decreased, that is, the kidney initially had a high resistance which fell as the renal volume decreased.

In some experiments the renal venous hæmatocrit was continuously measured by a conductivity method.[7] As might be expected, the hæmatocrit increased with a rise in pressure and fell with a drop in pressure. This effect was less pronounced in the autoregulatory range than below it, a finding indicating that, in the autoregulatory range, the kidney does not sequester plasma as pressure increases. When autoregulation was abolished by cyanide, the vascular distensibility and the distensibility of the kidney both decreased.

These results conflict with those in which intrarenal pressure did not vary with renal resistance.[8] The fact that renal volume changes parallel renal flow changes is in accord with the possibility that the normal kidney exhibits autoregulation of flow because extravascular fluid compresses some low-pressure vessels. This mechanism has previously been considered unacceptable.[9]

I would like to thank Mr. O. F. Brown for his technical assistance.

Allen M. Scher
Department of Physiology and Biophysics,
School of Medicine,
University of Washington,
Seattle 5, Washington.

[1] Winton, F. R., Trans. Fourteenth Congresso Internaz. de Fisiol., 264 (1932).
[2] Shipley, R. E., and Study, R. S., *Amer. J. Physiol.*, **167**, 676 (1951).
[3] Ochwadt, B., *Pfluger's Arch. ges. Physiol.*, **262**, 207 (1956).
[4] Pappenheimer, J. R., and Kinter, W. B., *Amer. J. Physiol.*, **185**, 377 (1956).
[5] Thompson, D. D., Kavaler, F., Lozano, R., and Pitts, R. F., *Amer. J. Physiol.*, **191**, 493 (1957).
[6] Miles, B. E., Ventom, M. G., and de Wardener, H. E., *J. Physiol.*, **123**, 143 (1954).
[7] Waugh, W. H., *Circ. Res.*, **6**, 363 (1958).
[8] Goodwin, R. S., and Sapirstein, L. A., *Circ. Res.*, **5**, 532 (1957).
[9] Winton, F. R., Harvey Lecture, **47**, 21 (1951–52).

Carbohydrate Metabolism in Hypervitaminosis A.

Excess vitamin A ingestion increases bleeding tendency[1], depresses basal metabolism[2] and increases excretion of neutral 17-ketosteroids in urine of albino rats[3]. The Qo_2 of liver slices of hypervitaminotic A rats is lower than that of control rats (Ray, Amal and Sadhu, D. P., unpublished observations), the weight of the thyroid is diminished and that of the adrenal increased[2]. In an attempt to elucidate the mechanism of hypometabolism induced in hypervitaminosis A, liver was studied for glycogen and fat contents and diaphragm as an index of glucose utilization in peripheral tissues.

Twelve young albino rats of 55 - 60 gm. weight were fed 30,000 I.U. vitamin A ('Arovit' Roche) daily for 10 days and were fed with twelve control rats and were killed by decapitation. A small piece from the upper part of the right lobe of the liver was taken for estimation of glycogen and fat content by a method previously described[4]. The diaphragm was divided into two halves and each hemidiaphragm was used for studying glucose utilization and glycogen synthesis[5] and the values compared with that of the pair-fed control rats. In hypervitaminosis A liver glycogen is decreased from the control value of $15{\cdot}1 \pm 1{\cdot}8$ (standard deviation) mgm. per gm. liver tissue to $12{\cdot}2 \pm 0{\cdot}93$ mgm. while the fat percentage is increased from control $8{\cdot}1 \pm 0{\cdot}71$ to $10{\cdot}1 \pm 0{\cdot}72$. Diaphragms show a decrease of glucose utilization from the control value of $0{\cdot}31 \pm 0{\cdot}02$ mgm. 100 mgm. wet diaphragm per hour to $0{\cdot}213 \pm 0{\cdot}05$ mgm., while glycogen synthesis which is $0{\cdot}163 \pm 0{\cdot}024$ mgm./100 mgm. wet diaphragm per hour in the control rats is decreased to $0{\cdot}068 \pm 0{\cdot}013$ mgm. in the hypervitaminotic A rats.

These experiments show that the depression of metabolism is not restricted to liver alone, but that in muscles is also depressed, in spite of hyperthyroxinaemia in hypervitaminosis[6] A.

We are grateful to Voltas Ltd. (India) and to Hoffman La Roche for a generous grant of 'Arovit' and to Principal K. C. Mukerjee for advice and interest.

Amal Ray
D. P. Sadhu
Department of Physiology and Nutrition,
Bengal Veterinary College,
Calcutta-37, India.

[1] Poole, J. C. F., *Quart. J. Exp. Physiol.*, 43, 427 (1958).
[2] Sadhu, D. P., and Brody, S., *Fed. Proc.*, 6, 420 (1947); *Amer. J. Physiol.*, 149, 400 (1947).
[3] Ray, Amal, and Sadhu, D. P., *Ind. J. Physiol. and Allied Sci.*, 11, 1 (1957).
[4] Sadhu, D. P., *Nature*, 177, 1236 (1956).
[5] Garner, R. J., and Roberts, R., *Biochem. J.*, 59, 224 (1955).
[6] Sadhu, D. P., and Kruscott, B. Lionel, *Endocrinol.*, 43, 120 (1948).

Effect of Thioctic Acid on Gain in Body-Weight by Turkey Poults

Thioctic acid (lipoic acid, DL-6-8 dithiooctinoic acid) is known to be a component of certain enzyme systems. It is also required as a growth factor by *Streptococcus fœcalis* in certain synthetic media, if this organism is to oxidize pyruvate successfully. Certain other bacteria, such as *Escherichia coli*, that oxidize pyruvate also require thioctic acid[1]. It has been tested for its ability to stimulate growth in higher animals; but the results have not been uniform. Positive results were obtained by DeBusk and Williams[2] with rats and chicks. In their experiments a growth response was obtained whether the basal ration was a practical corn-soybean-alfalfa meal type or a purified sucrose–alcohol extracted casein-gelatin type. Feed efficiency was also improved with both the chicks and the rats.

Briggs and Fox[3] afterwards reviewed the literature up to 1957 and initiated another experiment with chicks. They could obtain no evidence of a growth stimulation when semi-purified or practical diets were supplemented with thioctic acid. These workers concluded that thioctic acid could not be considered as an animal growth factor.

Kratzer *et al.*[4] at a later date did obtain a slight growth response to thioctic acid with turkey poults; but the response was not statistically significant. There is a possibility that the turkey poult may differ from the chick in its requirements or in its ability to synthesize micronutrients. The present experiment was set up in an attempt to clarify further the role of this factor in turkey poult nutrition.

A practical poult starter ration, currently in use at this laboratory, was supplemented with thioctic acid at a level of 7 mgm./kgm. This poult starter contained ground wheat and barley, soybean meal, fish meal, meat scrap and alfalfa meal. It was fortified with vitamin and mineral supplements in accordance with general recommendations for this type of diet. Procaine penicillin was added at 9 mgm./kgm. Each dietary treatment was replicated

Table 1. Effect of Thioctic Acid on Poult Growth to Six Weeks of Age

Dietary treatment	Body-weight (gm.)	
	Males	Females
Poult starter	1,152	998
Poult starter plus thioctic acid (7 mgm./kgm.)	1,160	971