# Effects of acute carbon monoxide poisoning on the P-wave and QT interval dispersions

## *Akut karbonmonoksit zehirlenmesinin P dalga ve QT aralığı dispersiyonları üzerine etkileri*

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## ABSTRACT

**Objective:** The aim of our study was to investigate atrial conduction and ventricular repolarization inhomogeneities using P-wave dispersion (Pwd) and QT dispersion (QTd) analyses in acute carbon monoxide (CO) poisoning.
**Methods:** Sixty patients were retrospectively included in this case-controlled study. Thirty acute CO poisoning patients were assigned to the Group with acute CO poisoning (ACOP). Patients who did not have acute CO poisoning were assigned to the control group (Group C, n=30). Anthropometric measurement, body mass index, electrocardiogram (ECG) and serum electrolyte levels were recorded in all patients. Also, carboxyhemoglobin (COHb) levels were recorded in Group ACOP. Pwd, QT interval and QTd durations were measured. Corrected QT (QTc) and QTc dispersion (QTcd) intervals were determined with the Bazett formula. Independent samples t and Chi-square tests were used for statistical analysis.
**Results:** No statistically significant difference was found between the age, gender distribution, anthropometric measurement, serum electrolytes, PR and QT durations between the groups. The Pwd (56.33±17.11 msec vs 28.33±11.16 msec, p<0.001) and QTd (63.33±26.69 msec vs 42.16±7.84 msec, p<0.001) were significantly longer in Group ACOP than in Group C. In addition, QTc and QTcd durations of Group ACOP were also found to be significantly longer than in Group C (p<0.001).
**Conclusion:** In our study, we found in ECG analyses of patients with acute CO poisoning that the Pwd, QTc and QTcd durations were significantly prolonged when compared with control group. For this reason, patients with acute CO poisoning need close attention because of arrhythmias, which can be related to increased QTcd and Pwd durations.
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**Key words:** Acute carbon poisoning, P-wave dispersion, corrected QT interval, corrected QT dispersion

## ÖZET

**Amaç:** Çalışmamızın amacı, akut karbonmonoksit (CO) zehirlenmesi olan hastalarda atriyal ileti ve ventriküler repolarizasyon homojenlik bozukluklarını P dalga dispersiyonu (PDD), ve QT dispersiyonu (QTd) aracılığı ile araştırılmasıdır.
**Yöntemler:** Retrospektif ve vaka-kontrollü olarak planlanan çalışmamıza, 30 akut CO zehirlenmesi olgusu ile (Grup ACOP), 30 sağlıklı olgu (Grup C) dâhil edildi. Grup ACOP'da acil servise başvuru anında çekilmiş standart 12 derivasyonlu elektrokardiyografi (EKG) kayıtları değerlendirildi. EKG kayıtlarından PDD, QT aralığı, QTd süreleri belirlendi. Düzeltilmiş QT aralığı (QTc) ve QTc dispersiyonu (QTcd) süreleri Bazett formülü kullanılarak belirlendi. Grup ACOP'da ve Grup C'de EKG kaydı ile eş zamanlı olarak alınmış kan örneklerinden serum elektrolit değeri ile Grup ACOP'da COHb değerleri belirlendi. İstatistik analiz için bağımsız örneklerde t ve Ki-kare testleri kullanıldı.
**Bulgular:** Gruplar arasında yaş, cinsiyet dağılımları, antropometrik ölçümleri, serum elektrolit düzeyleri PR ve QT süreleri açısından anlamlı farklılık yoktu. Grup ACOP'da PDD (56.33±17.11 msn'ye karşın 28.33±11.16 msn, p<0.001), ve QTd (63.33±26.69 msn'ye karşın 42.16±7.84 msn, p<0.001) süreleri Grup C'den anlamlı olarak daha uzundu. Ek olarak, QTc ve QTcd süreleri de Grup ACOP'da Grup C'den anlamlı olarak daha uzun saptandı (p<0.001).
**Sonuç:** Akut CO zehirlenmesi olgularında uzamış PDD, QTc ve QTcd süreleri nedeniyle atriyal ve ventriküler aritmi riski yüksektir. Bu olguların yoğun bakım ünitelerindeki takibi sırasında, oluşabilecek atriyal ve ventriküler aritmilere karşı dikkatli olunmalıdır.
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**Anahtar kelimeler:** Akut karbonmonoksit zehirlenmesi, P dalga dispersiyonu, düzeltilmiş QT süresi, düzeltilmiş QT dispersiyonu

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## Introduction

Carbon monoxide (CO) is a colorless, odorless, tasteless, non-irritant, highly toxic gas which produced by incomplete combustion of hydrocarbons (1, 2). Acute CO exposure at high concentrations is known to be lethal and is the most common cause of morbidity and mortality by poisoning (1, 3-5). Acute CO poisoning includes brain, heart and kidneys (2, 6). Acute coronary syndrome, myocardial damage, dysfunction, ischemia, infarction, fibrosis, atrial thrombus, arrhythmias, and cardiac arrest are the cardiovascular changes reported in acute CO poisoning (1, 3, 6-13). Previous reports with small number of patients with acute CO poisoning indicated that in some of the cases; attacks of atrial fibrillation (AF), premature beats, episodes of sinus tachycardia and alterations in ventricular repolarization were observed (14-15). Recent studies showed electrocardiographic (ECG) changes related to arrhythmia risk such as increased QT, QT dispersion (QTd) duration and branch blocks in acute CO poisoning (16-20).

P-wave dispersion (Pwd) is associated with the inhomogeneous and discontinuous distribution of the sinus impulses in the atria, and is defined as the difference between the maximum P-wave duration and the minimum P-wave duration. P-wave dispersion is an easy, simple and non-invasive ECG indicator of risk of occurrence of atrial arrhythmias such as AF (1, 21).

Although AF attacks were observed in some cases with acute CO poisoning, the incidence of AF was not well defined and there is no data published on the impact of acute CO poisoning on Pwd.

The hypothesis of this study is that, similar to corrected QT interval and QT dispersion, the Pwd duration can also increase during acute CO poisoning.

Therefore, we aimed to investigate atrial conduction and ventricular repolarization inhomogeneities using Pwd and QTd analyses in subjects with acute CO poisoning, and compare them to those with control group.

## Methods

### Patients

Following the approval of the hospital Ethics board, 60 patients who had applied to our hospital between May 2005 and April 2009 were retrospectively included into this case-controlled study. Thirty patients admitted to the emergency department with a diagnosis of acute CO poisoning were included in acute CO poisoning (ACOP) Group. On the other hand, 30 patients who applied to anesthesia polyclinic for preoperative evaluation with similar demographic characteristics of Group ACOP, and did not have acute CO poisoning history, were assigned as the control group (Group C).

Patients who were pregnant, pediatric (under 15 years) and with body mass indexes below 20 and above 30, having a story of serious edema, chronic alcoholic liver disease, nephrotic syndrome, renal failure (serum creatinine level >1.5 $mg.dL^{-1}$), electrolyte disorder, diabetes mellitus, hypothyroidism, hyperthyroidism, known psychiatric disorders, coronary artery disease, Chagas's disease, cardiomyopathy, atrial and/or ventricular hypertrophy on ECG, cardiomegaly, valvular disease, cardiac failure or chronic disease, patients with excessive smoking and alcohol consumption and with medication causing QT interval prolongation were excluded from the study.

### Electrocardiography

Standard 12 derivation ECG recordings obtained with a paper speed of 25 mm/sec and a deflection of 10 mm/mV of patients participating in the study were analyzed (Hewlett Packard®, Pagewriter 300pı). In group ACOP, the ECG recordings were obtained before treatment and during the first 10 hours in emergence service. Heart rate was calculated using mean RR time.

### Analysis of QT dispersion

The QT interval was defined as between the beginning of QRS complex and the point where T waves descend onto the TP isoelectric line. When a U wave interrupted the T wave before returning to baseline, the QT interval was measured to the nadir of the curve between the T and U waves (20, 22). The corrected QT interval (QTc) was calculated using the Bazett formula; QTc (ms)=QT measured/√RR (where RR is the RR interval). Prolonged QTc interval was defined as a duration of more than 440 ms. The QTd value was determined as the difference between the longest and shortest QT intervals in the 12 ECG leads. Extended QTd was defined as longer than 60 ms. The QTc dispersion (QTcd) duration according to heart rate was identified with the Bazett formula; QTcd (ms) = QTd measured/√RR.

### Analysis of P-wave dispersion

The beginning of P-wave was defined as positive deflection from the isoelectric line, and the end as the point when the positive deflection returned to the isoelectric line (1, 21). Derivations where the beginning and end of P-waves were not obvious were excluded from the study. Pwd was the difference between the longest and shortest P-wave durations. Increased Pwd was defined as Pwd duration longer than 40 ms.

Subjects who had less than 9 derivations assessed on the ECG were excluded from the study. All ECG measurements were evaluated three times by two experts who were not aware of which group the subject belonged.

### Laboratory analyses

The initial blood levels of sodium, potassium, magnesium, chloride and calcium were measured in the emergency department. In group ACOP, the initial arterial carboxyhemoglobin (COHb) levels were obtained before treatment in emergency service.

### Statistical analysis

Statistical Package for the Social Sciences (SPSS) version 10.0 (SPSS, Inc®; Chicago, Illinois, USA) was used for data analysis. Independent samples t-test was used to compare continuous variables and Chi-square test was used for categorical variables. Pearson's correlation coefficients were determined for relationship of COHb levels with QTc, QTcd, and Pwd. A $p<0.05$ was considered statistically significant.

## Results

### Clinical characteristics of patients

There was no significant difference between Group ACOP and Group C in terms of age, weight, height, body mass index, mean serum sodium, potassium, calcium, chloride, magnesium levels and gender distributions (Table 1 and 2).

In Group ACOP, the mean time delay for admitting to the hospital was 3.6±1.6 hours and the mean measured COHb level was 10.1±5.5% at the admission. In Group ACOP, mean exposure time to COHb was determined as 135.3±138.4 minutes, and 18 (60%) patients had history of loss of consciousness at the time of their admission to the hospital.

### Electrocardiographic findings

In Group ACOP, 4 patients had sinus tachycardia on the ECG records taken at emergency service. All the patients in Group C were in sinus rhythm. No patient in either group had atrioventricular block; nor did any of the patients demonstrated branch block pattern. There was no significant difference between Group ACOP and Group C according to ECG rhythm characteristics ($p=0.056$). The average heart rate of Group ACOP was found to be significantly higher than that of Group C ($p<0.001$) (Table 3). No significant differences were observed in terms of PR ($p=0.434$) and QT ($p=0.365$) intervals between groups (Table 3).

### QT dispersion

The QTc, QTd and QTcd durations of Group ACOP were significantly longer than of Group C ($p<0.001$) (Table 4). In Group ACOP the QTc interval value was above 440 ms in 9 patients (30.0%), while no QTc interval extension was observed in any of the patients in Group C ($p<0.002$). The QTcd interval value was found to be above 60 ms in 20 cases in Group ACOP (66.7%) and none of the cases in Group C ($p<0.001$).

### P-wave dispersion

The minimum P-wave, maximum P-wave and Pwd duration of Group ACOP were found significantly longer than those of Group C ($p=0.002$, $p<0.001$ and $p<0.001$, respectively) (Table 4). The Pwd duration value was found to be above 40 ms in 22 patients in Group ACOP (73.3%) and only in 1 patient in Group C (3.3%) ($p<0.001$).

Pearson's correlation analysis had revealed a moderately significant positive relation was present between QTc, QTcd, Pwd interval, heart rate and COHb levels ($p<0.001$) (Table 4).

**Table 1. Demographic and anthropometric data of the study groups**

| Variables | Group C (n=30) | Group ACOP (n=30) | p* |
|---|---|---|---|
| Age, years | 27.36±7.14 | 29.16±10.47 | 0.441 |
| Gender, F / M, n | 15/15 | 16/14 | 0.796 |
| Height, cm | 166.60±8.59 | 168.23±7.47 | 0.436 |
| Weight, kg | 67.26±10.10 | 68.83±8.07 | 0.510 |
| BMI, kg/m$^2$ | 24.14±2.38 | 24.28±2.26 | 0.822 |

Data are presented as mean±SD and proportions
*Independent samples t-test and Chi-square test
ACOP - acute carbonmonoxide poisoning, C - control, MI- body mass index, F - female, M - male

**Table 2. Biochemical data of the study groups**

| Variables | Normal Values | Group C (n=30) | Group ACOP (n=30) | p* |
|---|---|---|---|---|
| Sodium, mmol.l$^{-1}$ | 136-157 | 142.23±3.09 | 142.83±2.96 | 0.444 |
| Potassium, mmol.l$^{-1}$ | 3.5-5.5 | 4.29±0.49 | 4.18 ± 0.37 | 0.321 |
| Chloride, mmol.l$^{-1}$ | 98-110 | 105.06±3.08 | 104.97±2.81 | 0.906 |
| Calcium, mg.dl$^{-1}$ | 8.4-10.2 | 9.56±0.43 | 9.45±0.54 | 0.386 |
| Magnesium, mg.dl$^{-1}$ | 1.7-2.5 | 2.22±0.22 | 2.14±0.17 | 0.148 |

Data are presented as mean±SD values
*Independent samples t-test
ACOP - acute carbonmonoxide poisoning, C - control

**Table 3. Electrocardiographic data of the groups**

| Variables | Group C (n=30) | Group ACOP (n=30) | p* |
|---|---|---|---|
| Heart rate, beats.min$^{-1}$ | 74.06±11.19 | 87.33±18.02 | <0.001 |
| PR interval, msec | 155.20±13.76 | 158.46±18.02 | 0.434 |
| Max. P-wave duration, msec | 95.66±14.06 | 113.66±17.90 | <0.001 |
| Min P-wave duration, msec | 67.33±12.57 | 57.3 ±11.42 | 0.002 |
| P-wave dispersion, msec | 28.33±11.16 | 56.33±17.11 | <0.001 |
| QT interval, msec | 352.90±26.81 | 360.5 ±37.28 | 0.365 |
| QTc interval, msec | 385.66±16.41 | 429.63±27.56 | <0.001 |
| QTd interval, msec | 42.16±7.84 | 63.33±26.69 | <0.001 |
| QTcd interval, msec | 46.03±9.24 | 76.75±33.14 | <0.001 |

Data are presented as mean±SD values
*Independent samples t-test
ACOP - acute carbonmonoxide poisoning, C - control, Max. - maximum, Min. - minimum, QTc - corrected QT interval, QTcd-corrected QT dispersion, QTd - QT dispersion

### In-hospital arrhythmias

It was determined that 5 (16.6%) patients of Group ACOP had arrhythmias during their intensive care follow-up. One of them had ventricular (3.3%) and 4 (13.3%) of them had atrial arrhythmias. Three of the patients having supraventricular tachycardia were treated with beta-blockers, the others had atrial extrasystoles. Extended Pwd duration was determined in all patients who developed atrial arrhythmias. One of the patients having ventricular arrhythmia had died in the intensive care unit. This

**Table 4. The correlations between electrocardiographic measurements and COHb levels**

| Variables | r* | p* |
|---|---|---|
| Heart rate | 0.520 | <0.001 |
| P min duration | -0.203 | 0.120 |
| P max duration | 0.485 | <0.001 |
| P-wave dispersion | 0.585 | <0.001 |
| PR duration | 0.001 | 0.992 |
| QT duration | -0.072 | 0.584 |
| QTc duration | 0.573 | <0.001 |
| QTd duration | 0.520 | <0.001 |
| QTcd duration | 0.605 | <0.001 |

*Pearson correlation analysis
COHb - carboxyhemoglobin, Max - maximum, Min - minimum, QTc - corrected QT interval, QTcd - corrected QT dispersion

patients' ECG records were taken at emergency service, and QTc, QTcd and Pwd intervals were measured as 443 msec, 109 msec and 60 msec, respectively.

## Discussion

In our study, in the ECG recordings of patients with acute CO poisoning, the QTc, QTd, QTcd and Pwd durations were found to be significantly longer when compared with those of the control group. There was a positive significant an association between acute CO poisoning and Pwd.

The QT and QTc intervals are indicators of ventricular repolarization on ECG. Prolonged QT interval reflects impaired myocardial refractoriness. Prolongation of QT and QTc durations may cause arrhythmia, polymorphic ventricular tachycardia ventricular fibrillation, torsades de pointes and sudden death (1, 23). Studies similar to our study, though limited, demonstrated the effects of acute CO poisoning on QT and QTc interval. Onvlee-Dekker et al. (16) reported that acute CO poisoning may cause unexplained syncope and QT prolongation. However, Gürkan et al. (18) showed that acute CO poisoning did not prolong QT interval. Similarly, we also did not find significant difference between the QT intervals of the study groups. On the other hand, Gürkan et al. (18) showed that corrected QT intervals of the acute CO poisoning patients were significantly longer than those of the control group. We also found the durations of QTc significantly prolonged in Group ACOP.

The QTd and QTcd reflect the physiological variability of regional ventricular repolarization. Increased QTd and QTcd durations were related to heterogeneity of regional ventricular repolarization and are accepted as markers for arrhythmia and sudden death (1, 24). Data on acute CO poisoning effects on QTd and QTcd duration are limited. Recent studies reported that QTd and cQTd durations were increased in the acute CO poisoning (16, 18, 19). Also Sarı et al. (1) showed that chronic CO exposure increased QTd and QTcd durations. Similar to these studies, in our study, we found that the durations of QTd and QTcd significantly prolonged in adult patients with acute CO poisoning.

Prolonged Pwd duration is an easy, simple and non-invasive ECG indicator of atrial arrhythmia such as AF in different patient groups (1, 21, 23-29). It has been reported that acute increases in ambient air pollution were associated with increased risk of paroxysmal AF episodes (30). Sarı et al. (1) also showed that chronic CO exposure increased P max and Pwd durations. However, the association between acute CO poisoning and Pwd has not been investigated before. In this study, we found that the minimum and maximum P-wave durations, and Pwd were significantly prolonged in adult patients with acute CO poisoning when compared with those of patients in the control group. Previous studies showed that increase in Pwd could be due to coronary ischemia (28) and obesity (31). For this reason, in our study we did not include patients with BMI above 30 or patients with coronary ischemia history.

In our study, we found a positive correlation between COHb level and QTc, QTcd, Pmax, Pwd duration. This finding may lead us to a relation between acute CO poisoning and the development of ventricular and atrial arrhythmias. Similar to our study, Yelken et al. (20) has demonstrated that COHb level was correlated with QT intervals and the clinicians should possibly avoid QT prolonging drugs and carefully monitor the QT, QTc, QTcd intervals in patients at high risk of cardiac disability due to high levels of COHb after CO poisoning.

Increased Pwd, P maximum, QT, QTd, QTcd durations in acute CO poisoning may be due to the effects of CO on the myocardium, which probably cause inhomogeneous impulse formation/conduction in the atria and the ventricles (1). On the other hand, CO poisoning may cause acute coronary syndromes and myocardial ischemia (3, 32). As has been previously shown, Pwd prolongation due to coronary ischemia may be an explanation for Pwd prolongation in CO poisoning (28).

### Study limitations

One of the limitations of our study is having no knowledge about the CO levels of the control group because they might be exposed to CO in different ways, such as via air pollution and passive smoking.

Another important limitation of our study is the manual calculation of Pwd on paper ECG. Although several studies have demonstrated a low error rate in the measurement of Pwd on paper printed ECGs (26, 33); others suggested that manual measurement of Pwd performed on standard paper-printed ECGs obtained at a standard signal size and paper speed is of limited accuracy (26, 34). Different manual methods of P-wave analysis (using digitally stored ECGs displayed on a high-resolution computer screen, or a high-resolution digitizing board with a specialized software package) were reported to be mutually consistent and acceptable (26, 34). Therefore, these methods may be an alternative to improve the precision of Pwd measurement.

## Conclusion

We found that the Pwd, QTc, QTd and QTcd durations were significantly increased in acute CO poisoning when compared with those in the control group. Moreover, COHb level is correlated with Pwd, QTcd and QTc. This implication deserves further studies for clarifying the possible linkage between acute CO exposure and atrial and / or ventricular arrhythmias. We believe that during the intensive care therapy of the patients with acute CO poisoning who have extended QTc, QTcd and Pwd durations, more attention should be applied for their high tendency to arrhythmias.

**Conflict of interest:** None declared.

## References

1. Sarı İ, Zengin S, Özer O, Davutoğlu V, Yıldırım C, Aksoy M. Chronic carbon monoxide exposure increases electrocardiographic P-wave and QT dispersion. Inhal Toxico 2008; 20: 879-84.
2. Prockop LD, Chichkova RI. Carbon monoxide intoxication: an updated review. J Neurol Sci 2007; 262: 122-30.
3. Monahan K, Eshaghian A, Dixon J. An unusual CO-conspirator in a case of acute coronary syndrome. Cardiovasc Toxicol 2008; 8: 45-6.
4. Gorman D, Drewry A, Huang YL, Sames C. The clinical toxicology of carbon monoxide. Toxicology 2003; 187: 25-38.
5. Fak AS. Carbon monoxide poisoning and cardiovascular toxicity. Anadolu Kardiyol Derg 2005; 5: 194-5.
6. Yıldırım C, Günay N, Büyükaslan H, Küçükdurmaz Z, Bozkurt S. A case of carbon monoxide poisoning with thrombus in the heart: a case report. Inhal Toxicol 2005; 17: 797-801.
7. Henry TD, Lesser JR, Satran D. Myocardial fibrosis from severe carbon monoxide poisoning detected by cardiac magnetic resonance imaging. Circulation 2008; 118: 792.
8. Kalay N, Özdoğru İ, Çetinkaya Y, Eryol NK, Doğan A, Gül I, et al. Cardiovascular effects of carbon monoxide poisoning. Am J Cardiol 2007; 99: 322-4.
9. Aslan S, Uzkeser M, Seven B, Gündoğdu F, Acemoğlu H, Aksakal E, et al. The evaluation of myocardial damage in 83 young adults with carbon monoxide poisoning in the East Anatolia region in Turkey. Hum Exp Toxicol 2006; 25: 439-46.
10. Henry CR, Satran D, Lindgren B, Adkinson C, Nicholson CI, Henry TD. Myocardial injury and long-term mortality following moderate to severe carbon monoxide poisoning. JAMA 2006; 295: 398-402.
11. Davutoğlu V, Günay N, Koçoğlu H, Günay NE, Yıldırım C, Çavdar M, et al. Serum levels of NT-ProBNP as an early cardiac marker of carbon monoxide poisoning. Inhal Toxicol 2006; 18: 155-8.
12. Satran D, Henry CR, Adkinson C, Nicholson CI, Bracha Y, Henry TD. Cardiovascular manifestations of moderate to severe carbon monoxide poisoning. J Am Coll Cardiol 2005; 45: 1513-6.
13. Varol E, Özaydın M, Aslan SM, Doğan A, Altınbaş A. A rare cause of myocardial infarction: acute carbon monoxide poisoning. Anadolu Kardiyol Derg 2007;7: 322-3.
14. Carnevali R, Omboni E, Rossati M, Villa A, Checchini M. Electrocardiographic changes in acute carbon monoxide poisoning. Minerva Med 1987; 78: 175-8.
15. San Lorenzo IS, Chiesa M, Gamba P, Toniolo A. Cardiologic aspects of carbon monoxide poisoning. Cardiologia 1989; 34: 439-46.
16. Onvlee-Dekker IM, De Vries AC, Ten Harkel AD. Carbon monoxide poisoning mimicking long-QT induced syncope. Arch Dis Child 2007; 92: 244-5.
17. Parenti N, Binetti N, Lenzi T. "Reversible" left bundle branch block in acute carbon monoxide poisoning. Intern Emerg Med 2006;1: 172-3.
18. Gürkan Y, Canatay H, Toprak A, Ural E, Toker K. Carbon monoxide poisoning - a cause of increased QT dispersion. Acta Anaesthesiol Scand 2002; 46: 180-3.
19. Macmillan CS, Wildsmith JA, Hamilton WF. Reversible increase in QT dispersion during carbon monoxide poisoning. Acta Anaesthesiol Scand 2001; 45: 396-7.
20. Yelken B, Tanrıverdi B, Çetinbaş F, Memiş D, Süt N. The assessment of QT intervals in acute carbon monoxide poisoning. Anadolu Kardiyol Derg 2009; 9: 397-400.
21. Duru M, Seyfeli E, Kuvandık G, Kaya H, Yalçın F. Effect of weight loss on P-wave dispersion in obese subjects. Obesity 2006; 14: 1378-82.
22. Cowan JC, Yusoff K, Moore M, Amos PA, Gold AE, Bourke JP, et al. Importance of lead selection in QT interval measurement. Am J Cardiol 1998; 61:83-7.
23. Hume-Smith HV, Sanatani S, Lim J, Chau A, Whyte SD. The effect of propofol concentration on dispersion of myocardial repolarization in children. Anesth Analg 2008; 107: 806-10.
24. Shimizu H, Ohnishi Y, Inoue T, Yokoyama M. QT and JT dispersion in patients with monomorphic or polymorphic ventricular tachycardia/ ventricular fibrillation. J Electrocardiol 2001; 34: 119-25.
25. Yıldırım N, Topaloğlu S, Bozboğa S, Öcal A, Sarıçam E. Diurnal variation of P-wave dispersion in chronic ischemic heart diseases. Coron Artery Dis 2006; 17: 707-10.
26. Dilaveris PE, Gialafos JE. P-wave dispersion: A novel predictor of paroxysmal atrial fibrillation. Ann Noninvasive Electrocardiol 2001; 6: 159-65.
27 Andrikopoulos GK, Dilaveris PE, Richter DJ, Gialafos EJ, Synetos AG, Gialafos JE. Clinical and electrocardiographic predictors of recurrent atrial fibrillation. Pacing Clin Electrophysiol 2000; 23: 352-8.
28. Dilaveris PE, Andrikopoulos GK, Metaxas G, Richter DJ, Avgeropoulou CK, Androulakis AM, et al. Effects of ischemia on P-wave dispersion and maximum P-wave duration during spontaneous anginal episodes. Pacing Clin Electrophysiol 1999; 22: 1640-7.
29. Dilaveris PE, Gialafos EJ, Sideris SK, Theopistou AM, Andrikopoulos GK, Kyriakidis M, et al. Simple electrocardiographic markers for the prediction of paroxysmal idiopathic atrial fibrillation. Am Heart J 1998; 135: 733-8.
30. Rich DQ, Mittleman MA, Link MS, Schwartz J, Luttmann-Gibson H, Catalano PJ, et al. Increased risk of paroxysmal atrial fibrillation episodes associated with acute increases in ambient air pollution. Environ Health Perspect 2006; 114: 120-3.
31. Seyfeli E, Duru M, Kuvandık G, Kaya H, Yalçın F. Effect of obesity on P-wave dispersion and QT dispersion in women. Int J Obes (Lond) 2006; 30: 957-61
32. Dzielski T, Wierzbicka-Chmiel J, Chmiel A. Acute myocardial ischemia after carbon monoxide exposure: a case report. Pol Merkur Lekarski 2006; 20: 696-7.
33. Ciaroni S, Cuenoud L, Bloch A. Clinical Study to investigate the predictive parameters fort he onset of atrial fibrillation in patients with essential hypertension. Am Heart J 2000; 139: 814-9.
34. Dilaveris PE, Gialafos JE. P-wave duration and dispersion analysis: methodological considerations. Circulation 2001; 103: E111-1.