seems "the greatest aim of our science" (p. 637); in 1896 historical inquiry in different regions with subsequent comparison of the processes of growth in each becomes the indispensable prerequisite to the discovery of laws, hence represents the immediate goal (p. 279); by 1920 the uniqueness of the several cultures is a dominant idea (p. 286); and in 1932 culture appears so complex that any generalizations about it are either truisms or spurious (p. 257f.).

Actually, the difference between the simpler data of physics and the complex phenomena of geography had impressed itself on Boas very early in his career (pp. 639–647). He thus came to distinguish as coordinate and mutually complementary the desire to merge a host of facts in a simple formula and the desire fully to understand a particular phenomenon in its unique individuality. The recurrent warning against identifying cultural features that are not at bottom comparable (pp. 258, 263) illustrates his tendency to apply to ethnography the geographer's rather than the physicist's point of view.

Boas is, indeed, preeminently scientific in his treatment of culture, but merely because here, too, he is concerned solely with ascertaining the truth, not with tickling aesthetic tastes or indulging in unchecked fancies. Hence the persistent coldness to ambitious schemes of historical reconstruction. "We desire to find uncontestable evidence of transmission, not alone the possibility or plausibility of transmission" (p. 459). Hence also his refusal to make common cause with either extremist faction in the perennial dispute over independent evolution versus diffusion; both schools, he contends, proceed from arbitrary assumptions (p. 282). And, precisely as in linguistics, his mind is not cribbed by conventional ruts. He gives to the threadbare theme an original twist by demonstrating convergence as an unsuspected third reality (pp. 263, 275, 299). As for diffusion, to ascertain its occurrence or direction—the task which exhausts most relevant efforts—is merely the first step. We must ask, which traits are borrowed, which are rejected, and why, and what further changes the innovation may stimulate (p. 291). The study of cultural transference thus merges in cultural dynamics. Characteristically, Boas has shifted his position at different periods, stressing real history as a corrective of evolutionary schemes and emphasizing the processes of growth when historical schematism was in retreat (p. 311).

Though Boas has been continuously interested in methodology, he has not elaborated comprehensive treatises on the order of Graebner's or Wilhelm Schmidt's, preferring to expound his standpoint in occasional papers carefully worked out according to the exigencies of the moment. This predilection inevitably leaves lacunae and even sporadic disharmonies. But this is of small moment. Counsels of perfection are cheap; what counts is the concrete practice of science. As Mach says of the founders of physics. "Noch ohne alle Methode, welche ja durch ihre Arbeit erst geschaffen wird, und die ohne Kenntnis ihrer Leistung immer unverstanden bleibt, fassen sie und bezwingen sie ihren Stoff and prägen ihm die begrifflichen Formen auf."

In the anthropological science of his time Boas has been the great exemplar, fearless of authority, relentlessly self-critical, driven by a sacred thirst to ever new Pierian springs, gaining ever deeper insights into the nature of man.

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SPECIAL ARTICLES

RELATIVE OVERGROWTH OF THE CENTRAL NERVOUS SYSTEM IN VITAMIN A DEFICIENCY IN YOUNG RATS[1]

THE EXPLANATION OF THE NEUROLOGICAL LESIONS OCCURING IN THIS DEFICIENCY

THE relation of vitamin A to growth and function of the nervous system has remained unsolved although several laboratories have reported neurological lesions as a consequence of vitamin A deficiency.

We have been unable to produce neurological lesions by vitamin A deficiency in rats after normal growth had occurred up to an age of 10–12 weeks although the epithelial changes characteristic of the deficiency were invariably produced. However, ataxia and paralysis may be regularly produced in young rats if the deficiency is established at a sufficiently early age. Our procedure has been such as to prevent any considerable storage of vitamin A during the first three weeks of life and placing the rats on a completely deficient diet at 21 days of age. Ataxia and paralysis appear at about 50 days of age, shortly before cessation of growth, and are attended by degeneration of the peripheral nerves and of nerve fibers in various tracts of the spinal cord and in the cerebellar peduncles. The pattern of degeneration in the spinal cord is irregular, chiefly of ascending tracts and can not be correlated with our rather meager knowledge of the order of myelination which is wholly post-natal in the rat. The explanation has been found to be a relative overgrowth of the central nervous system resulting in mechanical damage and degeneration of nerve fibers. The earliest and most striking manifes-

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tations are within the spinal canal and involve both anterior and posterior nerve roots.

The gross manifestations are: (1) extensive and striking herniations of the nerve roots into the intervertebral foramina distorting and, to some degree, displacing laterally the spinal ganglia; (2) herniations into the bodies of the vertebrae (ventral wall of spinal canal) which become symmetrically deeply pitted and filled with irregularly coiled nerve roots. The localization of these pits is probably due to the vascular cushion ventral to the nerve roots which enter the spinal ganglia. We believe that the veins in this location yield to the crowding and thus determine the regions of bone resorption; (3) increase in size of the contents of the cranium as evidenced thus far by—(*a*) the presence of herniations of the cerebrum, cerebellum, and posterior colliculus into the transverse venous sinus. These herniations presumably occur at the sites of arachnoidal villi. The most notable herniations are those of the cerebellum which enter the transverse sinus where the superior cerebellar veins open into it; (*b*) distortion of the brain, made conspicuous by moulding of the mid-brain and cerebellum and partial obliteration of the cisterna magna; (*c*) changes in the contours of the fossae of the floor of the skull, due to bone resorption.

Thus far no striking gross changes have been noted in the peripheral nerves. In some instances the optic nerves close to the eyeball have shown a symmetrical globoid enlargement.

As it is well known that growth of bone ceases with the establishment of vitamin A deficiency, the obvious explanation of the unequal growth is that the growth of the central nervous system continues after the growth of the skeleton has ceased. We have endeavored to test this by two procedures—(1) the study of rats whose growth has been retarded at an equal rate and degree through inadequate diet with full vitamin complement; (2) a study of rats retarded by riboflavin deficiency. In spite of the facts that these control rats paralleled in rate of weight increase and skeletal growth the A-deficient rats, no neurological lesions were produced and dissection showed normal relations of the nervous system to spinal canal and cranium. This was also true in rats where the stunting was severe and prolonged.

Experiments indicate that the unequal growth of bone and nerves occurs between the fortieth and sixtieth days of age. In a few experiments we have found that if vitamin A in the form of carotene is added to the diet at 42 days of age and the diet restricted in amount so that the growth parallels littermates in continued A deficiency, no manifestations of nervous lesions occur. The rats retained on the deficiency became paralyzed on about the fiftieth day. On postmortem examination at 63 days of age, those given carotene showed either no changes in the central nervous system or only very slight evidences of beginning herniation of nerve roots, in contrast to the striking lesions found in the litter-mates maintained on the deficiency for the entire period.

The unequal growth is most strikingly exhibited in the lumbar and sacral regions. It is manifest, to some degree however, throughout the entire length of the spinal cord. Rough estimates would indicate that nerve trunks taking exit in the lower lumbar and sacral foramina may be 4–6 mms longer than the distance from their origins to the foramina of exit.

It is possible that the compression of the cerebrum and cerebellum may be responsible for degeneration of descending fibers in the cord. At present the evidence points to a marked predominance of ascending tract lesions. The consequence of the herniations is obviously essentially that of more or less complete transection of the nerve roots and the few microscopic studies made support this view. Evidences of regeneration of nerve fibers proximal to the cell of origin have been found to take place during the deficiency. Observations in gross indicate that small excrescences comparable to amputation neuromata are present. Extensive formations of this sort have been seen after a period of extended repair on diets containing carotene.

Elucidation of the significance of the above observations is being attempted through a series of experiments designed to give information concerning the normal growth relations of the central nervous system and bony enclosure. As no readily discernible changes in nerve cells of the central nervous system or spinal ganglia are to be found other than those explainable through mechanical factors, an obvious conclusion is that the growth and physiology of the nervous system is independent of vitamin A, although the possibility that vitamin A deficiency accelerates the growth of the central nervous system can not be definitely eliminated without further work.

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EFFECTS OF STEROID GLYCOSIDES AND CORTIN ON INSULIN CONVULSIONS AND BLOOD SUGAR

PREVIOUS work from this laboratory has shown that extracts of beef adrenal cortex can affect both the blood sugar level and the electrolyte pattern of normal and adrenalectomized animals. Because our preparations were not pure substances, it could not be determined whether the effects were produced by one or several hormones. Nevertheless, our work presented evidence that the adrenal cortex was intimately con-