Usefulness of Measuring QT Dispersion for Anesthesia and Surgical Procedures

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Abstract

Electrocardiography (ECG) is a useful monitoring method during anaesthesia. Among the numerous ECG parameters, QT dispersion (QTd) is an indicator of instability during ventricle repolarization. According to previous reports, an increase of QTd may be occurred any patients with sympathetic instability. However, almost all such non-cardiac patients have a physical status of 1 or 2 according to the American Society of Anesthesiology. Therefore, anaesthesiologists should be aware whether the patient’s condition may increase QTd, and should be able to treat unexpected arrhythmia during the perioperative period.

Increases in QTd occur during various perioperative anesthetic managements or surgical procedures. Several reports have indicated that stabilizing autonomic excitation with opioids, volatile anesthetics, β-blockers, or magnesium can prevent unexpected ventricular arrhythmia associated with increased QTd during anesthetic induction. Unexpected ventricular arrhythmia can occur intraoperatively during laparoscopic surgery under general anesthesia. Moreover, the QTd increases significantly before anesthetic induction in patients with major depression, and modified electroconvulsive therapy further increases the QTd.

The disadvantage of QTd is that it cannot be monitored during the intraoperative period. However, anaesthesiologists should understand the meaning of QTd increases, and measuring the QTd preoperatively may facilitate safe management during the perioperative period.

Keywords: Anesthesia; QT interval; QT dispersion; Ventricular arrhythmia; Autonomic instability

Introduction

Electrocardiography (ECG) can provide a lot of information, such as the condition of cardiac conduction activity, hemodynamic response, and origin of cardiac constriction or type of arrhythmia. To assess the cardiac condition from ECG, several parameters are used to detect unexpected cardiac events, including ventricular arrhythmia, during the perioperative period [1].

This review focuses on the significance of measuring the QT dispersion (QTd) for safely managing anesthesia.

QT Dispersion

The QT interval indicates the depolarization and repolarization of ventricles, and elongation of QT is a potential risk factor for ventricular arrhythmia and sudden cardiac death [1].

On the other hand, QTd, a value obtained by subtracting the minimum QT interval from the maximum QT interval, as determined using a recorded 12-lead ECG, is an indicator of instability during ventricle repolarization [2] and may correlate with ventricular arrhythmia induced by autonomic instability [3-5].

Usually, increased QTd in patients without obvious cardiac illness is not a serious condition. However, QTd is considered to be associated with unexpected ventricular arrhythmias [6] and severe cardiac adverse events [7-12] in patients with unidentified cardiac substrate damage or myocardial conductive abnormality. Typically, the normal acceptable range of heart rate-corrected QT dispersion (QTcd), calculated by Bazett's formula, is <50 ms.

A recently published study showed that QTd >58 ms increased the risk of cardiovascular mortality by 3.2-fold in healthy individuals [13], and that patients with QTd ≥ 80 ms have a 4-fold risk for cardiac death compared to those with QTd values <30 ms [14]. Therefore, increased QTd may be a predictor of intraoperative sudden cardiac events caused by inherent autonomic instability. Accordingly, it is important to understand the intraoperative alterations of QTd to avoid unexpected cardiac events during anesthesia.

Previous reports regarding the increase of QTd

According to previous reports, the increase of QTd is associated with cardiovascular diseases such as cardiomyopathy (hypertrophic cardiomyopathy and dilated cardiomyopathy), acute myocardial infarction with ventricular fibrillation, Brugada syndrome, and subarachnoid hemorrhage in the cerebral basilar artery, and with hypomagnesemia. A recent study indicated that QTd is a noninvasive tool for identifying patients with hypertrophic cardiomyopathy and a propensity for non-sustained ventricular tachycardia [15]. Patients with coronary artery disease (CAD) have a significantly increased QTd, which may indicate an increased risk of arrhythmogenesis in these patients. Primary percutaneous coronary intervention for patients with CAD has been shown to be effective for reducing the
QTd, which is an important arrhythmogenic parameter that functions as a marker for successful reperfusion [16-18].

Although the aforementioned reports showed an increase of QTd due to CAD, there are also several reports on increases of QTd in patients with non-cardiac diseases such as hypertension, diabetes mellitus, and obesity. Tanindi et al. [19] reported that the QTd was increased in pre-hypertensive patients compared with normotensives, with different patterns of nocturnal blood pressure decreasing significantly. Franzoni et al. [20] reported that women with anorexia nervosa had a greater QTd than women with a normal body weight. Dagli et al. [21] reported that patients with central serous chorioretinopathy showed increased QTd. Further, approximately 40% of patients with systemic lupus erythematos had an increase of QTd [22]; these patients may have undergone general anesthesia. However, almost all of the aforementioned non-cardiac patients were categorized with a physical status of 1 or 2 according to the American Society of Anesthesiology, because of their general condition. Furthermore, the authors did not assess the QTd preoperatively; thus, the perioperative risk of unexpected cardiac events remains. Therefore, anesthesiologists should know whether the patient’s condition may be associated with increased QTd, and should be able to treat unexpected arrhythmia during the perioperative period.

The relations between anesthesia, surgical procedures, and QTd are described in the following section.

Relationships between QTd, anesthesia, and surgical procedures

Several reports have indicated that QTd is increased as a result of various perioperative anesthetic managements or surgical procedures. These are collated with sympathetic nervous stimulation caused by mechanical or electrical procedures, hypoxia, or CO2 consumption.

As a possible condition of the increase of QTd during the perioperative period, we have focused on the following: (1) anesthetic induction, (2) laparoscopic surgery, and (3) modified electroconvulsive therapy (mECT).

Anesthetic induction (mechanical sympathetic stimulation)

At the induction of anesthesia, especially during tracheal intubation, the sympathetic nervous system is strongly stimulated. As a result, the patient’s heart rate and blood pressure markedly increase. Moreover, arrhythmia occurs at induction, especially ventricular arrhythmia, which may result from sympathetic stimulation by intubation, hypoxia, and/or CO2 consumption (e.g., hypercarbia).

Several reports have indicated that the increase of QTd may occur simultaneously with anesthetic induction, and to reduce this increase caused by sympathetic stimulation, various approaches of anesthetic induction are being assessed. All volatile anesthetics, especially isoflurane and desflurane, have been found to prolong the QTc and increase the QTcd [23]. On the other hand, neither remifentanil nor fentanyl appears to prolong the QT, whereas the QTd is decreased after anesthetic induction and does not increase after tracheal intubation in patients receiving remifentanil compared with fentanyl. Overall, a remifentanil infusion may be the opioid-based treatment regimen of choice in patients at risk for dysrhythmias [24]. Further, while various catecholamines, such as epinephrine, dopamine, and phosphodiesterase inhibitors, increase the QTd, β-blockers have been demonstrated to reduce sympathetic activity and QTd [25-27]. Further, lidocaine also reduces the QTd [28]. Kiraci et al. showed that QTd is increased during tracheal intubation in patients with CAD, and reported that magnesium sulfate may be useful for these patients [29].

Taken together, these reports indicate that stabilizing autonomic excitation with opioids, volatile anesthetics, β-blockers, and magnesium can prevent unexpected ventricular arrhythmia associated with an increase of the QTd at the induction of anesthesia.

Laparoscopic surgical procedures (autonomic stimulation by the insufflation of CO2)

Laparoscopic surgery is performed by inflating CO2 gas into the abdominal cavity. Inflated CO2 is absorbed from the peritoneal membrane by diffusion to complete its transition into the systemic vascular system. The consumption of CO2 is allowed to activate sympathetic nerve activity, and induces the increase of catecholamine to 2–3 times [30]. In a recent report, 47% of patients who underwent laparoscopic surgery had an intraoperative arrhythmia, and 30% of these had bradycardia [31]. Therefore, we previously investigated the measurement of QTd in laparoscopic surgery performed under general anesthesia. As a result, statistically significant increases of QTd occurred during CO2 insufflation, and these were higher than in the abdominal wall lift-up group [32].

Moreover, the QTd also increased significantly during CO2 insufflation in both elderly and younger groups [33]. However, the increases of QTd were significantly greater in the elderly patients compared to in younger patients from 120-150 min after CO2 insufflation. In particular, the longer duration of CO2 insufflation with head-up tilt was suggested to be associated with the increase of QTd in elderly patients.

Hence, it can be considered that laparoscopic surgery under general anesthesia in elderly patients may induce unexpected arrhythmias intraoperatively.

Modified ECT (electrical sympathetic stimulation)

Modified ECT (mECT) is used to treat severe psychiatric disorders by inducing stimulation of the autonomic nervous system with initial parasympathetic outflow, which is immediately followed by a sympathetic response. These responses may induce initial bradycardia, arrhythmias, and hypertension. Various antidepressants and antipsychotics may prolong the QT interval and increase the QTd prior to mECT. In fact, prolongation of QT and QTd in patients taking these drugs prior to mECT was found to be enhanced by electrical stimulation under anesthesia in our previous report [34]. The effects of an electrical stimulus by mECT on the QT and QTd are of considerable interest [35]. Tezuka et al. designed a study to investigate the effects of electrical stimulation caused by mECT on the RR interval, QT, QTc, QTd, and QTcd under anesthesia with 1 mg/kg of propofol and sufentanyl [34]. In that report, ventricular premature complex and tachycardia were observed in 26 of 30 cases after electrical stimulation. Moreover, in 90% of patients, the baseline values of QTcd were higher than the normal limit, and these increased significantly immediately to 5 min after the electrical stimulus was applied. Thus, the authors concluded that the QTcd increased significantly before anesthetic induction in patients with major depression. Moreover, we have found preventive effects of administering 0.5 μg/kg or 1.0 μg/kg of remifentanil prior to mECT on ventricular arrhythmia along with stabilization of hemodynamic changes after electro-stimulation, according to observed alterations in the QTc (under review).
Accurate ECG measurements immediately after mECT are difficult to obtain. However, ECG approximately 1 min after electro-stimulation can be recorded accurately. Therefore, these studies are informative to understand the autonomic instability caused by mECT.

Conclusions

Whether QTd can become a surrogate marker is still unclear. The reproducibility of all QTd measurements is inferior to that of conventional ECG indices in healthy subjects [36]. Some studies have reported that QTd depends on the amplitude and width of the T-wave loop, and that it does not reflect the heterogeneity of the repolarization time [37,38]. However, there are several reports regarding the usefulness of assessing QTd in ill patients during the intraoperative period. Disadvantages of QTd include that it cannot be monitored intraoperatively. Hence, we cannot predict sudden cardiac events by monitoring of the QTd during surgery. However, the relevance of various ECG parameters has not been recognized as the useful indicators. I recommend that anesthesiologists should aim to understand the meaning of QTd increases, and conclude that measuring the QTd preoperatively may facilitate avoiding unexpected events to ensure safe management during the perioperative period.

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Conflicts of interest

The authors declare no conflicts of interest.

References


