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Giant Negative T Waves After Maxillofacial Surgery

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A patient developed strongly negative T waves following anesthesia for maxillofacial surgery. The electrocardiogram was normal preoperatively, except for a single premature ventricular depolarization, and no abnormalities were noted during the operation. Postoperatively, the T wave gradually inverted in almost all leads and approached the criterion of −10 mm for giant negative T waves in V_3 2 days postoperatively. The T wave returned to normal approximately 4 months later. Although the T-wave inversion in this patient may have been caused by surgical trauma to the sympathetic nerve supply to the heart or by myocardial infarction, the exact cause remains undetermined.

The giant negative T wave is an electrocardiographic abnormality that can be associated with surgery or general anesthesia. Because substantial T-wave inversion also occurs after myocardial infarction, especially subendocardial infarction, it is important to determine the cause of such T-wave changes. Several reports have described giant negative T waves, but the mechanisms underlying this electrocardiographic disturbance are not established. This report describes a patient who developed strongly negative T waves in almost all leads following maxillofacial surgery.

CASE REPORT

The patient was a 50-kg, 68-yr-old male with cancer of the right lower jaw. He was scheduled for a right radical neck dissection, segmental resection of the mandible, and reconstruction with an A-O plate. The medical history revealed pulmonary tuberculosis at the age of 24 yr and recurrent anginal attacks since the age of 50 yr. The patient was taking nifedipine and ubidecarenone, and he reported that he had not experienced an anginal attack for several years.

Multifocal premature ventricular contractions (PVCs) were revealed on the ambulatory electrocardiogram (ECG). He was given disopyramide for 10 days before surgery to control this arrhythmia. Laboratory data were within normal limits (Table 1). The preoperative ECG showed a left axis deviation (−50°) and a single PVC (Figure 1). There were no obvious abnormalities on the echocardiogram (Table 2). The chest x-ray showed radiopaque areas bilaterally in the upper lung regions, consistent with old pulmonary tuberculosis (Figure 2A). Ophthalmoscopic examination (Figure 3) revealed retinal arteriolosclerosis (Scheie's classification H2, S1).

Table 1. Preoperative Laboratory Data

Hematology			Pulmonary function		
WBC[a]	5,500	/mm³	FVC	3.58	L
RBC	410 × 10⁴	/mm³	$FEV_{1.0}$	2.73	L
Hb	13.2	g/dl	%VC	94.8	%
Ht	39.4	%	$\%FEV_{1.0}$	84.4	%
Blood chemistry			Urinalysis		
AST	16	U/L	Specific gravity	1.015	
ALT	15	U/L	pH	5.8	
GGT	16	U/L	Glucose	(—)	
LH	211	U/L	Ketones	(—)	
Protein	7.3	g/dL	Protein	(—)	
ALB	4.1	g/dL	Blood gas analysis		
A/G	1.28		pH	7.421	
CK	31	U/L	P_{CO_2}	39.3	mm Hg
Na^+	42	mEq/L	P_{O_2}	78.3	mm Hg
K^+	4.2	mEq/L	HCO_3^-	25.5	mEq/L
Cl^-	102	mEq/L	BE	1.7	mEq/L

[a] WBC, white blood cells; RBC, red blood cells; Hb, hemoglobin; Ht, hematocrit; AST, asparate aminotransferase; ALT, alanine aminotransferase; GGT, γ-glutamyl transpeptidase; LH, lactate dehydrogenase; ALB, albumin; A/G, albumin/globulin ratio; CK, creatine phosphokinase; FVC, forced vital capacity; $FEV_{1.0}$, forced expiratory volume in one sec; P_{CO_2}, arterial carbon dioxide tension; P_{O_2}, arterial oxygen tension; BE, base excess.

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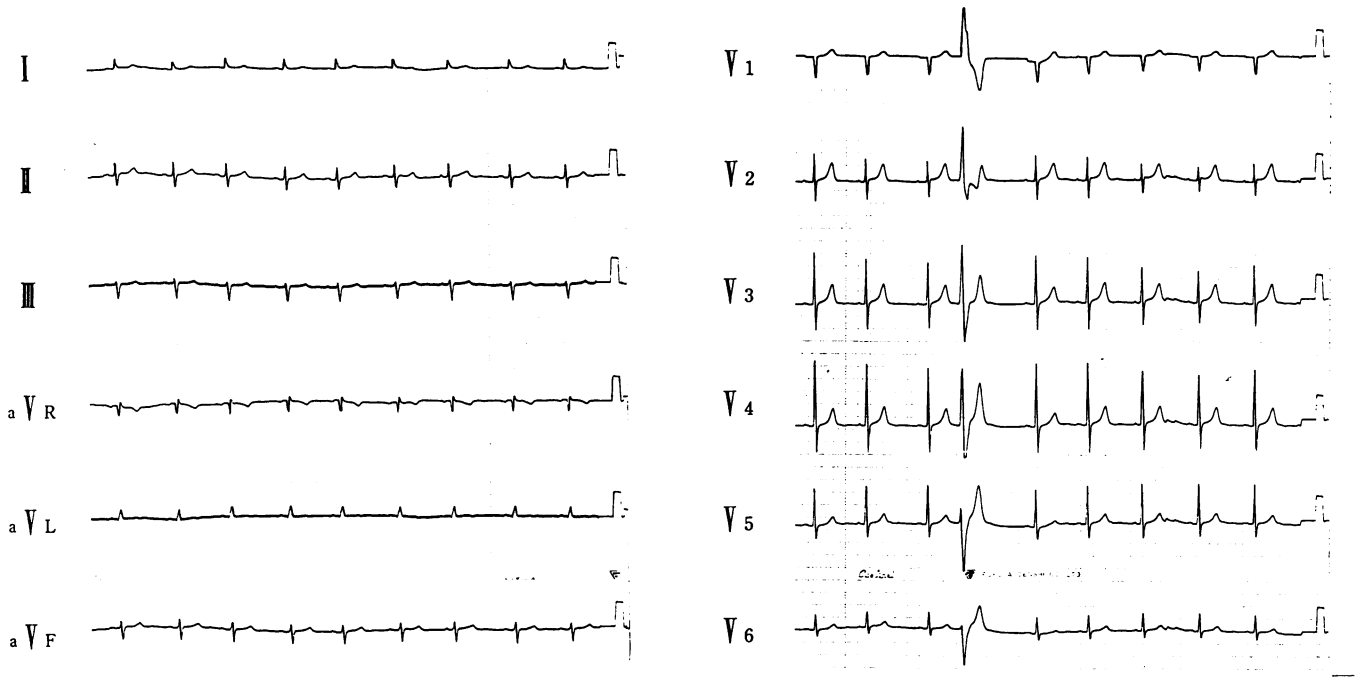

Figure 1. Preoperative electrocardiogram.

Nifedipine and ubidecarenone were taken until the day before surgery, and 150 mg disopyramide was given on the morning of the operation. The patient was premedicated intramuscularly with 0.3 mg atropine and 50 mg meperidine. After 20 min the patient was brought to the operating room. The blood pressure was 160/90 mm Hg and the heart rate was 105 beats/min. Following the insertion of a nasogastric tube and transtracheal and topical anesthesia of the upper airway with lidocaine, nasotracheal intubation was gently performed under intravenous sedation with diazepam. Diltiazem was administered intravenously to control blood pressure until the endotracheal tube was placed.

Table 2. Echocardiographic Findings

	Preoperative	*2 Days Postoperative*	*3 Months Postoperative*
AOD[a] (mm)	28	31	28
LAD (mm)	22	23	29
LVDd/s (mm)	46/34	48/33	48/32
LVS (mm)	12	10	10
LVPW (mm)	15	10	10
CO (ml)	50	64	67
EF (%)	52	59	62
LV wall motion	normal	normal	normal
MR	(−)	(−)	mild
AR	(−)	(−)	(−)
TR	(−)	(−)	(−)

[a] AOD, aortic dimension; LAD, left atrial dimension; LVDd/s, left ventricular dimension (diastole/systole); LVS, left ventricular septum; LVPW, left ventricular posterior wall; CO, cardiac output; EF, ejection fraction; LV, left ventricle; MR, mitral regurgitation; AR, aortic regurgitation; TR, tricuspid regurgitation.

Anesthesia was maintained with nitrous oxide (6 L/min), oxygen (3 L/min), and sevoflurane (1% to 2%) under assisted ventilation. Prilocaine with felypressin was injected for local anesthesia at the onset of surgery. The procedure progressed uneventfully, and no remarkable changes in blood pressure and heart rate were seen intraoperatively (Figure 4). Although occasional premature atrial contractions were observed, no PVCs or abnormalities consistent with myocardial ischemia were noted.

Postsurgically, oxygen was delivered by face mask at a flow of 1 to 2 L/min. A sheet of isosorbide dinitrate tape was applied to the patient's chest to control blood pressure. The tape was left in place for 3 hr, after which it was removed and the disopyamide reinstituted.

At approximately 9 pm (6 hr after the end of surgery) T-wave changes were observed on the ECG monitor, and a 12-lead ECG was ordered. T-wave inversion was observed in leads I, II, III, $_aV_F$, and V_3 to V_6. The blood pressure was 136/74 mm Hg, the heart rate was 75 beats/min, and there was no clinical evidence of myocardial ischemia (eg, chest pain). A sheet of isosorbide dinitrate tape was reapplied.

By the first postoperative day, the T wave in lead V_2 became inverted, and the QT interval was slightly prolonged (0.40 sec). Except for a slight elevation of creatine

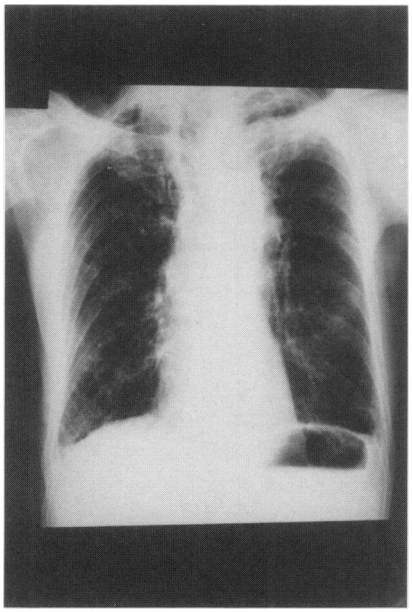

Figure 2A. Preoperation chest X-ray.

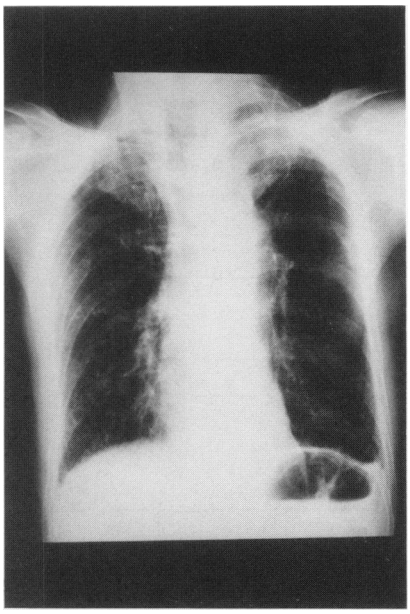

Figure 2B. Postoperation chest X-ray.

kinase (CK) and CK-MB (the muscle-brain isoenzyme of CK), laboratory data were normal (Table 3). Nifedipine and ubidecarenone were resumed, and isosorbide dinitrate tape was maintained (1 sheet/day).

On the second postoperative day, the ECG showed greater prolongation of the QT interval (0.48 sec) and an abnormal QTc (0.58). The deepest inversion of the T wave, 0.9 mV, was present in lead V_3. No other abnormalities, such as QS patterns or ST elevations, were evident on the ECG. There also were no abnormalities on the echocardiogram (Table 2) or the chest x-ray (Figure 2B).

Oxygen supplementation and isosorbide dinitrate tape was maintained, respectively, for 11 days and 18 days postoperatively. The T-wave inversion, which was deepest on the second and third postoperative days, began to subside on the fourth day. A normal QT interval and positive T waves in some leads had returned by 4 wks after surgery, and the patient was discharged on the same medication he had been taking preoperatively. A follow-up ECG about 4 months later (111 days) was normal (Figure 5). While the color flow of the Doppler echocardio-

Figure 3. Preoperative ophthalmoscopy.

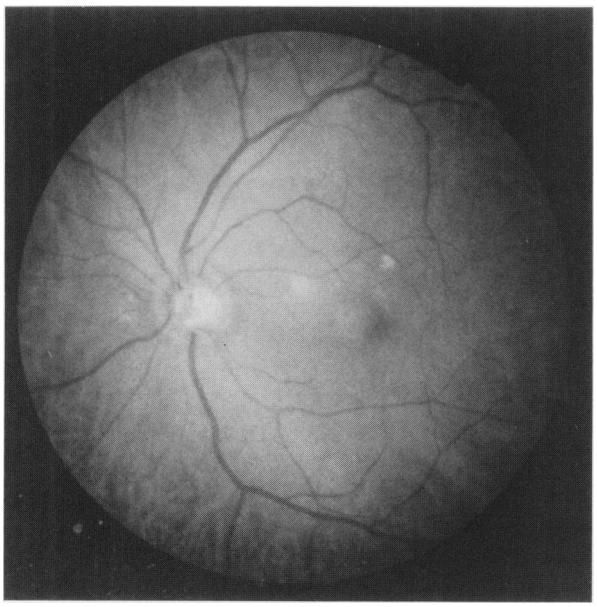

Table 3. Postoperative laboratory data

Hematology			*Blood Chemistry*		
WBC[a]	9,400	/mm^3	AST	25	U/L
RBC	375 × 10^4	/mm^3	ALT	12	U/L
Hb	12.0	g/dL	GGT	13	U/L
Ht	35.7	%	LH	213	U/L
			CK	181	U/L
			CK–MB	5.9	%
			Na^+	137	mEq/L
			K^+	4.0	mEq/L
			Cl^-	100	mEq/L

[a] WBC, white blood cells; RBC, red blood cells; Hb, hemoglobin; Ht, hematocrit; AST, asparate aminotransferase; ALT, alanine aminotransferase; GGT, γ-glutamyl transpeptidase; LH, lactate dehydrogenase; CK, creatine phosphokinase; CK-MB, creatine phosphokinase; NA^+, sodium; K^+, potassium; Cl^-, chloride.

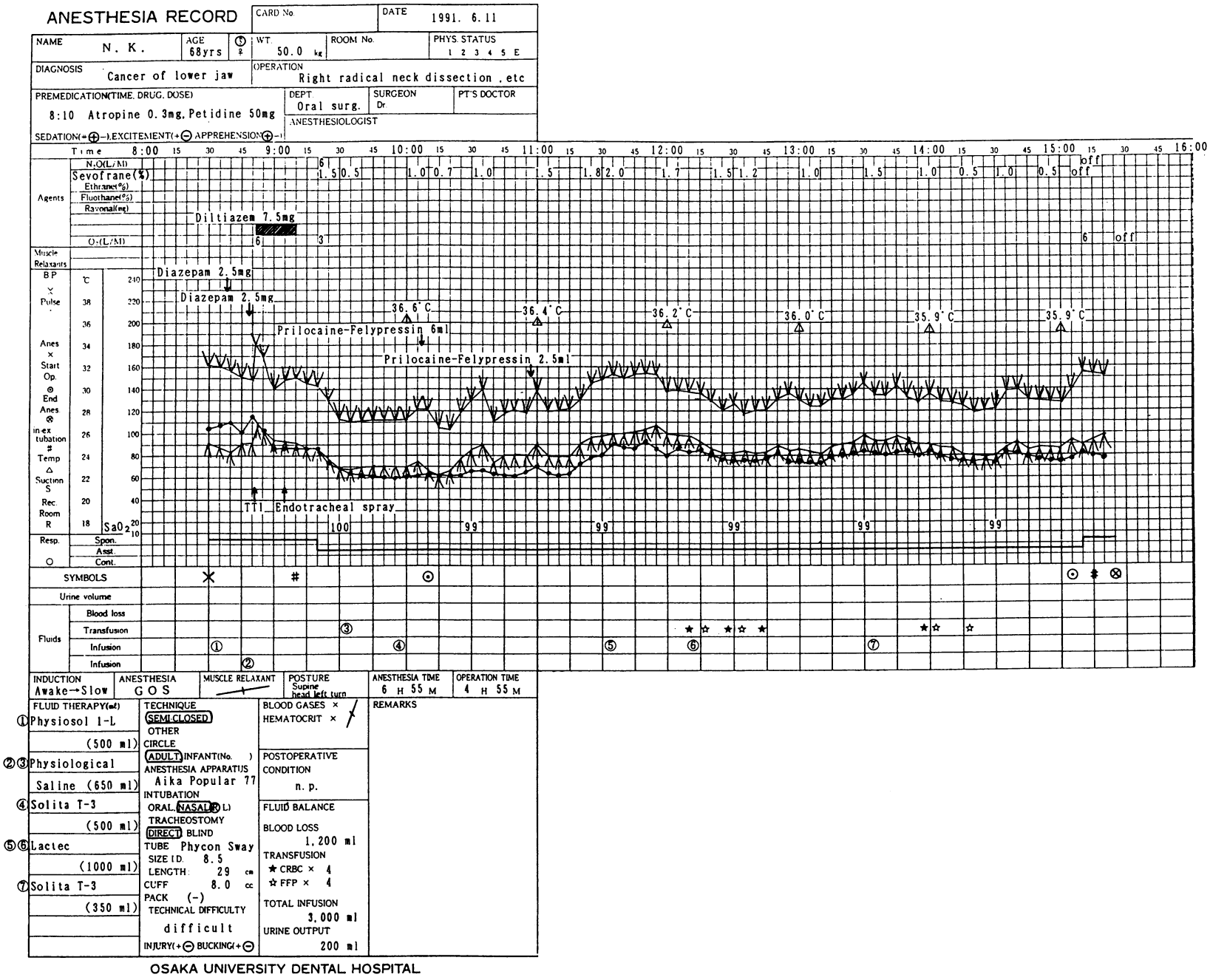

Figure 4. Anesthesia record.

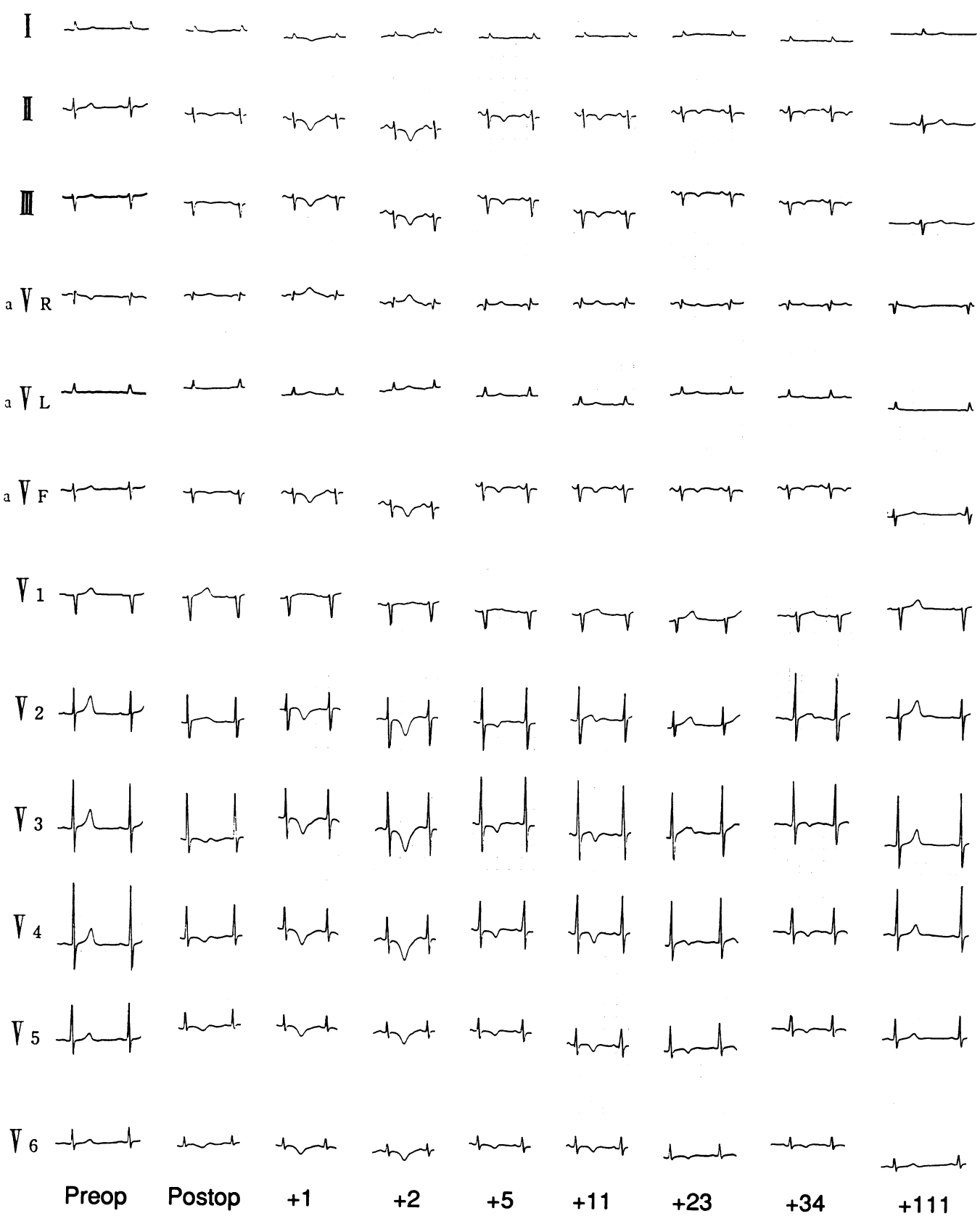

Figure 5. Electrocardiographic (ECG) changes with time. Preop, preoperative ECG tracing; postop, recording 6 hr after surgery. Additional postoperative ECG recordings are referenced by the number of days after surgery (+ number).

gram revealed mild mitral regurgitation, all other findings were within normal limits (Table 2). Throughout the entire postoperative period, neurological examinations were uniformly negative.

DISCUSSION

The ECG abnormalities in this case, the inverted T waves and prolonged QT intervals, were temporary and not associated with any clinical symptoms or findings indicative of cardiac ischemia. The possible causes for such abnormalities are ischemic heart disease (coronary artery disease)[1–5]; metabolic disturbances (potassium or calcium imbalances)[2,6]; cerebral disturbances[7–10]; drug effects[2,11]; surgical damage to the sympathetic innervation of the heart[12–17]; hypertrophic cardiomyopathy[18,19]; bradycardia[20,21]; Stokes–Adams attacks associated with complete heart block[22]; and right ventricular hypertrophy and right bundle branch block.[2]

Ischemic Heart Disease

The T-wave inversion with prolongation of the QT interval is believed to result from delayed repolarization in the epicardial region. It can be seen in patients during the early stages of an acute myocardial infarction.[1] Giant negative T waves due to myocardial ischemia are narrow, sharp, and symmetrical (the so-called "coronary type" of inversion).[2] This phenomenon can occur in transmural infarction or in subendocardial infarction.[3] In the case described here, however, there were no confirmatory cardiovascular symptoms, such as chest pain, elevations in serum enzymes, or abnormalities on the echocardiograph. Consequently, it is not likely that the patient had a myocardial infarction.

Sasa et al[4] showed that the following factors are related to a transitory myocardial infarction-like ECG pattern in postoperative patients: an advanced age (above 70 yr), preoperative ECG abnormalities, a cardiothoracic ratio (CTR) over 50%, and an intraoperative heart rate above 100 beats/min and systolic blood pressure below 100 mm Hg. These findings suggest that, in elderly patients with myocardial hypertrophy or coronary artery disease, intraoperative conditions that increase myocardial oxygen demand and/or decrease coronary perfusion might cause T-wave inversion from myocardial ischemia. In our case, intraoperative hemodynamics were rather stable (Figure 4). Nevertheless, the history of angina since the age of 50 yr and multifocal PVCs before surgery are suggestive of long-standing cardiac ischemia. In addition, the T-wave pattern was consistent with cardiac ischemia, because the waves were narrow and symmetrical. Inversion of T waves in leads I, II, III, $_aV_F$ and V_2–V_6 generally indicate anterolateral and inferior cardiac ischemia. Accordingly it appears that ischemic heart disease is of the possible causes of the T-wave inversion.

On the other hand, Kuramoto et al[5] proposed that myocardial ischemia could be caused by disseminated intravascular coagulation (DIC) or by an elevated hematocrit because of coronary insufficiency based on hypercoagulation and hyperviscosity. They described these conditions as "acute reversible myocardial infarctions." As these conditions are reversible, the ECG changes are transient and the rise in serum enzymes slight or absent. However, we do not believe that these conditions are involved in our case, because there were no findings of DIC (eg, hemorrhage or thrombocytopenia) or increased hematocrit.

After the T waves returned positive, the color flow Doppler echocardiogram revealed the presence of mild mitral regurgitation, but there were no findings of an enlarged left atrium and ventricle. The relationship between these findings and the giant negative T wave is unknown.

Metabolic Disturbances

The giant negative T wave can occur in electrolyte disorders, especially with hypokalemia, and T- and U-wave fusion might create the impression of QT prolongation.[2,6] The classical metabolic cause of an extended QT is hypocalcemia. The alterations cannot be attributed to metabolic disturbance alone, but the effect of both cerebral and ionic changes acting together may well be responsible.[2] However, there were no electrolyte abnormalities in our patient (Tables 1 and 3).

Cerebral Disturbances

Similar ECG changes can be observed in patients with cerebral vascular accidents, especially suffering from subarachnoid hemorrhage.[7–10] Usually, necropsy reveals no evidence of coronary vascular disease in such instances. Of course, there were no clinical symptoms nor signs of a cerebral vascular accident in our case.

Drug Effects

Quinidine and procainamide can alter T-wave and QT morphology,[2,11] but their effects are rarely so prominent as recorded here. These drugs were not administered to our patient.

Surgical Damage to the Sympathetic Innervation of the Heart

Postoperative T-wave inversion has been reported predominantly in patients following abdominal surgery. With

respect to oral surgery, the giant negative T waves may be seen after radical neck dissection or radical lymph-node dissection of the neck.[12–14] Hugenholtz[15] reported similar ECG abnormalities after right radical neck dissection and raised the possibility that the surgical procedures and extensive manipulation of the carotid artery destroy sympathetic nerve fibers innervating the heart. Yanowitz et al[16] and Vincent et al[17] showed that either right stellate ganglionectomy of left stellate stimulation gave similar ECG changes and suggested that such alterations were due to changes in sympathetic tone, leading to a shift in the relative duration of ventricular repolarization. The interference of the sympathetic nerves by surgery may have caused the ECG changes in our patient. The following questions remain: was the surgical procedure really extended to such a deep region as to impinge upon the sympathetic nervous system, and if so, why were there no reactions, such as severe or persistent hypotension, pronounced bradycardia, and striking ECG alterations at the moment of the surgical injury?

Hypertrophic Cardiomyopathy

The ECG pattern characterized as "giant" inversion of T waves (usually defined as 10 mm or more in depth) in the lateral precordial leads was used to define patients with a particular morphological form of hypertrophic cardiomyopathy.[18,19] Our patient does not fit this classification.

Bradycardia

Scherf[20] showed that in patients with myocardial disease, T-wave inversion became more obvious after a long R-R interval, and he suggested that the larger diastolic load might affect repolarization. Szilagyi and Solomon[21] confirmed that the alteration in the T waves from beat to beat depended on the diastolic filling period. Bradycardia, however, was not an issue in this case.

Stokes-Adams Attacks Associated with Complete Heart Block

Deep, obtuse, negative, asymmetrical, and broad T waves, which are usually maximal in leads V_2 and V_3, have been encountered in patients with Stokes–Adams attacks and loss of consciousness. Lenègre and Moreau[22] suggested that this T-wave pattern after a Stokes–Adams attack indicated specifically that ventricular fibrillation, and not standstill, was responsible for the attack. Cerebral reflexes were thought to be mainly responsible for those changes.[2] Again, this cause bears no relationship to the present case.

Right Ventricular Hypertrophy and Right Bundle Branch Block

Abnormalities in right depolarization may be involved in the production of giant negative T-wave changes encountered in conditions associated with enlargement of the right ventricle.[2] Our patient, however, did not have an enlarged heart.

CONCLUSION

In conclusion, a number of antecedent elements may be involved in the differential diagnosis of inverted or giant T waves. With regard to the present case, all but myocardial ischemia and physical damage to the sympathetic nervous system supply to the heart can be ruled out. It may be that a combination of these factors and/or some unknown etiology was causative. Although the exact reason for T-wave inversion during or after anesthesia and surgery may be difficult to uncover, its close association with myocardial ischemia and infarction demands that appropriate preventive and corrective measures be taken whenever it occurs.

REFERENCES

1. Goudsouzian N, Karamanian A: Physiology for the Anesthesiologist, 2nd ed. Norwalk, CT, Appleton-Century-Crofts, 1984:39.

2. Jacobson D, Schrire V: Giant T wave inversion. Br Heart J 1966;28:768–775.

3. Conti CR: Introduction to Clinical Cardiology. New York, Raven Press, 1991:120.

4. Sasa H, Tsuboi H, Sone T, Minakuchi K, Okumura Y, Kimura T, Kanamori I, Hachisuka K, Yamaguchi A, Miyaji M: Clinical significance of transitory myocardial infarction-like electrocardiographic pattern in postoperative patients. Heart 1983;15:669–678.

5. Kuramoto K, Matsushita S, Shimada H, Mifune J, Sakai M, Iwasaki T, Maki N, Murakami M: The cause of reversible myocardial infarction-like electrocardiographic pattern. Intern Med (Japan) 1979;43:562–568.

6. Aber CP, Jones EW: Steroid-induced T wave changes in heart block. Br Heart J 1965;27:56–61.

7. Cropp GJ, Manning GW: Electrocardiographic changes simulating myocardial ischemia and infarction associated with spontaneous intracranial hemorrhage. Circulation 1960;22: 25–38.

8. Hugenholtz PG: Electrocardiographic abnormalities in cerebral disorders. Report of six cases and review of the literature. Am Heart J 1962;63:451–461.

9. Fentz V, Gormsen J: Electrocardiographic patterns in patients with cerebrovascular accidents. Circulation 1962;25:22–28.

10. Kreus KE, Kemilä SJ, Takala JK: Electrocardiographic changes in cerebrovascular accidents. Acta Med Scand 1969; 185:327–334.

11. Fox TT, Weaver J, March HW: On the mechanism of the arrhythmias in aberrant atrio-ventricular conduction (Wolff–Parkinson–White). Am Heart J 1952;43:507–520.

12. Ban I, Matsuura H, Hirose I, Sakuda S, Akita R, Shono S: The giant negative T wave following surgery. J Jpn Dent Soc Anesthesiol 1977;5:341–351.

13. Umemura A, Shida T, Adachi H, Ueda Y, Masui Y, Shirasu R, Hamada Y, Niki H, Okano H: A giant negative T wave during general anesthesia. J Jpn Dent Soc Anesthesiol 1984;12:159–166.

14. Fukushima T, Adachi H, Shida T, Ueda Y, Umemura A, Niki H: Pre- and postoperative giant negative T wave. J Clin Anesth 1985;9:869–871.

15. Hugenholtz PG: Electrocardiographic changes typical for central nervous system disease after right radical neck dissection. Am Heart J 1967;74:438–441.

16. Yanowitz F, Preston JB, Abildskov JA: Functional distribution of right and left stellate innervation to the ventricles. Circ Res 1966;18:416–419.

17. Vincent GM, Abildskov JA, Burgess MJ: Q-T interval syndromes. Prog Cardiovasc Dis 1974;16:523–530.

18. Yamaguchi H, Ishimura T, Nishijima S: Hypertrophic nonobstructive cardiomyopathy with giant negative T waves (apical hypertrophy): Ventriculographic and echocardiographic features in 30 patients. Am J Cardiol 1979;44:401–412.

19. Fernando A, Petros N, James S, Shaughan D, Robert L, William JM: Clinical significance of giant negative T waves in hypertrophic cardiomyopathy. J Am Coll Cardiol 1990; 15:965–971.

20. Scherf D: Alterations in the form of the T waves with changes in heart rate. Am Heart J 1944;28:332–347.

21. Szilagyi N, Solomon SL: Variation in the form of the T wave in a case of partial heart block. Am Heart J 1959; 58:637–644.

22. Lenègre J, Moreau P: Le bloc auriculo-ventriculaire complet. Ses causes et ses lésions. Soc Med Hop Paris 1962; 113:767–770.