place in joints if the direction of force transmitted through them varies from the normal and the articulation is made to perform a function for which it was not originally fitted. I refer to changes occurring in the joints in knock-knee, bow-legs, lateral curvature, in cases of shortening of one leg by fracture or disease, &c. I am well aware that both rheumatoid and rheumatic arthritis, and especially the former, are very prone to affect a joint damaged in its anatomical detail, and consequently in its physiology, by occupation or injury, and this has been pointed out very clearly by Dr. Hensley of Bath, and it may in some few cases be difficult to state whether the condition is one whose causation is solely traumatic, or whether added to this we have in addition a factor whose mode of action is much more complex; yet I would assert that in the very large majority of instances of the condition which I have called chronic traumatic arthritis there is no difficulty in recognising it either clinically or in the dead body, and that therefore we should no longer include this definite group under a series of names which only serve as a cloak to ignorance, but give it a definite and explanatory title.

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OBSERVATIONS ON CERTAIN DISPUTED POINTS IN RENAL PATHOLOGY.¹

By A. G. AULD, M.D.

FIRST I shall describe what seems to be the true interpretation of the morbid changes found in the pia mater and in the skin, then certain lesions in the Malpighian bodies, and lastly I shall deal with some considerations based on anatomical data respecting the cause of the polyuria which is so constantly observed in the contracting form of Bright's kidney. I have been enabled, through the courtesy of Dr. Coats, to examine the pia mater of all brains examined in the Western Infirmary during a period of fifteen months. In each case the membrane was carefully hardened in Müller's fluid and alcohol, then dehydrated, and soaked for some days in Hamilton's freezing fluid A. From the resulting sections some valuable facts were obtained; and, in particular, I have been enabled to make out by means of high powers what seems to be the true nature of the hyaline-fibroid thickening of Gull and Sutton. As is well known, these authors, in their celebrated papers, contended that in Bright's disease the bloodvessels throughout the body, and especially those of the pia mater, had their outer coats thickened by the deposition therein of a hyaline fibroid material, probably derived from the blood. The muscular coat in such vessels they conceived to be atrophied, while the inner coat frequently, but by no means invariably, partook of hyperplastic change. This theory received a most damaging criticism from Johnson, who strongly maintained the truly hypertrophic condition of the muscular layer, while the so-called "hyaline fibroid" he alleged to be a delusive result of reagents, such as glycerine and acidulated mounting fluids. In 1873 Grainger Stewart pointed out that, while mounting media undoubtedly had some influence in producing the hyaline appearance, not a few cases exhibited a true fibrous thickening. Dickinson subsequently lent his adherence to this view, but the precise nature of the change is still the theme of debate. According to my observations, it may be stated generally that in Bright's disease all the coats are thickened. In the vast majority of cases the inner coat shows endarteritis. This change seems very constant in fatty kidneys in adults, though it is occasionally absent in contracting kidney. As regards the muscular coat, there can be no room for doubt that Johnson's contention is the correct one. Further, it was maintained by this eminent observer that a longitudinal layer of muscle-cells could be observed alongside the hypertrophied circular layer in some of these cases. It was asserted by Dr. Bryan Waller that this was an impossibility, and that Dr. Johnson mistook the cellular strata of the thickened inner coat for a layer of muscle cells. I think, however, that on looking at one of the specimens we will be satisfied that to the inside of the hypertrophied circular coat longitudinal muscular fibres

exist which can in no way be confounded with the proximate layers of the thickened intima. There are, however, some cases in which the muscular hypertrophy is far from being a marked feature, and when the disease is far advanced degenerative changes of an aggressive character attack the hypertrophied fibres. In the case of the outer coat it is plainly shown from these preparations that it is thickened, and the question is, What is the nature of this thickening? Now, after eliminating the possibility of amyloid disease by careful staining, I have invariably made out by means of very high powers the existence of branched corpuscles, with dividing nuclei, either embedded in or lying alongside the hyaline-fibroid formation. It is clear that this formation owes its origin to a slowly progressing inflammatory new formation of connective tissue, and that the effect of reagents is, after all, but unimportant. Not only so, but this corpuscular activity is manifest throughout the connective tissue of the pia mater generally, leading to a more or less general thickening.

As regards the *skin*, the sections show that the muscular coats of the arterioles are much thickened. This is generally to be observed. In one case, in which the oedema was a very marked feature, the derma is unduly cellular; clusters of leucocytes seem to be scattered throughout its substance. It seems as though some inflammatory process were at work, and hence we find here considerable support to the view of Cohnheim as to the cause of this form of oedema.

With regard to the lesions of the kidney itself in chronic Bright's disease, examination of a large number of kidneys has led me to recognise two great divisions. In one, the lesions of the glomeruli and of Bowman's capsule are very marked, and overshadow the other lesions; in the other class the reverse holds good.² Greenfield recognises the lesions of the capsule as pericapsulitis or cellular infiltration and tissue-formation occurring chiefly outside this structure, as hyaline thickening of the capsule itself, and as endo-capsulitis or the formation of concentric laminae of cells and tissue inside the capsule between it and the glomerular tuft. Greenfield regards the cells lining the capsule to be capable of originating this formation. Now, in acute glomerulitis it is undoubted that layers of epithelial cells are formed inside the capsule, derived from germination of its lining epithelium. But in chronic glomerulitis this is no longer seen. Instead we have the following changes:—1. The epithelial cells covering the glomeruli swell and assume a peg-top or columnar shape (well seen in the section placed under the microscope); they then fall into the periglomerular space, and assume a rounded form. 2. A similar desquamation of the cells lining the capsule occurs, but without the intervention of the columnar shape. 3. The connective tissue nuclei, and likewise the nuclei of the capillary walls of the Malpighian tufts, germinate, multiply, and give rise to a new growth of tissue between the capsule and the tuft, the capillaries undergoing hyaline thickening the while. In one specimen we see a band of new tissue stretching across the peri-glomerular space to which numerous epithelial cells have attached themselves—a phenomenon which has been questioned by Ziegler and some others. 4. Not infrequently the capsule itself becomes slit up into several layers, giving the appearance of two or more capsules for part of its circumference. This dissecting process is accomplished by cells from the surrounding connective tissue. In other and more advanced cases the capsule appears striated and concentrically thickened, and blends with the sclerosed glomerular tuft. It is difficult to see why these lesions should occur in some cases and not in others; they are by no means confined to scarlatinal cases, and, moreover, in post-scarlatinal nephritis which has assumed a chronic form they are occasionally absent.

Lastly, I propose to advance a few original considerations respecting the cause of the polyuria which is such a characteristic symptom of contracting kidney—considerations based, I may say, almost entirely on the histological appearances which this kidney presents. It is the universal custom to ascribe the excess of urine to one or other, or else a combination, of the following factors. Increase of the blood pressure generally; increase of the blood pressure locally in the glomeruli, arising from obliteration of capillary areas; inability of the tubular epithelium to absorb the water passing from the glomeruli; and increased permeability of

¹ A paper delivered, with microscopic demonstrations, before the Glasgow Pathological and Clinical Society, Jan. 11th, 1892.

² I purposely omit here particular reference to changes in the parenchyma.

the glomerular wall. On each and all of these alleged causes the facts of pathological histology seem to me to cast grave doubt. As regards the third of these—the inability of the glandular epithelium to absorb the water,—I do not believe that such an absorption normally occurs; for if it did, we should have the extraordinary phenomenon of a cell simultaneously secreting and absorbing a waste product. Many arguments could be adduced against this theory, but it does not vitally concern my position. Consider first the normal secretion of urine. It is accomplished by two great systems, from two great sources. One of these is the glomerular system, which secretes the watery part of the urine *plus* the salts; the other is the tubular system, which secretes a fluid rich in urea and extractives. This is conclusively proved by the experiments of Nussbaum on amphibians, and by the experiments of Heidenhain. With respect to the glomerular system, the view of Heidenhain seems unquestionably the correct one, that the Malpighian body is in reality a secreting gland and not a mere filtrating apparatus, otherwise it is inconceivable how, after section of the medulla, the injection of sodium acetate into the blood is accompanied by a copious secretion of urine which is of course unattended by any rise in the blood pressure. And, indeed, in the case of granular kidney, Broadbent has given cases in which the polyuria was marked without any rise in the arterial tension. The epithelium of the tuft is therefore pre-eminently a water-secreting epithelium. Now, what is observed on examining microscopically sections of granular kidneys? It is found that the glomeruli are agglomerated together in more or less wedge-shaped areas of sclerotic tissue, the intervening spaces being occupied by dilated tubules whose basement membrane has escaped much alteration, but whose vascular supply is abnormally abundant. Now, in the majority of cases, 80 per cent. of the Malpighian corpuscles in these sclerosed areas are totally functionless. How, on any pressure theory, can the few remaining tufts manage to secrete the large quantity of water which flows from the kidney, especially when, in addition, it is considered that the tubules from the patent glomeruli are not infrequently snared by the cirrhotic tissue? It seems to me that this view must be abandoned, and the seat of the secretion of water sought for elsewhere. This I believe to be found in a peculiar modification or transformation of the tubular epithelium in the areas which have escaped destruction. It is a remarkable fact, acknowledged by all observers, that the normal glandular epithelium in this situation becomes converted in granular kidney into a thin, flat epithelium—an epithelium, in fact, resembling that normally lining the glomerular tuft. This may be readily seen in the sections. Is there, therefore, any reason to deny that this peculiar modification of the epithelium is but a compensatory process of nature whereby the elimination of water is effected? Further, these epithelial cells have bright-staining nuclei which proves them to be in an active state. If it be not conceded that this epithelium actually subserves a modified function—that of secreting a much larger percentage of water than normally,—I think it at any rate cannot be denied that it more readily permits a passage outwards of water from the underlying bloodvessels, and that it ceases to become a “water-inhibiting” epithelium, as in the normal gland.

This view is supported by the anatomical appearances, by the constant diminution of the urine in cases where the epithelium is gravely injured, and by many of the kindred facts of pathology.

Glasgow.

CONGENITAL ANOMALIES OF THE RETINAL VEINS.

By SYDNEY STEPHENSON, M.B., F.R.C.S. EDIN.

It is impossible, I suppose, to over-estimate the importance of recognising congenital variations of normal conditions, and nowhere throughout the body does this recognition assume greater clinical consequence than in the eye. Loring¹ has well said, “It is the same old story of the musician and his scales. Five-sixths of

the art of ophthalmoscopy are contained in a knowledge of the normal eye; the rest is a series of representations which can be read almost at sight.” In the eye, as in other parts of the body, that arrangement of the vessels which obtains in the majority of cases is regarded as representing the normal condition of affairs. It may be well, therefore, to preface this account of certain anomalous dispositions of the retinal veins by a short description of the usual mode of distribution of these vessels. The primary division of the central vein of the retina may take place either in the retro-ocular portion of the optic nerve, or (less frequently perhaps) upon the surface of the disc itself. Either mode of division results in the formation of a superior and an inferior primary vein. These veins again divide (usually upon the disc), and their branches undergo division and subdivision in a dichotomous fashion; but it is commonly held that no insculcation takes place between the various branches. The whole process of repeated dichotomous division gives rise to the picture of an arborescent system of non-anastomosing veins, which, springing from the optic disc as a centre, ramify in the innermost part of the nervous tunic of the eye. The smaller variations observed in this general plan of distribution are innumerable: no two eyes ever present exactly the same arrangement of the retinal vessels. (The veins, it will be noticed, have been described as pursuing a course contrary to that taken by the blood current; this has been done simply for the sake of lucidity.) In accordance with the fact that, throughout the body generally, the arrangement of veins appears to be less stable and uniform, less fixed, so to speak, than that of the arterial system, one meets with anomalies of the retinal veins more frequently than of the retinal arteries. I have, for instance, seen eight examples of an anomalous disposition of the retinal veins during the past eighteen months; while during the same period I have failed to meet with a single anomalous retinal artery.

Turning now to the literature of the subject, one finds that the number of recorded cases is not great. In the *Medical News* (Philadelphia) of Sept. 11th, 1886, Dr. B. Alex. Randall published a case in which the central vein was connected with an aberrant vein by means of a cross-branch; the communication took place across the optic disc. The same author, moreover, recorded, in the Transactions of the American Ophthalmological Society for 1888, four new cases, and incidentally he mentioned the existence of a sixth case which he had recently encountered. Dr. Randall's communications are illustrated by drawings of the conditions he describes. In Vol. IX. of the Transactions of the Ophthalmological Society of the United Kingdom, Mr. Adams Frost, under the title of “Abnormal Course of a Retinal Vein,” described a venous abnormality, which he had met with in the right eye of a young woman; while in Vol. X. of the same publication an account of “an abnormal retinal vein” is given by Mr. Louis Werner. Both these authors give pictures illustrative of their respective cases. Under the somewhat ill-conceived title of “Bifurcating Retinal Veins,” I described, in Vol. XI. of the Transactions of the Ophthalmological Society, two examples of the condition in question. The patients were shown at a meeting of the Society; and illustrations of the anomalies will be found in the Transactions.

It appears, then, from the above catalogue that ten examples of the condition discussed have been placed on record. Accompanying this communication will be found sketches of six anomalous retinal veins which I have met with lately. In this number I do not, of course, include those cases mentioned above as having been shown by me to the Ophthalmological Society; for that matter, examples of arrangements similar to those then exhibited have been met with since, and are included among the sketches now presented.

In connexion with this matter of anomalous veins certain questions naturally present themselves. What forms do these anomalies assume? Are the arrangements seen reducible to any sort of order; or are they, on the other hand, wholly irregular? Judging from my own cases and the cases placed on record by others, I should say that the anomalous arrangements fall into one or other of three classes, with this reservation, however: that the classification here outlined must, in the absence of more extended observations, be regarded as more or less provisional in its nature. The first class includes cases in which two of the primary veins are united by

¹ Text-book of Ophthalmoscopy.