often ascribed to hypercalcemia remit during the sodium-EDTA infusion except evanescently in patient No. 1. The eventual return of hypercalcemic levels to normal in patient No. 2 is not attributed to EDTA. With the exception of patient No. 3, no apparent modification of survival was effected by the procedure.

Summary. 1. A sodium salt of ethylene diamine tetra acetic acid at pH 7.4 has been administered rapidly intravenously to 5 patients with hypercalcemia from bone destruction. 2. Decrease in serum calcium concentration has been produced but this is of remarkably short duration and unassociated with clinical benefits. 3. Two instances of renal tubular vacuolization following administration of sodium-EDTA have been observed.

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Effect of Electroshock Therapy on Blood Lactic Acid and Pyruvic Acid. (20646)

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Electroshock therapy (EST) is widely used in the treatment of certain mental disorders. However, the biochemical changes in blood constituents resulting from such traumatic electrical stresses to the body are little known. Our immediate interest in this problem was centered around the changes in lactic acid and pyruvic acid levels appearing in the blood after the extreme muscular excitation induced by EST. The evidence available in the literature concerning the effect of EST on the lactic acid and pyruvic acid levels, as well as on other constituents, in the blood is both fragmentary and inconclusive(1-3). In most cases reported, the time intervals of blood sampling of patients receiving EST were chosen arbitrarily and consequently have given rise to conflicting and unsystematic data. We undertook this investigation to ascertain precisely the changes occurring in both lactic acid and pyruvic acid levels of the blood following EST.

Methods. Ambulatory patients, ranging from 20 to 40 years of age and carrying a diagnosis of chronic schizophrenia, were employed in this study. Patients having a relatively quiet awakening after EST were

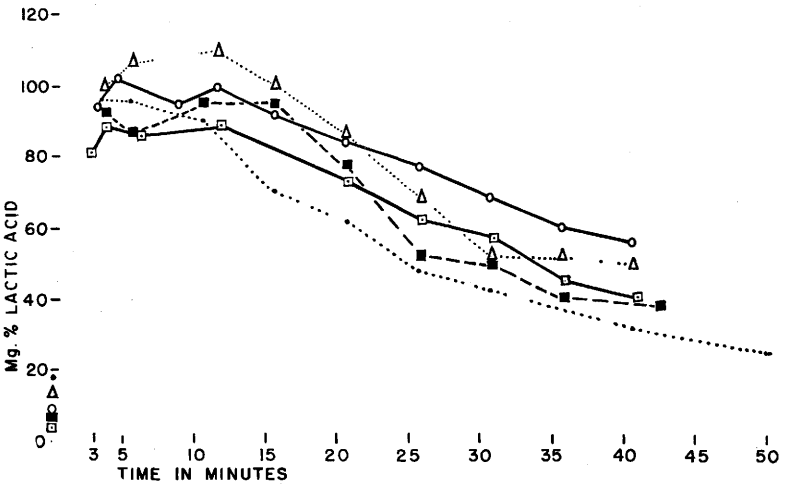

FIG. 1. Various symbols represent lactic acid values of different patients. Pre-shock values of each patient are shown on the ordinate. Abscissa represents the time from onset of shock.

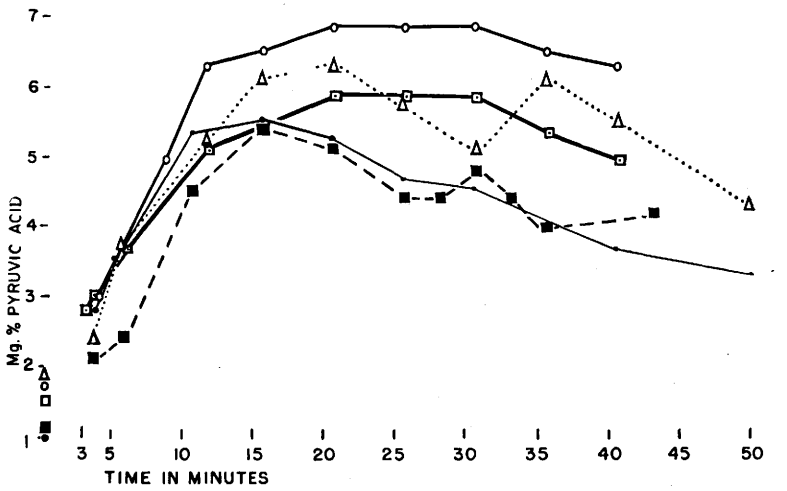

FIG. 2. Same as Fig. 1, except the values are for pyruvic acid.

selected. Each subject was given 3 to 6 grains of sodium amytal by mouth prior to shock. EST was administered by the "Electronicraft" machine with the initial current at 125 milliamperes and with automatic resistance compensation. Maximum current of 350 milliamperes was reached within 2½-3 seconds. The current was reduced to 250 milliamperes in 5 seconds then was gradually decreased depending on the response of the patient and was abruptly terminated during the later part of the clonic phase of the convulsion. The average length of the total treatment was 35 to 40 seconds. Samples of venous blood were collected without the aid of a tourniquet and immediately placed in cold 10% trichloroacetic acid. A pre-shock sample was removed and samples were taken following the clonic phase of the convulsion at approximately one-half minute intervals up to 3 minutes and every 5 minutes thereafter. The method used for the determination of lactic acid was that of Barker and Summerson(4). Lithium lactate, which is used by most workers as a standard for this method, appeared to pick up water and thus furnished only relative results. Consequently, we employed as our lactic acid standard a sample of non hygroscopic calcium L(+) lactate of known water content which was a mixture of hydrates: H_2O, 23.4%; ash

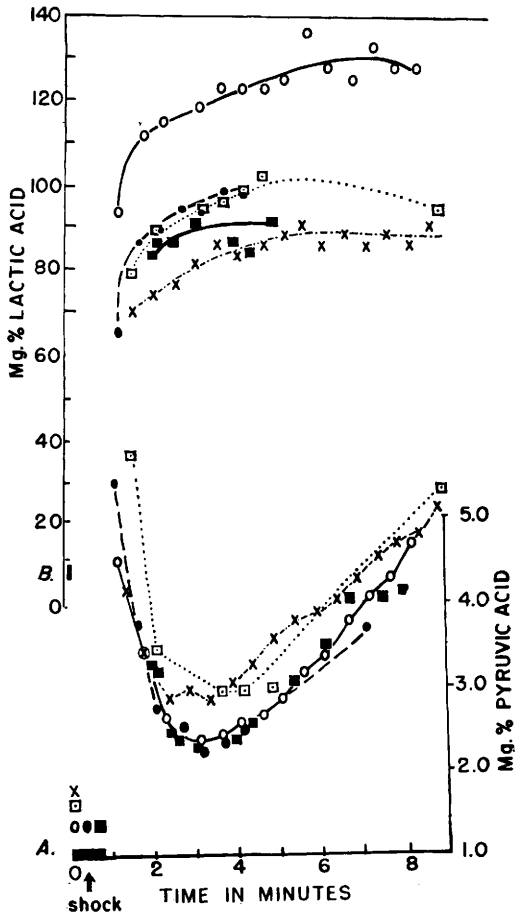

FIG. 3. Time scale (abscissa) is enlarged to show drop in blood pyruvic acid levels and corresponding rise of blood lactic levels. Various symbols in lower left corner (marked A) represent pre-shock levels of pyruvic acid. Bar on the left ordinate (marked B) represents variation in pre-shock levels of lactic acid.

($CaCO_3$) calculated, 35.5%, theory, 35.2%. The lactic acid determinations were made on the same day the blood samples were collected, since on standing the lactic acid concentration increased significantly. The method for determining pyruvic acid was that of Friedmann and Haugen(5) as modified by Fister(6). Samples for pyruvic acid determination were stored at 5°C for 24 hours in order to destroy interference by acetoacetic acid. The Friedmann and Haugen procedure(5) and the Lichtenbelt and Florijn method(7) (heating the fresh deproteinized blood filtrate in a boiling water bath for 5 minutes) gave similar results.

Results. The blood lactic acid level for each patient, following EST (Fig. 1), rose sharply to a peak within 3 to 5 minutes, then declined approaching the normal pre-shock level usually within 1½ hours. The concentrations at peak levels increased approximately 6-fold over pre-shock levels. The blood pyruvic acid reached a peak approximately three times the pre-shock level after about 20 minutes (Fig. 2).

When blood samples were drawn at 20 to 30 second intervals, it was observed that both the lactic acid and pyruvic acid concentrations rose immediately following EST. However, after thirty seconds the pyruvic acid level dropped, falling to a minimum at the same time that the lactic acid reached its maximum (Fig. 3). The variation among patients of these two metabolites, at any particular time after shock, was quite large. However, as shown in Fig. 4, the ratio of lactic acid to pyruvic acid (L/P), at any particular time, was quite constant. It seems clear that unless a comprehensive picture of the fluctuations of the levels of lactic acid and pyruvic acid (or any other metabolite) is obtained, an interpretation of data based on arbitarary spot-checking of the concentrations of these metabolites in the blood can only lead to confusing and conflicting results. Since during the first three minutes there was a drop in pyruvic acid levels with a corresponding rise in lactic acid levels,

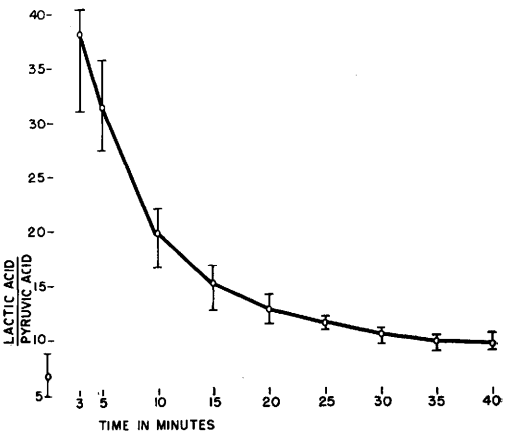

FIG. 4. Points (circles) represent mean ratio of lactic acid/pyruvic acid for same patients illustrated in Fig. 1 and 2. Bars represent largest deviation from the mean. Circle located on the ordinate represents mean ratio of pre-shock levels.

it would appear that the Embden-Meyerhof scheme represented the dominating event during this time interval. Also, since after the first 3 minutes, the keto acid levels increased as the hydroxy acid levels decreased, and since the concentrations of these metabolites then returned to normal pre-shock levels, it would appear that the aerobic phase predominated during this time interval. It is of considerable interest that the L/P ratio, at any particular time after shock, of all patients studied fell within a small range.

Further investigations are in progress to determine the relationship between the variations in the concentrations of lactic acid and pyruvic acid, or their ratios, as an index of the relative intensity of the convulsion in EST.

Preliminary data obtained on the blood levels of glucose, sodium, potassium, and calcium, indicated that the concentration of these metabolites varied considerably over a sixty minute period following EST, rising and falling several times during this time interval.

Summary. A simultaneous measurement has been made of lactic acid and pyruvic acid changes in the blood of neuropsychiatric patients before and after extreme muscular excitation induced by electroshock therapy (EST). The blood lactic acid level of all patients following EST rose sharply to a peak (six times pre-shock level) within 3 to 5 minutes and then returned to the normal level after 1 to 1½ hours. The blood pyruvic acid level of all patients rose immediately following EST for thirty seconds (4-6 mg %), then fell to a minimum (2-3 mg %) within 3 to 5 minutes and rose again to a peak three times the pre-shock level 20 min after EST.

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Influence of Hibernation on Survival Time and Weight Loss of X-Irradiated Ground Squirrels.* (20647)

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Numerous studies on the influence of temperature on the radio-sensitivity of various biological systems have been conducted and it has been concluded(1) that temperature changes within the range which can be tolerated by most living systems do not directly modify radiation reactions. However, the tissue injury resulting from the interaction of the products of ionizing radiations with cellular constituents is delayed when the temperature of the organism is maintained below normal following irradiation and survival is thereby prolonged. Although this effect has been demonstrated in several types of cells and tissues by a number of investigators, its occurrence in hibernating mammals(2-4) is

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