Methemoglobinemia should be considered a possibility in any cyanosis without apparent cardiovascular or pulmonary etiology, especially if the cyanosis appears out of proportion to other signs and symptoms of hypoxia. Methemoglobin is a dark pigment, and the color of blood with high levels will appear chocolate brown and will not change upon agitation with room air. As in our case, Pa_{O_2} may be high, even though the blood's total O_2-carrying capacity is decreased markedly causing tissue hypoxia.

The lethal blood level of methemoglobin is probably between 70 and 90%.[6–8] One case proved to be fatal at a level of 74% and a hemoglobin concentration of 8.7 g/dl.[9] Undoubtedly, the lethal level is dependent on other factors such as duration of methemoglobinemia before treatment, the general physical condition of the patient, and the hemoglobin concentration. The amount of functioning hemoglobin present in g/dl is figured by subtracting the nonfunctional methemoglobin from the total hemoglobin concentration. We call this the "effective hemoglobin concentration" and believe it gives a more accurate indication of the severity of the problem than does a simple methemoglobin percentage. By this method, the patient mentioned above had an "effective hemoglobin concentration" of 2.3 g/dl (8.7 g/dl − [0.74] [8.7 g/dl]), and our patient had essentially the same level at 2.4 g/dl. The fact that this low a level of functioning hemoglobin was sustained without obvious sequalae of hypoxia is fortunate and is probably due to the rapidity with which the situation was reversed and to the high FI_{O_2} that the infant was given throughout the episode.

Specific therapy in methemoglobinemia is aimed at restoring the O_2-carrying capacity of the blood. Methylene blue in an iv dose of 1–2 mg/kg acts rapidly *in vivo* as an electron carrier in the reduction of methemoglobin to normal hemoglobin. Blood transfusion is another rapid and simple measure that may be necessary to provide additional functioning hemoglobin to the bloodstream. As in our case, exchange transfusion may be considered when high levels of methemoglobin are present in an infant.[10]

Acute methemoglobinemia can be a serious, even life-threatening, reaction to the use of benzocaine-containing products in infants and small children. It can be diagnosed with relative certainty without time-consuming laboratory studies, and, in most cases, can be effectively treated with methylene blue and/or blood transfusion. Even if methemoglobin levels are very high, prognosis appears excellent if the problem is recognized early and therapy begun quickly.

References

1. Goluboff N, MacFayen DJ: Methemoglobinemia in infant, associated with application of tar–benzocaine ointment. J Pediatr 47:222–226, 1955
2. Hughes JR: Infantile methemoglobinemia due to benzocaine suppository. J Pediatr 66:797–799, 1965
3. Sherman JM: Methemoglobinemia owing to rectal-probe lubrication. Am J Dis Child 133:439, 1979
4. Steinberg JB, Zepernick, RG: Methemoglobinemia during anesthesia. J Pediatr 61:885–886, 1962
5. Nathan DG, Oski, FA: Hematology of Infancy and Childhood. Philadelphia, WB Saunders, 1974, pp 381, 383
6. Miller DR, Pearson HA: *In* Smith's Blood Diseases of Infancy and Childhood. St. Louis, CV Mosby, 1978, p. 365
7. Schaffer AJ, Avery ME: Diseases of the Newborn. Philadelphia, WB Saunders, 1977, p 627
8. Finch CA: Medical progress; Methemoglobinemia and sulfhemoglobinemia. N Engl J Med 239:470–478, 1948
9. Bucklin R, Myint MK: Fatal methemoglobinemia due to well water nitrates. Ann Intern Med 52:703–705, 1960
10. Rudolph AM: Pediatrics, New York, Appleton-Century-Crofts, 1977, p 1175

Anesthesiology
59:464–467, 1983

Temperature-related T-Wave Changes during Thoracotomy

JUDD WESTOVER, M.D.,* AND LAWRENCE J. SAIDMAN, M.D.†

Electrocardiographic changes due to whole body hypothermia[1,2] and hyperthermia[3,4] are well known. Much less appreciated are electrocardiographic (EKG) changes in animals due to heating and cooling of the epicardial surface of the heart.[5–8] The only possible clinical paradigm of these latter animal experiments is to bathe the heart

* Clinical Instructor.
† Professor and Chairman, Department of Anesthesiology.
Received from the Department of Anesthesiology, University of California Medical Center, San Diego, California. Accepted for publication April 14, 1983.

Address reprint requests to Dr. Saidman. Department of Anesthesiology, University Hospital. 22S West Dickinson Street, H-770, San Diego, California 92103.

Key words. Heart: electrocardiography. Temperature: heating. Anesthesia: thoracic.

0003-3022/83/1100/0464 $01.00 © The American Society of Anesthesiologists, Inc.

Downloaded from http://asa2.silverchair.com/anesthesiology/article-pdf/59/5/464/626248/0000542-198311000-00020.pdf by guest on 21 September 2023

in either warm or cold solution as would be done during irrigation of an open-chest saline irrigation procedure. In this report we describe a patient who had giant T-waves develop on the EKG following irrigation of an open-chest with warm saline.

Report of a Case

A previously healthy 36-year-old man was admitted to an outlying hospital following a high-speed motor vehicle accident. There had been no loss of consciousness, and the patient was stable at the scene, with an arterial blood pressure of 130/50 mmHg and a heart rate of 72 beats/min. An upright chest roentgenogram revealed multiple rib fractures and a slightly widened mediastinum, and an aortogram revealed a circumferential tear of the descending aorta just distal to the left subclavian artery. The patient was evacuated by helicopter to our hospital for surgical repair of his thoracic aorta. During the 30-min flight, he received propranolol 7 mg iv in 0.5-mg increments to decrease myocardial contractility and blood pressure and thereby decrease stress upon the torn aorta.

On arrival at our hospital, he was alert and stable. Vital signs and laboratory values were within normal limits. Anesthesia consisted of halothane and oxygen supplemented with pancuronium and fentanyl. The thoracic aorta was repaired with a Dacron graft via a left thoracotomy, during which unilateral ventilation of the right lung was performed. A Gott shunt was placed temporarily during the repair. The patient remained stable during the course of the surgical repair, although esophageal temperature fell to 34.5° C.

Prior to chest closure, the surgeon requested warm saline to irrigate the operative field. One liter of saline, which subsequently was demonstrated to have a temperature of 44° C, was placed into the open chest cavity. Within seconds a dramatic increase in the T-wave amplitude was noticed (fig. 1). There were no accompanying hemodynamic changes or arrhythmias. With an $F_{I_{O_2}}$ of 1.0, *p*Ha was 7.40, Pa_{O_2} 347 mmHg, Pa_{CO_2} 30 mmHg, base excess 5 mEq/l, and potassium 4.8 mEq/l. The surgeon was informed of the EKG changes, the saline was aspirated from the chest cavity, and the T-wave changes resolved within minutes. Another liter of warm saline, at a temperature similar to that previously used, was placed in the chest cavity and was followed by a recurrence of the T-wave changes. These too resolved shortly after the saline was removed from the chest.

Following closure of the chest, the patient was transferred to the trauma unit, and after extubation of the trachea the next day, went on to a rapid and uneventful recovery. A postoperative 12-lead EKG revealed nonspecific ST-T wave abnormalities, and a CPK level was 2,260 IU with 2% MB fraction, which was felt to be consistent with a cardiac contusion. Subsequent EKGs have been normal.

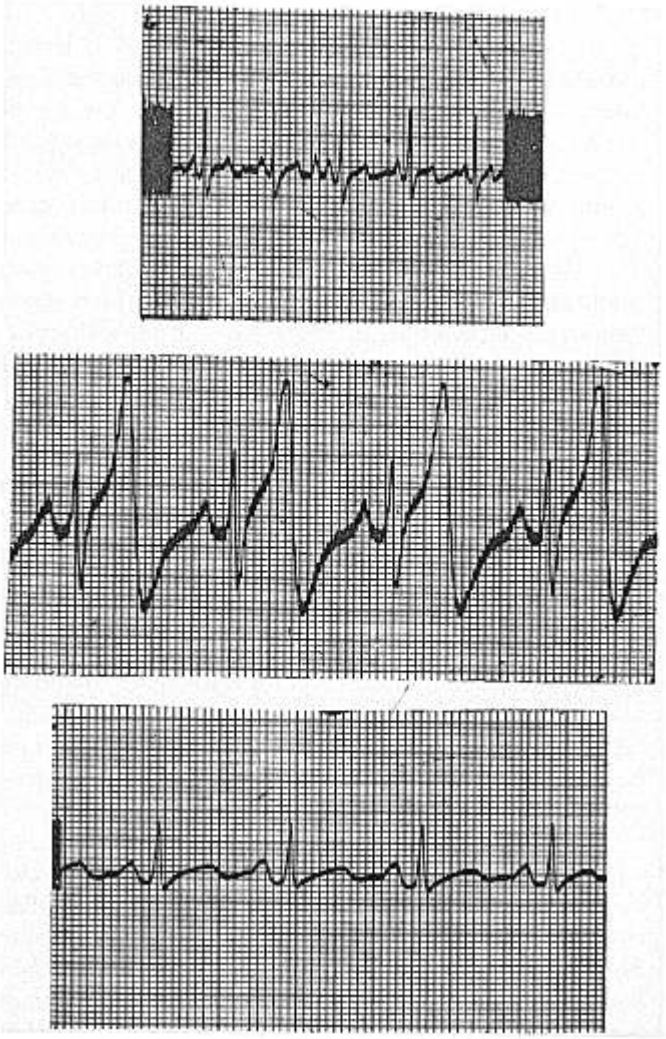

FIG. 1. Lead II EKG (*A, top*) preoperative, paper speed 10 mm/s, (*B, middle*) intraoperative in left lateral decubitus position, chest open following warm saline irrigation, paper speed 25 mm/s, and (*C, bottom*) 20 min after 1*B* with chest closed, paper speed 10 mm/s. The change in the QRS from 1*A* to 1*B* had occurred prior to the saline irrigation and was associated with the lateral decubitus position and the open chest.

Discussion

Alterations in core temperature can affect cardiac electrical activity. Hyperpyrexia, in the absence of electrolyte or acid base alteration, has been associated with sinus tachycardia, prolongation of the QT interval, nonspecific ST-T wave changes, and occasional ST segment depression.[3,4] The dysrhythmias seen during episodes of anesthetic-associated malignant hyperthermia are thought to be due primarily to increased sympathetic tone, hypoxia, hypercapnea, and metabolic/respiratory acidosis.[9] Hypothermia produces sinus bradycardia, PR and QT prolongation, and T-wave inversion.[2] Atrial and ventricular dysrhythmias appear at 30° C, while temperatures below 28° C produce ventricular fibrillation.[1] The hypothermic J-wave (Osborn wave, camel hump), an elevation of the J-segment at the QRS-ST junction, usually appears at 29° C[2] but is nonspecific and occurs with subarachnoid hemorrhage and in normal subjects.[10] Our patient was mildly hypothermic (esophageal temperature of 34.5° C) but had demonstrated no EKG abnormalities. Thus, when normal saline at a temperature of 44° C was placed in his chest cavity, in contact with the epicardial surface of the heart, an endocardial to epicardial temperature gradient of 9–10° C was established. The lead II EKG promptly showed new tall positive T-waves that resolved on removal of the warm saline.

Downloaded from http://asa2.silverchair.com/anesthesiology/article-pdf/59/5/464/626248/0000542-198311000-00020.pdf by guest on 21 September 2023

Electrocardiographic effects of localized temperature changes or temperature gradients were studied by several investigators in the early years of cardiac electrophysiology. There are many reports of precordial T-wave inversions following chest wall cooling with ice packs[7,11] and with the ingestion of ice water.[12] No correlation was found between plasma potassium levels and T-wave changes elicited by chest wall cooling.[13] Hellerstein and Liebow studied the effects of heating and cooling canine endocardial and epicardial surfaces with localized applications of saline, while recording EKGs from both intracavitary and epicardial electrodes.[5] Either endocardial cooling with injections of 8° C saline or epicardial heating with 45° C saline produced a tall positive T-wave in the epicardial leads. Conversely, endocardial heating or epicardial cooling were associated with broad, negative T-waves. Ackman *et al.* perfused the pericardial sac of dogs with saline at various temperatures and produced changes similar to those described above over the entire epicardial surface.[6] These changes occurred immediately, and recovery to baseline required 10 s to 2 min. No dysrhythmias were noted except for transient bradycardia when cold saline was injected into the right ventricle. This latter phenomenon recently has been reported in humans during thermodilution cardiac output measurement.[14]

The genesis of these T-wave changes is as follows: Ventricular depolarization spreads from the endocardium to the epicardium and results in the normally positive QRS complexes seen in the inferior and lateral precordial leads. Repolarization spreads in the opposite direction, from the epicardial to the endocardial surface, and the resulting normal T-wave is also positive in those leads where the QRS is positive. The T-wave corresponds with phase 3 of repolarization and is dependent on potassium flowing out of the cell along an electrochemical gradient.[15] This potassium channel is temperature sensitive, and heating an area of the myocardium will increase the intensity and rate of repolarization.[16] Electrodes overlying a heated epicardial area will see a relatively more positive field during this time and thus show a more positive T-wave, as was demonstrated in our patient. On the other hand, heating the endocardium or cooling the epicardium will enhance endocardial repolarization relative to that of the epicardium. Repolarization will spread from the relatively warmed endocardial surface and result in a negative T-wave in leads facing the relatively cool surface of the epicardium.

Myocardial temperature gradients also could occur during rapid iv administration of cold blood or fluid, in addition to the direct application of hot or cold materials to the epicardial surface. Altered repolarization caused by temperature gradients may increase the opportunity for reentrant tachyarrhythmias. Ventricular extrasystoles, ventricular tachycardia, and ventricular fibrillation have occurred following transfusion of cold blood, although the most likely causes in these cases were core hypothermia, or possibly hyperkalemia or citrate intoxication.[17]

The differential diagnosis for the sudden appearance of tall T-waves includes some serious entities, such as acute hyperkalemia, hypokalemia with metabolic alkalosis (giant U-waves), cerebral vascular accident, acute hemopericardium, coronary insufficiency, and the hyperacute phase of myocardial infarction.[18] Although some of these are difficult to rule out intraoperatively, in our patient we were able to inspect the heart directly and to eliminate metabolic causes with blood gas and electrolyte analysis. This young man was at little risk for coronary insufficiency, although his elevated CPK MB fraction and nonspecific ST-T wave changes after operation do not rule out subendocardial myocardial infarction. The temporal relationship between the appearance of these tall T-waves and the warm saline irrigation on two separate occasions make the cause in this case fairly obvious. Obviously, the temperature of irrigation fluid should be checked before administration, to avoid this puzzling and possibly dangerous entity.

References

1. Trevino A, Razi B, Bellen BM: The characteristic EKG of accidental hypothermia. Arch Intern Med 127:470–473, 1971
2. Osborn JJ: Experimental hypothermia: respiratory and blood pH changes in relation to cardiac function. Am J Physiol 175:389–398, 1953
3. Kew MC, Tucker RB, Bersohn I: The heart in heatstroke. Am Heart J 77:324–335, 1969
4. Costrini AM, Pitt HA, Gustafson AB: Cardiovascular and metabolic manifestations of heat stroke and severe heat exhaustion. Am J Med 66:296–302, 1979
5. Hellerstein HK, Liebow IM: Factors influencing the T wave of the EKG. An experimental study employing intraventricular and extraventricular leads. Am Heart J 39:35–55, 1950
6. Akman LC, Silber EN, Miller AJ, Katz LN: Effects of heating and cooling the entire epicardial surface. Am J Physiol 159:492–498, 1949
7. Smith FM: Some observations on the effect of heat and cold on the ventricle and the T deflection of the electrocardiogram. Heart 10:391–397, 1923
8. Rogers MC, Abildskov MD, Preston JB: Cardiac effects of stimulation and block of the stellate ganglion. ANESTHESIOLOGY 39:525–533, 1973
9. Gronert GA: Malignant hyperthermia. ANESTHESIOLOGY 53:395–423, 1980
10. Abbott JA, Cheitlin MD: The nonspecific "camel hump" sign. JAMA 235:413–414, 1976
11. Ashman R, Ferguson FP, Gremillion AT: The effect of cycle length changes on the form and amplitude of the T deflection of the electrocardiogram. Am J Physiol 143:453–461, 1945
12. Dowling CV, Hellerstein HK: Factors influencing T wave of electrocardiogram: Effects of drinking iced water. Am Heart J 41:58–77, 1951
13. Kaminer B, Bernstein RE: Electrocardiographic and plasma potassium responses elicited on cooling the chest wall of man. Circulation 15:559–567, 1957

Downloaded from http://asa2.silverchair.com/anesthesiology/article-pdf/59/5/464/626248/0000542-198311000-00020.pdf by guest on 21 September 2023

14. Nisikawa T, Dohi S: Slowing of heart rate during cardiac output measurement by thermodilution. ANESTHESIOLOGY 57:538–539, 1982
15. Berne RM, Levy MN: Cardiovascular Physiology, St. Louis, CV Mosby, 1981, pp 5–22
16. Surawicz B: The pathogenesis and clinical significance of primary T wave abnormalities, Advances in Electrocardiography. Edited by Schlant RD, Hurst JW. New York, Grune and Stratton, 1972, pp 377–421
17. Boyan CP, Howland WS: Cardiac arrest and temperature of bank blood. JAMA 183:58–60, 1961
18. Goldberger AL: Myocardial Infarction: Electrocardiographic Differential Diagnosis. St. Louis, CV Mosby, 1979, pp 186–194

Anesthesiology
59:467–469, 1983

Transient Large Upright T-Wave on the Electrocardiogram during Multiple Monitored Electroconvulsive Therapy

GWENDOLYN GRAYBAR, M.D.,* JOHN GOETHE, M.D.,† THOMAS LEVY, M.D.,‡ JOHN PHILLIPS, M.D.,§ JOHN YOUNGBERG, M.D.,* DONALD SMITH, M.D.¶

Large T-waves on the electrocardiogram (ECG) frequently are associated with hyperkalemia and can be a manifestation of intracranial injury.[1] Although many ECG alterations accompany electroconvulsive therapy (ECT),[2–4] large upright T-waves have not been reported.

REPORT OF A CASE

A 42-year-old woman was admitted with the diagnosis of unipolar affective disorder (major depressive illness). Her weight had decreased from 73 kg to 31 kg. An extensive medical and neuropsychiatric workup revealed no organic basis for her depression or weight loss. She had been hospitalized for a similar episode 4 months prior to the present admission. Initial 12-lead ECGs were interpreted as within normal limits. An ECG taken prior to the fifth ECT session revealed a new finding of mild T doming, suggesting hyperventilation and/or drug effect.

Multiple Monitored Electroconvulsive Therapy (MMECT) is a procedure for administering electroconvulsive therapy, which allows multiple treatments to be given in a single therapy session. The voltage is varied automatically to provide a constant current of 800 milliamps through a bipolar square wave pulse. Typically, the maximum energy administered during MMECT is 69 joules; the average energy is 17.5 joules, compared with an average of 50–100 joules in standard ECT.

During each session, anesthesia was induced with thiopental 150–350 mg and *d*-tubocurarine 3 mg, followed by succinylcholine 100–120 mg iv to facilitate endotracheal intubation. Nitrous oxide 60% and succinylcholine 0.2% as a continuous iv infusion were given for maintenance of anesthesia. A peripheral nerve stimulator was utilized to insure maximal relaxation prior to each stimulus. Hyperventilation was instituted a minute prior to each stimulus. Large, upright T-waves were noticed first during the fifth session. Simultaneous ECG and EEG tracings returned rapidly to normal after the seizure and were not associated with apparent hemodynamic compromise (fig. 1). Therefore we elected to follow her next treatment session with a twelve lead ECG, as well as serial potassium and CPK levels (fig. 2).

DISCUSSION

ECG findings in patients with neurologic problems are similar to those described in this report. Burch and Phillips[1] reported a series of 55 patients with a variety of central nervous system conditions including tumors, subarachnoid hemorrhages, and intracerebral hemorrhages, with ECG changes believed to be secondary to their CNS lesions. Seventeen of the 55 patients showed the more classic ECG findings of prolonged QT and large inverted T-waves. Thirty-eight had prominent upright T-waves, prolonged QT, prominent U-waves and TU fusion. The QRS complex was unchanged. Greenspahn's group[5] reported a patient with deep T-wave inversions simulating acute nontransmural myocardial infarction following cerebral concussion alone. The EEG, CT scan, enzymes, echocardiogram, and technetium pyrophosphate scan in their patient revealed neither CNS nor cardiac pathology.

An imbalance in sympathetic tone to the heart is believed to be the mediating mechanism in ECG findings with CNS lesions.[6] Yanowitz *et al.*[7] found in dogs that right stellate ganglionectomy or left stellate stimulation would produce prolonged QT intervals and increased amplitude of T-waves. Left stellate ganglionectomy or right stellate stimulation produced increased T-wave negativity without measurable change in the QT interval. In contrast, Rogers' *et al.*[8] showed in cats right or left stellate ganglion block resulted in diphasic T-waves of lower amplitude. Stimulation of the right stellate ganglion in the cat increased the amplitude of the T-waves, while stimulation of the left stellate produced deeply inverted

* Associate Professor, Department of Anesthesiology.
† Associate Professor, Department of Psychiatry.
‡ Assistant Professor, Department of Medicine.
§ Professor, Department of Medicine.
¶ Assistant Professor, Department of Anesthesiology.

Received from the Departments of Anesthesiology, Psychiatry, and Medicine, Tulane University School of Medicine, New Orleans, Louisiana, Accepted for publication April 15, 1983.

Address reprint requests to Dr. Graybar: Department of Anesthesiology, Tulane Medical Center, 1430 Tulane Avenue, New Orleans, Louisiana 70112.

Key words: Anesthesia: electroshock. Heart: enlarged T-waves.

0003-3022/83/1100/0467 $00.95 © The American Society of Anesthesiologists, Inc.

Downloaded from http://asa2.silverchair.com/anesthesiology/article-pdf/59/5/464/626248/0000542-198311000-00020.pdf by guest on 21 September 2023