this sort was not observed after administration of any dose of ether or nembutal. Eserine, on the other hand, does enhance two-neuron arc discharges. This is in accord with the excitant action of eserine on the knee jerk reported by Schweitzer and Wright.[4] If the two-neuron arc discharges have previously been enhanced by morphine, they are still further enhanced by a subsequent injection of eserine. Multineuron arc discharges in previously untreated preparations are little affected by eserine. If, however, eserine is injected after the multineuron arc discharges have been depressed by morphine, enhancement of these discharges occurs—an effect similar to that exerted by large doses of morphine.

Summary. 1. In the spinal cat, small doses of morphine (5 mg per kg) enhanced two-neuron arc reflex discharges and depressed those traversing multineuron arcs. After the injection of larger doses (15 mg per kg), depression of multineuron arc discharges was followed by enhancement of these discharges.

2. Nembutal depressed both two-neuron and multineuron arc discharges. These effects were unaltered by previous injection of eserine or morphine plus eserine, and increased progressively with the dose of nembutal.

3. Ether depressed both two-neuron and multineuron reflex arc discharges.

4. Eserine enhanced two-neuron arc discharges, but had little effect on multineuron arc discharges. If, however, morphine had been injected previously, resulting in enhancement of two-neuron and depression of multineuron arc discharges, eserine caused enhancement of both types of reflex arc discharges.

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Hypertensin Destruction by Oxidized Cytochrome.

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The level of hypertensin in blood plasma depends on the relative velocity of its production and destruction reactions.[1]

I. Renin + Hypertensinogen—→Hypertensin (Anaerobic reaction)

I. Hypertensin + Enzyme ⟨ (Protease)—→Amino Acids (Anaerobic reaction); (Oxidase)—→ X (Aerobic reaction)

Under certain conditions, the first reaction is inhibited by some substances found in Vitamin A concentrates,[2] and by some quinones, as methylnaphthoquinone that can stop it completely.[3,4]

So far, the second reaction has been considered to depend on an anaerobic proteolysis only. In fact, most of the organs, mainly the intestinal mucosa, the kidney and the liver destroy hypertensin at different speeds. *In vitro* it has been found that hypertensin is destroyed by renal and intestinal proteinase,[5] by an amino polypeptidase found in renal extracts[6] and in blood plasma,[7] by tyrosinase[8] and by carboxypolypeptidase.[9]

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[4] Cruz-Coke, E., and Croxatto H., *Bol. Soc. Biol.*, Santiago, 1945, in press.

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[7] Croxatto, H., and Croxatto, R., *Bol. Soc. Biol.*, Santiago, 1945, in press.

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TABLE I.
Average reduction rate of renal filtration in 13 toads' kidneys.

Hypertensin	60.5%
Hypertensin + reduced cytochrome	70.8%
Hypertensin + oxidized cytochrome	0.

The correlation between the start of the hypertensin activity on the organism and the ischemic and anoxemic phenomena in the kidney, led us to consider that respiration played an important part in the destruction of hypertensin. We investigated this effect, using oxidized cytochrome prepared according to Keilin and Hartree[10] on a Loewen-Trendelenburg[11, 12] preparation, and in a perfused toad's kidney using Plaza de los Reyes technique.[13] To measure the hypertensin activity, the rate of renal filtrate from this preparation was used as a test in a Ringer's circuit without calcium.

Table I. shows the average reduction of this renal filtrate rate in 13 tests. This reduction proves that oxidized cytochrome inhibits the effects of the hypertensive vaso constrictor properties of hypertensin in the kidneys of toads.

TABLE II.

Time of Reduction of oxidized cytochrome, in presence of:	
Renal tissue	24 hrs.
Hypertensin	24 "
Renal tissue + hypertensin	15 min.

Oxidized cytochrome cannot be reduced by renal tissue or hypertensin separately, but, when renal tissue and hypertensin are present together cytochrome is reduced as shown in Table II.

This reduction of cytochrome in the presence of renal tissue and hypertensin is parallel with the disappearance of the vaso constrictor properties of hypertensin (Fig. 1).

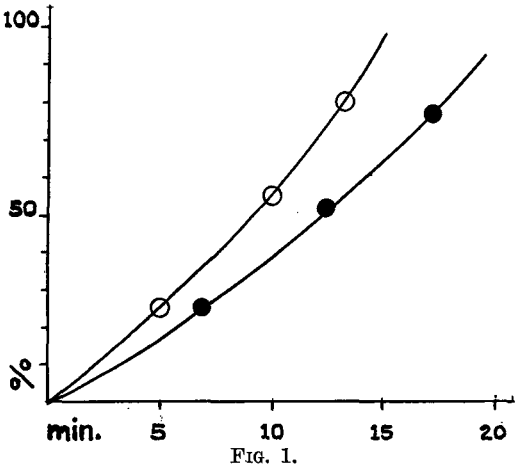

FIG. 1.
Shows parallelism of reduction of cytochrome and inactivation of hypertensin.
○ Reduction of cytochrome.
● Inactivation of hypertensin in the presence of cytochrome.
Ordinates in Figs. 1, 2, 3 and 4 represent percentage reduction in hypertensin.

The action of oxidized cytochrome on destruction of hypertensin by renal tissue was also investigated in the cat. The destruction of hypertensin by anaerobic proteolysis is usually a rather slow process and takes about an hour's time, unless unusually high amounts of enzyme are used. When oxidized cytochrome is added to renal tissue, a rapid de-

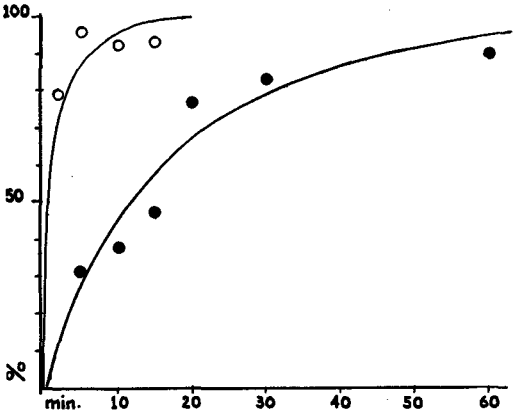

FIG. 2.
Shows that when oxidized cytochrome is added, hypertensin is destroyed much faster.
● Control, *i.e.*, destruction of hypertensin without oxidized cytochrome.
○ Destruction when oxidized cytochrome is added.

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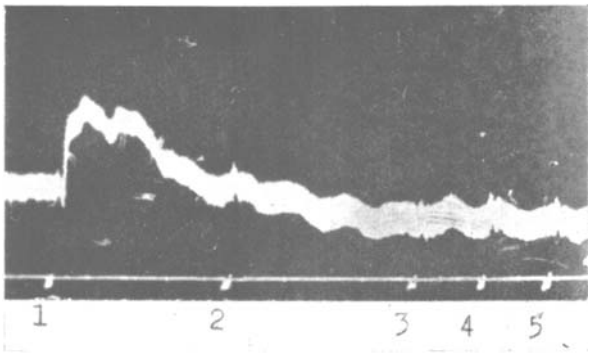

FIG. 3.
This figure represents a typical experiment in which 0.1 cc hypertensin and 0.2 cc renal extract, incubated for different lengths of time, were injected as follows:

No.	Incubation time in seconds	Elevation of carotid arterial pressure mm
1	2	17
2	2	3
3	5	1
4	10	3
5	15	2

At No. 1 no cytochrome was added and the pressure was sharply increased. At 2, 3, 4 and 5 cytochrome was added, and no such increase occurred.

struction of the pressor hormone occurred in a few minutes when tested in the cat. Fig. 2 and 3 graphically show these results.

When 1 cc of N/100 of cysteine or of ascorbic acid was added to the hypertensin renal extract and cytochrome, the hypertensin destruction was sharply retarded as seen in Fig. 4. It is evident that cysteine and ascorbic acid retarded considerably the destruction of hypertensin in both cases *i. e.* with or without cytochrome.

In these experiments renin was prepared according to Swingle, Taylor, Collings and Hays.[14] The hypertensin was obtained by the method of Braun Menendez, Fasciolo, Leloir and Munoz,[15] as modified by Cruz-Coke, Gonzalez, Marish and Hulsen.[16]

It may be of interest to note that the oxygen consumed by the kidney cortex of hypertensive dogs was less than normal,[17] but normal in rats.[18]

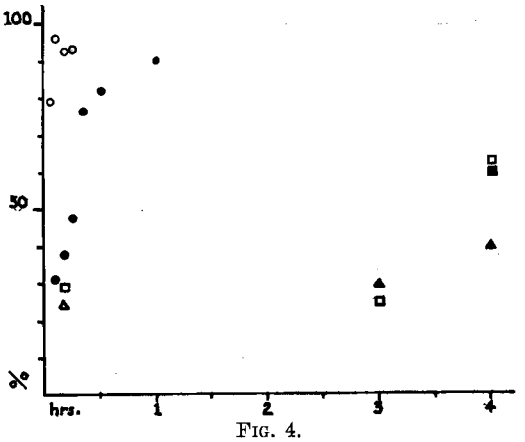

FIG. 4.
This figure shows the destruction of hypertensin when each of the following was added to renal extract:
● renal extract alone (control)
○ cytochrome
■ ascorbic acid
□ " " and cytochrome
▲ cysteine
△ " and cytochrome

Conclusions. The destruction of hypertensin by renal extracts (hypertensinases) was

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[15] Braun, Menendez E., Fasciolo, J. C., Leloir, L. F., and Munoz, J. M., *J. Physiol.*, 1940, **98**, 283.

[16] Cruz-Coke, E., Gonzalez, F., Marish, M., and Hulsen, W., *Science*, 1945, in press.

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tested by two methods: (1) the rate of filtration in the toad's kidney and, (2) the rise of arterial pressure in the cat.

In both cases this destruction of hypertensin was markedly accelerated by oxidized cytochrome and sharply retarded by reducing agents such as ascorbic acid and cysteine.

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Pityrosporum ovale—A Lipophilic Fungus. Thiamin and Oxaloacetic Acid as Growth Factors.*

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The cultivation of *Pityrosporum ovale* by the addition of fats to wort agar has been reported previously[1] and corroborated by Emmons.[2] Of a number of fats and fatty acids, oleic acid seemed the most favorable. In subsequent experiments[3] it was found that if oleic acid was supplied, this organism would sometimes grow feebly on an inorganic solution, similar to Currie's medium, containing NH_4NO_3, KH_2PO_4, $MgSO_4$ and dextrose. Substitution of asparagin as a nitrogen source caused increased growth. If thiamin was added, some growth was regularly obtained even with NH_4Cl as a nitrogen source, and there was increased yield with asparagin. It seemed that further study might clarify the growth requirements of this fungus and throw some light on the function of thiamin in its metabolism.

As a basal media a modified Currie's solution was used. It contained KH_2PO_4, 1 g $MgSO_4$, 0.25 g per liter. Either asparagin 1.25 g or NH_4Cl 2.5 g was added, and a parallel series of flasks containing in addition 50 g of dextrose was usually prepared. A solution of the soluble constituents was distributed in amounts of 50 cc in Erlenmeyer flasks and autoclaved. One-half cc of oleic acid was then added to each flask. Brom cresol green was used as an indicator. Solutions of the supplements to be tested were added by pipette in the following amounts: B_1 5 gamma, ethyl oxalacetate 5 cc of a 5% solution, L-Keto-glutaric acid 2 cc of an M/10 solution. The pH of these media before the addition of oleic acid and the supplements varied from 4.4 to 4.8. The pH was not changed with B_1, but rose to 5 with the ethyl oxalacetate and the keto-glutaric acid. The growth did not seem to be influenced by the pH within the range tested.

Stock cultures were maintained on wort agar tubes to which oleic acid had been added, as previously described. The growth appears, first as tiny cream-colored colonies which later fuse, becoming dry and wrinkled with the color varying from cream to tan or orange. This growth was removed, ground with distilled water to make a heavy suspension. The suspension was then centrifuged, washed several times with distilled water and diluted for use. (0.1 cc of packed fungus cells to 4.9 cc of water). Two or 3 drops of this suspension were used as inoculum. Growth was permitted for 4-6 weeks at room temperature. At this time the growth was harvested and the dry weight obtained. This was done by centrifuging the mold specimen with an equal volume of 95% ethyl alcohol. The sediment was then washed with 50% alcohol and finally

* The writer wishes to acknowledge her indebtedness to Professor Edgar A. Miller for advice and guidance in conducting these experiments.

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