojedyncze narzędzie diagnostyczne w wykrywaniu nieprawidłowości RV. Elektrokardiografia to powszechne, tanie, nieinwazyjne i łatwe do wykonania badanie uzupełniające, które może mieć istotne znaczenie w algorytmie diagnostycznym różnych chorób przebiegających z zajęciem RV.

Słowa kluczowe: elektrokardiografia, zatorowość płucna, nadciśnienie płucne, zawał prawej komory, arytmogenna kardiomiopatia prawej komory

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