

## SHORTENING OF THE PR INTERVAL IN PERICARDITIS AFTER CORONARY BYPASS SURGERY

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**Background:** Pericarditis, following pericardiotomy, is a well-known complication of cardiac surgery. The diagnosis of postpericardiotomy pericarditis (PP) is based on the electrocardiography (ECG) changes — the ST segment elevation in combination with a depression of the PR interval. However, in some cases, the ECG changes are difficult to distinguish from the changes associated with ST segment elevation acute coronary syndrome. In such cases, the diagnosis of pericarditis is made by excluding acute coronary syndrome, for which additional expensive diagnostic tests are performed. **Aims:** the purpose of the study is to identify a pattern in the change in the PR interval, which is detected in patients with acute pericarditis who underwent pericardiotomy during coronary bypass surgery. **Methods:** The observational study included 47 patients after coronary bypass surgery. We compared ECG of two groups of patients after coronary artery bypass grafting — 25 patients who demonstrated the ECG signs of acute pericarditis and 22 patients without those. **Results:** In most patients with PP after coronary bypass surgery, the characteristic ECG signs of acute pericarditis were accompanied by a transient shortening of the PR interval by 0.04 sec. **Conclusion:** The absence of such dynamics in patients after coronary artery bypass grafting without the ECG signs of pericarditis may indicate that a transient shortening of the PR interval may be an additional easily available ECG sign of acute PP.

**Keywords:** acute pericarditis; postpericardiotomy pericarditis; electrocardiography; electrocardiography in pericarditis; shortening of the PR interval; electrocardiography signs of pericarditis.

**For citation:** Dundua DP, Khabazov RR, Hricheva NA. Shortening of the PR Interval in Pericarditis After Coronary Bypass Surgery. *Journal of Clinical Practice*. 2022;13(2):5–11. doi: <https://doi.org/10.17816/clinpract108032>

Submitted 18.05.2022

Revised 30.05.2022

Published 18.06.2022

### BACKGROUND

Pericarditis, a well-known clinical syndrome, is the inflammation of the pericardial layers caused by infection, trauma, or damage [1–3]. The inflammatory response of the visceral pericardium to surgery, postpericardiotomy pericarditis (PP), is also a well-known phenomenon [1, 3]. It manifests as pain in the chest, which, unlike anginal pain, is more localised in the cardiac region, aggravated by breathing or movement, sometimes indistinguishable from anginal pain in coronary heart disease. Cardiac auscultation also suggests acute pericarditis. The appearance of a ternary murmur, the so-called pericardial friction murmur, is believed to confirm the diagnosis of dry, non-exudative pericarditis [4]. Auscultatory data in PP have low-informative value because the presence of drainage in the pericardial cavity creates additional noise artefacts; thus, a pericardial friction murmur is often impossible to auscultate.

Electrocardiography (ECG) plays the main role in the diagnosis of acute PP [5, 6]. Acute PP is characterised by typical ECG dynamics in the form of concordant ST-segment elevations in several leads, whereas acute myocardial ischaemia is characterised by discordant changes when ST-segment elevations in the ischaemia zone are combined with ST-segment depressions in the zone opposite to the myocardial ischaemia. Nevertheless, the differential diagnostics of acute pericarditis and acute ischaemic myocardial injury according to ECG criteria is often difficult because the ECG signs of acute PP and acute transmural myocardial ischaemia are sometimes very similar. Differential diagnostics of acute coronary syndrome and acute pericarditis in patients with a history of cardiac surgery is extremely important.

Postoperative myocardial infarction, a life-threatening condition, must not be omitted [7, 8]. The definitive diagnosis of acute pericarditis is made by

ruling out acute ischaemic injury. This requires cardiac imaging, namely, echocardiography (echoCG), radioisotope myocardial scintigraphy and analysis of the activity of cardio-specific enzymes (highly sensitive troponins) over time [9–11]. However, these diagnostic techniques significantly increase the treatment cost and often delay the stay in the intensive care unit.

Another important specific ECG criterion for acute pericarditis is the combination of *ST*-segment elevation with the depression of the *PQ* segment in the standard and left-chest leads [5] because these criteria are pathognomonic for acute pericarditis. However, the follow-up of patients after coronary artery bypass grafting (CABG) showed that *PQ* segment depression in PP did not occur in all patients with concordant *ST*-segment elevation, and the assessment of the *PQ* segment in patients with PP is often complicated because of a noticeable shortening.

This study aimed to clarify whether the shortening of the *PQ* interval is an additional characteristic trait of PP. If this is the case, then the detected ECG phenomenon may be of clinical significance. The presence of an accessible and reliable additional ECG criterion in acute pericarditis after CABG will allow differentiating acute pericarditis from acute myocardial ischaemia, avoiding additional costly studies.

**The study aimed** to identify a pattern in the change in the *PQ* interval in patients with acute pericarditis, who underwent pericardiotomy during CABG. To achieve this aim, we compared the dynamics of *PQ* intervals in patients with ECG signs of acute PP after CABG with that in patients who underwent CABG and did not have ECG signs of acute PP.

## METHODS

### Study design

A retrospective study was conducted.

### Eligibility criteria

**Inclusion criteria.** The study included 60 consecutive patients with chronic coronary heart disease who underwent CABG and had 12-lead ECGs that were of high quality for analysis.

**Exclusion criteria.** Patients with acute postoperative myocardial infarction ( $n=2$ ), mitral valve replacement ( $n=3$ ), paroxysms of atrial fibrillation before, during, or after CABG ( $n=5$ ), acute complete left His bundle branch block ( $n=2$ ) and pacing ( $n=1$  patient) were excluded. Moreover, patients with initial ECG changes in the form of grade 2 and 3 atrioventricular block, complete left His bundle branch block, *ST*-segment

elevation before surgery, with an initially shortened *PQ* interval, and pacing were also excluded. Patients requiring the infusion of sympathomimetic amines on day 2 after CABG, amiodarone, beta-blockers and non-dihydropyridine calcium channel antagonists were also excluded.

### Medical intervention

All patients, before and after CABG, underwent standard 12-leads ECG, two-dimensional echoCG and general clinical tests, including determination of the concentration of blood plasma troponin T before and after CABG, electrolyte composition and acid–base balance of the blood. The diagnostic criteria for acute coronary syndrome were a typical pain syndrome, characteristic ECG dynamics, echoCG-detected new zones of local myocardial contractility disorders and increased highly sensitive troponin T levels in blood plasma, which exceeded the upper limit of normal values by 20 times (a conditional limit adopted in the clinic).

ECG was recorded with a sweep rate of 25 mm/s and a standard calibration of 1 mV=10 mm before surgery, on the day of surgery, by the end of day 1 after surgery and 7–12 days after CABG.

In the analysis of *ST* and *PQ* segments, the *PQ* interval was measured manually. The displacement of the *ST* and *PQ* segments from the isoline was calculated in millivolts (mV). The *ST*-segment position was assessed from the end of the previous T wave to the beginning of the next P wave.

The criteria for diagnosing postoperative pericarditis included ECG changes in the form of *ST*-segment elevations of  $\geq 1$  mV in  $\geq 2$  leads and *PQ* segment depression of  $\geq 1$  mV in standard leads, except for the aVR lead (where the *PQ* segment elevation is equivalent to depression in other leads).

The diagnosis of acute myocardial infarction was ruled out based on the clinical presentation, ECG dynamics (such as the absence of reciprocal changes and appearance of new pathological Q waves), absence of an increase in plasma highly sensitive troponin T of  $>20$  times the upper limit of normal values and the absence of new zones of impaired contractility with echoCG.

### Ethical considerations

Before the study, all patients gave informed consent to the processing of personal data.

### Statistical analysis

Predictors for differences between *ST* elevation and non-*ST* elevation groups were examined. The critical

significance level ( $p$ ) for the study was 0.05. Qualitative variables (predictors) such as sex and history of arterial hypertension, diabetes mellitus and infarction were examined using Pearson's chi-square test, adjusted for continuity and odds ratios. Quantitative variables such as age, heart rate (HR) before and after surgery and at discharge and  $PQ$  before and after surgery and at discharge, were examined using the Mann–Whitney test for unrelated samples. The Wilcoxon test was used to compare predictors over time (linked samples). Statistical calculations were performed using IBM SPSS Statistics for Windows version 26 (IBM Corp., Armonk, NY, USA).

## RESULTS

### Study participants

For the final analysis, 47 patients were selected from among 60 enrolled patients with chronic coronary heart disease. Group 1 consisted of 25 patients with signs of postoperative pericarditis, whereas group 2 (comparison group) consisted of 22 patients without signs of postoperative pericarditis. The average patient age was 64 (range, 50–70) years.

The majority of the patients in both groups had arterial hypertension, and less than one-third of the patients had type 2 diabetes mellitus (Table 1). The

groups were comparable in age and a history of myocardial infarction. Group 1 had male predominance, but the groups did not differ statistically in terms of sex.

### Primary results

According to the chi-square test, for all studied predictors, such as history of myocardial infarction, arterial hypertension and diabetes mellitus, no significant differences were found between the groups (significance level strictly  $>0.05$ ). The dependence of the  $ST$ -segment elevation on the patient's sex was also not proven (Table 1).

$ST$ -segment elevation by more than 1 mV at the J point (up to a maximum of 3 mV; Fig. 1) was noted in more than two leads in group 1.

In 23/25 (92%) patients with signs of acute pericarditis on the ECG after CABG, significant  $PQ$  interval shortening on day 2 after surgery was revealed, with an average of 0.04 s from the baseline (median values 0.18 s before surgery and 0.14 s on day 2).

In group 2, no changes in the duration of the  $PQ$  interval were found (Table 2). The shortening of the  $PQ$  interval in group 1 was transient. In almost all patients, before discharge from the hospital, the duration of the  $PQ$  interval returned to the baseline. The shortening of the  $PQ$  interval in group 1 was sometimes very

Table 1

Main characteristics of patients in the compared groups

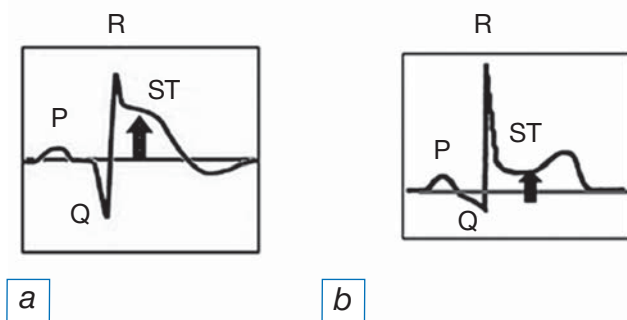
Indicators		ST elevation		Non-ST elevation	
		Quantity	%	Quantity	%
Sex	f	4	16.0	7	31.8
	m	21	84.0	15	68.2
Arterial hypertension		23	92.0	21	95.5
Diabetes mellitus		8	32.0	4	18.2
History of myocardial infarction		12	48.0	9	40.9

Table 2

Heart rate and duration of the  $PR$  interval in the compared groups

Indicator	ST elevation			Non-ST elevation		
	Median	$Q_1$	$Q_3$	Median	$Q_1$	$Q_3$
Age, years	64	60	70	63	55	67
Heart rate (HR) before surgery	63	58	71	68	63	75
$PQ$ before surgery	0.18	0.16*	0.20	0.16	0.16	0.18
HR after surgery	76	68	86	81	75	88
$PQ$ after surgery	0.14	0.12	0.16	0.16	0.14	0.18
HR at discharge	77	72	85	80	74	89
$PQ$ при выписке	0.16	0.14	0.18	0.16	0.15	0.16

**Note:** \*  $p=0,046$ .  $Q_1$ ;  $Q_3$  — interquartile interval. HR — heart rate.



**Fig. 1.** Typical electrocardiogram in acute coronary syndrome with ST segment elevation (a) and acute pericarditis (b): a — for acute coronary syndrome, the curved, monophasic elevation of the ST segment on the isoline is more characteristic; b — in acute pericarditis, the elevation of the ST segment has a saddle-shaped shape, its degree is less pronounced, while depression of the PR interval is noted.

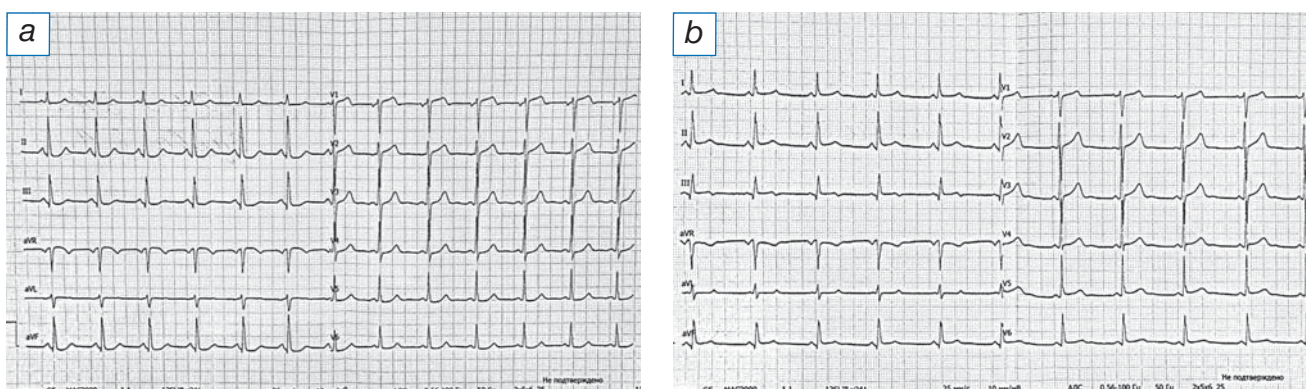
pronounced that it often did not allow the interpretation of the position of the PQ segment relative to the isoline (Fig. 2). Quantitative variables such as age, HR before and after surgery and at discharge, PQ before and after surgery and at discharge showed no relationship.

As shown in Table 2, the only significant change in the PQ interval was its shortening compared with the initial value in patients with ECG signs of PP on day 2 after CABG. In group 2 (without signs of pericarditis), no significant changes in the PQ interval duration were found when compared with the initial one. The median values of the PQ interval before CABG, day 2 after CABG and before discharge did not change and amounted to 0.16 s. According to Table 2, no significant difference in the duration of the PQ interval on the ECG before surgery was found between the groups (the median values of the PQ interval were 0.18 and 0.16 s

in groups 1 and 2, respectively). Upon discharge, the median values of the PQ interval were 0.16 s in both groups.

## DISCUSSION

ECG signs of acute pericarditis are well studied and described. Known differential diagnostic criteria present that ECG can distinguish acute coronary syndrome with ST elevation and pericarditis [1, 3, 5]. Usually in pericarditis, ST-segment elevations are not as pronounced as in acute coronary syndrome, and elevation is often recorded in standard leads. Unlike patients with acute myocardial ischaemia, the ST-segment elevation is unidirectional (concordant) and is not accompanied by reciprocal changes, such as ST depression in other leads. Pain in pericarditis differs from anginal pain; it arises or disappears with a change in body position, and is associated with breathing. However, patients who had undergone heart surgery represent a special group in which the classic signs of PP, such as pericardial friction murmur or breathing-associated pain, may be absent. Owing to analgesia and general anaesthesia, anginal symptoms may also be not pronounced [7, 8]. Thus, the differential diagnostics of postoperative acute coronary syndrome and PP is sometimes difficult. Competent interpretation of ECG changes is very important, and additional ECG signs of acute PP can be used in the differential diagnosis. A very specific but an insensitive symptom of pericarditis is the depression of the PQ segment in leads with ST-segment elevations [5]. However, in patients who underwent pericardiectomy in association with CABG surgery, PQ segment shortening by an average of 0.02 s is also often noted. In the available literature, no study has described such an ECG sign.



**Fig. 2.** Electrocardiogram of the patient: a — on the day of coronary bypass surgery: ST and PR segments on the isoline; the duration of the PR interval is 0.134 seconds; b — on the 2nd day after surgery: in addition to the elevations of the ST segment in leads  $V_2$ – $V_3$ , attention is drawn to the shortening of the PR interval to 0.116 seconds, which does not allow to assess its position relative to the isoline.

Our findings may indicate the presence of a connection between acute inflammation of the pericardium and changes in atrioventricular conduction.

The *PQ* interval is the total time of impulse conduction through the atria, AV node and bundle of His. Its shortening may be the result of pacemaker migration from the sinus node to the atria [5, 11]. However, in this case, the configuration and duration of the P wave also changed. In our cases, the configuration of the P wave did not change. The *PQ* interval also depends on the HR and blockade of atrioventricular conduction. As regards the HR, no differences were found between the groups initially, on day 2 and before hospital discharge. The difference could be accidental if in patients with pericarditis the initial *PQ* interval would be shortened; however, the indicator was within the normal range (median 0.18 s) and did not significantly differ from the control values.

If the duration and axis of the P wave are unchanged, then the *PQ* interval shortening is probably associated with an acceleration of conduction along the atrioventricular node, and this may be a consequence of local, i.e., pericardial, inflammatory causes. If the cause is in the atrioventricular node, there must be either nervous influences (irritation of the pericardial ganglia) or humoral influences because the atrioventricular node is located quite far from the pericardium.

According to our observations, *PQ* segment shortening is noted against the signs of acute PP and disappears after the resolution of pericarditis. This may imply that the observed ECG phenomenon is a characteristic of acute pericarditis. In patients with PP, the level of systemic inflammation should be expected to be increased. However, we did not study the level of inflammatory markers in the compared groups.

### Study limitations

This study has some limitations. First, we relied only on ECG signs of PP compared with other studies, and magnetic resonance imaging of the heart was not performed. This does not enable ruling out other causes of ECG changes. Second, we only evaluated the ECG of PP patients and did not compare this with the ECG of patients with pericarditis of other origins (idiopathic or viral). Thus, a multicentre study including a large number of patients with PP and pericarditis of other origins is desirable.

### CONCLUSION

Thus, in most patients with PP who underwent CABG, along with the characteristic ECG signs of acute

pericarditis, there is a transient *PQ* interval shortening by an average of 0.04 s. The absence of such dynamics in patients after CABG and without ECG signs of pericarditis may indicate that a transient *PQ* interval shortening may be an additional diagnostic ECG sign of acute PP along with other specific ECG signs of acute pericarditis. *PQ* interval shortening may be an additional ECG phenomenon that allows differential diagnostics between pericardial inflammation and acute myocardial ischaemia in patients with PP after CABG.

### ADDITIONAL INFORMATION

**Authors' contribution.** *D.P. Dundua* — making the design of the study, collection of clinical data, literature search and analysis, writing of the manuscript; *R.R. Khabazov* — discussion of the study results, collection and processing of clinical data, editing the manuscript; *N.A. Hricheva* — collection of clinical data, preliminary analysis. The authors made a substantial contribution to the conception of the work, acquisition, analysis, interpretation of data for the work, drafting and revising the work, final approval of the version to be published and agree to be accountable for all aspects of the work.

**Funding source.** The study was funded by the Federal Research Clinical Center of Specialized Medical Care and Medical Technologies of the Federal Medical Biological Agency of Russia.

**Competing interests.** The authors declare that they have no conflicts of interests related to this study.

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