mucicarmin or mucihematin in the cells. Using a special technic of fixation the same author demonstrated metachromatic granules in synovial cells with Toluidin blue. Our own attempts to stain "mucin" in vitreous humor were all futile. Furthermore the capsule of group A hemolytic streptococci, which at least to a large extent is composed of hyaluronic acid, can only be stained by mucin stains with special technics.[12]

It is not known whether the normal endothelium produces hyaluronic acid in small amounts. However, the usual exudates and transudates of the serous cavities are not viscous.

Conclusion. Hyaluronic acid in high concentration has been isolated from the chest fluid of a patient with a malignant tumor, probably an endothelioma. The polysaccharide acid in the original fluid migrates in an electric field at pH 7.8 at essentially the same speed as the isolated pure polysaccharide acid, indicating the existence in the fluid of the free acid and not of a protein complex.

11057 P

Non-Osseous Origins of Serum Phosphatase.

AARON BODANSKY.

From the Laboratory Division, Hospital for Joint Diseases, New York.

Associated increases of serum cholesterol, serum bilirubin and serum phosphatase have been observed in diseases of the liver with various degrees of obstruction of the biliary passages; *dissociated effects* have also been observed in other liver diseases.[1, 2] We have sought to reproduce analogous conditions and effects in experimental animals, in order to be able better to relate these dissociated effects to the underlying tissue changes.[3-6]

The effects of intravenous injections of gum acacia were of interest in this connection. We have obtained a marked *increase* of serum

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[2] Flood, C. A., Gutman, E. B., and Gutman, A. B., *Arch. Int. Med.*, 1937, **59**, 981; this paper reviews some earlier work.

[3] Bodansky, A., and Jaffe, H. L., PROC. SOC. EXP. BIOL. AND MED., 1934, **31**, 1179.

[4] Bodansky, A., *Proc. International Physiol. Cong.*, Leningrad, 1935, 253.

[5] Bodansky, A., *Enzymologia*, 1937, **3**, 258.

[6] Bodansky, A., *Proc. International Physiol. Cong.*, Geneva, 1938.

TABLE I.

Effects of a Single Intravenous Injection of 1.2 g of Gum Acacia per Kilo of Body Weight.
Some figures are in italics to show the time of occurrence of the more significant effects.

Day	Hematocrit, %	Sedimentation rate mm/hr	Cholesterol, mg/100 cc: Total	Esters	Free	Esters, % of total Cholest.	Proteins: Albumin, %	Globulin, %	A/G ratio	Total, %	Phosphatase units/100 cc
					Dog 652.						
—8	55	0	139	98	41	70	4.0	2.5	1.6	6.5	1.5
—4	53	2	144	105	39	73	3.8	2.5	1.5	6.3	1.8
1	48	*38*	*100*	*69*	*31*	69	3.1	*1.9*	1.6	*5.0*	7.2
3	51	31	110	78	32	71	2.9	2.3	1.3	5.2	*9.0*
6	55	8	122	85	37	70	3.2	2.6	1.2	5.8	7.2
10	50	2	134	93	41	69	3.0	2.7	1.1	5.7	2.6
14	49	8	153	108	45	71	3.5	2.3	1.5	5.8	2.3
29	52	1	178	125	53	70	3.8	2.5	1.5	6.3	2.6
					Dog 811.						
—8	55	0	156	111	45	71	4.3	2.1	2.0	6.4	2.3
—4	54	0	148	105	43	71	4.1	2.2	1.9	6.3	2.2
1	51	*33*	*100*	*67*	*33*	67	3.3	*1.5*	2.2	*4.8*	7.2
3	50	8	128	93	34	72	3.4	1.8	1.9	5.2	*8.0*
6	52	0	105	69	36	66	3.9	1.8	2.2	5.7	6.8
10	49	1	120	86	34	72	3.3	*2.2*	1.5	5.5	3.1
14	50	1	141	102	39	72	4.1	1.6	2.6	5.7	3.3
29	51	0	129	90	39	70	4.2	1.8	2.3	6.0	3.2

phosphatase activity, with a striking decrease of cholesterol and of the ratio of cholesterol esters to the total cholesterol—a ratio which is generally considered one of the significant indices of liver function.

Experiment 1. Two dogs, weighing about 12 kilos each, were fasted for 20 hours before the intravenous injection of 1.2 g of gum acacia (in 8% solution) per kilo of body weight. Table I includes the more significant results of a month's observation; the time of occurrence of maximum effects is of interest:

Anorexia, retching and vomiting, one or more days after the injection must be mentioned, particularly in relation to the serum protein changes. The *hematocrit reading* decreased slightly, and the *sedimentation rate* increased markedly within 24 hours. *Serum albumin* reached within 24 hours a level near which it remained for 10 days; *serum globulin* reached its minimum on the 1st day and rose to a maximum on the 10th day; the *total protein* values represented a summation of these effects. *Total serum cholesterol* decreased to a minimum within 24 hours, largely at the expense of the ester fraction. *Serum phosphatase* rose to a maximum on the 3rd day.

Recovery to the initial values was substantially completed at various times before the end of the experiment, except for the serum phosphatase which remained significantly higher. In one dog the hematocrit reading remained slightly below the initial value and the cholesterol ester declined slightly after reaching the initial level; in the other dog, both cholesterol fractions rose above the initial levels.

Experiment 2. Examination of Table I indicated immediate effects much greater than those observed after 24 hours. The experiment was repeated, with the same dogs. The results are given in Table II. The values observed after 2 and 6 hours were of particular interest.

The total serum proteins and the globulin fraction reached their minimum values within 2 hours; serum albumin decreased relatively less, and the subsequent changes were less definite than those of the globulin fraction. *Cholesterol esters* declined drastically within 2 to 6 hours; free cholesterol decreased in one dog and remained substantially constant in the other. Serum phosphatase rose about 50% within 2 hours.

Summary and Conclusions. Serum globulin participated relatively more in the changes of serum protein content, causing at first a decrease and then an increase of the albumin/globulin ratio before the final return to initial values. Cholesterol esters decreased absolutely and relatively more than the free cholesterol fraction. Serum phosphatase activity rose promptly, continued to rise for 3 days and

TABLE II.

Effects of a Second Intravenous Injection of Gum Acacia Solution, One Month After the First.

Hour	Hematocrit, %	Sedimentation rate mm/hr	Cholesterol, mg/100 cc: Total	Esters	Free	Esters, % of total Cholest.	Proteins: Albumin, %	Globulin, %	A/G ratio	Total, %	Phosphatase units/100 cc
					Dog 652.						
0	52	1	178	125	53	70	3.8	2.5	1.5	6.3	2.6
2	43	36	127	90	37	71	3.0	1.6	1.9	4.6	4.0
6	44	41	81	45	36	56	2.9	1.9	1.5	4.8	4.9
24	45	32	126	81	45	64	3.1	1.8	1.7	4.9	8.2
					Dog 811.						
0	51	0	129	90	39	70	4.2	1.8	2.3	6.0	3.2
2	48	3	68	34	34	50	3.7	1.4	2.6	5.1	4.4
6	47	10	99	57	42	58	3.8	1.5	2.5	5.3	5.3
24	50	0	124	83	41	67	3.9	1.7	2.3	5.6	6.2
48	54	0	134	97	37	73	4.0	1.6	2.5	5.6	6.1

was still above the initial levels a month after the injection of gum acacia; we consider this a manifestation of the reaction of the liver to injury. In absence of criteria which would enable us to identify in the serum the "alkaline" phosphatases of various origins, we regard the rise of serum phosphatase in liver involvements—particularly when there is no evidence of obstruction of the biliary passages—as an indication that the liver is a source of serum phosphatase. We assume that the "alkaline" phosphatase in the serum represents the sum total of contributions from various organs and tissues capable of producing "alkaline" phosphatase, the contribution of each being increased by an injury to which it is able to react.

This study, including histological investigations, is being continued.

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Cure and Prevention of Vitamin E-Deficient Muscular Dystrophy with Synthetic α-Tocopherol Acetate.

G. C. KNOWLTON, H. M. HINES AND K. M. BRINKHOUS.

From the Departments of Physiology and Pathology, College of Medicine, State University of Iowa.

It is now well established that adult rats reared from birth or weaning on a vitamin E-deficient diet eventually develop a muscular dystrophy.[1-3] It has also been shown that muscle changes, characteristic of dystrophy, can be detected before overt symptoms appear.[4] The criteria offered were (1) increase in the concentration of water and (2) of chlorides of the muscle, (3) decrease in the maximum strength, (4) focal hyaline necroses of muscle fibers, and (5) decrease in the creatine concentration of the muscle. Using the first 4 criteria it has been shown that wheat germ oil would prevent the development of the dystrophy.[4] It was later shown that wheat germ oil or its vitamin E-active concentrate would induce recovery from

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2 Burr, G. O., Brown, W. R., and Moseley, R. L., PROC. SOC. EXP. BIOL. AND MED., 1937, **36**, 780.

3 Ringsted, A., *Biochem. J.*, 1935, **29**, 788.

4 Knowlton, G. C., and Hines, H. M., PROC. SOC. EXP. BIOL. AND MED., 1938, **38**, 655.