The changes in the renal ganglia in bright's disease / by Robert Saundby.

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THE CHANGES IN THE RENAL GANGLIA IN BRIGHT'S DISEASE.*

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IN the *American Journal of Medical Sciences* for July 1880, there is a very able paper, by Drs. Da Costa and Longstreth of Philadelphia, entitled "Researches on the State of the Ganglionic Centres in Bright's Disease."

They give the particulars of nine cases of Bright's disease, with microscopical details of the changes in the kidneys and semi-lunar ganglia. They divide their cases into three groups: the first includes two cases of well-marked contracting kidney; the second, three cases of the mixed type of chronic Bright's disease; and the third, one case of large white kidney, one of recent nephritis after typhoid fever, and two of chronic Bright's disease, with large coarse diffusely diseased kidneys.

It is impossible to avoid feeling somewhat confused by the heterogeneous composition of these groups; and, in order to place the results of their investigations in a clearer light, I have ventured to summarise the conclusions which I think may be fairly drawn from these nine cases.

(a.) In acute or subacute nephritis (Case IX), the only change met with in the ganglia was an increase of the cell-elements of the stroma.

(b.) In the large white kidney (Case VII), the connective tissue of the ganglia was loose and swollen, and its cell-elements increased. The ganglionic cells were generally normal, degenerative changes being present in about one third. There were no vascular changes.

(c.) In the mixed forms (Cases III, IV, V, VI, and VIII), the connective tissue was increased, and its cell elements were numerous; the ganglionic cells were more or less fatty and pigmented; the blood vessels were dilated and thickened in two cases.

(d.) In typical contracting kidney (Cases I and II), the stroma of the ganglia was increased in amount, but contained few cell-elements; the ganglonic cells were generally shrunken, deformed, fatty, and pigmented; the blood-vessels were dilated and hypertrophied.

After giving these descriptions, the authors proceed to consider the relations of the ganglionic lesions to the renal disease; and, after stating that they may be regarded as either causes, concomitants, or results of one another, accept the view that the changes in the ganglia stand in some way as causes to certain forms of Bright's disease, especially the contracting form. In explanation of this view, they propound the following theory of the *modus operandi*. "The specific cause, whatever may be its unknown form or character, acts in such a manner on the ganglion-cells presiding as centres of innervation to the kidney, and whose fibres, distributed to the vessels of these organs, regulate, not only the calibre of the vascular trunks by changing the state of contraction of their muscular fibres, but also probably the conditions of osmotic action between the blood and the tissues, that the collective phenomena known as Bright's disease are brought about."

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They also express the opinion that the hypertrophy of the heart is the result of similar changes in the cardiac ganglia, though they offer no evidence in support of it.

I have examined the semilunar ganglia in fifteen cases of Bright's disease, viz.: Acute Bright's disease (1); Large white kidney (3); Mixed large white and waxy kidney (1); Mixed fatty and contracting kidney (7); Small contracting kidneys (3). I possess microscopical preparations and drawings of each of these.

Fig. 1 is a drawing of part of a semilunar ganglion from a case of acute nephritis in a boy, aged 5, which was probably postscarlatinal; but my knowledge of the case is limited to the fact that it came to the *post mortem* room with the diagnosis "acute nephritis". In this preparation, the only change to be observed is the increase of round-cell elements in the stroma. The ganglionic cells stained well; they are well formed, and show no trace of pigment. The vessels were normal.

Fig. 2 shows the state of the ganglia in a case of large white kidney in a young man, aged 28, who died under my care. The stroma is very much increased, the cell-elements are fairly numerous; the ganglionic cells are pale, but for the most part free from pigment, and well formed. The vessels are not thickened.

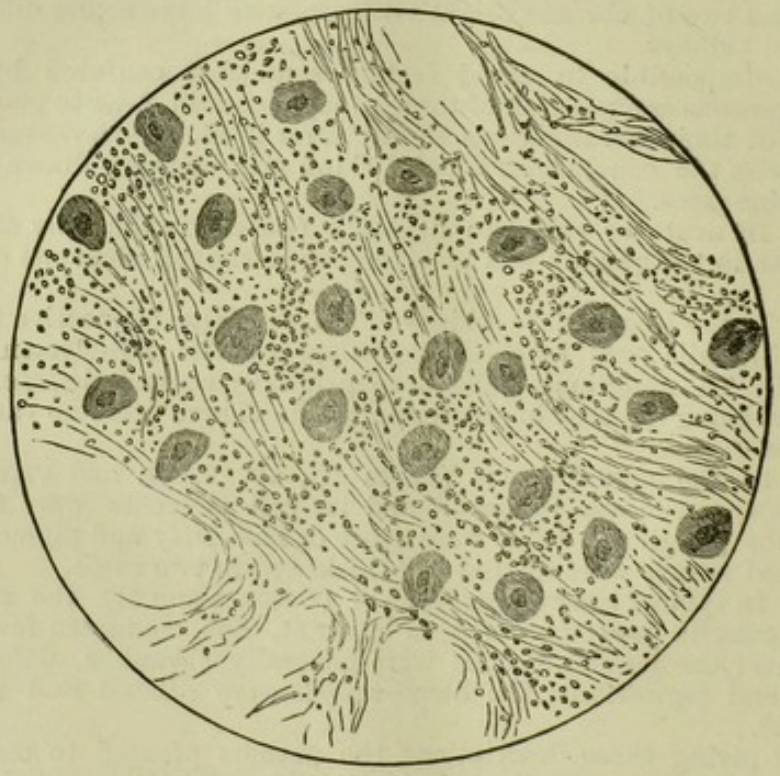

Fig. 1.

In the mixed forms, the lesions in the ganglia occupy an intermediate position between those seen in Figs. 2 and 3. The stroma was increased, its cellular elements were abundant; the ganglionic cells were more or less deformed, undergoing various degrees of pigmentary degeneration; the vessels were dilated and hypertrophied in two cases.

Fig. 3 is taken from the ganglion of a typical case of contracting kidney, which had been under my observation for two years. The kidneys weighed together four ounces. The stroma of the ganglia was much increased in breadth, but contained very few cell-elements, either round or spindle-shaped; the ganglionic cells were few in number, shrunken, and undergoing advanced pigmentary degeneration. The vessels were dilated and hypertrophied in every case.

It will probably be allowed that the vascular dilatation and hypertrophy formed part of the general affection of the vessels in

granular kidney, so that they need not be again referred to in discussing the significance of these changes. The process appears to begin in the stroma of the ganglion, and in its acuter forms is characterised by infiltration of this tissue with lymphoid cells; this is followed by increase in the connective tissue, and finally by the formation of a hyaline stroma, poor in cell-elements or nuclei. The ganglionic nerve-cells are affected secondarily, and as a result of this chronic disturbance of nutrition. They undergo pigmentary degeneration, which destroys their protoplasm, and converts them into masses of dark granules obscuring the nuclei, which are also probably eventually destroyed. These observations confirm the accuracy of the description in the original paper, but I must dissent from the use of the term "fatty" to express the change in the ganglionic nerve-cells. In my preparations, the change is that generally called in this country pigmentary degeneration, a common lesion of nerve-cells in chronic diseases of the nervous system, and probably a normal factor in the senile decay of these elements; but I am disposed to think this difference to be merely verbal. The point upon which I join issue with Drs. Da Costa and Longstreth is on the significance of these lesions.

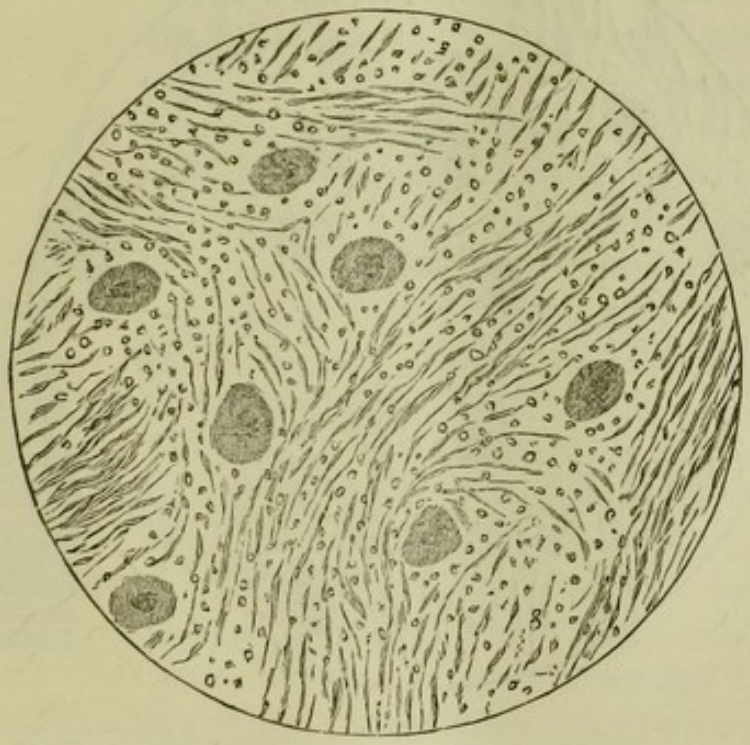

Fig. 2.

As I have already shown by quotation, they regard the ganglionic lesions as the cause of Bright's disease, or some form of it, especially the contracting kidney. We might reasonably inquire why it was that, having found changes in so many different types of Bright's disease, they are disposed to restrict this relation to the contracting kidney. It is not for me to suggest an answer to this question; but this restriction makes their meaning less clear and their reasoning less logical.

I think we cannot escape from the conclusion that the ganglionic lesions stand in the same relation to all the forms of Bright's disease. There is nothing to suggest that they are more likely to be primary in one form than in another; so that, if we hesitate to regard acute Bright's disease as primarily a disease of the renal ganglia, any objections which weigh with us in this case should hold good in all. It may be thought that the insidious mode of origin of the contracting kidney makes it a fairer field for speculation than the other forms of Bright's disease; but I would earnestly protest against the assump-

tion that the obscurity of a problem justifies the introduction of crude hypotheses resting on ambiguous facts and doubtful analogies. A moment's reflection, moreover, will assure us that we really know no more of the actual mode in which the kidney is affected by, for example, the poison of scarlatina, than we do of that of gout.

But I may be asked, Are not Addison's disease and Graves's disease analogous cases? To this I would answer, that it has never been proved that the changes in the sympathetic ganglia in these still very obscure diseases are primary, and that quite similar changes have been described in the ganglia in diffuse eczema (Marcacci), pseudo-hypertrophic muscular paralysis, and pernicious anæmia (Brigidi), gliosarcoma of the brain (Morselli), general paralysis of the insane (Poincaré and Bouchet), cholera (Pio Foá), and diabetes. Moreover, Giovanni (*Patologia di Simpatica.* Milan, 1876) found cellular infiltration of the sympathetic ganglia in an immense variety of visceral and general diseases, showing that structural changes in the organs is very generally accompanied by signs of irritation in the ganglia. One of my cases of contracting kidney, in which the

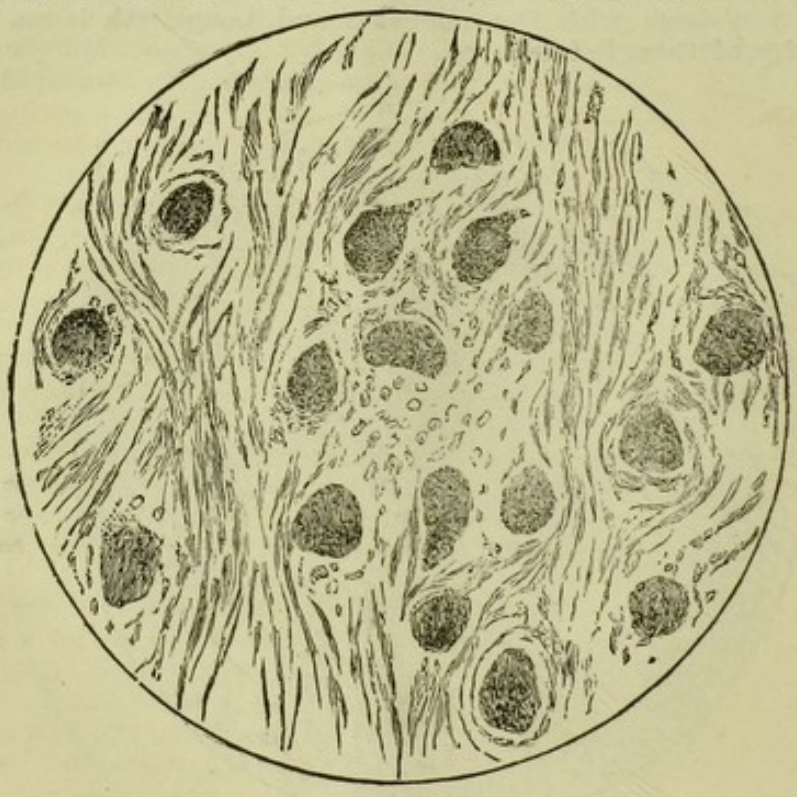

Fig. 3.

ganglionic changes were in every way typical, was a death after ovariotomy, in which the pelves of the kidneys were dilated, and there was reason to believe that the disease originated from pressure on the ureters. If this were so, the ganglionic changes would only be the results, or at most the concomitants, of the renal mischief.

It is probable that, in all inflammations, the vaso-motor centres may be affected practically immediately in point of time; and it is possible that structural alterations take place in the ganglia as early as in the inflamed part. It is also quite possible, that some poisons may act directly upon the ganglia, and not reflexly through the tissues of the organs; but this is a point upon which we have no definite information. No doubt, there is a tendency to ascribe greater influence to primary functional disturbance of nerve-cells than we were used to do; and, for my part, I cannot but think this tendency exists to an inordinate degree, considering the very slender basis of fact upon which it rests.

Though I dissent from their conclusions, I wish to acknowledge the accuracy of the observations of Drs. Da Costa and Longstreth, and the importance of their contribution to the data of Bright's disease.