The patient had classic ECG changes of hypothermia (sinus bradycardia, prolonged PR interval, prolonged QT interval, and Osborn waves). These changes occurred in hypothermia resulting from sepsis, without exposure being a factor. Documentation of Osborn waves in this clinical setting supports the theory that they result as a direct consequence of myocardial cooling.

Because of the usefulness of induced hypothermia in neurologic and cardiovascular surgery, and because hypothermia due to exposure is common, the ECG changes (including sinus bradycardia, prolonged PR interval, prolonged QT interval, and Osborn waves) have been carefully described and have been the subject of intense investigation in both human and animal studies. The ECGs of patients suffering from accidental hypothermia were first published by Tomaszewski in 1938. He noted a change in the ST segment near the R wave resembling a "domed T wave." This elevation of the J point with accompanying positive deflection has been termed "Osborn wave" as a result of Osborn's investigations in 1953 into the mechanism of the change. The term J-wave has also been employed. However, since proof is lacking as to the mechanism, and since, as previously pointed out, confusion can occur over J-wave and J point, we have elected to use the eponym Osborn wave.

Significantly, however, most of the reports and studies involve either induced hypothermia or cases of exposure. We present here a case of fatal hypothermia, with characteristic ECG changes, as a consequence of sepsis.

**CASE REPORT**

A 63-year-old black man was admitted after being found comatose at home.

On physical examination he was comatose with rectal temperature 34.4° C; blood pressure, 140/100; pulse rate, 100 beats per minute. Scleral icterus and conjunctival edema were noted. There were rales in the bases of both lung fields. A grade 3/6 systolic crescendo-decrescendo murmur was heard at the left lower sternal border and S₃ and S₄ gallop sounds were noted. No focal neurologic abnormalities were appreciated. The admission ECG (Fig 1) revealed sinus rhythm with a rate of 65, axis of +00, PR .20 sec, QRS .08 sec, and QT .40 sec.

The etiology of his coma was presumed hepatic encephalopathy, and he was treated with lactulose and protein restriction. Blood cultures grew a Clostridial species and an Enterococcus sensitive to ampicillin and tobramycin with which he was treated. No source for infection was found. By the seventh hospital day, he was normothermic, and he continued to improve, becoming alert by the 19th hospital day. However, on the 21st hospital day, the patient again became hypothermic and unresponsive. Evaluation revealed no definite source for infection. Cultures were obtained, and the patient was empirically treated with oxacillin and tobramycin. Temperatures remained below 34.4° C despite a heating blanket. On the 21st hospital day, the serum potassium level was 3.5 mEq/L, calcium value, 8.4 mg/100 ml, and albumin, 2.6 g/100 ml. An ECG on this day (Fig 2) revealed sinus bradycardia with a rate of 47, axis +30, PR .24, QRS .07, QT .60, and Osborn waves in V₁ and V₄. He became progressively bradycardic and died later the same day.

**DISCUSSION**

An extensive review of the literature yielded only one prior case report with hypothermic ECG changes not related to exposure. Thompson et al present a case of a 20-year-old man with fatal adult respiratory distress syndrome and with ECG changes developing over a two-day period.

As the case presented here and the case reported by Thompson et al illustrate, ECG interval prolongation and the Osborn wave occur in hypothermia resulting from an "internal" disease state without "external" environmental cooling. Indeed, in the case we report, a heating blanket was employed for two days prior to death. Significant in a discussion of the origin of the Osborn wave is its occurrence in this clinical setting. Although prolonged intervals with cold are supported and partially explained by previous electrophysiologic

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**Figure 1.** Admission ECG showing sinus rhythm, rate 65, axis +00, PR .20 sec, QRS .08 sec, and QT .40 sec.
studies,5-8 the mechanism of the Osborn wave is still unknown and very much a matter of debate. Gould and Reddy9 have shown that cold isotonic saline applied locally to the His bundle causes delayed conduction. In earlier studies, hypothermia was shown to decrease resting membrane potential and decrease the action potential, slowing depolarization and repolarization, in preparations of animal heart muscle.5-7 Electron microscopic studies of rat myocardium have shown widening of the plasma membrane at the intercalated discs with lowered temperature, possibly resulting in less negative transmembrane potentials.8

The apparent genuine occurrence of permanent Osborn waves in a patient with subarachnoid hemorrhage without hypothermia has prompted the suggestion that hypothalamic or neurogenic factors may be an etiologic factor.9-10 Significantly, the occurrence of Osborn waves in a patient hypothermic from sepsis adds a point of evidence against a neurogenic effect and in favor of a direct effect of cooling. The basic hypothalamic neurochemical processes in hypothermia from sepsis are the opposite of those in exposure or induced hypothermia. In sepsis with hypothermia, the hypothalamic set point for thermoregulation is moved downward, and there is a cooling effect by peripheral vasodilatation and decreased metabolic rate. In exposure, however, the set point is unchanged, but the hypothalamic drive tends to cause vasoconstriction and shivering with an increased metabolic rate.11

It is, therefore, important to note that in spite of the different mechanisms involved, the direction of the temperature gradient is the same in exposure as in sepsis, with cooling occurring from the surface inward.

Correspondingly, the ECG changes are identical. This is one more piece of evidence toward a fuller understanding of the mechanism of the Osborn wave.

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Figure 2. Electrocardiogram on day of death showing sinus bradycardia with rate 47, axis +30, PR .24 sec, QRS .07 sec, QT .60 sec, and Osborn waves (marked with arrows). Rectal temperature was below 34.4° C.