Intracoronary ECG guided PCI in the contemporary catheterization laboratory. Part 1

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ABSTRACT

Intracoronary ECG was invented in 1974 and in 1985 it was shown for the first time that intracoronary wire could be used as an ECG unipolar lead to detect ongoing ischemia in humans. Intracoronary ECG still remains highly underused technique for guiding percutaneous coronary interventions. This article summarizes studies dating from 1974 through 2016 showing intracoronary ECG usefulness in finding zones of ischemia, predicting myocardial necrosis, exploring vital myocardium, predicting myocardial recovery during primary PCI and finding new possibilities for developing better strategies for PCI. Still very cheap and easy to perform, it however requires additional time and deserves better integration in the catheterization laboratory.

Key words: intracoronary ECG, percutaneous coronary intervention, ischemia

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STRESZCZENIE

Wewnątrzwieńcowe EKG zarejestrowane za pomocą jednobiegunowego prowadnika wprowadzonego do tętnicy wieńcowej w celu oceny niedokrwienia wynaleziono w 1974 roku, a po raz pierwszy zostało wykonane u człowieka w 1985 roku. Wewnątrzwieńcowe EKG wciąż jest bardzo rzadko stosowaną techniką diagnostyczną podczas przezskórnej interwencji wieńcowej (PCI). Artykuł ten podsumowuje badania z okresu 1974–2016, pokazując że wewnątrzwieńcowe EKG może być przydatne w znalezieniu strefy niedokrwienia, przewidywaniu martwicy mięśnia sercowego, detekcji żywotnego miokardium, prognozowaniu zdrowienia mięśnia sercowego po pierwotnej PCI, jak również w poszukiwaniu nowych strategii podczas PCI. Nadal bardzo tanie i łatwe do wykonania, mimo iż wymaga dodatkowego czasu, to zasługuje na większe zainteresowanie przez pracujących w pracowniach hemodynamiki.

Słowa kluczowe: EKG wewnątrzwieńcowe, przezskórna interwencja wieńcowa, niedokrwienie

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How to perform intracoronary ECG?

Intracoronary ECG (IC-ECG) is performed using coronary wire, which is the conductor of electrical signal, and the crocodile clips at its distal end connected to a unipolar lead. The guiding catheter serves as an isolator. Balloon catheter can be used for isolation of the coronary wire or coronary polymer-covered wire can be used. Polymer covered wire can additionally be isolated with microcatheter [1–3]. The size of recording (exploring) electrode is the least 3 cm of every workhorse coronary guidewire. This conclusion was verified by checking the change in signal using a microcatheter; i.e., when the catheter tip started to cover the radiopaque distal 3 cm part of the wire, the signal configuration (amplitude, shape) changed. By pulling back the microcatheter, the signal does not change unless the final 3 cm are not covered.

Intracoronary ECG can be used to monitor for ischemia at real time during the procedure, using continuous tracings that is the best way for detecting ischemia, because the time interval from the real event to detecting it on intracoronary ECG is not known. This means that real-time tracing can help in choosing the best treatment strategy. Intracoronary ECG should be performed in every side branch of the treated artery in order to detect regional ischemia — mapping the whole region for ischemia (Figure 1).

Morphology of intracoronary electrocardiogram

Intracoronary ECG detects epicardial electrical signal [4]. Intracoronary ECG could be described as deep S waves and biphasic T waves. There are no "normal" R, S and T waves. They should be interpreted as dynamic changes throughout the whole percutaneous procedure. In myocardial ischemia, ST changes before, during and after the intervention should be interpreted.

R wave amplitude during exercise could detect ischemia [5]. A few studies of peripheral ECG show different results concerning R wave amplitudes. One of them showed that R wave amplitude is positional and the other showed that R wave amplitude is increased during ischemia in precordial and intracoronary leads. Other studies showed that R/S ratio amplitude before and after stenting correlates with post procedural levels of troponin.

ST changes during angioplasty are predictors of ischemia [6] and ventricular arrhythmia during procedure [7]. During myocardial ischemia QT interval is shortened and this could be seen firstly on intracoronary ECG and later on peripheral ECG [8]. The relationship between T-wave peak-to end interval (TPE) and intracoronary ST-segment recovery has been studied. TPE values decreased significantly after primary PCI in patients with intracoronary ST-segment resolution (80.9 ± 22.8 ms vs. 65.8 ± 14.4 ms; $p < 0.001$), whereas they did not change significantly after PCI in patients without intracoronary ST-segment recovery.

Comparing intracoronary and surface electrocardiogram

It is well known that voltage in electrocardiogram depends on the distance between electrodes and cardiomyocytes. Therefore, naturally the intracoronary electrocardiogram should be more sensitive than surface electrocardiogram.

There are few studies that compare sensitivity of both intracoronary and surface ECG for detecting ischemia. In conclusion, intracoronary ECG has better sensitivity for detecting ischemia, finding ischemia in 14% more patients [2, 4, 9–11].

Not only intracoronary ECG is more sensitive for detecting ischemia, but also it is more sensitive in detecting ST changes with higher voltage [9–11].

In these studies, 16–45% of the patients' ischemia is detected using only intracoronary ECG. It should be noted that there are few studies that show patients' ischemia only on surface ECG [2, 6, 11]. Two studies show lower sensitivity for detecting ischemia by intracoronary ECG in RCA than in LAD and LCx [6, 11]. This is mostly caused by the fact that finding ischemia on intracoronary ECG is very much dependent on skills of the operator performing the ECG mapping. It is also dependent on good contact between the coronary wire and ECG cable; sometimes the presence of more metal elements e.g.,

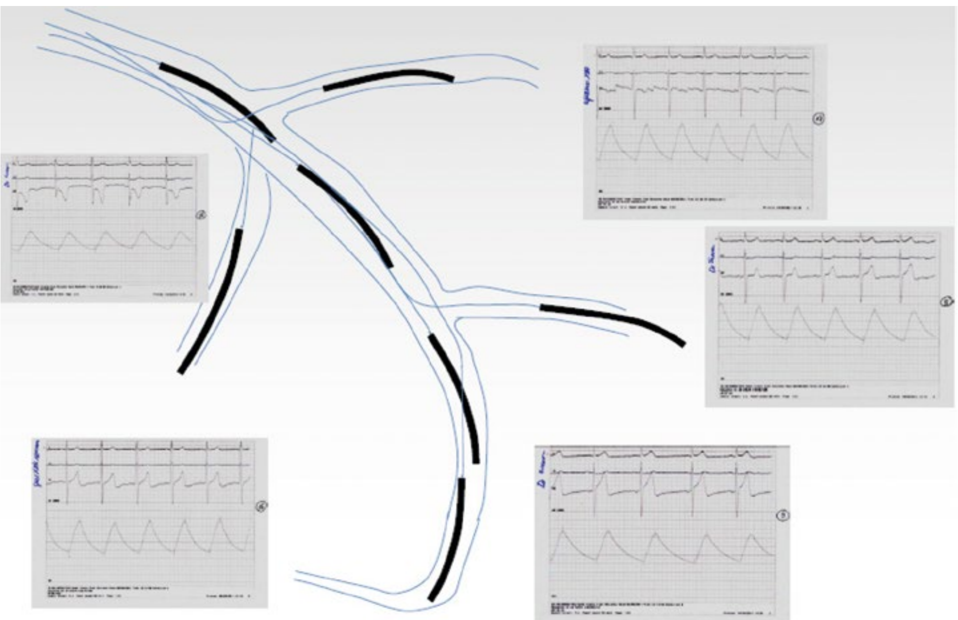

Figure 1. Example of intracoronary ECG mapping of the main branch and all of the side branches in order to search for ischemia in this local territories

stents, or more than one wire can distort the electric signal and insulation balloon or catheter is needed.

Intracoronary ECG and regional ischemia

Intracoronary ECG is the gold standard for detecting ischemia; it shows that precordial V2 from surface ECG is the most sensitive for detecting ischemia in LAD, but not sensitive for detecting ischemia in Lcx [12]. As we know, left circumflex territory is poorly presented in surface ECG. The intracoronary ECG is usually better than surface ECG, because the latter has unfavorable traditional external lead positions, and i.c. ECG has identical sensitivity in the territories of all coronary arteries [10].

Wust *et al.* showed that there are functional collaterals that alleviate ischemia during vessel closure with balloon dilatation [13, 14].

Elevation of cardiac biomarkers after coronary angioplasty reflects periprocedural myocardial damage and is associated with adverse cardiac events. One study assessed whether periprocedural myocardial damage that occurs despite successful PCI could be rapidly and easily identified by intracoronary ST-segment recording with the use of a catheter guidewire. After PCI, intracoronary ST-segment shift of 1 mm from baseline was considered significant. Significant intracoronary ST-segment shift after PCI was present in 40 patients (37%; group A) and absent in the remaining 68 (63%; group B). Procedural myocardial damage was documented in 37 group A patients (93%) and in 13 group B patients (19%; $p < 0.001$); significant ECG changes were found on standard ECG after intervention in only 5 patients (13%) and 1 patient (1%) ($p < 0.05$). Sensitivity of intracoronary ST-segment shift for predicting myocardial damage was 74%, and specificity was 95%, with positive and negative predictive values of 93% and 81%, respectively. On multivariate analysis, intracoronary ST-segment shift was the sole independent predictor of myocardial damage (odds ratio, 54.1; 95% confidence interval, 12.1 to 240; $p < 0.0001$). At a median follow-up of 12 ± 5 months, major coronary event–free survival was significantly worse in group A patients (log-rank test $\chi^2 = 4.0$; $p < 0.05$) [15].

Intracoronary ECG can detect silent ischemia after thrombolytic therapy [16]. During balloon angioplasty, chest pain is caused not only by ischemia but also by stretching of the vessel wall [17]. Intracoronary ECG proves that adenosine-induced chest pain is not caused by ischemia [18]. After aminophylline chest pain decreases without changing ST-segment, proving that P1 receptors are responsible for chest pain during ischemia.

Intracoronary ECG is used to show that perfusion balloons decrease ischemia during angioplasty [8, 19]. Intracoronary ECG is used to detect ischemia during septal angioplasty for treatment of HOCM [20].

Intracoronary ECG could be used for ischemic stress test with atrial rapid pacing after angioplasty [21]. Studies showed that ischemia detected by intracoronary ECG correlates with rise of left ventricular end-diastolic pressure [22], and that there is dependence between hyperemia and ischemic tolerance [23], and other studied investigated the mechanisms of abnormal Q waves in HCM on surface ECG [24].

Intracoronary ECG can detect ischemia during angioplasty; therefore, it could also detect myocardial damage. Residual ischemia on intracoronary ECG is expressed as residual ST-segment elevation after the intervention and as a rise in cardiac biomarkers for myocardial damage as troponin elevation. In that study, intracoronary ECG has positive predictive value of 93% and negative predictive value of 81% [25]. Intracoronary ECG surpasses surface ECG in detecting ischemia and is an independent factor besides patient characteristics, type of lesion and type of intervention.

Percutaneous interventions induce proinflammatory and prothrombogenic factors that could induce distal embolization. Up to 44% of patients after PCI have increased markers for myocardial necrosis, which correlates with increased mortality [26, 27]. Intracoronary ECG could monitor ischemia and ischemia could be detected despite TIMI 3 coronary flow. This could help in choosing the best strategy for treating the patient.

The width of initial QRS complex (pre-angioplasty) correlates positively with troponin rise after angioplasty (OR 1.05, CI 1.01–1.1, $p = 0.016$) [28]. The difference between initial QRS width and its final width also positively correlates with rise of post-angioplasty troponin (OR 18909, CI 19.633–22000, $p = 0.005$) [28].

In patients with NSTEMI and multivessel disease, intracoronary ECG can detect the target lesion with mapping and measuring the ischemia in different regions [29].

Primary angioplasty

In primary infarct angioplasty, resolution of ST-segment changes on IC-ECG during intervention predicted a low peak of creatine kinase values and better left ventricular function at 6 months [30]. The IC-ECG, however, was only slightly better and not significantly different in sensitivity and specificity from the surface ECG for detecting viability in these patients. This study, however, only undertook a limited comparison of the two modalities, as it used a non-continuous method of monitoring both surface ECG and IC-ECG, and used different time points for the acquisition of IC-ECG and surface ECG recordings [30].

ST-segment-resolution (STR) on surface ECG is a good surrogate for myocardial reperfusion in patients with acute ST-segment-elevation-myocardial-infarction (STEMI). In a study, STR on IC-ECG was a measure for predicting of myocardial injury evaluated by cardiac MRI (CMR). Early intracoronary-ECG ST-segment resolution (early IC-STR > 1 mm) correlated with smaller scar mass ($p = 0.003$), nonviable myocardial mass ($p < 0.001$), and microvascular obstruction (MVO) ($p = 0.004$) on CMR at day 3. Ejection fraction (EF) was also better at day 3 ($p = 0.026$) and 90 ($p = 0.039$). Poor early IC-STR (IC-STR < 30%) was conversely associated with larger scar mass ($p = 0.017$), nonviable myocardial mass ($p = 0.01$), and MVO ($p = 0.021$) at day 3. This was also associated with worse EF at day 90 ($p = 0.044$). The degree of early IC-STR (defined by IC-STR > 1 mm or < 30%) successfully predicts myocardial damage following primary-PCI for an acute STEMI [31].

In other study, intracoronary ECG was a predictor of microvascular obstruction during primary PCI detected by cardiac magnetic resonance. Intracoronary ST-segment resolution correlated with MVO ($p = 0.005$). Furthermore, IC-STR (intracoronary ST segment resolution) correlated with infarct-mass, non-viable-mass, peak creatinine kinase and end-systolic-volume at day 4. Intracoronary ST-segment resolution also correlated with favorable left ventricular end-diastolic-volume at day 90 ($p = 0.022$). On multivariate analysis, IC-STR was an independent predictor of MVO [32].

In other study, peak-to-peak QRS voltage was used as marker of myocardial viability detected by delay-enhanced magnetic resonance. Peak-to-peak voltage significantly decreased with nonviable myocardium ($p < 0.001$ for all comparisons). When receiver-operating characteristic analysis was used to compare nonviable with viable segments, the optimal discriminating voltages were 4.6, 2.2, and 0.78 mV for, respectively, HP-0.5, HP-30, and HP-100 filters, with a sensitivity of 92%, 94%, and 99% and a specificity of 70%, 79%, and 69%. The amplitude of the IC-EGMs discriminates viable from nonviable left ventricular segments. Because this technique is simple and inexpensive and provides real-time results, it is potentially useful to aid decision making in the catheterization laboratory [33].

Conclusions

Intracoronary ECG is easy to be performed, utilizes the tools that are not expensive, and performs better in detecting ischemia and finding its location than surface ECG; all of these reflect in many applications. Coronary interventions are expected to be fast and exact and adding a new method will inevitably slow down the intervention. Moreover, sterile connectors are needed. Sometimes the quality of the tracing is not good enough, but could be improved at the expense of time and adding isolators. This technique could help making better decisions about treating the target vessel and provide additional real-time information about myocardial damage.

References

1. Hashimoto K., Corday E., Lang T.W. *et al.* Significance of ST-segment elevations in acute myocardial ischemia. Evaluation with intracoronary electrode technique. Am. J. Cardiol. 1976; 37: 493–500.
2. Meier B., Killisch J.P., Adatte J.J. *et al.* Intracoronary electrocardiography during transluminal coronary angioplasty. Schweiz. Med. Wochenschr. 1985; 115: 1590–1593.
3. Uetani T., Amano T., Kumagai S. *et al.* Intracoronary Electrocardiogram Recording With a Bare-Wire System Perioperative ST-Segment Elevation in the Intracoronary Electrocardiogram Is Associated With Myocardial Injury After Elective Coronary Stent Implantation JACC Cardiovasc. Interv. 2009; 2: 127–135.
4. Friedman P.L., Shook T.L., Kirshenbaum J.M. *et al.* Value of the intracoronary electrocardiogram to monitor myocardial ischemia during percutaneous transluminal coronary angioplasty. Circulation 1986; 74: 330–339.
5. Bonoris P.E., Greenberg P.S., Castellanet M.J. *et al.* Significance of changes in R wave amplitude during treadmill stress testing: angiographic correlation. Am. J. Cardiol. 1978; 41: 846–851.
6. Kwan T., Feit A., Alam M. *et al.* ST-T alternans and myocardial ischemia. Angiology 1999; 50: 217–222.
7. Shah D., Subramanyan K. Significance of ST alternans during percutaneous transluminal coronary angioplasty. Indian Heart J. 1995; 47: 248–252.
8. Safi A.M., Kwan T., Feit A. *et al.* Use of intracoronary electrocardiography for detecting ST-T, QTc, and U wave changes during coronary balloon angioplasty. Heart Dis. 2001; 3: 73–76.
9. Abboud S., Cohen R.J., Selwyn A. *et al.* Detection of transient myocardial ischemia by computer analysis of standard and signal-averaged high-frequency electrocardiograms in patients undergoing percutaneous transluminal coronary angioplasty. Circulation 1987; 76: 585–596.
10. Piessens J., Vrolix M., Sionis D. *et al.* The value of the intracoronary electrogram for the early detection of myocardial ischaemia during coronary angioplasty. Eur. Heart J. 1991; 12: 1176–1182.
11. Pande A.K., Meier B., Urban P. *et al.* Intracoronary electrocardiogram during coronary angioplasty. Am. Heart J. 1992; 124: 337–341.
12. Berry C., Zalewski A., Kovach R. *et al.* Surface electrocardiogram in the detection of transmuralmyocardial ischemia during coronary artery occlusion. Am. J. Cardiol. 1989; 63: 21–26.
13. Wustmann K., Zbinden S., Windecker S. *et al.* Is there functional collateral flow during vascular occlusion in angiographically normal coronary arteries? Circulation 2003; 107: 2213–2220.

14. Seiler C., Fleisch M., de Marchi S.F. *et al.* Functional assessment of collaterals in the human coronary circulation. Semin. Interv. Cardiol. 1998; 3: 13–20.
15. Balian V., Galli M. Intracoronary ST-Segment Shift Soon After Elective Percutaneous Coronary Intervention Accurately Predicts Periprocedural Myocardial Injury. Circulation 2006; 114: 1948–1954.
16. Taylor G.J., Katholi R.E., Womack K. *et al.* Increased incidence of silent ischemia after acute myocardial infarction. JAMA 1992; 268: 1448–1450.
17. Tomai F., Crea F., Gaspardone A. *et al.* Mechanisms of cardiac pain during coronary angioplasty. J. Am. Coll. Cardiol. 1993; 22: 1892–1896.
18. Mehta A.B., Mardikar H.M., Hiregoudar N.S. *et al.* Adenosineinduced chest pain: Is it due to myocardial ischaemia? Clinical, electrocardiographic, haemodynamic and metabolic study. Indian Heart J. 1997; 49: 267–270.
19. Lambert C.R., Bikkina M. Functional characteristics of a new coronary perfusion balloon: Comparison with the Flowtrack 40 in closed chest swine. Cathet. Cardiovasc. Diagn. 1995; 35: 176–180.
20. Kuhn H., Gietzen F., Leuner C. *et al.* Induction of subaortic septal ischaemia to reduce obstruction in hypertrophic obstructive cardiomyopathy. Studies to develop a new catheter-based concept of treatment. Eur. Heart J. 1997; 18: 846–851.
21. Jain A. Early detection of myocardial ischemia after successful percutaneous coronary angioplasty. Cardiology 1997; 88: 533–539.
22. Paraskevaidis I.A., Kyriakides Z.S., Kassimatis A.K. *et al.* Diastolic aortic pressure rise during percutaneous transluminal coronary angioplasty: An index of left ventricular systolic dysfunction. Br. Heart J. 1995; 74: 242–246.
23. Tanaka T., Nonogi H., Miyazaki S. *et al.* Enhancement of coronary reactive hyperemia in patients with ischemic myocardial tolerance during angioplasty. Heart Vessels 1996; 11: 242–247.
24. Koga Y., Yamaga A., Hiyamuta K. *et al.* Mechanisms of abnormal Q waves in hypertrophic cardiomyopathy assessed by intracoronary electrocardiography. J. Cardiovasc. Electrophysiol. 2004; 15: 1402–1408.
25. Balian V., Galli M., Marcassa C. *et al.* Intracoronary ST-segment shift soon after elective percutaneous coronary intervention accurately predicts periprocedural myocardial injury. Circulation 2006; 114: 1948–1954.
26. Nienhuis M.B., Ottervanger J.P., Bilo H.J. *et al.* Prognostic value of troponin after elective percutaneous coronary intervention: a meta-analysis. Catheter Cardiovasc. Interv. 2008; 71: 318–324.
27. Cavallini C., Savonitto S., Violini R. *et al.* Impact of the elevation of biochemical markers of myocardial damage on long-term mortality after percutaneous coronary intervention: Results of the CK-MB and PCI study. Eur. Heart J. 2005; 26: 1494–1498.
28. Vassilev D., Dosev L., Rigatelli G. *et al.* Prediction of troponin elevation by means of intracoronary electrocardiogram during percutaneous coronary intervention of coronary bifurcation lesions (from COronary SIde Branch Residual IschemiA and COllateralization Assessment Study; COSIBRIA & Co Study). DOI: 10.5603/KP.a2016.0057.
29. Hishikari K., Yonetsu T., Lee T. *et al.* Intracoronary electrocardiogram ST-segment elevation in patients with non-ST-segment elevation myocardial infarction and its association with culprit lesion location and myocardial injury. EuroIntervention 2014;10: 105–112.
30. Balian V., Galli M., Repetto S. *et al.* Intracoronary ST segment evolution during primary coronary stenting predicts infarct zone recovery. Catheter Cardiovasc. Interv. 2005; 64: 53–60.
31. Wong D.T., Leung M.C., Das R. *et al.* Intracoronary ECG ST-Segment recovery during primary percutaneous intervention for ST-Segment Myocardial Infarction: Insights from a cardiac MRI study. Catheter Cardiovasc. Interv. 2012; 80: 746–753.
32. Wong D.T., Leung M.C., Das R. *et al.* ECG during primary percutaneous coronary intervention for ST-segment elevation myocardial infarction predicts microvascular obstruction and infarct size. Int. J. Cardiol. 2013; 165: 61–66.
33. Petrucci E., Balian V., Bocchieri A. Real-time assessment of myocardial viability in the catheterization laboratory using the intracoronary electrograms recorded by the PTCA guidewire in patients with left ventricular dysfunction comparison with delayed-enhancement magnetic resonance imaging. J. Am. Coll. Cardiol. Intv. 2014; 7: 988–996.

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