hydrogen-deuterium exchange is 3:1 (primary:secondary) which is the dotted line in Fig. 3. The results show that the distribution is more nearly 1:1. This indicates that deuterium exchanges preferentially at the secondary position. A quantitative measure of this effect must await further detailed information about the electron dissociation patterns of the individual deuterated compounds.

References

1. FARKAS and FARKAS. *J. Am. Chem. Soc.*, **61**, 1336 (1939).
2. TAYLOR, H. S. *Frontiers in Chemistry*. New York: Interscience Pub. (1945).
3. TURKEVICH and FRIEDMAN. Studies of the Catalytic Reduction of Acetone. Presented at the 116th meeting ACS, Atlantic City, Sept. 18–23, 1949.
4. MORIKAWA, BENEDICT, and TAYLOR. *J. Am. Chem. Soc.*, **57**, 592 (1935); *ibid.*, **58**, 1445, 1795 (1936).
5. FARKAS. *Trans. Faraday Soc.*, **35**, 906 (1939).
6. MELVILLE and BOLLAND. *Proc. Roy. Soc. (London)*, **A160**, 384 (1937).
7. TURKEVICH. *Brookhaven Conference Report* (1948).
8. TURKEVICH, *et al. J. Am. Chem. Soc.*, **70**, 2638 (1948).

Adrenal Cholesterol in the Scorbutic Guinea Pig

M. Jane Oesterling[1] and C. N. H. Long

Department of Physiological Chemistry, Yale School of Medicine, New Haven, Connecticut

Many different stress situations have been shown to evoke in the adrenal of normal animals a response similar to that which occurs following injection of adrenocorticotrophic hormone (ACTH). This response is characterized by a marked fall in the adrenal concentrations of both ascorbic acid and cholesterol. Among the stimuli that have been studied in this regard are the following: hemorrhage (*1*), exposure to cold (*2*), electrical stimulation of peripheral nerves (*2*), burns (*3*), and infection (*4*). Considerations that have been elaborated elsewhere (*5, 6*) appear to justify the use of this response as an indication of the secretion of cortical hormones.

In the present work a study has been made of the effect of stress caused by vitamin C deficiency on the level of cholesterol in the adrenal. Paired-feeding was used in order to distinguish the effect of the vitamin deficiency per se from that of the low calorie intake which accompanies the deficiency in its advanced stages. Two stages of the vitamin deficiency have been studied, both of which are characterized by severe adrenal depletion of vitamin C. The first stage is designated as "early scurvy" and the second as "late scurvy." In the former, inanition has not become a complicating factor in the disease, whereas in the latter, very low food intake accompanies the deficiency. The adrenal is approximately 95% depleted of its vitamin C in the first stage and 98% in the second.

Early scurvy is herein defined as that stage of scurvy occurring when the growth curve reaches a plateau, there is beginning failure of appetite, and the concentration of ascorbic acid in the adrenal is less than 10 mg %. This condition was reached in the present experiments by 17 days after the last vitamin C injection. The concentrations of ascorbic acid in various tissues of early scurvy animals compared with those of late scurvy and normal guinea pigs are shown in Table 1.

TABLE 1

TISSUE VITAMIN C LEVELS IN NORMAL, EARLY SCURVY, AND LATE SCURVY GUINEA PIGS
(Mg/100 g)

	Normal guinea pigs		Early scurvy† pigs		Late scurvy‡ pigs
	Before ACTH	After ACTH§	Before ACTH	After ACTH§	
Adrenal	164.7 ± 7.0*	90.4 ± 4.9	6.02 ± 2.00	6.33 ± 0.85	1.87 ± 0.53
Brain	20.4 ± 0.8	18.0 ± 1.0	8.93 ± 1.10	8.10 ± 0.52	3.21 ± 0.10
Testis	36.5 ± 1.1	34.7 ± 1.9	6.76 ± 0.62	6.42 ± 0.31	2.90 ± 0.32
Spleen	53.8 ± 1.9	44.6 ± 1.7	3.84 ± 0.66	3.69 ± 0.25	.66 ± .12
Kidney	9.19 ± 0.38	7.81 ± 0.79	.59 ± .07	.66 ± .04	.29 ± .06
Liver	19.2 ± 1.1	20.2 ± 2.2	.51 ± .11	.72 ± .15	.25 ± .05
Whole blood	0.62 ± 0.07	0.59 ± 0.05	.07 ± 0.01	.09 ± 0.01	.04 ± 0.02

* Standard error of mean.
† Sacrificed 17 days after last vitamin C injection.
‡ Sacrificed 27–37 days after last vitamin C injection.
§ Sacrificed 6 hr after intraperitoneal injection of 1 mg/100 g of body weight.

Late scurvy is defined for the purposes of this experiment as that stage of scurvy occurring when the vitamin C of such tissues as brain and testis, which are the most slowly depleted of any tissues analyzed in this work, has reached a level below which further significant reduction is difficult to demonstrate; i.e., 80% depletion for brain tissue and 90% for testis. This stage was reached 27 days after the last vitamin C injection. At this time there has been rather rapid weight loss for 9 or 10 days. Gross signs of scurvy are apparent.

Guinea pigs varying in weight from 350 to 500 g, caged individually, were kept at an environmental temperature of 82° ± 2° F and fed a diet (*7*) containing calf meal, 91%; wheat germ, 3%; dried brewers yeast, 3%; and cod liver oil, 3%. Vitamin C was supplied by daily subcutaneous injections of neutralized ascorbic acid (Cevalin #319, Lilly) equivalent to 5 mg/100 g of body weight. Only animals which grew well and remained free of respiratory infections were used.

In order to ensure a state of "tissue saturation," each animal was given a preliminary series of daily injections for at least one week prior to beginning the deficient regime. Each animal on the deficient regime

[1] Marion Talbot Fellow of the American Association of University Women during the initial part of this work and Alexander Brown Coxe Memorial Fellow of Yale University during the remainder. Present address: Karolinska Institutet, Fysiologiska Institutionen, Stockholm 60, Sweden.

TABLE 2

ADRENAL CHOLESTEROL
(Percentage)

Before ACTH			After ACTH§	
Pair-fed controls	Early scurvy†		Pair-fed controls	Early scurvy
5.56 ± 0.20*	7.09 ± 0.22	$p < 0.01$	4.06 ± 0.39	4.04 ± 0.38
Pair-fed controls	Late scurvy‡			
5.72 ± 0.48	2.56 ± 0.42	$p < 0.01$		

* Standard error of mean.
† Sacrificed 17 days after the last vitamin C injection.
‡ Sacrificed 27–37 days after the last vitamin C injection.
§ Sacrificed 6 hr after intraperitoneal injection of 1 mg/100 g of body weight.

was paired with a control animal of approximately the same weight, and each control animal was carefully limited in food intake to the amount eaten on the previous day by the deficient animal. An interval of 12–18 hr was allowed to elapse between the last vitamin C injection and the time of sacrificing each control animal.

When ready to be sacrificed, each animal was anesthetized with Nembutal (5 mg/100 g of body weight dissolved in normal saline) injected intraperitoneally. Blood for vitamin C analysis was obtained by heart puncture while the animal was still breathing. The abdomen was opened immediately thereafter, and the left adrenal rapidly removed for analysis by the method of Roe and Kuether (*8, 9*). The right adrenal was used for cholesterol analysis by the method of Schoenheimer and Sperry, as described by Sperry (*10*). The following tissues were also analyzed for vitamin C by the method of Roe and Kuether: liver, kidney, spleen, brain, and testis.

It has been found that in early scurvy the adrenal cholesterol concentration, rather than being reduced, is significantly higher than that of the controls (Table 2). The adrenal at this stage, although 95% depleted of vitamin C, responds to injection of ACTH (*11*) by showing a 42% reduction in cholesterol concentration after 6 hr ($p < 0.01$). Adrenal ascorbic acid was not further reduced in the scorbutic animals by injection of ACTH (Table 1). However, in the control animals a highly significant reduction was noted ($p < 0.001$).

In late scurvy, there is a marked decrease in adrenal cholesterol compared with that of the pair-fed controls (Table 2). This observation is at variance with a report of Baldwin, Longenecker, and King (*12*), who found no significant difference in adrenal cholesterol concentration[2] between the animals "in the last stages of scurvy" (1.70%) and the pair-fed controls (1.64%), both groups showing remarkably low levels. In view of the fact that the adrenal cholesterol may be lowered in response to a wide variety of stress situations, it is suggested that some unnamed stress may have been operating in the experiments of Baldwin *et al.* The use of chloroform anesthesia by these workers may have been in part responsible for the extremely low cholesterol values, since volatile anesthetics are likely to lead to respiratory anoxia, unless very carefully administered, and such a condition causes rapid reduction of adrenal ascorbic acid and cholesterol. The use of an anesthetic such as Nembutal dissolved in normal saline and warmed to body temperature before intraperitoneal injection has been found to be much the safer procedure (*2*).

Other explanations of the conflicting results are possible; for example, younger animals were used by Baldwin *et al.*, and the ability to withstand severe restrictions in food intake is much less in the young than in older animals. It should be emphasized that the present study was not concerned with terminal fasting, the control animals being far from moribund at the time they were sacrificed. It seems clear that the degree of stress imposed by the restricted food intake is much less than that imposed by the vitamin deficiency in its advanced stage.

[2] The adrenal cholesterol is reported by Baldwin *et al.* in terms of its percentage of the total adrenal lipid, the scurvy animals showing an average of 8.3, and the pair-fed controls, 6.9. The adrenal lipid for the two groups was 20.5% and 23.7%, respectively. Thus, the adrenal cholesterol expressed in g/100 g of adrenal tissue is $20.5 \times 8.3/100 = 1.70$ for the scurvy animals and $23.7 \times 6.9/100 = 1.64$ for the pair-fed controls.

References

1. SAYERS, G., *et al. Endocrinology,* **37**, 96 (1945).
2. LONG, C. N. H. *Recent Progress in Hormone Research,* **1**, 99 (1947).
3. HARKINS, H. N., and LONG, C. N. H. *Am. J. Physiol.,* **144**, 661 (1945).
4. PINCHOT, G. B., CLOSE, V. P., and LONG, C. N. H. *Endocrinology,* **45**, 135 (1949).
5. LONG, C. N. H. *Bull. N. Y. Acad. Med.,* **23**, 260 (1947).
6. ———. *Federation Proc.,* **6**, 461 (1947).
7. MENDEL, L. B., and HUBBELL, R. B. *J. Nutrition,* **10**, 557 (1935).
8. ROE, J. H., and KUETHER, C. A. *J. Biol. Chem.,* **147**, 399 (1942).
9. KUETHER, C. A., TELFORD, I. R., and ROE, J. H. *J. Nutrition,* **28**, 347 (1944).
10. SPERRY, W. M. *J. Clin. Path.,* Tech. Suppl., **2**, 91 (1938).
11. SAYERS, G., WHITE, A., and LONG, C. N. H. *J. Biol. Chem.,* **149**, 425 (1943).
12. BALDWIN, R. A., LONGENECKER, H. E., and KING, C. G. *Arch. Biochem.,* **5**, 137 (1944).

A Universal Motion Analyzer Applied to Psychomotor Performance

Karl U. Smith and Robert Wehrkamp

Department of Psychology,
University of Wisconsin, Madison

It has been recognized for some time that improved methods are needed for motion analysis and dimensional measurement of motor skills and psychomotor activities involved in typical domestic, industrial, and military performances. This report describes methods of motion analysis which provide: (*a*) automatic registration of elapsed time in the performance of any simple or complex motor task; (*b*) separate automatic registration of the different components of travel time and manipulation, or time involved in the