ORIGINAL INVESTIGATION

Osborn (J) Wave Appearance on the Electrocardiogram in Relation to Potassium Transfer and Myocardial Metabolism during Hypothermia

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ABSTRACT

The genesis of the J wave during hypothermia has been attributed to injury current, delayed ventricular depolarization and early repolarization, tissue anoxia, and acidosis. To our knowledge, no studies have addressed the appearance of the J wave in relation to the myocardial K^+ transfer and metabolism during hypothermia. Dogs ($n = 9$) were progressively cooled, blood samples were taken from the aorta and coronary sinus, and myocardial tissue samples were obtained for adenosine triphosphate (ATP), creatine phosphate (CP), and glycolytic intermediate determination. In every instance, the appearance of the J wave was preceded by a net loss of K^+ from the myocardium. In one dog, there was no myocardial K^+ loss and the J wave was absent. The J wave appeared when the esophageal temperature was between 27° and 24°C (26.6 ± 0.73°C). At that temperature, the animals were hypotensive and bradycardic, but arterial oxygen partial pressure, carbon dioxide partial pressure, and pH were within the physiologic range at that temperature. The myocardial ATP and CP from the hypothermic dogs was lower compared with the value obtained from dogs at 37°C ($p < .025$ and $p < .005$, respectively). The levels of the glycolytic intermediates, fructose-1,6-diphosphate, dihydroxyacetone phosphate, and pyruvate, were lower and the level of lactate was higher compared with those from the normothermic dogs (not significant; $p < .007$, $p < .02$, $p < .001$, respectively). These findings suggest that the appearance of the J wave on electrocardiography during cooling is a result of depression of the metabolic process concerned with maintenance of the partition of ions across the cell membrane, as evidenced by decreased myocardial energy content and K^+ loss during the hypothermic state.

Key Words: Osborn J wave, potassium, adenosine triphosphate

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The characteristic deflection located between the end of the QRS and the beginning of the ST segment is usually referred to as the J wave (Osborn wave). It appears when the body temperature drops to approximately 25°C.[1] The appearance of the J wave was first described by Tomaszewski in 1938[2] and was induced experimentally in dogs by Gosse-Brockhoff and Schoelel in 1943.[3] The J-wave appearance on the electrocardiogram (ECG) has been attributed to multiple events and conditions, which are described in the comprehensive article by Gussak and colleagues.[4] These authors present a historical review of the clinical and experimental data regarding J wave and address possible mechanisms for its development. To our knowledge, no studies have addressed the appearance of the J wave in relation to the myocardial potassium transfer or the myocardial energy state during hypothermia.

MATERIAL AND METHODS

Nine mongrel dogs of both sexes were anesthetized intravenously with sodium thiamylal (Surital) 35 mg/kg. Supplemental anesthesia was given as required during the experimental period. After induction of anesthesia, the animals were intubated and ventilated with the Harvard respiratory pump (Harvard Apparatus Co. Inc., Holliston, MA), using ambient air. The left carotid artery and external jugular vein, femoral artery, and femoral vein were surgically exposed for catheterization. Under fluoroscopic guidance, heparinized catheters were placed in the coronary sinus and at the aortic root through the external jugular vein and carotid artery, respectively. The femoral artery and vein were cannulated with large-bore polyethylene catheters. Heparin (at 6 mg/kg initial doses and thereafter at 3 mg/kg doses) was given every 2 hours intravenously. The dogs were cooled in an ice bath. To speed the cooling process, blood from the animal was circulated extracorporally through a half-inch stainless steel coil immersed in an ice water bath (at approximately 4°C) using a rotary pump from the femoral artery to the femoral vein. Core temperatures were monitored with esophageal and rectal probes connected to the telethermometers (Model 46 TVC, Yellow Springs Instrument Co., Yellow Springs, OH).

Arterial blood pressure and ECG were monitored constantly on an Electronics for Medicine DR-8 recorder (White Plains, NY). The aortic catheter was connected to a Statham pressure transducer (Model P-23Db, Statham-Gould, Oxnard, CA). A standard ECG with the addition of one precordial lead was used to record cardiac electrical activity and the appearance of the J wave. The appearance of the J wave was determined by the agreement of three observers using the clearest ECG channel.

The dogs were cooled slowly over a period of several hours to temperatures as low as 18°C. Heart rate, arterial pressure, core temperatures, blood gases, pH, and potassium and hemoglobin concentrations were measured and recorded periodically during the cooling process. Paired blood samples from the aorta and coronary sinus were obtained and analyzed immediately with a calibrated Copenhagen ABL4 Radiometer (Copenhagen, Denmark). With each measurement, the presence or absence of the J wave was noted and recorded on ECG. The net myocardial K^+ loss was determined by the difference between the aortic root blood potassium (Ao $[K^+]$) and the coronary blood potassium concentration (CS $[K^+]$).

After the appearance of the J wave, several blood samples were obtained and ECG recordings were made over the next 15 to 20 minutes. Myocardial tissue samples were then obtained through a left thoracotomy with a large core biopsy tool that had been precooled in liquid nitrogen. The samples were placed in liquid nitrogen and stored at −70°C.

Two additional anesthetized dogs were operated on without undergoing the cooling process, and myocardial samples were obtained and stored as described above. To avoid sacrificing more dogs, adenosine triphosphate (ATP), creatine phosphate, and glycolytic intermediate myocardial contents levels were comparable to data from concurrent experiments in anesthetized sham-operated normothermic dogs ($n = 4$). The combined data from these dogs served as controls. The myocardial tissue was homogenized while still frozen in 6% precooled, perchloric acid (4°C). The supernatant was used to measure ATP, creatine phosphate (CP), fructose-1,6-diphosphate, dihydroxyacetone phosphate (DHA-P), pyruvate, and lactate levels.

The ATP and CP were determined with the method described by Lamprecht and Stein,[5] and the lactate and pyruvate were determined by the method of Marbach and Weil.[6] Fructose-1,6-diphosphate and DHA-P were assessed by the method of Bucher and Hohorst.[7] The results are expressed as mean ± standard error, and statistics were performed with the two-tailed Student's *t*-test for dependence and independence as appropriate. A value of $p < .05$ was considered to be significant.

RESULTS

J waves were induced in eight of the nine hypothermic dogs (Figure 1). The J wave appeared at esophageal temperatures between 27° and 24°C (26.6° ± 0.73°C). We observed a net potassium loss from the myocardium when the J wave became apparent. The average difference (Ao $[K^+]$ − CS $[K^+]$) when the J wave appeared was −0.325 ± 0.092 in mEq/L. The one dog that did not develop a J wave was cooled to a temperature of 18°C before it fibrillated and died. In this particular animal, no net loss of myocardial potassium was observed.

When the J wave appeared, the animals were hypotensive and bradycardic and had a higher Ao oxygen partial pressure than at baseline (Table 1). These values are comparable to those reported by other investigators.[8,9] We found lower levels of both ATP and CP in the myocardium of hypothermic dogs compared with those in the normothermic dogs. The myocardial content levels of the glycolytic intermediates were lower, whereas the lactate levels were higher in the hypothermic dogs compared with the normothermic dogs (Table 2).

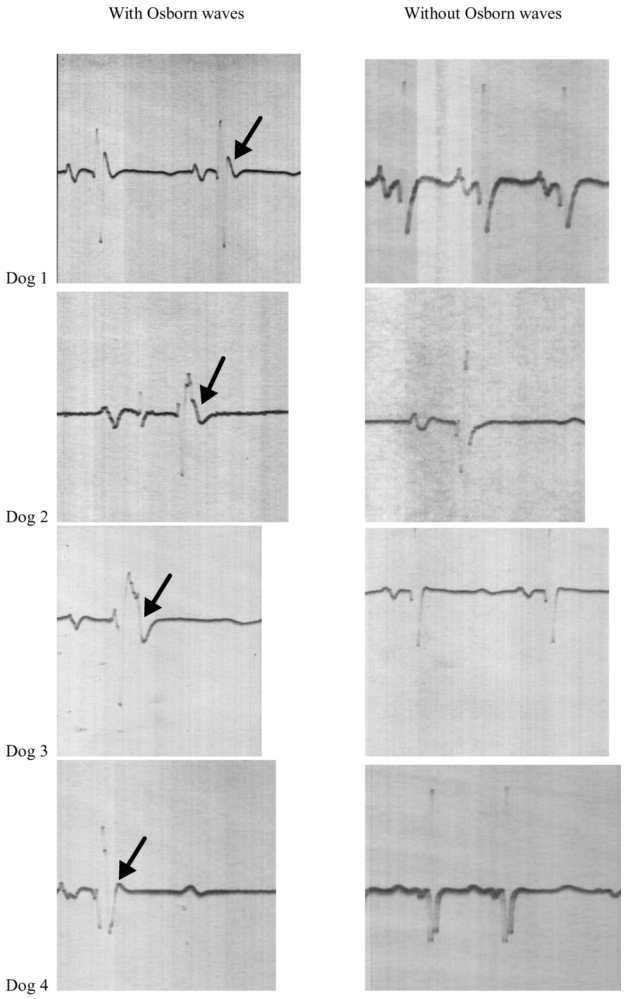

FIGURE 1 Examples of dogs with and without the presence of the Osborn wave. The *arrows* point to the J-wave deflection.

TABLE 1 Changes in Heart Rate, Mean Aortic Pressure, Arterial Blood Gases, and pH during Cooling at 36°C, 30°C, and the Temperature at which the J Wave Appeared (26.6° ± 0.73°C)

	36°C	*30°C*	*26.6 ± 0.73°C**
HR (bpm)	132 ± 13.1	102 ± 7.52*	55 ± 6.14****
AoP (mm Hg)	130 ± 8.31	96 ± 5.88**	75.2 ± 7.43***
aPo_2 (mm Hg)	110 ± 8.08	133 ± 10**	164 ± 16.2
$aPco_2$ (mm Hg)	34.9 ± 2.60	36.6 ± 3.34	38.11 ± 3.65
pH	7.365 ± 0.021	7.319 ± 0.019	7.319 ± 0.022

AoP = mean aortic pressure; $aPco_2$ = aortic carbon dioxide partial pressure; aPo_2 = aortic oxygen partial pressure; HR = heart rate.
Statistical analysis with the paired *t*-test revealed a significant decrease in heart rate mean aortic pressure (36°C vs 30°C vs when the J wave appeared). There was a significant increase in aortic Po_2, whereas the aortic Pco_2 and pH did not change significantly during the cooling.
* $p < .01$.
** $p < .02$.
*** $p < .002$.
**** $p < .0005$.

DISCUSSION

Since the initial description of the J wave in 1938,[2] there have been many attempts to determine its etiology. Some theories have included conduction defect,[2] acidosis,[10] current of injury,[1] myocardial anoxia,[11] and atrial repolarization.[12] A number of these theories have since been discredited. In 1958, Emslie-Smith proposed that the J wave could be due to "imbalanced electrical activity of the heart when depolarization ends and repolarization begins."[13,14] This theory is based on observations that depolarization and repolarization slow at different rates when mammalian myocardium is cooled.[13,14] The depolarization phase of the action potential was slightly prolonged by decreasing temperature, but the repolarization phase was greatly prolonged.[13,14] Coraboeuf and Weidmann hypothesized that a "pump" contributing directly to the value of the resting potential by throwing out cations (Na^+) might be seriously depressed when metabolism is slowed in hypothermia.[15] With our knowledge of active ion transport (ie, Na^+-K^+ adenosine triphosphatase [ATPase]), this theory seems intuitive and is, in fact, the concept of our study. We found the energy content of hypothermic myocardium to be reduced, as evidenced by diminished levels of ATP and CP. This was true regardless of the presence of a J wave. We also found a net potassium loss from the myocardium only in the presence of a J wave. Interestingly, net loss of K^+ from the myocardium is the opposite of that found in the peripheral circulation in which K^+ concentration decreases with increasing hypothermia.[16,17] It appears that the diminished energy of the hypothermic myocardium has affected its ability to maintain the partition of ions across the cell membrane. This could be due to reduced activity of the energy requiring Na^+-K^+ ATPase and the other active transport mechanisms, such as Ca^{++} ATPase.[4]

In the dog that did not develop a J wave and had no net K^+ loss, the myocardial ATP and CP levels were similar to those found in the other hypothermic dogs ($n = 8$). This animal fibrillated at 18°C without the development of a J wave. Interestingly, Osborn made a similar observation that dogs cooled below 25°C without exhibiting a J wave usually fibrillated.[1]

TABLE 2 Adenosine Triphosphate, Creatine Phosphate, and Glycolytic Intermediate Myocardial Content from Normothermic and Hypothermic Dogs after the J Wave Appeared (26.7° ± 0.73°C)

	Normothermic	*Hypothermic*	p *Value*
ATP	5.943 ± 0.31	4.881 ± 0.265	< .025
CP	9.365 ± 0.475	5.943 ± 0.475	< .005
FDP	0.246 ± 0.032	0.221 ± 0.36	NS
DHA-P	0.320 ± 0.012	0.257 ± 0.012	< .01
Pyruvate	0.235 ± 0.019	0.149 ± 0.010	< .02
Lactate	7.258 ± 0.283	9.72 ± 0.316	< .001

ATP = adenosine triphosphate; CP = creatine phosphate; DHA-P = dihydroxyacetone phosphate; FDP = fructose-1,6-diphosphate; NS = not significant.
Statistical analysis with nonpaired two-tailed Student's *t*-test. The values are expressed as mean ± standard error of the mean. The metabolites are presented as μg/g of wet tissue.

Since a large portion of the energy required for the function of membrane ATPase is produced in cytoplasm via glycolysis, it has been observed that glucose uptake is reduced by about 50% at temperatures between 25° and 20°C.[18] In hypothermic intact animals, Maur and colleagues concluded that the impairment in glucose use represents decreased oxidative metabolism of carbohydrates, there being no apparent decrease in the formation of lactic acid via pyruvic acid.[19] As previously indicated, we found the energy content of hypothermic myocardium to be decreased, as evidenced by diminished ATP and CP. The glycolytic intermediate myocardial content was lower

compared with that found in normothermic hearts (see Table 2). Lactic acid was higher in the hypothermic myocardium, which might be explained by curtailment of the oxidative carbohydrate use. Thus, it is possible that decreased pyruvate conversion to acetyl coenzyme A may be responsible for its conversion to lactate.

We believe our findings to be consistent with the theory of Coraboeuf and Weidmann.[15] The decrease in cellular energy disrupts active ion transport across the membrane and leads to slowed repolarization and the inability of the cell to reclaim the equivalent amount of K^+ lost during repolarization. This change in electrical activity of the heart at the transition from depolarization to repolarization may well account for the J wave. It appears that the J wave is associated with net potassium loss from the hypothermic myocardium.

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