body temperature had declined to about this temperature level (Table III). Evidence that 36° C is a temperature in some way important to human thermoregulation has been obtained by others. For example, the results obtained by Burton and Bazett[1] in experiments in which subjects were immersed in relatively warm water showed that metabolic heat production increased above basal level as rectal temperature approached 36° C. DuBois[2] found 36° C to be approximately the lower limit of a series of rectal temperature measurements obtained on a large group of hospital patients. Careful observation under a variety of experimental conditions might elucidate in greater detail the significance to human thermoregulation of this seemingly important deep body temperature level.

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Tolerance Curve of Vitamin C.*

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The suggestion has been made that intravenous injection of vitamin C, preoperatively, might be of value in preventing the resultant traumatic shock.[1-3] Oral administration has a more prolonged effect upon the blood ascorbic acid concentration and results in greater retention of the vitamin.[4-9] This study was made in an attempt to determine the quantity of synthetic ascorbic acid which, on ingestion, would produce a high concentration of the vitamin in the blood plasma and maintain an increase for a relatively long period of time.

The results of 80 tolerance curves are herein reported. With a lapse of at least one week between each observation, 12 young college women were given 250, 500, 750 and 1000 mg of ascorbic acid, orally. All tests began early in the morning, with omission of breakfast. Lunch was limited, consisting only of vitamin C-free foods. Fasting bloods were taken just before ingestion of the test dose, and subsequent blood samples were drawn each hour thereafter for 6 to 10 hours, depending upon the amount of synthetic vitamin given. Determination of the vitamin C content of the blood plasma was made by the Farmer-Abt micro-method.[10]

The 80 fasting values thus obtained ranged from 0.36 mg to 1.36 mg of ascorbic acid per 100 cc of plasma, the average concentration being 0.79 mg %. During the 3-month period

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the fasting values changed very little for any individual. The response to a test dose varied from subject to subject, but neither the amount of increase in plasma ascorbic acid concentration nor its duration were directly related to the concentration of the vitamin in the fasting sample. Tolerance curves of subjects with the lower initial blood plasma levels showed as much rise above fasting as those in which the initial values were higher. In some cases the plasma level remained on a plateau for one to 3 hours after the peak was reached. In other instances the drop from the peak was more pronounced.

The maximum increase in the ascorbic acid concentration occurred one to 4 hours after a test dose was given. After ingestion of 250 mg of vitamin C the plasma levels of most subjects reached their highest point in one or 2 hours. Following the administration of 500, 750, or 1000 mg, peaks were attained, in most instances, in 2 or 3 hours.

A return of the plasma ascorbic acid to approximately the initial level took place, in the majority of cases, 6 hours after 250 mg of the synthetic material was given. Following the administration of 500, 750, or 1000 mg of the vitamin, a slight elevation was maintained for several more hours.

The average increases in the plasma vitamin C content at each hour after the ingestion of 250, 500, 750, and 1000 mg of ascorbic acid are plotted in Fig. 1. Comparison shows that ingestion of 250 mg of synthetic vitamin C did not have as great or as long-lasting an effect as a larger amount. The tolerance curves obtained after the ingestion of 500, 750, and 1000 mg of ascorbic acid are analogous. When these average curves are compared, it is interesting to note that there is less than 0.1 mg variation at any point throughout the 10 hours.

The ingestion of 500 to 1000 mg of synthetic ascorbic acid resulted in similar in-

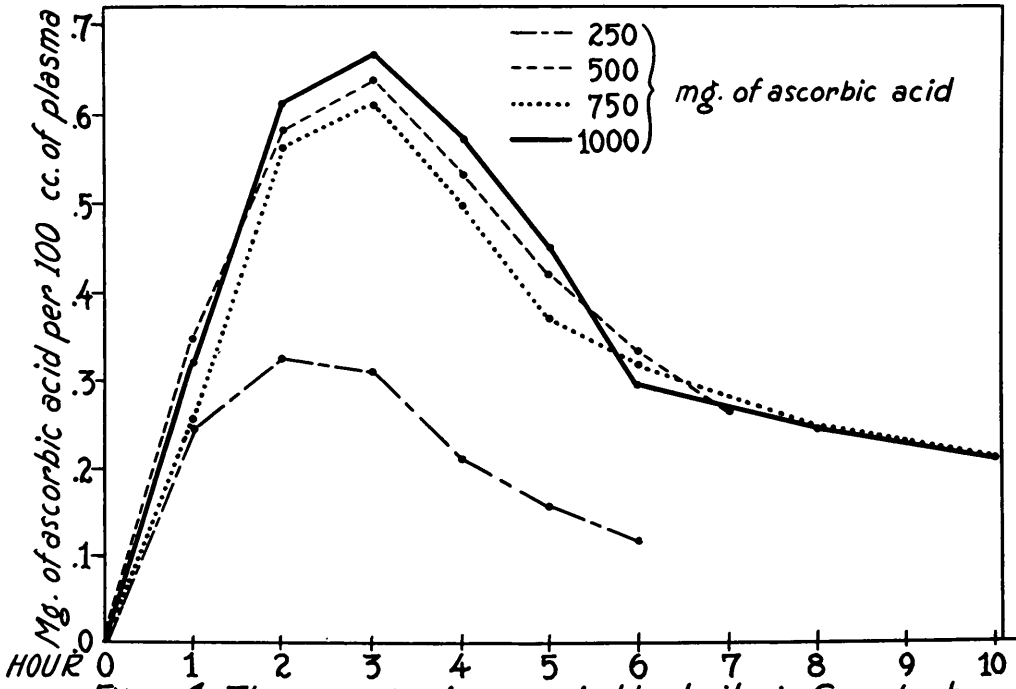

Figure 1. The average increase in blood vitamin C content following ingestion of large amounts of ascorbic acid; 4 curves, each based upon 20 individual observations, the result of giving 250, 500, 750, and 1000 mg. of synthetic ascorbic acid.

creases in the plasma concentration of the vitamin. However, data furnished by concurrent observations[11] on the amount of ascorbic acid *excreted* by the subjects of this study, showed that with the greater intake of the vitamin, more was retained over a 12-hour period.

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Effect of Protein Depletion on Plasma Proteins in the Dog Measured by Electrophoretic Analysis.

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Electrophoresis has become a valuable tool for studies of the effect on plasma proteins of disease and other abnormalities in man or experimental animals. Immune sera[1] and sera of patients suffering from diseases such as multiple myelomatosis,[2] pneumonia, peritonitis, acute lymphatic leukemia, etc.[3,4] show electrophoretic patterns differing from the normal. Although most diseases produce a rise in α globulin content of the sera, a rise in β_2 and γ fractions but not in α globulin has been reported in rheumatic fever.[4] In many of these studies the alteration of plasma proteins may have been caused in part by protein depletion as well as by other metabolic shifts attributed to disease. There have been no reports published on the effect of protein depletion alone on the plasma electrophoretic pattern although there is need for such data on the plasma of normal and experimental animals, particularly the dog. In this animal, which has been used extensively in studies on hypoproteinemia and plasma protein regeneration, it has been demonstrated by chemical precipitation methods[5–7] that protein depletion results in a loss of albumin greater than that of the other plasma proteins. In the following are presented the results of studies of the electrophoretic patterns of plasma of dogs before and after depletion, both by protein-free feeding and by plasmapheresis.

Methods. The dogs were depleted of protein by a protein-free diet[8] for 6 to 10 days, after which plasmapheresis was performed for 2 to 5 days. During these days, one-quarter of the blood volume of the dog was removed daily, the cells being returned suspended in saline. This period of plasmapheresis was followed by protein-free feeding alone until the plasma protein concentration was relatively constant at 4.0 to 4.5 g per 100 ml of plasma.

The plasma was analyzed for total nitrogen and non-protein nitrogen by the micro Kjeldahl method. The technic of Howe (1921) as modified by Robinson, Price, and Hogden,[9]

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