signs and symptoms did not develop as fast as when phosphate was added. In addition, these dogs showed spasticity of the jaw muscles and extremities. At autopsy there were, in addition, hemorrhages in the esophagus, more extensive hemorrhages in the small intestines which contained digested blood, hemorrhages in the colon, and slight edema in the walls of the gastrointestinal tract.

Similar experiments were done with young albino rats. Adenine alone (0.5 or 1.0 per cent) or adenine plus phosphate (0.5 or 3.0 per cent) was added to the normal diet. At autopsy the kidneys showed a marked increase in size and weight. The entire surface appeared greyish mottled and hemorrhagic. The cut surface bulged, and the medulla was usually hemorrhagic.

The syndrome produced by adenine points to multiple avitaminosis. It shows all the symptoms described in experimental pellagra in dogs (black tongue). The opalescence of the cornea, the spasticity, and the ataxia observed are characteristic of riboflavin and vitamin A deficiency.

Adenine, or its metabolites, probably produced the syndrome by combining with constituents of the vitamins or their precursors, thus preventing their utilization in the animal. An interference with the formation and activity of alloxazine adenine dinucleotide, of the phosphopyridine dinucleotides (coenzymes I and II), and of nucleoproteins can be assumed. It is of interest to note that a rise in blood pressure resulted and that autopsy revealed extensive damage to the kidney.

These experiments demonstrate the existence of a direct relationship between purine metabolism and avitaminosis. A disturbance of the former may cause changes characteristic of various avitaminoses under dietary conditions which provide for normal vitamin requirements.

If the mechanism demonstrated in these experiments is applicable to human beings, the occurrence of pellagra, which is by far the most frequent variety of avitaminosis occurring in North America, might be reduced and the treatment aided by the elimination of foods containing adenine in a form producing the multiple avitaminosis. To that end the adenine content of certain foods will be determined.

These experiments are being continued and extended to other purines and purine derivatives. A detailed report, including the microscopic findings, will be published elsewhere.

Elasticity of the Aortic Wall

ALLEN L. KING

Department of Physics, Dartmouth College

The rubber-like characteristics of the aortic wall (*4, 11*) suggest that the wall material is essentially elastomeric. On applying the general theory for ideal elastomers (*5*), a relation is obtained between relative volume and fluid pressure within a cylindrical tube (*7*). Some data by Hallock and Benson (*3*) on segments of the descending human aorta for several age groups were analyzed by means of this relation. In it, the *effective* thickness, e_o, was treated as an adjustable parameter and computed for each age group. As anticipated, these values of e_o were smaller than actual thicknesses, such as measured by Krafka (*8*) on strips of aortas. It was suggested, however, that part of this discrepancy is due to a thickening of the aortic wall when peripheral chains are cut. That thickening on cutting does occur was pointed out recently by Remington, Hamilton, and Dow (*9*). Segments of canine aortas were found to decrease in length by as much as 30 per cent on removal (*10*), probably in this case due to a shortening of the longitudinal chains. In general, then, intact aortic walls are thinner and less firm than the removed segments.

In their discussion of aortic size, Bazett, Cotton, Laplace, and Scott (*1*) use values of the ratio of thickness to internal radius of undistended aortas as a function of age which were computed from data obtained by Kani (*6*). The estimated error in Kani's measurements, however, is greater than the apparent decrease of the ratio with age as reported by Bazett and co-workers for aortas. It is interesting to note that Kani's values for thoracic aortas yield a ratio of thickness to *external* diameter of 0.088 for the thinnest parts and 0.095 for the thickest parts, with a maximum variation with age of ± 0.008, only half the estimated average deviation of measurements for any one age group. The corresponding ratio, e_o/d_o, from the results found in Table 1 (*7*) are given below:

RATIO e_o/d_o FOR SEVERAL AGE GROUPS

Age group	20–24	29–31	36–42	47–52	71–78
e_o/d_o	0.094	0.081	0.077	0.073	0.052

The general decrease in the ratio, e_o/d_o, beyond the age of 20 years suggests that accumulating collagenous fibers and other deposits in the aortic tissues, along with the enlargement of the vessel, cause a thinning out of the elastomeric constituents. Apparently the ratio of actual thickness to diameter remains nearly constant at approximately 0.09, so that by the 75th year almost 50 per cent of the aortic wall is effectively non-elastomeric. On the other hand, the product, $e_o d_o$, is constant at 15 mm.^{2} to within an average deviation of 0.2 mm.^{2} over the age range of 20–80 years, as though the materials within the vessel wall that render it elastic really do not disappear but, rather, become more thinly dispersed with time (*7*). Elastin and similar constituents of blood vessel walls are relatively stable substances (*2*).

From this analysis of aortas the aging process appears, at least in part, to consist of a gradual, effective shortening of molecular chains within the aortic wall. Such an effect can take place either by the establishment of cross-linkages between chains or by introducing fix-points in the form of collagenous fibers and fatty aggregates.

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