ACID-BASE STATUS AND GLYCOLYTIC PARAMETERS IN CHRONIC DISCOID LUPUS ERYTHEMATOSUS AND ICHTHYOSIS PATIENTS

ACID-BASE STATUS AND GLYCOLYTIC PARAMETERS IN CHRONIC DISCOID LUPUS ERYTHEMATOSUS AND ICHTYOSIS PATIENTS

by

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The study of acid-base status in patients with skin diseases may be regarded at the first sight as a rather strange idea. Nevertheless, a series of data mentioned in the previous papers indicate the occurrence of serious metabolic disturbances in chronic discoid lupus erythematosus (DLE) and ichtyosis patients. This could also indicate changes in the acid-base status with consequences in the evolution of these diseases.

With the aim to clarify certain aspects of this kind a number of 19 DLE and 11 ichtyosis patients from the previously reported group were investigated.

Determination of acid-base status parameters was performed on capilary blood samples from finger pulp. The blood was collected in heparinized capilary tubes without pressing the subject s finger. No arterializing procedures were applied.

A complex AVL type Gas check system was utilized in the determination of parameters.

Variation of acid-base equilibrium was followed in the patients before the onset of therapy and then after different time intervals according to the stages of clinical evolution.

Considering the fact that the acid-base status data are mathematically correlated, the acid-base equilibrium was graphically presented in case of each patient on a Siggaard-Andersen acid-base chart. This allowes to have a suggestive image of equilibrium state in case of main parameters and permits the visual evaluation of each case separately (See the annexed charts).

Some of the most suggestive cases show relatively slight variations as against the normal area.

The general trend noted in acid-base status of the patients before therapy was rather normal or with a tendency towards a slight alkalosis, while the equilibrium was shifted toward a slight acidosis after a period of treatment.

Cases of uncompensated alkalosis (pH exceeding 7,45) were noted in certain instances. The cases with slight alkalosis represent about 1/3 from the total of DLE patients.

P_{CO_2} and HCO_3^- values are generally normal.

The decrease in P_{CO_2} and HCO_3^- values are characteristic to the period following therapy while the pH is maintained between the normal ranges.

In order to make a more precise evaluation of the significance of these variations the distribution of P_{CO_2} values as well as the HCO_3^- / H_2CO_3 ratio has to be followed in the investigated patients (fig. 1 and 2).

HCO_3^- / H_2CO_3 ratio is slightly increased before therapy in both DLE and ichtyosis indicating the slight alkalosis status. This increased value is own both the increase in the HCO_3^- and to a slight decrease in H_2CO_3, as it can be noted in Tab. 3. While the P_{CO_2} values decrease to under normal levels in DLE patients after therapy, the values of the HCO_3^- / H_2CO_3 ratio are diminished towards normal figures (never dropping under this level).

HCO_3^- values are well correlated with base excess (BE) and buffer base (BB).

It may be assumed that the decrease in BE and BB, as indicated by their after therapy averages, are due to the decrease in bicarbonate.

The explanation of these decreases occurring in HCO_3^- and P_{CO_2} might consist in an increased rate of HCO_3 elimination in kidney, compensated by the lung hyperventilation. Unfortunately, no data are available tu support the diminution of the urine acidification process.

Attempts to correlate these data with the clinical criteria concerning disease severity, history, antecedents, associated diseases, previous therapies, etc., showed no significant connections to be applied to the concrete cases.

That is why the disturbances occurring in the acid-base status can be correlated only with the general changes of both hystopathological and metabolical nature characterizing these diseases.

In this context the dermal vascular impairements which proved to be related to the immunochemical processes mentioned in the previous works may be taken into account. Following the allergic reactions as well as the formation and sedimentation of immune complexes, the walls of dermal vessels are subject to permeability changes and to changes in the reactivity to chemical mediators. These result in a general state of vasoplegia which, implies disturbances in the blood circulation stasis on the one hand, and on the other hand it results in adverse conditions (hypoxia, presumed local acidosis) at the level of dermal tissues which generate metabolic changes (alterations in the metabolical pathways of cellular energy generation).

These assumptions may be indeed correlated with the phenomena of acroasphyxia also observated in a number of the DLE patients.

It seems also reasonable to admit that in some particular cases, the alkalosis should be regarded as compansatory process towards the presumed tissue acidosis.

The serum activity of phosphohexoseisomerase (PHI) and serum pyruvate concentration were evaluated, among other parameters in order to emphasize the cellular metabolic alterations with a possible role in the pathogeny of DLE and ichtyosis.

Phosphohexoseisomerase is an enzyme playing a key role in the anaerobic degradation of the carbohydrates via the Embden Meyerhoff Parnass pathway; its activity is implicated both in the utilization of glucose as such (subsequently converted to glucose - 6 - P by hexokinase) and of the glucose originated from the degradation of glycogen to glucose - 1 - P and glucose 6 - P.

Owing to its key role in the glycolysis the activity of PHI is a sensitive indicator in the glucose metabolism i. e. in the anaerobic glycolysis intensity.

The pyruvic acid is the end product of the anaerobic degradation of carbohydrates representing meantime the connection with the next step, the oxydative decarboxylation and Krebs cycle. The pyruvic acid concentration also indicates the intensity of anaerobic degradation and the next stages in which it is used as a substrate.

Determinations of PHI activity were performed with standard reagents (Boehringer kit - PHI - mono-test).

Evaluation of pyruvate concentration was performed with an enzymatic method (Boehringer kit - Pyruvat test).

Determinations were also carried out on a group of 10 aparently healthy blood banking donors.

The results are mentioned in the fig. 4 and 5.

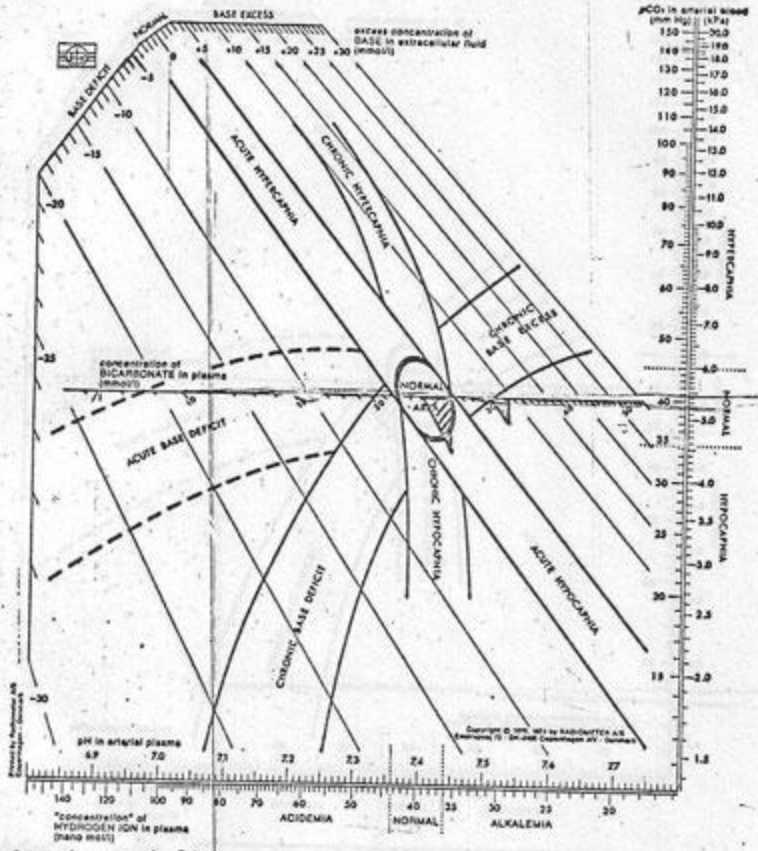
Evident increases in PHI activity and pyruvate concentration were noted in both DLE and ichtyosis.

In interpretation of the alterations occurring in PHI activity, the fact that the assay of PHI was performed on serum has to be taken into account. Nevertheless, the evident increases noted could be the indicator of an increased cellular activity. This could suggest modifications in the intensity of certain glycolysis sequences, a fact which is also supported by the increased pyruvate concentration. More precisely a relative blocking of the glycolysis at the pyruvate-lactate level occurrs. This situation is supported both by the probable decrease in the activity of Zn-dependent pyruvate - decarboxylase, owing to the decrease in the level of plasma Zn concentration and to the decrease in the Krebs cycle intensity under anaerobiosis conditions.

An intensification of anaerobic carbohydrate degradation also occurrs, supported by the following factors : the necessity to obtain energy for the regeneration processes occurring in the lesions; unblocking of other metabolic steps in glycolysis, normally represed under aerobiotic conditions (Pasteur effect).

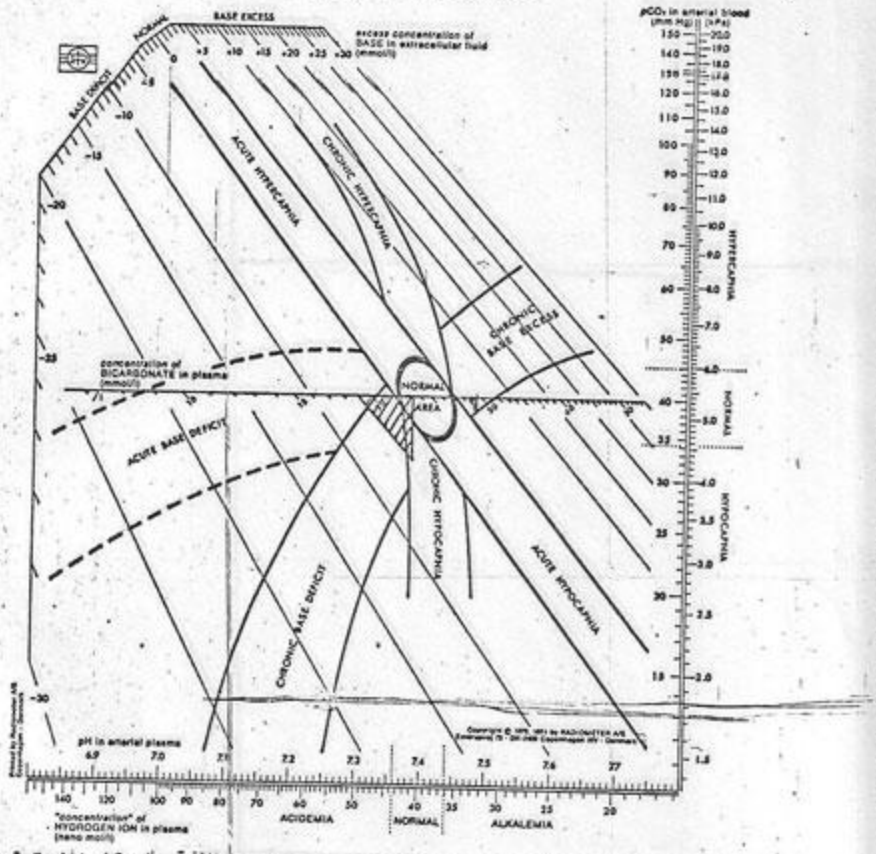
It may be noted that also the presented data on the acid-base status and the stages of anaerobic glycolysis are still reduced, the results are correlated and even at this stage of preliminary researches they suggest the importance of the metabolic modifications in the pathogeny of discuses under study. It is interesting to note the significance of the hypoxia state occurring in dermal lesions in the maintenance of the vicious circle generated by the consequence of the immunochemical reactions and disturbance of the metabolic processes involved in energy generation and utilization.

SIGGAARD-ANDERSEN ACID-BASE CHART



Patient identification 9.P.M.	pH	pCO_2	HCO_3^-	H_2CO_3	$\frac{HCO_3^-}{H_2CO_3}$	BE	BB
Before therapy.	7.53	36.5	31.3	1.09	28.7	+8.7	56.4
After 15 weeks of therapy.	7.44	33.0	24.2	0.99	24.4	+1.5	48.7

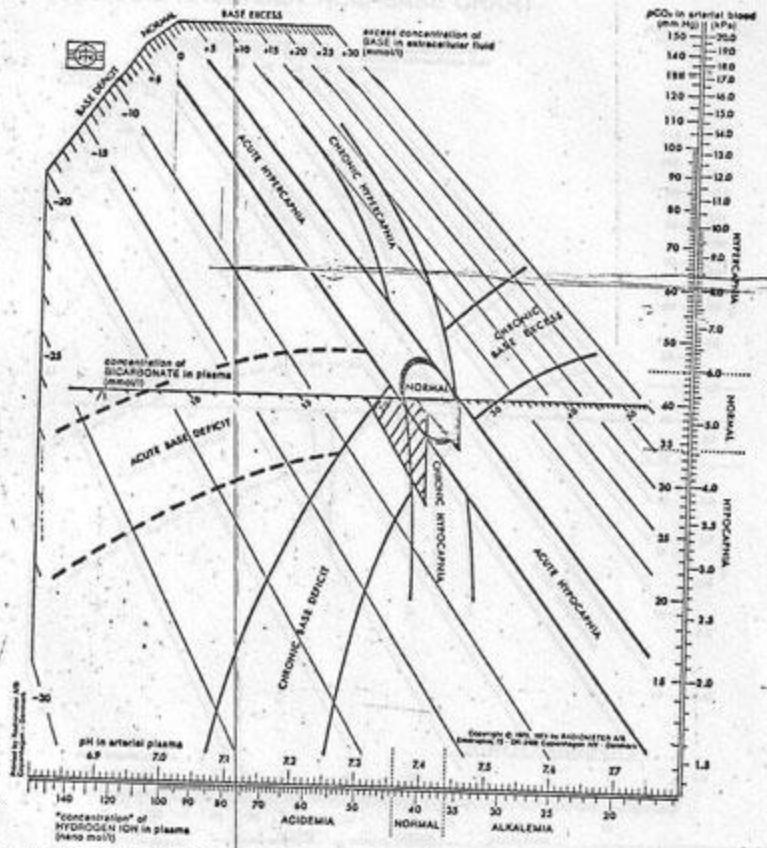
SIGGAARD-ANDERSEN ACID-BASE CHART



Patient identification

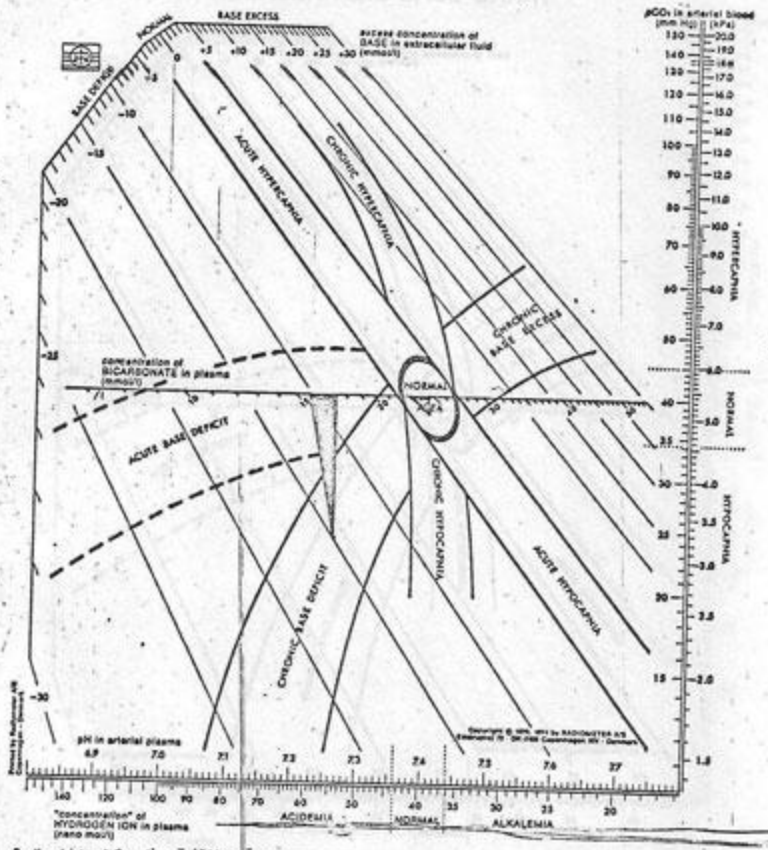
10.C.G.	pH	pCO_2	HCO_3^-	H_2CO_3	$\frac{HCO_3^-}{H_2CO_3}$	BE	BB
Before therapy.	7.48	38.5	29.7	1.15	25.8	+6.4	54
After 8 weeks of therapy.	7.38	32.0	19.0	0.96	19.8	-6.0	34

SIGGAARD-ANDERSEN ACID-BASE CHART



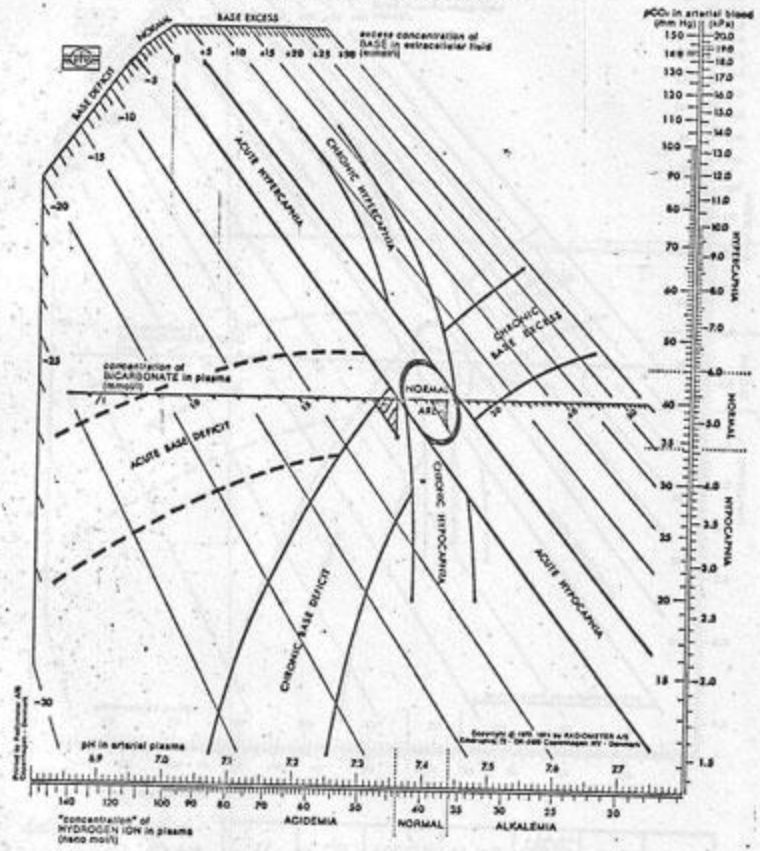
Patient identification 14. B.R.	pH	pCO_2	HCO_3^-	H_2CO_3	$\frac{HCO_3^-}{H_2CO_3}$	BE	BB
Before therapy.	7.45	33.5	23.2	1.0	23.2	+0.4	47.8
After 7 weeks of therapy.	7.40	27.5	19.5	0.82	23.8	-6.0	42.0

SIGGAARD-ANDERSEN ACID-BASE CHART



Patient identification 27.P.M.	pH	pCO_2	HCO_3^-	H_2CO_3	$\frac{HCO_3^-}{H_2CO_3}$	BE	BB
Before therapy.	7.26	24.0	16.0	0.72	22.2	-15	49
After 7 weeks of therapy.	7.40	38.0	23.2	1.14	20.6	-0.6	47.3

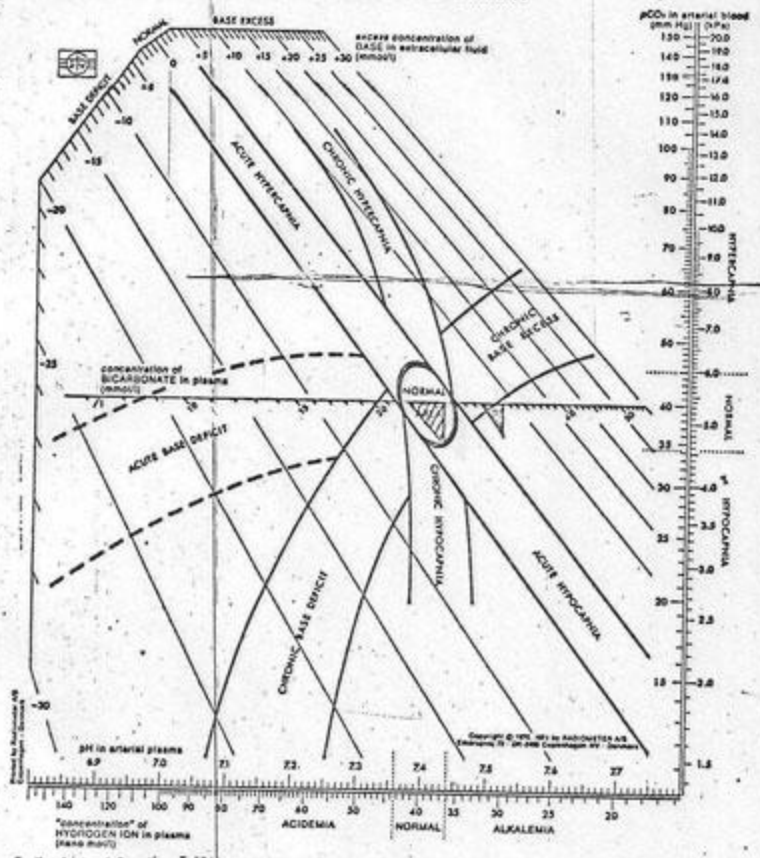
SIGGAARD-ANDERSEN ACID-BASE CHART



Patient identification

7.S.G.	pH	pCO_2	HCO_3^-	H_2CO_3	$\frac{HCO_3^-}{H_2CO_3}$	BE	BB
Before therapy.	7.43	36.4	25.0	1.09	22.9	+0.7	48
After 11 weeks of therapy.	7.35	35.0	20.0	1.05	19.05	-5.0	46

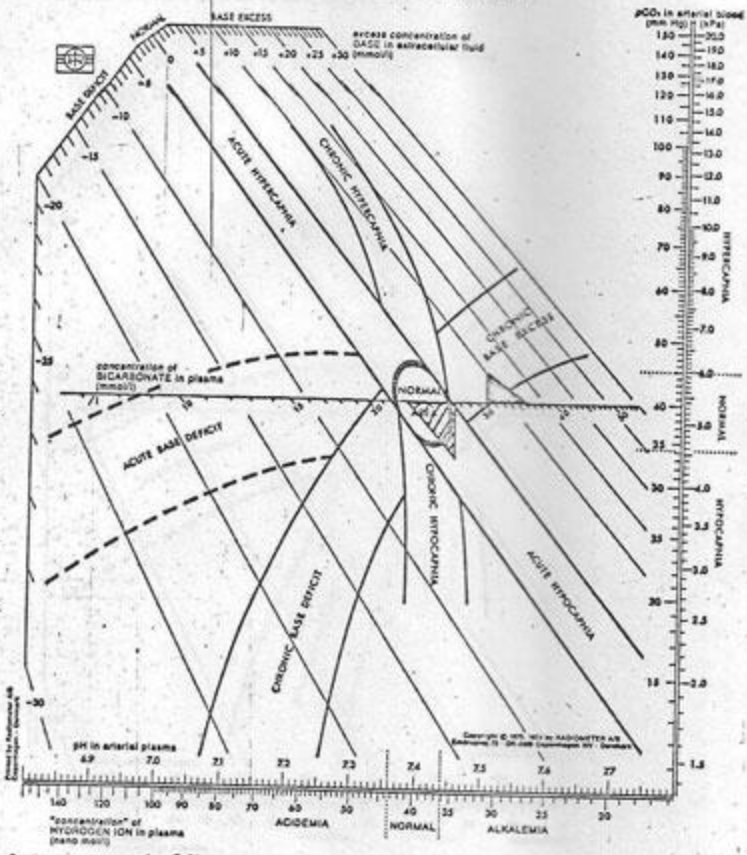
SIGGAARD-ANDERSEN ACID-BASE CHART



Patient identification

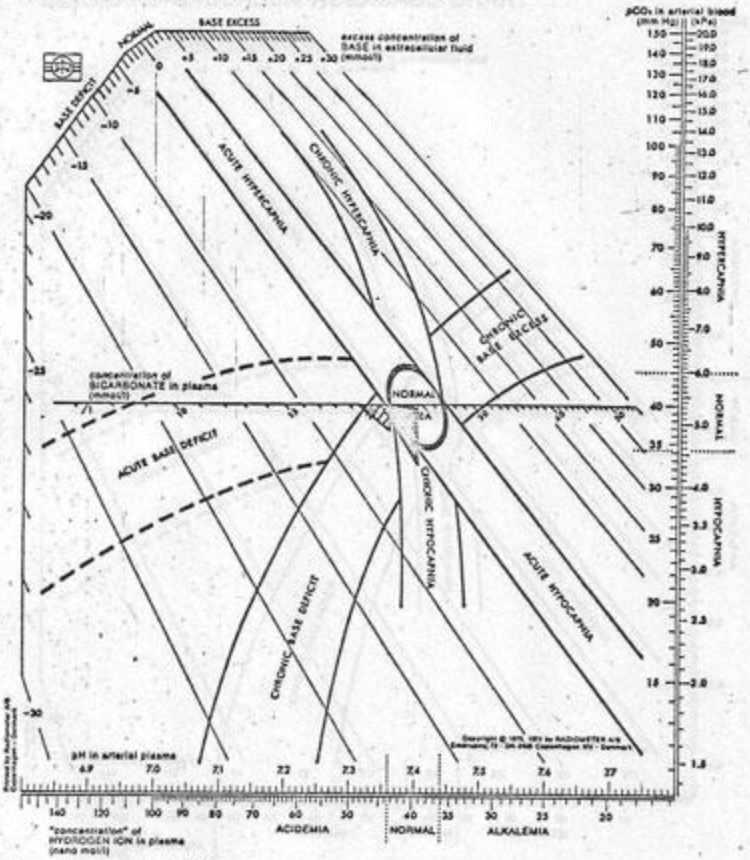
12.R.M.	pH	pCO_2	HCO_3^-	H_2CO_3	$\frac{HCO_3^-}{H_2CO_3}$	BE	BB
Before therapy.	7.52	35.5	30.4	1.06	28.69	+7.9	55.5
After 8 weeks of therapy.	7.43	34.5	23.0	1.03	223	-2.0	37

SIGGAARD-ANDERSEN ACID-BASE CHART



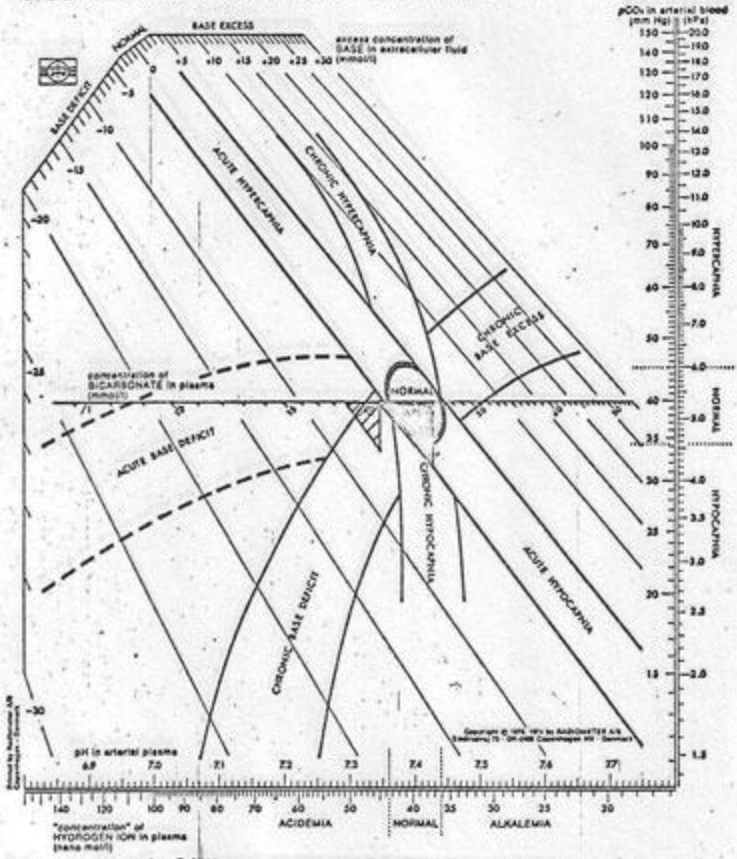
Patient identification 13.B.P.	pH	pCO_2	HCO_3^-	H_2CO_3	$\frac{HCO_3^-}{H_2CO_3}$	BE	BB
Before therapy.	7.50	44.4	36.2	1.33	27.2	+11.5	59.5
After 8 weeks of therapy.	7.45	33.0	23.0	0.99	23.2	-1.0	45.5

SIGGAARD-ANDERSEN ACID-BASE CHART



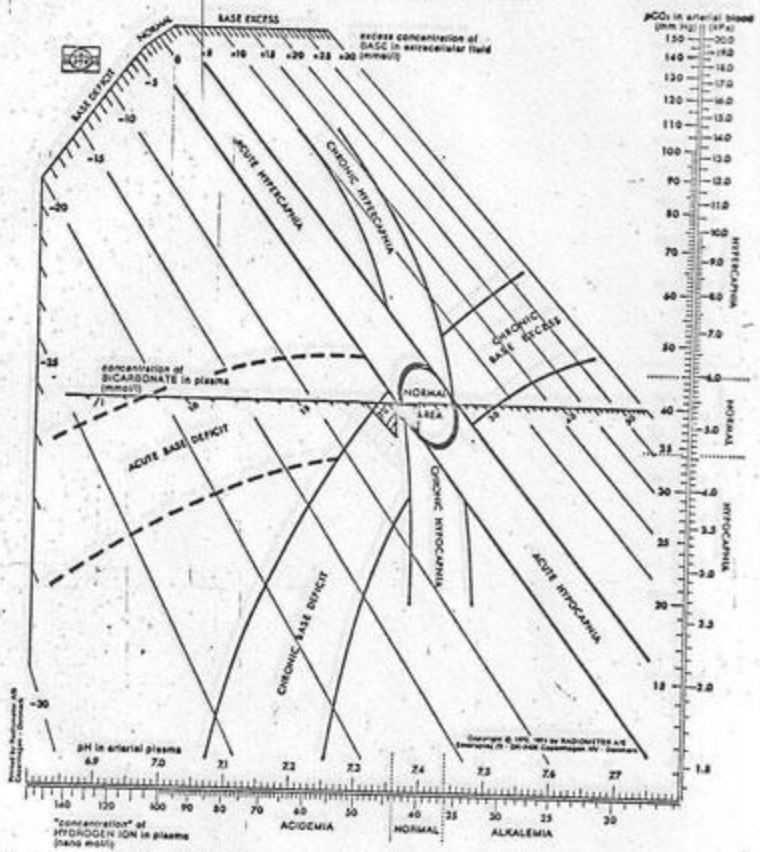
Patient identification 28.M.S.	pH	pCO_2	HCO_3^-	H_2CO_3	$\frac{HCO_3^-}{H_2CO_3}$	BE	BB
Before therapy.	7.40	32.0	22.0	0.96	22.9	-3.0	45
After 13 weeks of therapy.	7.36	36.9	19.4	1.10	17.6	-4.7	39.7

SIGGAARD-ANDERSEN ACID-BASE CHART



Patient identification 39.M.N.	pH	pCO_2	HCO_3^-	H_2CO_3	$\frac{HCO_3^-}{H_2CO_3}$	BE	BB
Before therapy.	7.43	31.4	21.1	0.94	22.4	-2.4	45.5
After 3 weeks of therapy.	7.35	33.7	19.0	1.01	18.8	-6.0	40.1

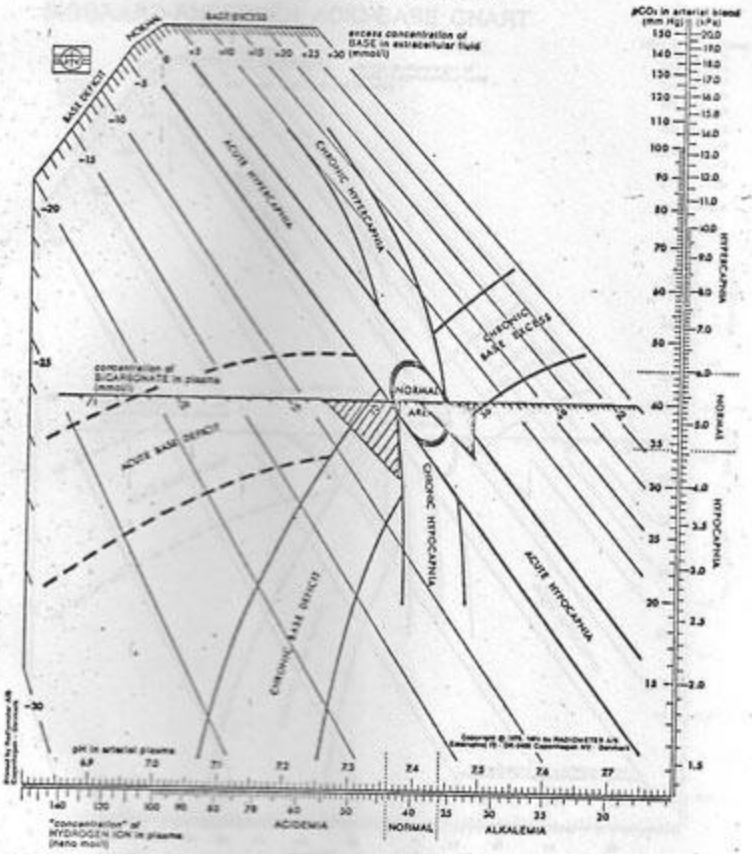
SIGGAARD-ANDERSEN ACID-BASE CHART



Patient identification

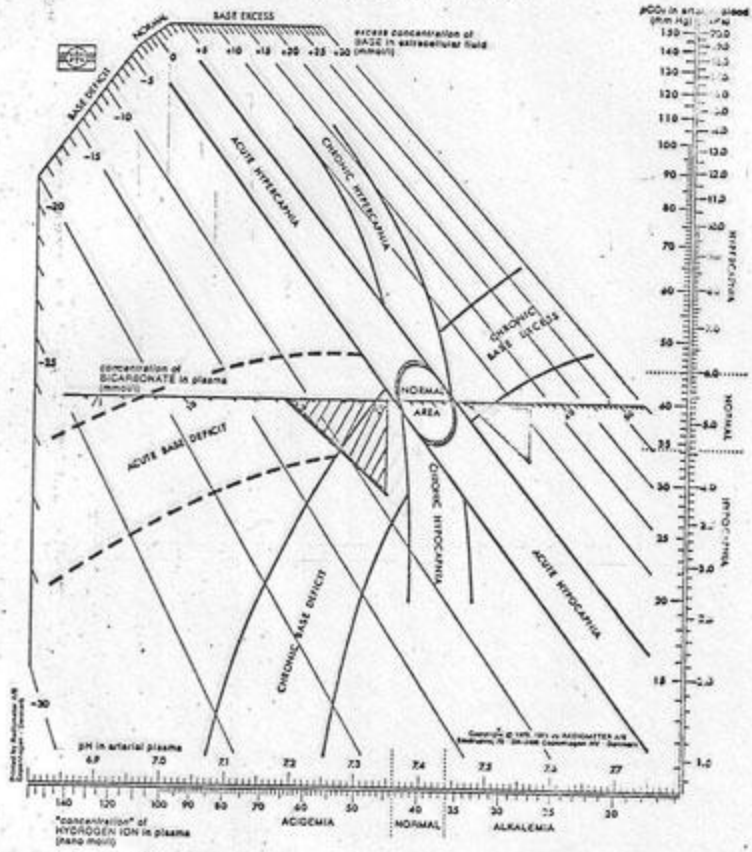
40.P.I.	pH	pCO_2	HCO_3^-	H_2CO_3	$\frac{HCO_3^-}{H_2CO_3}$	BE	BB
Before therapy.	7.39	35.6	20.9	1.07	19.5	-2.8	45.1
After 3 weeks of therapy.	7.35	35.4	19.1	1.06	18.0	-5.2	42.7

SIGGAARD-ANDERSEN ACID-BASE CHART



Patient identification 42.M.M.	pH	pCO_2	HCO_3^-	H_2CO_3	$\frac{HCO_3^-}{H_2CO_3}$	BE	BB
Before therapy.	7.49	33.0	24.5	0.99	24.7	+2.7	50.7
After 3 weeks of therapy.	7.37	30.4	17.0	0.91	18.7	-6.5	41.4

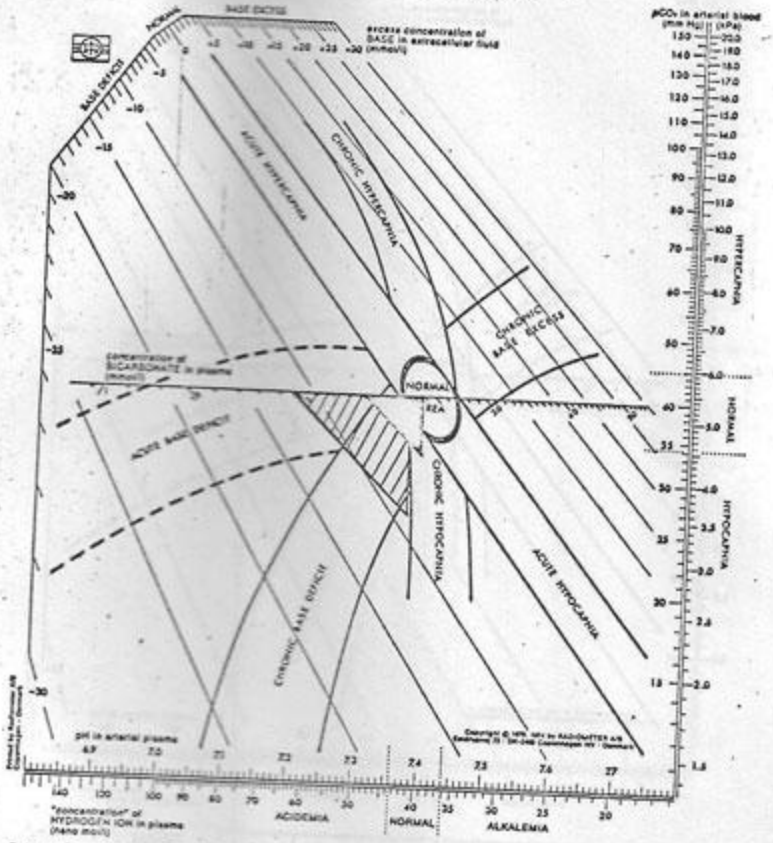
SIGGAARD-ANDERSEN ACID-BASE CHART



Patient identification 43.S.I.

	pH	pCO_2	HCO_3^-	H_2CO_3	$\frac{HCO_3^-}{H_2CO_3}$	BE	BB
Before therapy.	7.56	32.4	28.2	0.97	29.0	+7.4	55.4
After 3 weeks of therapy.	7.34	28.6	14.9	0.86	17.3	-9.0	38.9

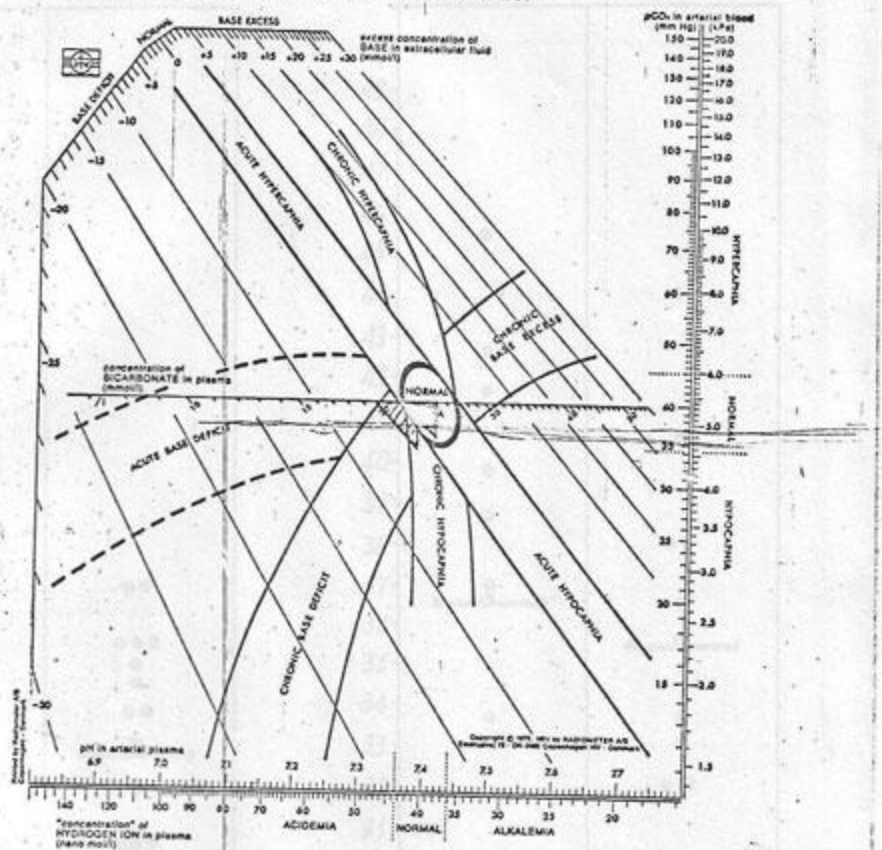
SIGGAARD-ANDERSEN ACID-BASE CHART



Patient identification

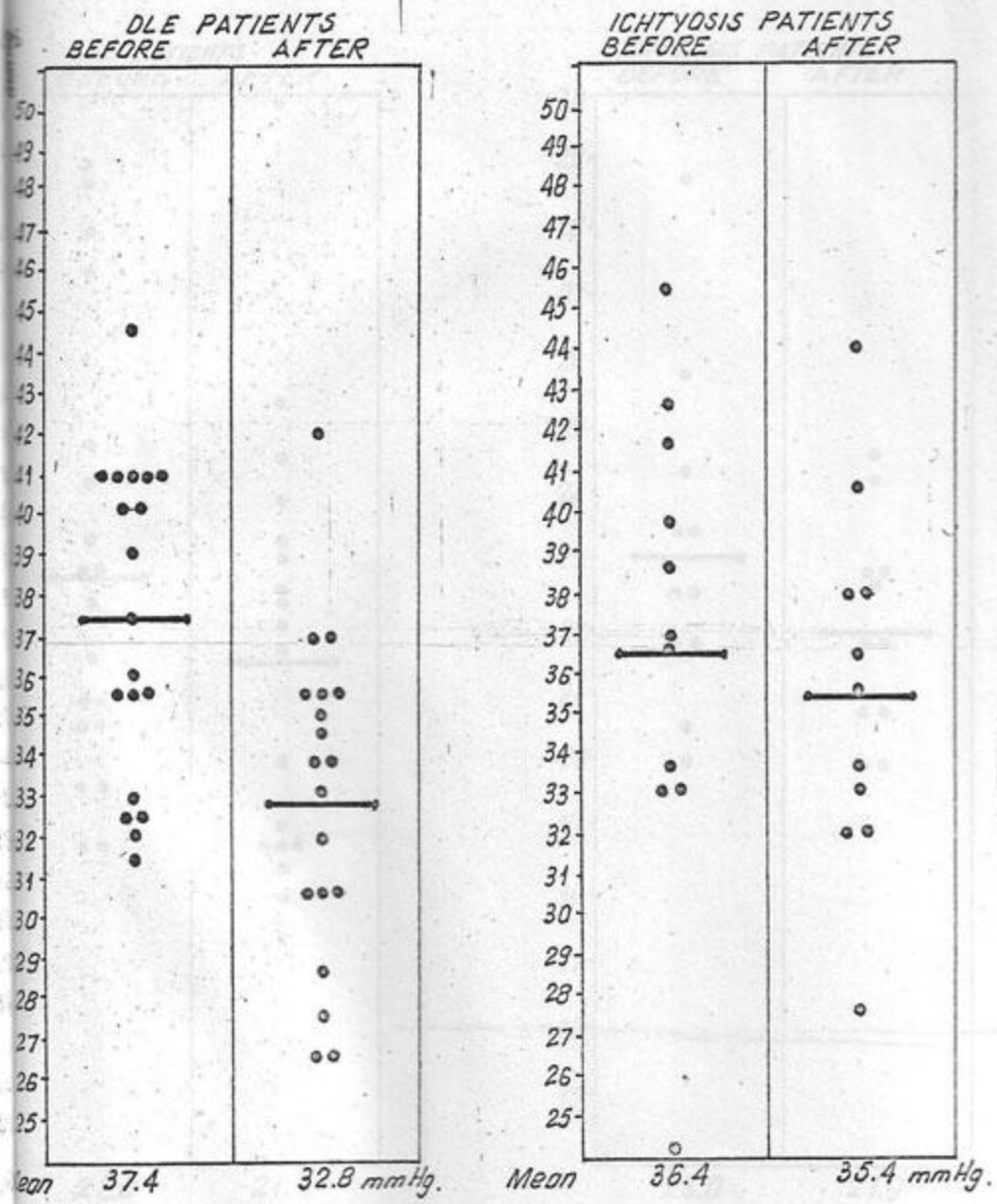
44.K.E.	pH	pCO_2	HCO_3^-	H_2CO_3	$\frac{HCO_3^-}{H_2CO_3}$	BE	BB
Before therapy.	7.39	32.4	19.1	0.97	19.6	-4.2	43.7
After 3 weeks of therapy.	7.37	26.4	14.8	0.79	18.7	-8.2	39.7

SIGGAARD-ANDERSEN ACID-BASE CHART



Patient identification 46.B.V.	pH	pCO_2	HCO_3^-	H_2CO_3	$\frac{HCO_3^-}{H_2CO_3}$	BE	BB
Before therapy.	7.41	35.3	22.0	1.06	20.7	-1.2	46.7
After 3 weeks of therapy.	7.38	33.5	20.0	1.0	20.0	-4.0	41.4

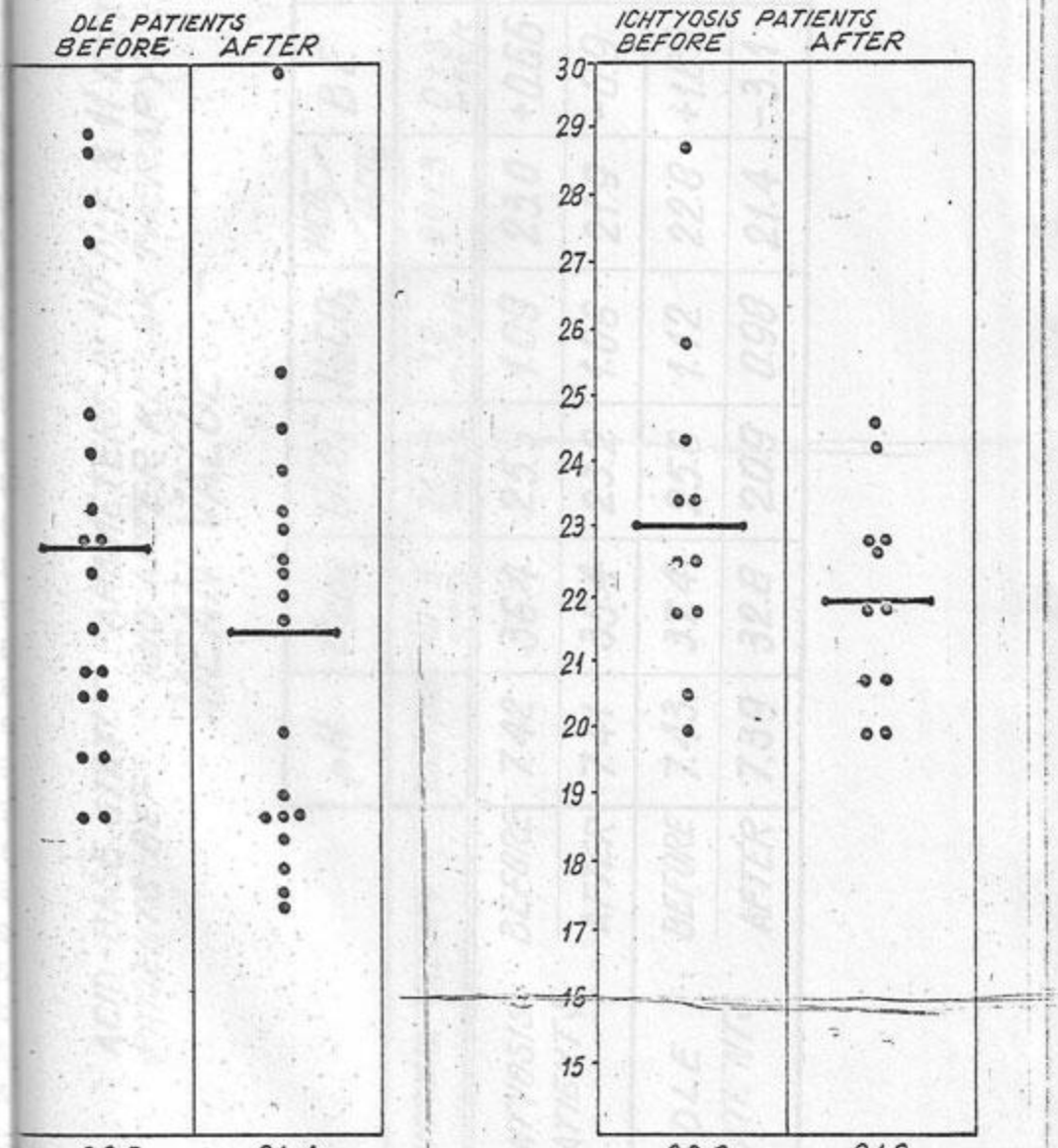
ICHTYOSIS PATIENTS BEFORE AND AFTER KOZAK THERAPY.



Normal range 40 ± 5 mm Hg.

Fig. 1

$CO_3]/[H_2CO_3]$ RATIO IN CHRONIC DISCOID LUPUS ERYTHEMATODES & ICTYOSIS PATIENTS BEFORE AND AFTER KOZAK THERAPY.



normal range 20 ± 3

Fig 9.

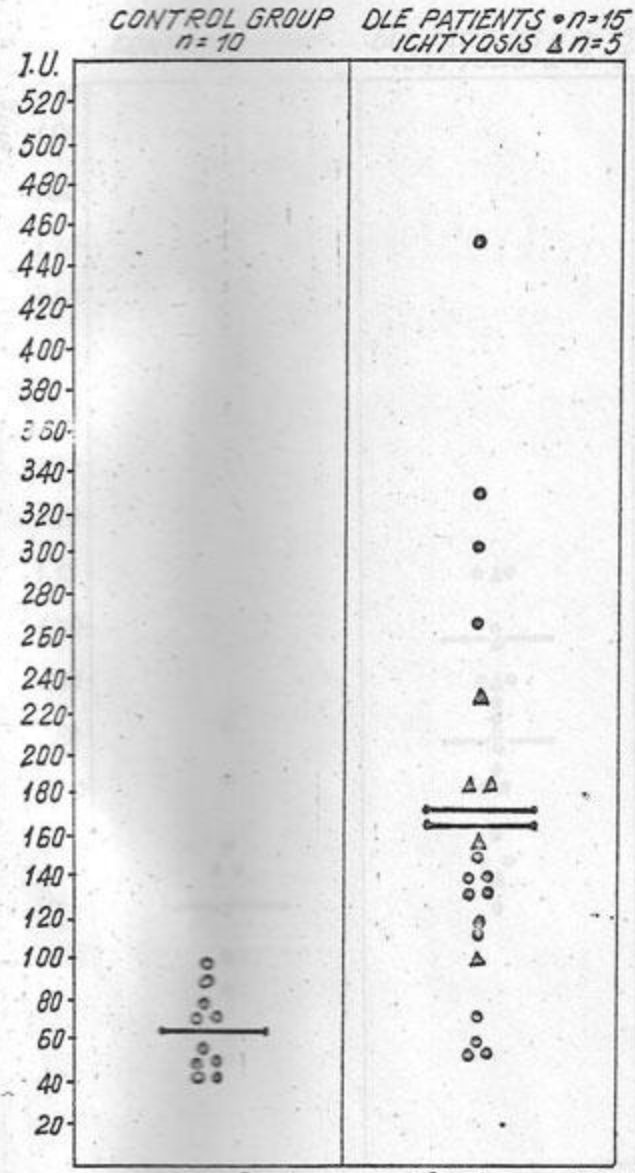
ACID-BASE STATUS PARAMETERS IN 19 DLE & 11 ICHTYOSIS PATIENTS BEFORE AND AFTER KOZAK THERAPY.

- MEAN VALUES -

		pH	P_{CO_2}	HCO_3^-	H_2CO_3	$\frac{HCO_3^-}{H_2CO_3^-}$	BE	BB
NORMAL VALUES		7.35-7.45	40±5 mmHg	24±3 mEq./l.	1.2 mEq./l	20±3	0±3 mEq./l	45±5 mEq./l
ICHTYOSIS PATIENTS	BEFORE	7.42	36.4	25.3	1.09	23.0	+0.66	50.2
	AFTER	7.41	35.4	23.2	1.06	21.9	-0.9	46.3
DLE PATIENTS	BEFORE	7.43	37.4	25.6	1.12	22.8	+1.89	49.8
	AFTER	7.39	32.8	20.9	0.98	21.4	-3.1	44.6

Fig. 3

P.H.I. ACTIVITY IN CHRONIC DISCOID LUPUS ERYTHEMATOSUS & ICHTYOSIS PATIENTS

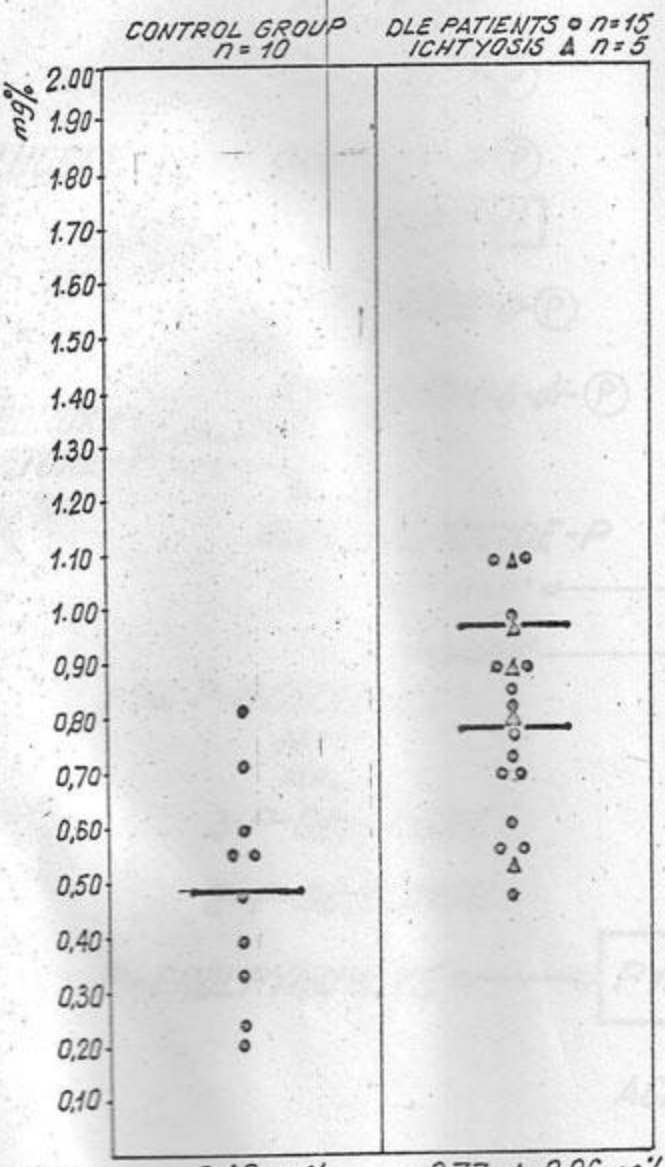


Mean 60.6 I.U. • 164.7 Δ 171.4 I.U.

Normal range 15 - 75 I.U.

Fig. 4

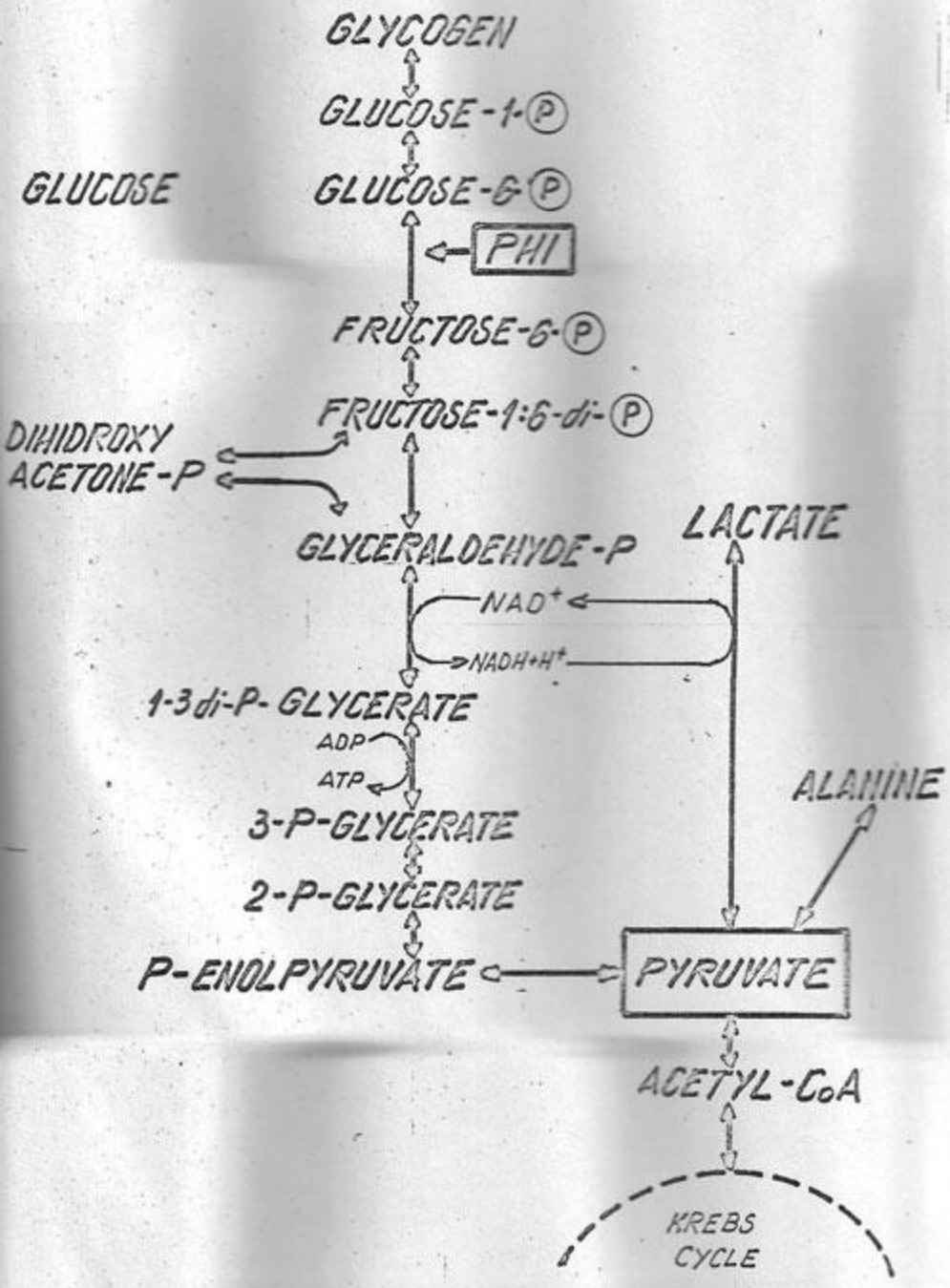
SERUM PYRUVATE VALUES IN CHRONIC DISCOID LUPUS ERYTHEMATOSUS & ICHTYOSIS PATIENTS.



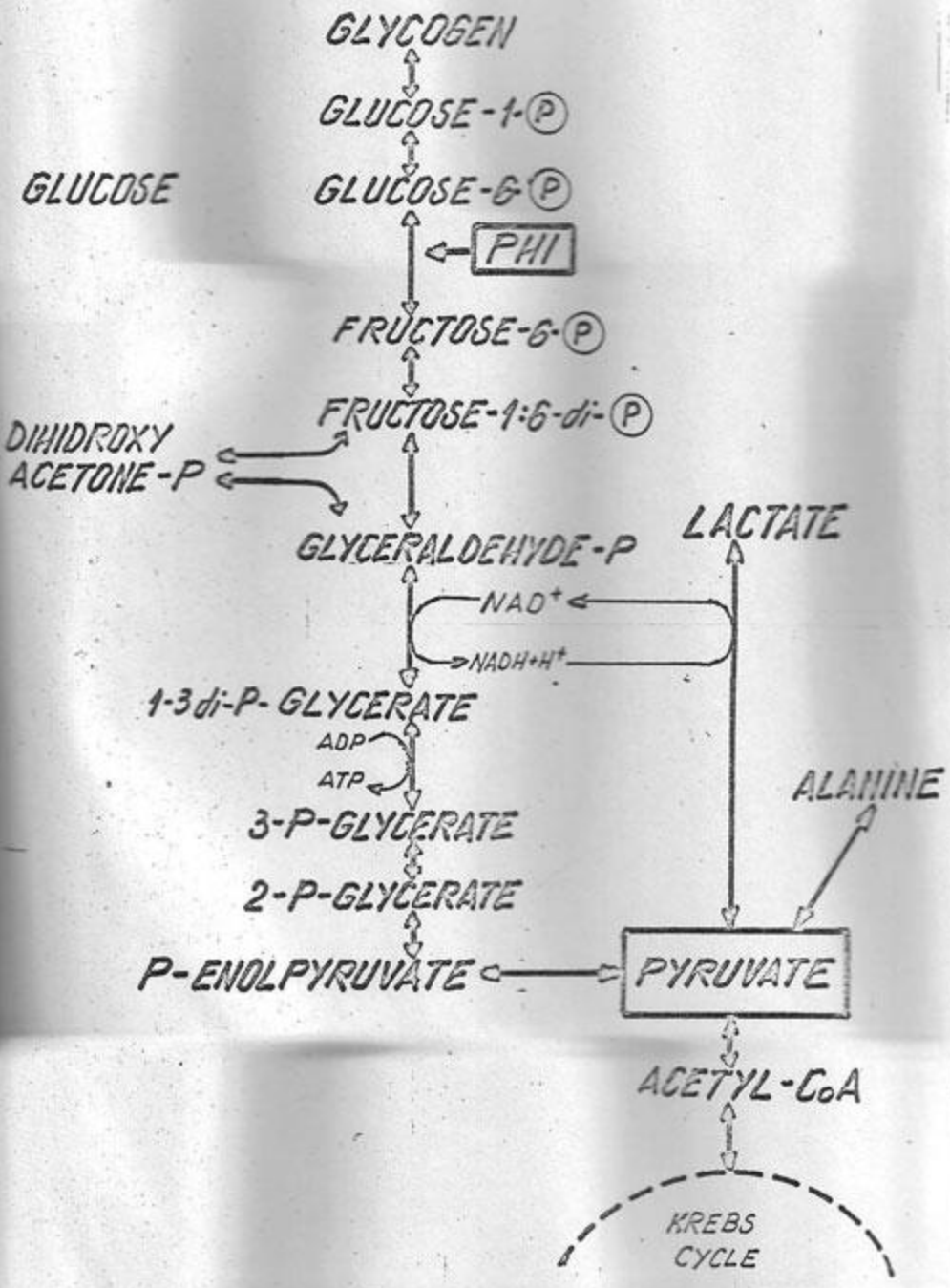
Normal range 0.36 - 0.59 mg. %

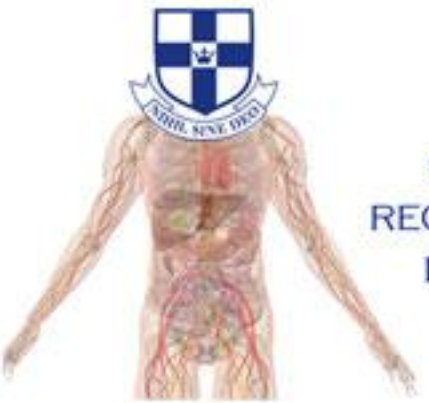
Fig. 5

EMBDEN-MEYERHOF PATHWAY



EMBDEN-MEYERHOF PATHWAY





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RECONSTITUIM SANATATEA
DIN INTERIORUL DVS..

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