REMARKS

ON

CHRONIC BRIGHT'S DISEASE; PARTICULARLY
THE CIRRHOTIC FORM.*By T. GRAINGER STEWART, M.D., F.R.C.P., F.R.S.E.,
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THE object of the present paper is to attempt to clear up the true pathology of that form of Bright's disease which is commonly described as the granular gouty, contracting or cirrhotic kidney; and to elucidate certain points as to its causes, mode of origin, symptoms, complications, and results.

The first point upon which it appears to me necessary to insist is, that the term "chronic Bright's disease" is not applicable to a single pathological process, but must be held to include all the forms of the malady, and not the individual forms alone, but their various combinations. Thus, I have watched one case of pure waxy or amyloid disease, the form associated with disease of the vessels, for a period of eleven years, and others for five or six years; while in regard to the inflammatory form, the disease proper to the tubules, I have had one case under observation for seven years and another for four. Cases in which the waxy and inflammatory forms existed I have also known to go on for several years. But in the presence of such an audience, it is not necessary further to insist upon this; for the experience of every physician who has closely studied the subject, must supply abundant illustration of what I am asserting. Still, granting these points, it must be admitted that, as the progress of the cirrhotic disease is eminently insidious and tardy, to it may be applied pre-eminently the term "chronic Bright's disease."

The second point which seems to require comment, is the error which appears to prevail that a granular condition of the surface, with contraction of the cortical substance, constitutes an unfailing evidence of the cirrhotic form. But my experience has satisfied me that in the waxy and inflammatory, as well as the cirrhotic form, a granular contracted kidney results, if life be sufficiently prolonged. I have traced the different stages of atrophy both in the waxy and in the inflammatory forms, and find that it is often possible to divine, with tolerable accuracy, the exact stage likely to have been reached in any individual instance. It may be suggested by some that, while this certainly does occur, it may, in all the forms, be due to one common process, such as the increase and subsequent contraction of the connective tissue; but my observations satisfy me that this is not the case, but that while in one form that is the cause, in others the change originates in ways entirely different.

Having made these preliminary remarks with the view of defining more clearly the opinions which I wish to maintain, I leave out of consideration the inflammatory affection of the tubules, and the waxy or amyloid disease of the vessels, and proceed to consider the pathology of the cirrhotic form. Three views with regard to the pathology of this affection at present find favour with different observers.

1. The first I shall consider is the theory of Sir William Gull and Dr. Sutton, that the disease is merely a local manifestation of a peculiar change of the blood-vessels, which they describe, and to which they give the name "arterio-capillary fibrosis." It may be best to give the summary of their conclusions in their own words. With regard to the microscopic appearances, their conclusions are as follows.

"1. The arterioles throughout the body, in that condition usually called chronic Bright's disease, with contracted kidney, are more or less altered.

"2. This alteration is due to a hyaline fibroid formation in the walls of the minute arteries, and a hyaline granular change in the corresponding capillaries.

"3. This change occurs chiefly outside the muscular layer, but also in the tunica intima of some arterioles.

"4. The degree in which the affected vessels are altered, and the extent to which the morbid change is diffused over the vascular system of the different organs varies much in different cases.

"5. The muscular layer of the affected vessels is often atrophied in a variable degree."

The general conclusions at which they have arrived they sum up as follows.

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"1. There is a diseased state characterised by a hyaline fibroid formation in arterioles and capillaries.

"2. This morbid change is attended by atrophy of the adjacent tissues.

"3. It is probable that this morbid change commonly begins in the kidneys, but there is evidence of it also beginning primarily in other organs.

"4. The contraction and atrophy of the kidney are but part and parcel of the general morbid changes.

"5. The kidneys may be but little if at all affected, whilst the morbid change is far advanced in other organs.

"6. This morbid change in the arterioles and capillaries is the primary and essential condition of the morbid state called chronic Bright's disease, with contracting kidney.

"7. The clinical history varies according to the organs chiefly and primarily affected.

"8. In the present state of our knowledge, we cannot refer the vascular changes to an antecedent change in the blood due to defective renal secretion.

"9. The kidneys may undergo extreme degenerative changes, without being attended by the cardio-vascular and other lesions characteristic of the condition known as chronic Bright's disease.

"10. The morbid state under discussion is allied to the conditions of old age, and its area may be said hypothetically to correspond to the area vasculosa.

"11. The changes, though allied with senile alterations, are probably due to causes not yet ascertained."

In seeking to determine the correctness of their opinions, I have first, by the kindness of Dr. Sutton, examined a number of preserved specimens, so as to satisfy myself of the exact appearances to which they have applied the term arterio-capillary fibrosis; and secondly, I have carefully examined the small vessels in every case of examination after death to which I have had access during the past three or four months. These cases are only twenty-three in number; and although I admit that such a number is totally inadequate to afford a foundation for any new view, it is, I submit, quite sufficient to test the correctness of views already advanced. All the more is this true in the present instance, as among the cases there was a considerable proportion of examples of Bright's disease.

The conditions which I have observed are—besides the presence of fatty granules, and of pigment on the surface of the vessels, and the occasional occurrence of small aneurisms in their walls, and of thrombi within them—first, a more or less marked thickening of the outer coat of the small arteries. This is in some cases so distinct as to attract attention by its wavy, fibrous appearance, and the sinuous outline of the vessel where no reagent has been applied. But these appearances become much more distinct when the specimens are placed for a short time in water, or in glycerine, or when a little dilute acetic acid is added.

In many cases, the application of these reagents produced an appearance as of thickening, certainly of increased prominence, of the external coat, even when the vessel without reagent appeared natural. But the appearance thus artificially produced differed from that of the true thickening, in that it was never wavy or fibrous-looking. I found no evidence of free exudation, or of the presence of any solid material in connection with the outer coat.

The condition thus described corresponds exactly to the appearances of the specimens of so-called hyaline fibroid disease which I examined. I have some difficulty in deciding what the elements were which so appeared thickened or became distinct by reagents; but I am satisfied that they are not any new material exuded, and I think that they include the tunica adventitia, and probably another sheath; whether lymphatic or a mere prolongation inwards of the pia mater, as Dr. Batty Tuke suggests, I am not prepared to say. With regard to the influence of various reagents, it appears to me that they produce their effects mainly by being imbibed into the space bounded by the delicate membrane. Secondly, I found in several instances distinct thickening of the middle coat, with increase of the muscular fibres. I found no evidence of atrophy of this coat in any case. Thirdly, I found the internal coat in some parts thickened. I have met with all the peculiarities above referred to in a single artery, the whole three coats being thickened; but I have never seen evidence of diminution of the lumen. With regard to the capillaries, I have failed to find any evidence either of thickening or of exudation upon their walls.

Of my series of 23 cases, there were 12 in which the vessels presented no abnormality. There were 5 which had the middle coat thickened, 3 to a marked extent, and 2 to a slight. There were 10 in which the outer coat was thickened, in 4 of which the middle coat was also thickened. Of the 12 in which the walls were natural,

there was—(1) one case of chronic peritonitis; (2) one of phthisis, with slight cirrhosis of kidneys; (3) one of cystic disease of kidney, fatal by uræmia; (4) one of slight cirrhosis of liver and kidneys; (5) one of tubercle of lungs, pleura, and peritoneum; (6) one of typhoid fever; (7) one of pleurisy and double pneumonia, and cerebral embolism; (8) one of cirrhosis of kidney, with aneurism; (9) one of phthisis, with combined waxy and inflammatory Bright's disease; (10) one of phthisis, with inflammatory and slight waxy disease; (11) one of insolation, with slight cirrhosis of kidney, with hypertrophy of heart; and (12) lastly, one of waxy Bright's disease.

Of the 5 cases in which the middle coat was hypertrophied, (1) one was a case of inflammatory Bright in the second, passing into the third stage; (2) one was another example of the same disease, fatal by hæmorrhagic apoplexy; (3) one was a case of cirrhosis of kidney, in which the kidneys were of about the normal size; in these it was well marked; (4) one of perinephritic abscess, with atrophy of one kidney and compensating hypertrophy of the other; (5) one of advanced cirrhosis of kidneys and of liver.

Of the 10 cases in which the outer coat was thickened, (1) one was a case of valvular heart-disease, with congestion of viscera; (2) one was a case of inflammatory Bright's disease, fatal in commencement of third stage; the middle coat was also thickened; (3) one was a case of acute alcoholism; (4) one was a case of carcinoma of the uterus, with hydronephrosis; (5) one was a case of typhoid fever; (6) one was a case of cirrhosis of kidney; the middle coat also was hypertrophied; (7) one was a case of perinephritic abscess; the middle coat here also was somewhat hypertrophied; (8) one was a case of inflammatory softening of the cord; (9) one was a case of commencing cirrhosis of kidneys and liver; and (10) one was a case of advanced cirrhosis of kidneys.

Let me now note the relationship between the cases of Bright's disease and the state of the vessels. There were 12 cases of Bright's disease, and of these the vessels were natural in 7. These 7 were—1 of waxy, 2 of waxy and inflammatory combined, and 4 of cirrhosis. Morbid conditions of vessels existed in the remaining 5 cases, of which 3 were cases of cirrhosis and 2 of inflammatory disease. One of the last-named had the outer coat natural. These facts seem to indicate—1st, that the waxy forms of Bright's disease, and the inflammation in its early stages, have no relationship to the state of vessels under consideration; 2nd, that, in regard to cirrhosis, although changes in the vascular walls were present in 3 cases, they were absent in 4. There seems, therefore, to be no constant relationship between the one condition and the other. Now turn to another aspect of the question. Of the 10 cases in which the outer coat was thickened, there were 6 in which there was no Bright's disease whatever; that is, in a majority of the cases in which the arterial fibrosis existed, there was none of the so-called corresponding renal lesion. We must therefore conclude that my facts are opposed to the view under consideration. But the facts seem to point to another relationship between renal disease and disease of the vessels. The middle coat of the arteries was hypertrophied in 5 cases, and 4 of them were cases of long-standing Bright's disease, while in the fifth one kidney was destroyed. Does not this tend to confirm Dr. George Johnson's observations that hypertrophy of the middle coat is a frequent result of renal disease? It is quite true that, according to his views, we might have expected it in a larger proportion of the cases; but it is to be noted that it occurred in none of the whole series that was not renal.

Turning now for a moment to the relationship between this condition of vessels and cardiac hypertrophy, we find that, of the 12 cases in which the vessels were normal, the heart is noted as hypertrophied in 4, and three of these had Bright's disease. Of the 5 cases in which the vessels had thickened middle coats, the heart was hypertrophied in 4, and all of them were cases of Bright's disease. Of the 10 cases in which the outer coat was thickened, it was hypertrophied in 5, of which 4 were cases of Bright's disease, and 1 had valvular affection, while it was normal in 3 and small in 2. These facts seem also to indicate that hypertrophy of the heart bears no special relationship to thickening of the outer coat of the vessels. For these reasons, as well as others to which I shall presently refer, I am unable at present to accept the theory of Sir W. Gull and Dr. Sutton.

2. The second theory is that maintained by Dr. George Johnson, both in his former publications and in the valuable course of lectures which he has been recently publishing in the JOURNAL of the Association. According to him, we have all the essential changes originating within the tubules. He finds, to quote his own words, "that the epithelium is opaque and granular, in a state of cloudy swelling; the tubes crowded and opaque, with degenerated and disintegrated epithelium; some contracted; others dilated in various degrees; some lined by transparent uninucleated cells; others filled with unorganised fibrine, rarely with blood, or with oil; lastly, the basement-membrane and the Malpighian capsules thickened, this thickening being often more apparent than real" (BRIT. MED. JOURNAL, Feb. 15th, 1873). It is thus, according to Dr. Johnson, all intratubular—not an affection of the connective tissue. His theory of the production of these changes is, that the cells lining the tubules undergo alteration in consequence of morbid nutrition due to blood-disease from gout or other malady; the work of elimination of these materials being thrown upon them. I fully recognise the accuracy of Dr. Johnson's anatomical description, but not as applied to the form of disease under consideration. I have not in these cases found changes in the epithelium, but have never failed to find increase of the connective tissue. I value Dr. Johnson's descriptions highly, as accurate delineations of the third stage of the inflammatory form of Bright's disease; but it does not accord with my experience of true cirrhosis.

3. The third view, which is supported by Dr. Dickinson and other authorities, appears to me to be correct. According to this view, the essential change is supposed to be situated in the intertubular connective tissue. The organ is at first enlarged in consequence of increased growth of connective tissue, and subsequently becomes atrophied, the vessels becoming obstructed, and the nutrition of the gland-elements interfered with. The obstruction of the vessels is due to the mechanical pressure of the hypertrophied connective tissue in the early stage, and afterwards to the cicatrix-like contraction of that material. This change begins always at the surface, and is associated with thickening of the capsule, and gradually spreads inwards till, in some cases, almost the whole cortical substance is destroyed. These alterations sufficiently explain the deficiency of the amount of urea excreted by the organ. It has been commonly assumed that this change is inflammatory in its nature; but of this I have no satisfactory evidence. Certainly the extreme slowness of its progress and the absence of free exudation seem to me to indicate that it is truly an hypertrophy.

It will be thus apparent that my further experience and study of this disease incline me to maintain the correctness of the view of its pathology which I had formerly expressed. While asserting that it consists essentially in an hypertrophy of connective tissue, I do not in the least doubt that the change is a result of constitutional causes. Among the reasons which compel this belief, are the following.

1. The disease almost constantly affects both organs.
2. It is pretty equally diffused throughout both.
3. In many cases, other organs, such as the liver and spleen, are similarly affected.
4. It is often associated with well recognised changes in the blood, such as gout and chronic lead-poisoning.

With a view to clearing up the history of cirrhotic kidney, I have prepared a table showing the results of a series of *post mortem* examinations made during the years of my pathologistship in the Edinburgh Royal Infirmary, and another showing the leading points of the history of twenty of the cases which I have treated in the wards of that institution.

The pathological cases are thirty-six in number; and, for convenience of reference, I have reduced to percentages the results found. It will be understood that the statements given apply only to the anatomical conditions found at the necropsies. There was some degree of dropsy in 25.5 per cent. The heart was hypertrophied in about 66.6; but of these there were about 8.4 per cent. in which other conditions than the renal disease probably had contributed towards the hypertrophy. Œdema and congestion of lungs existed in 52.8 per cent.; tubercles of lungs in 13.9 per cent. Of serous inflammations, pericarditis was present in 11.1 per cent.; pleuritis in 16.6 per cent.; peritonitis in no case. The liver was fatty in 22.2 per cent., cirrhotic in 16.6 per cent. The capsule of the spleen was thickened, and its substance fibrous, in 33.3 per cent. There were tubercular ulcers of the intestine in 8.33 per cent. Sanguineous apoplexy was present in upwards of 8.33 per cent.

Of a series of twenty cases which I have observed clinically, only three were women. Allowing for the preponderance of male over female patients in most hospitals, and for the fact that my male ward is larger than my female, it is clear that my cases afford further evidence of the generally recognised fact that the male sex is more frequently affected with this disease than the female.

With regard to age, the oldest was 70, and the youngest 21. Of the 20 cases, 5 were between 20 and 30, 0 between 30 and 40, 4 between 40 and 50, 7 between 50 and 60, 3 between 60 and 70, and 1 70. In the cases between 20 and 30, it was clear that the disease had existed for a considerable number of years; so that we have in these cases a confirmation of the fact that, while it is most common after 50, it is by no means rare in youth and early manhood.

With regard to habits, of the 20 cases, 12 were undoubtedly intemperate; most of them having been spirit-drinkers, some beer-drinkers,

and some promiscuous drunkards. Now, if we take all the forms of Bright's disease together, we find no very striking connexion between them and intemperance; but if we take, as here, the cirrhotic form alone, the relation is at once apparent. The cause assigned in 12 cases was the intemperance; in one of these, lead-poisoning co-operated with that cause; in another, gout. There was one other case in a patient not known to be intemperate, but whose illness was assigned to lead-poisoning. In the remaining seven cases, the cause was not known. It is further worthy of notice, that the three youngest victims of the disease were intemperate; and it appears to me probable that intemperance in early life specially tends to induce this malady.

It will be observed that I have not given heart-disease as a cause in any case. My reason for so doing is, that I regard the changes induced by passive congestion, in consequence of heart-disease, as distinct from those of true cirrhosis.

With regard to the mode of origin, it was in every case insidious. It never was marked in its early stages by dropsy and diminished urine, as is the inflammatory, nor by polyuria without dropsy, as is the waxy. Sometimes an apparently early symptom was frequent nocturnal micturition, but in a large number of cases there was nothing to attract notice until convulsion, or dimness of sight, or palpitation, or debility drew attention to the disease already well advanced. The quantity of urine was large in four cases; all of these were far advanced and had hypertrophy of heart; it was slightly increased in two; it was natural in five; and diminished in six; in three the quantity was not noted. In almost all the cases the colour was pale; in two it was occasionally bloody. The urine was acid in all but one. Its specific gravity was generally below the normal standard, but in some it was up to it. The amount of albumen varied, in many cases being a mere trace, in some very copious. In certain cases the quantity varied remarkably from time to time, both during the same day and on different days.

Hyaline and granular casts were found in a large proportion of cases, and in four instances in which the amount of urea was determined it was found diminished. The amount was determined and found diminished in other cases besides those included in the table. Three of the patients suffered from pains in the loins, only one to a considerable extent.

These facts confirm remarkably the ideas generally prevalent as to the conditions of the renal secretion in this form of Bright's disease.

Of symptoms connected with the alimentary canal, the most common were nausea, vomiting, and catarrh of the stomach. One or other of these was present in eleven of the cases, but not constantly, nor in the early stage. The blood presented in three cases a slight increase of white corpuscles. It is noted as natural in thirteen cases, and as poor in red corpuscles in one; but these remarks apply only to microscopic examination, and I have made no chemical analysis of the blood.

Hæmorrhages occurred to a serious extent in four cases, and slightly in one. The heart was hypertrophied in sixteen of the cases, the hypertrophy generally most marked in the left ventricle. In four the heart was natural. Vessels of the size of the radial were noted as thickened and sclerosed in thirteen. The pulse was, in almost all, full and hard. In two of the three cases, in which I have notes of the condition of the minute arteries, there was thickening due to hypertrophy of the middle coat. The degree of hypertrophy of heart and of alteration in the middle-sized arteries strictly corresponded to the advance of the disease, so that evidently from the operation of a common cause, or in consequence of the disease, hypertrophy took place.

From all I have seen of these conditions, both in this and in other forms of Bright's disease, I am satisfied that the changes, both in heart and arteries, are, in some way or other, consequences, as Bright and Johnson suppose, not mere concomitants of the affection. Statistics which I formerly published (see *Bright's Disease of the Kidneys*, 2nd ed., p. 308) shewed that, of cases of the inflammatory form fatal in the first stage, hypertrophy of the heart was present in 12.5 per cent.

Among the nervous symptoms, none is so common or so persistent as neuro-retinitis. This was present in eight of the twenty cases, and in several constituted the first symptom which attracted attention. Many cases had the oedematous conjunctiva, and one or two had hæmorrhage into that structure. The cerebral symptoms consequent upon the disease existed in seven of the twenty cases, convulsions occurred in six, severe headache in about the same number, and coma ushered in the fatal result in five. In several cases convulsions occurred occasionally, and afterwards disappeared. Of those fatal in the second stage, it occurred in 38.5 per cent.; and of those in the third, in 100 per cent. These statistics prove that the changes in the circulatory system are consequences of the renal affection.

I have hitherto believed that the old theory of embarrassment of circulation from impurity of blood explained the change; but I cannot

altogether refuse credence to the ingenious hypothesis of Dr. Johnson, which would refer it primarily to a protective action on the part of the small arteries. This hypothesis certainly affords an explanation of the hypertrophy of arteries which Dr. Johnson discovered; and I must say that no other explanation occurs to me, unless this thickening could be shown to promote the propulsion of the blood. But, before accepting Dr. Johnson's hypothesis, it would be necessary to explain the occurrence in certain cases of cardiac hypertrophy with normal vessels.

It appears to me that the sclerosis in the smaller arteries and the thickening of the outer coat of the minute vessels are both to be ascribed to increase of connective tissue, which may be a result of increased tension within the vessels, or, perhaps, a local manifestation of that tendency to increase of connective tissue, which is so marked in the kidneys. Had the theory of Gull and Sutton been that the renal disease was a local manifestation of a constitutional tendency to increase of connective tissue, those who are of my way of thinking would have had nothing to say against it.

Among the diseases of the respiratory system, the most common were congestion, oedema, and bronchitis. These occurred in nine of the cases. They are extremely common towards the end. Emphysema is not unfrequently associated with the condition, and pleurisy occurred as a complication in three of the twenty cases. There was slight dropsy in a considerable number of the cases, but it was persistently present in none. Occasional swelling of the feet, puffiness of the eyelids, and oedema of the conjunctiva, are symptoms very frequently present. In one case the patient made a temporary recovery from coma. Delirium occurred also in a few of the cases; mania in one.

Besides these consequent complications, there were several chronic degenerative diseases of the nerve-centres. One patient had glosso-labial paralysis and muscular atrophy, three had aphasia and slight hemiplegia. Of the fatal cases, nine in number, death resulted from exhaustion in one; from uræmia in five; from pulmonary oedema and congestion and pleurisy in three.

The study of these cases appears to me fully to confirm the views which I have elsewhere advanced as to the symptoms, clinical history, and complications of the disease; and I hope that this paper may be accepted by the profession as a slight contribution towards the consolidation of our knowledge of these affections.

CASES OF COLOTOMY.*

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HAVING had during the last three years occasion to perform the operation of colotomy in the left loin nine times, I wish to bring the particulars briefly before the members of the surgical section, being convinced that the operation is a most valuable one, and applicable with great advantage to many cases otherwise irremediable.

Of my nine cases, which all occurred in females, two operations were undertaken for cancer of the rectum, causing obstruction, which had existed many days before the patients came under my notice: both died. Three operations were performed for scirrhus in an earlier stage before obstruction had occurred, and of these one died and two recovered; one of these patients dying seven months afterwards, and the other being about and well seven months after the operation. Two operations were performed for syphilitic ulceration and stricture; both recovered, and are alive now. One operation was performed as a last resource in a patient worn out with extensive fistula and ulceration (probably syphilitic) before she came under my notice, and proved fatal. One operation was performed for the relief of a recto-vesical fistula, and was perfectly successful. The result, therefore, was four deaths and five immediate recoveries. (See Postscript.)

CASE I.—Sophia D., aged 49, married twenty-six years, having had four children, was admitted into the Hospital for Women in January 1871. For six months she had had constant inclination to go to stool, with little result; there was no blood passed, and no pain during the act. Three months before, she began to pass blood with great straining, and had been treated for piles. On admission, two inches up the rectum there was a ring of hard irregular growths, admitting the point of the finger; the bowels had not acted for five days, but were relieved by an enema and medicine.

Colotomy was performed on January 28th, and the patient was dis-

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