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Prolongation of the Q-T Interval and Sudden Cardiac Arrest Following Right Radical Neck Dissection

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Radical lymph node dissection of the neck with laryngectomy for carcinoma can result in various perioperative complications.[1,2] Among them, prolongation of Q-T interval and severe ventricular arrhythmias with cardiac arrest occasionally have been reported.[3,4] Such a life-threatening complication in a patient who underwent this operation on the right side of the neck and the consideration of published data,[3,5–7] stimulated this study. We investigated on the Q-T interval prolongation with regard to right or left radical neck dissection and the incidence of severe postoperative cardiac arrhythmias.

PATIENTS AND METHODS

Sixty patients scheduled for right or left radical neck dissection consented to be in this prospective study, which covered a period of 9 months. The patients were divided into two groups. Group 1 consisted of 32 patients who underwent right radical neck dissection. Group 2 consisted of 28 patients who underwent left radical neck dissection. Both groups were comparable in age, weight, ASA class, anesthetic technique, and duration of the operative procedure. The preoperative Q-T interval was normal in all patients. We compared preoperative and postoperative Q-T intervals and heart rates. The values were assessed after the first, second, sixth, and twelth postoperative hour, the first postoperative day in all patients and after 2 months in 21 patients. The Q-T interval was measured from the beginning of the Q wave (or the beginning of the R deflection) to the end of the T wave (whether upright or inverted). The Q-T interval then was corrected for heart rate using Bazett's formula ($Q\text{-}Tc = Q\text{-}T/\sqrt{R\text{-}R}$) where Q-T and Q-Tc are expressed in milliseconds (ms) and R-R interval in s. The upper limit of normal of the Q-Tc interval was taken as 440 ms.[4]

Plasma levels of potassium and calcium also were measured 1 h after surgery. All patients stayed in the recovery room for 36 h to have continuous ECG monitoring. Ventilation was controlled via a tracheostomy tube during the first postoperative night.

Intragroup data were analyzed using the paired t test and the intergroup data with the t test for two means. All results are expressed as means ± 1 SE.

RESULTS

Right radical neck dissection resulted in a large and significant increase of Q-T interval (fig. 1) after surgery, as compared with preoperative values ($P < 0.001$). This increase persisted in the 12 patients we were able to follow more than 2 months postoperatively ($P < 0.001$). In contrast, the same surgical procedure on the left side of the neck did not alter significantly the Q-T interval. Significant postoperative increase in heart rate was observed in both groups, as compared with preoperative values ($P < 0.02$).

During the first postoperative days, three of the 32 patients of Group 1 developed severe episodes of tachyarrythmia, causing sudden circulatory arrest. This tach-

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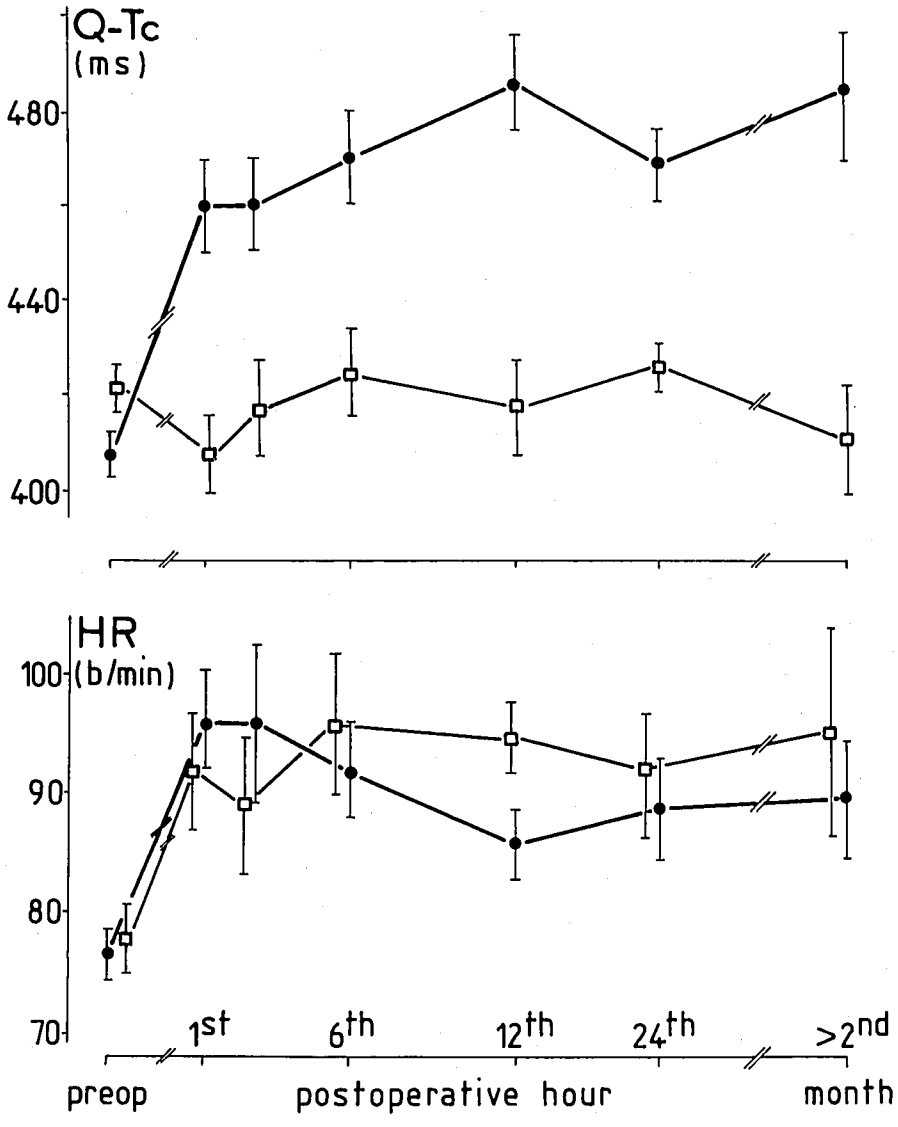

FIG. 1. Corrected Q-T interval (Q-Tc) and heart rate (HR) in relation to right (– ● –) and left (– □ –) radical neck dissection. Values plotted are means ± SE. Right dissection increased Q-Tc when compared with left radical neck dissection ($P < 0.001$). Both right and left dissection resulted in postoperative tachycardia when compared with preoperative heart rate ($P < 0.02$).

yarrhythmia consisted in an atypical ventricular tachycardia, known in France as "*torsades de pointes.*" In the first patient, the Q-T interval increase to 698 ms was followed by severe episodes of *torsades de pointes* requiring cardiac massage, 20 h postoperatively (fig. 2). The plasma potassium level was 3.6 mEq·l^{-1}. The arrhythmia was treated successfully by cardiac pacing and the iv infusion of potassium chloride. The Q-T interval returned to 499 ms. The patient had no further complications. In the second patient, the postoperative Q-T interval increased to 500 ms, while the plasma potassium was 3 mEq·l^{-1}. Ten hours postoperatively, typical episodes of *torsades de pointes* in close succession occurred. They were treated successfully by the iv administration of isoproterenol and KCl, after which the Q-T interval returned to 460 ms. In the third patient, the Q-T interval postoperatively reached 612 ms, and the plasma potassium level fell to 3.1 mEq·l^{-1}. After correction of hypokalemia, the Q-T interval returned to 534 ms. The immediate course was uneventful, but the patient suddenly died 10 days postoperatively. An autopsy was not obtained.

The plasma calcium and potassium levels in Group 1 and Group 2 were not significantly different the first hour postoperatively (4.32 ± 0.08 *vs.* 4.40 ± 0.08 mEq·l^{-1} for calcium and 3.94 ± 0.1 *vs.* 3.84 ± 0.08 mEq·l^{-1} for potassium).

There was no linear relation between the Q-T interval and plasma potassium level. Hypokalemia developed in seven Group 1 patients and in four Group 2 patients. Only patients from Group 1 developing a hypokalemia suffered severe cardiac complications, *i.e.*, two *torsades de pointes* and one unexpected death, possibly also related to a severe arrhythmia.

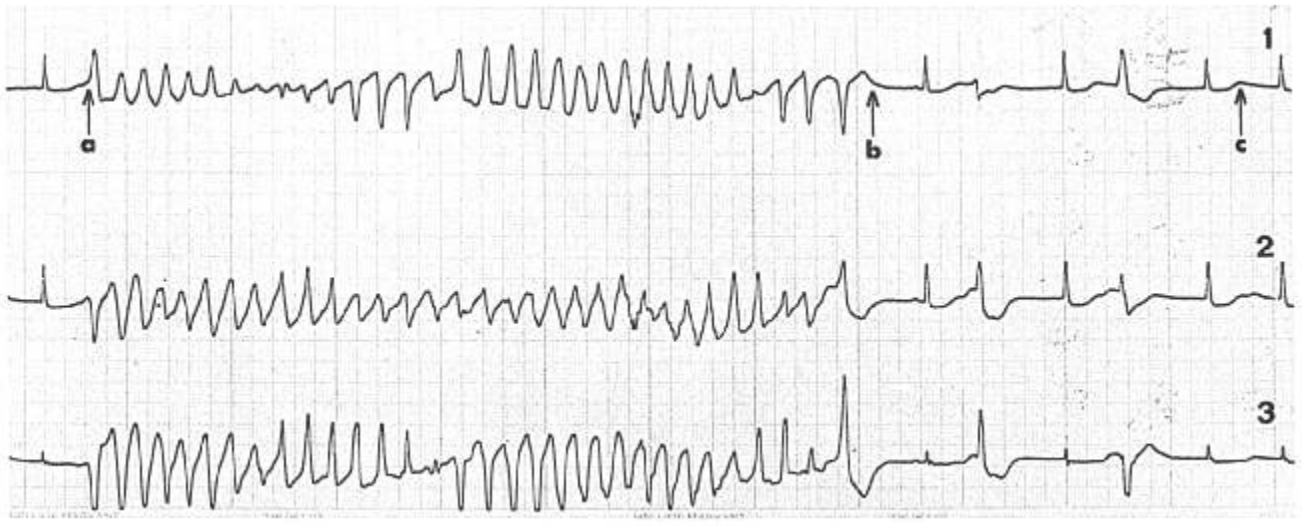

FIG. 2. Paroxysm of "*torsades de pointes*" in one patient. It is initiated by a ventricular premature systole falling on the vulnerable part of the preceding complex (*arrow* a) and ends spontaneously (*arrow* b). Q-T intervals are prolonged (*arrow* c). ECG leads 1, 2, and 3 are shown.

DISCUSSION

The effects of radical neck dissection on Q-T interval and development of severe cardiac arrythmias rarely have been described. In 1967, Hugenholtz[3] described a case of Q-T interval prolongation in a patient who had undergone radical right neck dissection. More recently, Moss and Schwartz[4] simply mentioned neck surgery as a possible cause of the long Q-T syndrome and sudden death with no documentation.

Postoperative prolongation of the Q-T interval and increase in heart rate are related to surgical trauma of the cervical autonomic nervous system. The surgical peeling of the vagus nerve during right or left radical neck dissection probably accounts for postoperatively tachycardia. Right radical lymph node dissection results in a significant and probably irreversible prolongation of the Q-T interval. Such a prolongation occurs neither after left radical neck dissection nor after right (or left) so-called functional dissection that removes only the lymph nodes and preserves the other structures (especially the sternocleidomastoid muscle, the internal jugular vein, and the accessory nerve). The alteration of right sympathetic outflow to the heart by neck dissection probably accounts for prolongation of the Q-T interval and severe arrhythmias. Indeed, a part of the afferent and efferent sympathetic fibers often are removed, although the dissection preserves the cervical sympathetic trunk itself. Several arguments substantiate this hypothesis. Experimental data suggest that right and left cardiac sympathetic nerves have different effects on cardiac excitability.[5] Ablation of the right stellate ganglion or stimulation of the left one prolongs the Q-T interval and lowers the ventricular fibrillation threshold.[6] Left stellate ganglionectomy or block constitutes a treatment of long Q-T interval syndrome.[4,7,8]

The prolongation of Q-T interval may result in a particular type of tachyarrhythmia, first described by Dessertenne[9] as *torsades de pointes*[9–13] and usually known in English-speaking countries as "atypical ventricular tachycardia." This type of arrhythmia is characterized by rapid (200–250 beats/min) and irregular paroxysms of ventricular activity with variations of QRS amplitude and polarity over 5–20 beats (fig. 2). It is usually self-limiting (the duration of an episode lies between several seconds and 1 min) but may degenerate to ventricular fibrillation. During an episode of *torsades de pointes* there is no cardiac output and, after several seconds of duration, syncope ensues. Prophylaxis of *torsades de pointes* can be achieved by administration of beta adrenergic blockers to prevent generation of ventricular extrasystoles, which induce episodes of *torsades* (R/T phenomenon). Treatment is based on initiation of a rapid ventricular rhythm with atropine, isoproterenol, or cardiac pacing in order to suppress the initiating ventricular extrasystoles that appear with low cardiac rates.[13]

The life-threatening ventricular arrhythmias associated with prolongation of the Q-T interval occurs especially in combination with hypokalemia. Hypokalemia by itself results in Q-T prolongation with prominent U waves.[14] Development of intraoperative and postoperative hypokalemia is favored by use of thiazides and corticosteroids, polyuria, and ventilatory alkalosis. In case of Q-T prolongation, hypokalemia should be avoided. If, in spite of correction of hypokalemia, a proplonged Q-T interval persists, treatment with beta

adrenergic blockers,[15] cardiac pacing,[16] left stellate ganglion pharmacologic blockade, or left cervicothoracic sympathetic ganglionectomy[4,8] may prove beneficial.

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