

and rat uterus. An average of 2.3 $\mu\text{g}/\text{mg}$ of dry platelets was found, equivalent to at least 1.7×10^{-9} $\mu\text{g}/\text{platelet}$. This is more than sufficient to account for all of the serotonin found in bovine serum. No other indolic or phenolic substances were found in the platelet extracts.

Addendum. After this paper was in press, we learned that M. Bracco and P. C. Curti (*Haematologica*, 1953, v37, 721) had identified 5-hydroxytryptamine in rabbit and sheep platelets by similar methods.

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Experimental Aortic Disease in Albino Rats Following Renal Operations.* (20856)

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Striking changes in the aortae of male albino rats were observed at autopsies performed from one to 13 months following renal operations previously shown(1-4) to induce hypertension and renal disease. The aortae were increased in diameter and length, moderately tortuous in some instances and showed complete or incomplete ring-like bands of calcification giving them an appearance rather like trachea. The rats at the time of autopsy were from four to sixteen months old. Table I gives the incidence of grossly visible lesions in experimental and control rats. Since mild degrees of the aortic lesion were observed in one male and one female rat of a group of untreated animals previously set aside to age relevant data from this group have been included in the table.

Fig. 1 shows one of the more severely affected aortas. The bend pictured was present

in the vessel during life so that it was appreciably displaced from the vertebral column. The lumpy, paler zones along the course of the aorta represent foci of medial calcification. Fig. 2 is a roentgenogram of this vessel. Fig. 3 is a magnified roentgenographic view of the same vessel compared with a control aorta from a rat of the same age. These diseased aortas appear very large in comparison with normal aortas after removal. Partly this is due to a genuine increase in size, attested to by curvature and beginning tortuosity, partly to failure of the diseased aorta to contract after removal.

Microscopic studies. Sections were stained with hematoxylin and eosin, by the van Gieson-Weigert and the von Kossa technics. The earliest stage of the lesion appeared to be associated with a deposition of finely granular basophilic material, identified as calcium salt by the von Kossa stain, in the spaces between the concentrically arranged elastic lamellae. Whether this deposition oc-

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TABLE I.

Procedure*	Age, mo (operation)	Age, mo (autopsy)	No. rats	Calcific aortic disease
$\frac{3}{4}$ nephrectomy, ♂	3	4-16	33	12
Renal constriction, ♂	2-3	4-13	11	6
Controls	2-3	10-14	22	0
Aging males	—	16-30	25	1
" females	—	15-30	22	1

* In the three-quarters nephrectomized group one kidney and half the other were removed. In the renal constriction group one kidney was removed and the other constricted with a figure of 8 ligature. The kidneys of the controls were exposed and briefly handled.

curred in the ground substance or in muscle cells was not determined. At a later stage the elastic lamellae sometimes contained calcium salts. With more severe degrees of the lesion there was destruction of muscle and elastic tissue with replacement by lumpy basophilic calcium-containing material. Ossification was not seen but a large mass of hyaline cartilage was found in one severely involved

aorta. The lesions in the 2 aging rats, not operated on, were of the same character. Both of these rats were over 2 years of age and had severe renal disease. No changes in the intima were visible. Calcification of the kidneys, with involvement of the basement membranes and cells of the proximal convoluted tubules, was seen in most of the rats with aortic disease though it was absent in a

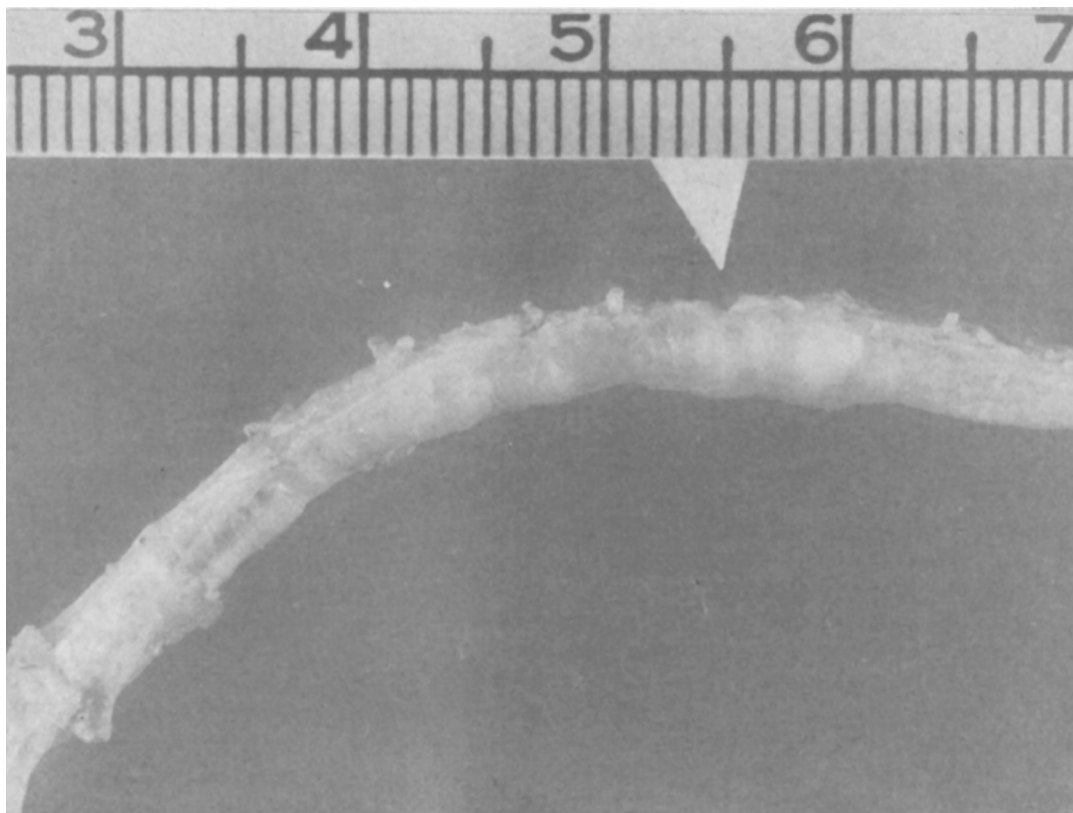


FIG. 1. Severely involved aorta. Most of the thoracic and abdominal portions are shown. The pale nodulations represent sites of medial calcification. 3X.

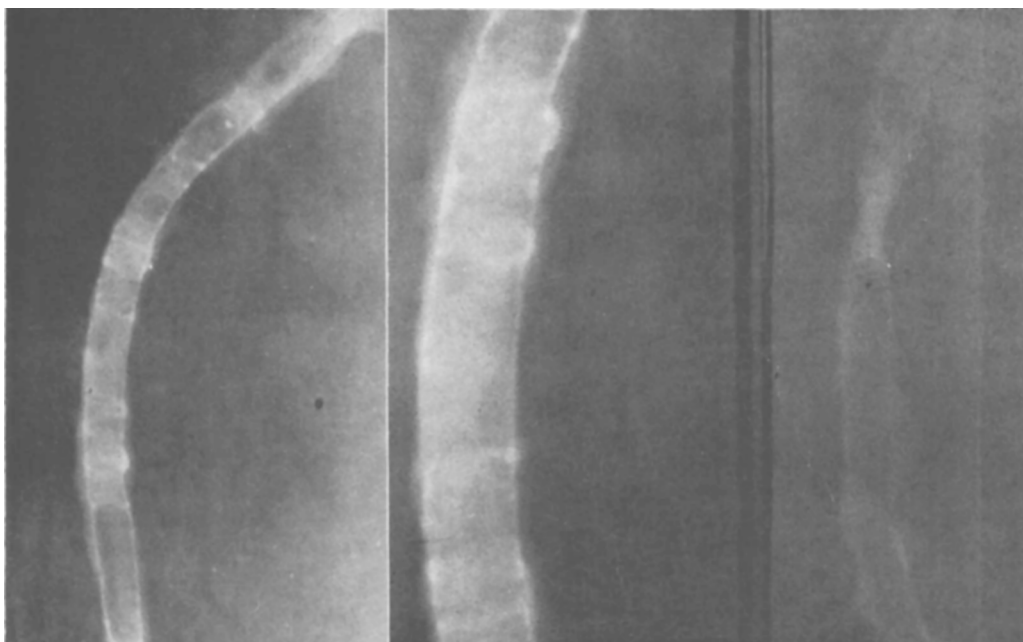


Fig. 2

Fig. 3

FIG. 2. Roentgenogram of aorta shown in Fig. 1. Ring-like and nodular calcification of wall. 1.25 $\times$.

FIG. 3. Roentgenogram of aorta shown in Fig. 1 and 2 compared with a control aorta from a rat of the same age. The opacity at the point of constriction of the control aorta is due to a drop of fluid. 3 $\times$.

few. All of the rats, however, had advanced renal disease of the type previously described (1,3) with dilatation of tubules and destructive changes in the glomeruli.

Discussion. With regard to the pathogenesis of the aortic lesions several possibilities present themselves. It has been shown by Pappenheimer (1) that albino rats, after three-quarters nephrectomy and the development of progressive destructive renal lesions, go on to develop secondary hyperparathyroidism in association with renal failure. Pappenheimer did not describe vascular lesions in his rats. In secondary as well as primary hyperparathyroidism the increased calcium-phosphorus ion product would be expected to favor metastatic calcification. Richardson (5), in a report of a case of chronic renal disease with secondary parathyroid hyperplasia and metastatic calcification, states that the common sites of calcification in chronic renal disease are the lungs, kidneys, subcutaneous tissue, heart and muscular arteries, while the stomach, contrary to the usual belief, is not

usually involved. Richards (6) reported a case of renal disease with secondary hyperparathyroidism in which calcification of blood vessels was very severe. The 44-year-old female subject of his report had severe hypertension and roentgenological evidence of calcification of the meningeal, basilar, common iliac, popliteal, radial and digital arteries. In addition there was advanced atheroma of the aorta plus medial degeneration and calcification. Parathyroid hormone can produce medial calcific lesions in the aortas of dogs, rats, and rabbits together with calcification of the lungs, kidneys, stomach and smaller blood-vessels, and, according to Hueper, there is prior degeneration of the muscle cells of the media with temporary preservation of the elastic elements (7).

The severity of the lesions in the rats of this series suggests that the aortas were predisposed to damage by some structural or chemical alteration not yet understood. It may be related to hypertension. All of the animals had hypertrophied hearts and at one

time or another had shown elevated tail systolic blood pressures. It appears that in addition to the destructive calcific lesion there is a separate histological process of adaptation to high blood pressure going on. Evidence for this is the hypertrophy of elastic tissue elements, with the development of short cross-over bands between concentric elastic lamellae, seen in aortas which did not show the calcific lesions as well as those which did. Development of these changes was associated with alterations of the shape of the stress-strain curve when the aortas were subjected to longitudinal stretch.

Summary. The development and incidence of medial calcific disease of the aorta follow-

ing operations on the kidneys of male albino rats is described.

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Synthesis of Amino Nitrogen from Ammonia in *Hymenolepis diminuta*.^{*} (20857)

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The rat tapeworm, *Hymenolepis diminuta*, is essentially independent of the protein in the diet of its host. Chandler(1) showed that no significant change in worm size, weight, or egg production could be induced under conditions of protein deprivation. Under such conditions it is quite probable that a considerable part of the amino nitrogen needed for growth and egg production comes directly from the host in the form of amino acids diffused from the intestinal mucosa. There is, however, the possibility that other sources of nitrogen contribute to these synthetic mechanisms. An investigation of the ammonia content of the rat intestine showed that a considerable amount was consistently present. The function of ammonia in intermediary protein metabolism is not very well understood; however, several workers have shown that it can contribute to the synthesis of certain amino acids and related substances(2,3). On the basis of these studies on mammalian tis-

sues an investigation of the role of ammonia in the protein metabolism of *H. diminuta* was begun.

Materials and methods. Mature tapeworms, raised in male albino rats of the Sprague-Dawley strain, were infected with *H. diminuta* cysticercoids according to the technic of Chandler(1). The worms were removed from the intestine by flushing with isotonic KCl. They were thoroughly washed and were incubated singly in a Krebs-Ringer-Phosphate (KRP) medium modified by the addition of 0.004 M (NH₄)₂CO₃ and 0.1 M sodium pyruvate, 0.1 M sodium alpha-ketoglutarate, 0.1 M sodium oxalacetate, 0.1 M sodium succinate, or 5% glucose. As a means of blocking the tricarboxylic cycle, M/30 sodium malonate was included in several of the experiments in which glucose served as the substrate. The incubations were carried out in air at 38°C. At the end of 90 minutes the worms were removed, weighed, homogenized, and the free amino acids extracted for chromatographic resolution. The paper chromatograms were run on Whatman No. 4 with phenol as the

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