The Electrical Activity of the Heart and Brain Under Acute Experimental Anoxia: The Protective Effect of Polarizing Solutions

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Abstract: The Electrical Activity of the Heart and Brain Under Acute Experimental Anoxia: The Protective Effect of Polarizing Solutions

Acute anoxia was produced in 80 dogs under anesthesia with sodium pentobarbital. Respiration was maintained with a Palmer's pump through an endotracheal tube. The anoxia was produced by stopping the pump and occluding the endotracheal tube. Polarizing solution (glucose-potassium-insulin) was perfused in 40 of the animals, starting three hours prior to the anoxia. The remaining 40 dogs comprised the control group. Peripheral electrocardiograms, as well as electroencephalograms, were simultaneously recorded before and during the period of anoxia. Recordings were continuously obtained until no more than one ventricular complex in a ten-second period was observed, even though the electrical brain activity had disappeared by this time.

Our present experimental work suggests the beneficial effect of polarizing solutions by delaying the anoxic degradation of the electrical phenomenon of the heart and the brain.

ADDITIONAL KEY WORDS: respiration, cardiac arrest, Kreb's cycle, oxidative phosphorylation process.

Methods

A series of 80 adult mongrel dogs were used. They were anesthetized with sodium pentobarbital (40 mg/kg of body weight) intraperitoneally and placed under positive respiration with a Palmer's pump.

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ELECTRICAL ACTIVITY OF THE HEART AND BRAIN

for the control group was 13.2 kg, and that of the treated one was 16.1 kg.

Results

THE ELECTRICAL ACTIVITY OF THE HEART UNDER ANOXIA

Previous studies carried out in our department,1 as well as present results, suggest that there may be a constant sequence in the degradation of the cardiac electrical phenomenon. It can be outlined as follows: (1) Between the second and fifth minute of anoxia the earliest signs of subendocardial ischemia are observed. (2) From the fifth to the eighth minute a lowering of the cardiac rate is observed, 40 to 60 beats per minute, with occasional periods of atrioventricular and intraventricular conduction disturbance. (3) Around the ninth minute, complete A-V block appears with a drop of the heart rate below 40 beats per minute; at this time a marked subepicardial injury develops with the duration of the QRS complexes, doubling that of the controls. (4) At a terminal stage bradycardia is extreme, and finally cardiac standstill occurs without passing through ventricular fibrillation. The average time for this is 19 minutes and 25 seconds (Bisten1 reports 19 minutes).

ANOXIA
Duration of the electrical phenomenon of the heart

GRAPH 1

The statistical analysis of the duration of the cardiac electrical phenomenon.

ANOXIA
Duration of the cerebral electrical phenomenon

GRAPH 2

The statistical analysis of the results showed a significant difference in the duration of the cerebral electrical activity under anoxia between the control and treated groups (207 and 362 seconds mean arithmetic, respectively).
FIGURE 1
This is a control experiment which shows complete disappearance of the brain electrical activity at the fourth minute, 23rd second period of anoxia. Subendocardial ischemia is simultaneously observed. EEG: Electroencephalogram. RFP: Right frontoparietal lead. RPO: Right parieto-occipital lead. LFP: Left frontoparietal lead. LPO: Left parieto-occipital lead. ECG: Electrocardiogram. L2: Lead 2.

FIGURE 2
This is a control experiment in which the brain electrical silence occurred at the third minute, fourth second interval of anoxia. Subendocardial ischemia can be recognized. EEG: Electroencephalogram. RFP: Right frontoparietal lead. RPO: Right parieto-occipital lead. LFP: Left frontoparietal lead. LPO: Left parieto-occipital lead. ECG: Electrocardiogram. L2: Lead 2.
ELECTRICAL ACTIVITY OF THE HEART AND BRAIN

500 ml. GKI (starting 3 hrs. prior the onset of anoxia)

anoxia

EEG Control

5 6'0"

LFP RFP

LPO

FIGURE 3

This experiment corresponds to a treated animal (see text). The brain electrical activity is still observed at the fifth minute of anoxia. The electrical activity has completely disappeared at the sixth minute and tenth second interval. Concomitant subendocardial ischemia is present. GKI: Glucose-potassium-insulin solution. EEG: Electroencephalogram. RFP: Right fronto-parietal lead. RPO: Right parieto-occipital lead. LFP: Left frontoparietal lead. LPO: Left parieto-occipital lead. ECG: Electrocardiogram. L2: Lead 2.

Though this succession of events was the same in the group treated with polarizing solution, a significant retardation of the cardiac arrest period was observed. We found an average time of 26 minutes and 48 seconds as compared with 19 minutes and 25 seconds for the control, nontreated group (graph 1).

THE ELECTRICAL ACTIVITY OF THE BRAIN UNDER ANOXIA

Under pentobarbital anesthesia the basal rhythm of the EEG presented a fast beta-wave type of activity (18 to 24 cps) with a superimposed theta frequency (3 to 7 cps). The voltage of these two components of the curve was 5 to 10 mv and 20 to 50 mv, respectively.

There were neither asymmetry nor synchronization between the cerebral hemispheres.

It is important to emphasize that the above-mentioned dominant rhythms, resulting from pentobarbital anesthesia, were similar in both the control and the treated groups.

CONTROL GROUP (figs. 1 and 2)

(1) At the thirtieth second of anoxia, a decrease in voltage was observed, particularly in the slow frequency rhythms. This change persisted for one minute.

(2) Between the second and third minute the theta activity was predominant due to a diminution of the beta components.

(3) At the third minute most of the cases showed transient slowed delta rhythms with a gradual vanishing of the EEG potentials until electrical silence was established. This occurred between the second (one single case) and the fifth minute, averaging 3 minutes and 27 seconds.

TREATED GROUP (figs. 3 and 4)

(1) Between the second and third minute a slower wave activity was seen, but with a higher voltage. The dominant frequencies were those of the 6 to 7 cps (theta waves).

(2) At the fourth minute a definite preponderance of the theta rhythm was observed, the frequency being 4 to 6 cps. On the other hand the beta activity, i.e., the fast low voltage type of waves (18 to 30 cps) was no longer recognized.

(3) From the fifth to the sixth minute large slow delta waves, below 4 cps, were
dominant. A progressive wane in amplitude was observed, until no electrical activity could be detected. This electrical silence occurred between the sixth and the eighth minute, averaging 6 minutes and 2 seconds.

The statistical analysis of our results, in both the control and the treated groups, reveals significant differences in the occurrence of brain electrical silence produced by anoxia (see graph 2).

**Discussion**

There is cumulative evidence in favor of the utility of polarizing solutions as a protective measure in the deterioration of Kreb's cycle due to anoxia. Experimental studies performed at this Institute have demonstrated, from a biochemical standpoint, how impairment of the oxidative phosphorylation process in infarcted dogs can be deleted or improved by the infusion of polarizing solutions. Another interesting observation supporting our findings is that of Hochrein and Lossnitzer who have been able to prevent the kalocytopenic effect of hypoxia through the administration of K and Mg salts.

Our present experimental work suggests the beneficial effect of polarizing solutions by delaying the anoxic degradation of the electrical phenomenon of the heart and the brain.

**References**

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