faces. On examination, we found that the perinæum had been completely ruptured, the rent extending through the external sphincter into the rectum. Owing to the urgent nature of the case, we had not noticed the rupture at the time when it occurred, and I can only account for it by supposing that, from the violence of the convulsive attack just at the moment of delivery, one of the limbs of the child must have been forced through the perinæum. The patient was afterwards operated upon very successfully by my colleague Mr. Coe.

NOTES OF A CASE OF INFLAMMATORY BRIGHT'S DISEASE: FATAL IN THIRD STAGE.

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CONSIDERING the views still prevalent in this country and abroad, and the new opinions which have lately been advanced in regard to Bright's disease, I think it desirable to put on record from time to time any individual cases which appear to throw light upon obscure questions connected with the malady. The case which I propose to bring under notice is of value, as affording strong evidence of the occurrence of atrophy, or the granular condition, in the advanced stage of nephritis. The Society is doubtless well aware that the leading German authors regard all examples of atrophied kidney as results of inflammation, while many of the English authorities have gone to the other extreme, and asserted that disease of the tubules never can give rise to atrophy. Now I maintain that, while a large majority of examples of the true granular kidney are results of cirrhosis, some are certainly also results of inflammation of the tubules—indeed, that this result necessarily follows if the patient live, and the disease last long enough. This opinion is, I am glad to find, now shared by at least one excellent clinical observer among the Germans, Professor Bartels of Kiel,* whose recently published lecture I regard as an important contribution to the subject.

The case is that of a man who was for seven years under my observation, and was closely watched during the whole period. An account of it was given up to the date of their publication in both editions of my book on *Bright's Disease*† and so crucial was the case in my opinion, that, had the *post mortem* revealed conditions other than those actually found, I should have felt myself under the necessity of altering my views in important respects. The part of the history already published I shall give only in outline, the later part and the necropsy a little more in detail.

Robert Pendreigh, originally a baker, afterwards a maltster, and ultimately a labourer in St. Margaret's Railway Works, was a perfectly healthy man until about the end of January 1865, and was then twenty-six years of age. At this date he became dropsical. The dropsy increased rapidly, and he observed that he had frequent calls to micturate, but passed little urine at a time, and that dark and bloody. He was admitted to the Infirmary on March 3rd, suffering from severe dropsy and uræmic convulsions. Under cupping, diuretics, and other treatment he somewhat improved, but soon again the symptoms increased in severity. Towards the end of April he came under my care. His face was then pale and pasty. He had general anasarca. The urine amounted to forty ounces daily, was pale but smoky, of specific gravity 1020, highly albuminous, and contained hyaline and fatty casts. There was much bronchitis, and some pulmonary oedema. Hot-air baths, acetate of potash, digitalis, and cream of tartar having proved unavailing, inhalations of oil of juniper were tried, with the result of producing immediate and free diuresis. The urine increased to upwards of one hundred ounces daily, and by this means, and pricking the dropsical skin, the dropsy soon yielded. He steadily improved, and was dismissed early in August quite well, except that the urine was albuminous.

In October of the same year, after exposure to cold, he had another attack as severe as the first, but less prolonged. In its course, also, he had at least one uræmic convulsion. From this time until 1871, I saw him occasionally. He thought himself quite well, and worked regularly, but the albuminuria continued.

On March 6th, 1871, he was readmitted to the Infirmary with an aggravation of his symptoms. His urine, very albuminous, varied from fifty to sixty ounces, and had, he stated, been recently increasing in quantity, especially during the night, when he had usually to rise four or five times. He had caught cold and had some degree of bronchitis,

with return of dropsy. The heart had become hypertrophied, beating between the sixth and seventh ribs, the first sound being much prolonged. The pulse was full and hard, and the radial arteries thickened. The urine was ninety ounces daily, of pale colour, one-half albumen, and there were some hyaline and granular casts. Under treatment he improved, and went out at the end of April.

On June 3rd of the same year, he returned, suffering from pleurisy and catarrh of the stomach, with a return of the symptoms. The urine was scanty, of specific gravity 1011, contained much albumen, and a few hyaline and granular tube-casts. By the end of June he was again better, the urine having risen to sixty or eighty ounces daily, of specific gravity varying from 1011 to 1019.

The last time that Pendreigh presented himself at the Infirmary was on January 12th, 1872. He then stated that from the time of his leaving in June he had been pretty well, and had worked regularly until the end of December, when he got wet, and was at once seized with symptoms like those of his previous attack. On admission, his eyelids, and, to a less extent, his face generally, were oedematous; his expression was anxious; and he was restless and had considerable dyspnoea, so that he could not lie down. His temperature was 100 deg. Fahr. There was no abnormality of the alimentary system, excepting some difficulty of swallowing and a considerable amount of ascites. The heart's impulse was diffused; the area of dulness could not be made out from the state of the lungs: the first sound was prolonged. The pulse was 80, regular and strong. The radial arteries felt firm to the touch. The capillaries and veins were natural. The white corpuscles of the blood were somewhat increased; but there was no evidence of enlargement of the spleen. The action of the left side of the chest was somewhat defective; and towards the base of that lung, both in front and behind, there was a degree of dulness. Auscultation all over the chest, and especially on the dull part, revealed prolongation of expiration, the character masked by crepitations, sibilæ, and snoring *râles*. The vocal resonance could not be determined. There was great dyspnoea; the patient at first maintaining the erect or semi-erect posture, afterwards lying down. The cough was frequent, but not severe. The sputum was copious, of loose consistence, and contained streaks of blood. There was some oedema of the legs, but less than on the face; and the nutrition of the body was not markedly defective. He complained of a dull aching in the lumbar and vesical regions. The urine was thirty to forty ounces daily, of a light straw-colour, of specific gravity 1014, and acid reaction. It contained no deposit. The coagulum of albumen was one-fourth of the urine. The patient's memory was defective, and it was difficult to keep up his attention. He was drowsy, and had difficulty in keeping awake; still he slept badly, and was constantly restless, though not noisy. Warm poultices were applied to the chest, and other treatment adopted, but without benefit. On the 15th, three days after admission, all the symptoms had increased in severity. The dyspnoea was very distressing. He could not swallow. The bladder was not distended, although he had passed no urine for twenty-four hours. On the evening of the 16th, he vomited a large quantity of a fluid resembling hare-soup. His dyspnoea and restlessness continued unabated. He spoke a good deal, often confusedly; but was not so drowsy as formerly. He had much difficulty in speaking. During the night he remained conscious, and he died on the morning of the 17th.

The *post mortem* examination was made by Dr. Pettigrew on the following day. There was no oedema, excepting to a slight extent about the face and ankles. The pericardium was natural, excepting that there were some milk-spots on its surface. The heart was much enlarged, and weighed 19½ ounces. The hypertrophy was mainly, but not exclusively, confined to the left ventricle. The valves were competent—here and there slightly atheromatous. The coronary arteries were tortuous, atheromatous, and in some parts contained calcareous plates. The muscular fibre of the heart was not examined microscopically; it appeared natural. The aorta was in some parts slightly atheromatous. Many of the smaller arteries, such as those at the base of the brain and the radials, were diseased; some were sclerosed, some atheromatous, and others calcareous. Both lungs were adherent to the thoracic walls to a greater or less extent; their substance was congested and oedematous, in some parts almost solid. The bronchi contained a large amount of mucus. Their mucous membrane was congested and thickened. The liver was rather below the normal size; its surface was slightly granular. On section, it felt tough and fibrous; and, on microscopic examination, the interlobular connective tissue was found increased. The spleen was normal. The stomach was in a state of chronic catarrh, its surface coated with mucus, its mucous membrane thickened, and a few minute extravasations existing throughout its substance. The intestines were in a similar condition. The kidneys were smaller than natural, and weighed each 4½ ounces.

* Klinische Studien ueber die verschiedenen Formen von Chronischen diffusen Nierenentzündungen. Von Prof. C. Bartels in Kiel.

† A Practical Treatise on Bright's Disease. Second edition, 1871, p. 56.

Their capsules stripped off readily. The surface was uneven and granular. There was little sebaceous-looking material to be seen from the surface. On section, the organs appeared somewhat congested, especially the cones. The cortical substance was relatively considerably diminished. Throughout it, and to a less extent in the cones, there were deposits of sebaceous-looking material. There was also a good deal of fat in the pelvis of the kidney. The ureters and bladder were natural. On microscopic examination of the kidneys, many of the tubules were found to contain fatty matter, and in some there were mineral impregnations. There were no cysts, nor was the connection increased as in cirrhosis. The arteries were thickened and prominent. Many tubules were normal; but here and there, throughout the whole organ, there were individual tubules full of opaque fatty matter. The size and form of the affected tubules varied much, some being of natural size and round, others smaller and angular, presenting, as it seems to me, the different stages of absorption of contents and consequent atrophy. The brain was pale and somewhat cedematous; it weighed nearly three pounds. The vessels at the base were at many parts atheromatous and calcareous.

I do not propose to comment upon all the points in this case—only to select two or three which appear of special interest. On looking back at this case, I feel convinced that a single process was in progress throughout its whole course. It will not be denied by any one, that at the commencement we had to do with an ordinary though severe case of inflammation of the kidney: and there was no break in the history. The albuminuria continued throughout. The tube-casts, the occurrence of dropsy when he caught cold, the gradual deterioration of the blood and the tissues generally, the hypertrophy of the heart, and the degeneration of the arteries, were all such as we expect to follow upon such an ailment when it neither proves fatal nor is fully recovered from.

The morbid anatomy exactly corresponds to what I have described as the third stage of the inflammatory form of Bright's disease, and of course confirms the accuracy of that description. I quite perceive that some observers might interpret the case differently. No one could regard it as an example of pure cirrhosis, but it might be held to be a combination of inflammation of the tubules with that process. This view appears to me untenable for the following reasons: 1. That the kidneys were not so reduced in size as they are in cases of fatal cirrhosis; 2. That the capsule slipped off readily, not carrying with it any portions of the renal tissue, as it does in cirrhosis; 3. That the surface was granular and uneven, but not to the extent met with in cirrhosis; 4. That the cortical substance was atrophic, and contained abundance of sebaceous-looking material; 5. That the stroma was not absolutely increased, while the blocked up tubules presented all stages of atrophy from the commencing to the most advanced—an atrophy apparently due to molecular absorption of the contents, rather than to any increased growth of connective tissue.

The symptoms attending the process were also of interest. While the albuminuria was persistent throughout, there were at first diminished flow of urine and dropsy. At a later period there was little dropsy, and the urine attained or exceeded the natural amount. The dropsy was throughout determined by the amount of urine secreted; and that secretion was, in the early stage, below the natural amount, because the obstruction in the tubes exerted a counter-pressure to the blood-pressure within the Malpighian bodies. In the later stages, when hypertrophy of the heart had occurred, and when comparatively few tubes were occluded, the blood-pressure was so increased as to induce a certain amount of diuresis. This appears to me one of the most beautiful and satisfactory explanations of pathological process with which I am acquainted.

Another point which is worthy of remark, is the extreme desirability of discovering some treatment by which the chronic inflammation which results in continued albuminuria should be removed. My experience shows me that the skim-milk treatment, so highly commended by Dr. Donkin, does not suffice to accomplish this; and while I have sometimes succeeded by means of counterirritation, with croton oil and with iodine, I have often failed. The internal use of belladonna also has in some cases appeared useful, but it is in too many cases valueless. I shall be very glad to hear the experience of members of the Society in regard to the point.

I may further remark that this case has a certain bearing upon the views recently advanced by Sir William Gull and Dr. Sutton. If my interpretation of the facts be correct, the thickening of the arteries and disease of the heart were complications consequent to the renal affection; and, if this be so in one form of chronic Bright's disease, it surely establishes a probability that in other forms the relationship will not be reversed—that, in other words, cirrhosis also induces, and is not a mere attendant upon, vascular disease.

ON DIPHTHERITIC ALBUMINURIA.*

By R. BROWNING, L.R.C.P.L. etc.

THE pathognomonic importance of albuminuria when occurring in the progress of true diphtheria is a practical point which, strangely enough, I find, after some painstaking, either totally passed over, or at least barely mentioned in the writings and *visà voce* cliniques of the highest authorities I have yet been able to consult.

In the absence of *post mortem* observations of my own, it is with some diffidence that I venture to put before you what I have reason to consider an equally novel and successful treatment of this momentous symptom. I would premise that by the term "true diphtheria" I wish to specify that dangerous disease known, unfortunately, but too well to most of us in this neighbourhood, characterised especially by the appearance of a false membrane on the surface of the pharynx, which membrane can be removed in strips by a forceps, and consists of numerous epithelial scales and cells, with some occasional pus-globules, all interspersed in the intestines of fibrinous filaments, to the exclusion of so-called "genuine croup," of "laryngitis stridulosa," and of any bad forms of tonsillitis, muquet, or local scarlatin lesion. [I have placed "genuine croup" in this *Index Expurgatorius* from deference to the opinion of most English authors; but many years since I was taught by Dr. Semple, at the St. Pancras Dispensary, that it was really and actually the diphtheritis of Bretonneau, and the same is held by most modern French writers.]

From what I have lately witnessed while watching two local epidemics of diphtheria, I am disposed to consider

a. That albuminuria is present in nearly all cases.

b. That its appearance is usually about the end of the first week after the diphtheritic membrane is developed, though sometimes earlier, more rarely later.

c. That coexistently with its appearance, a notable diminution of the quantity of the urine, and an increased excretion of urea occur (the latter sometimes equalling treble its normal amount); whilst lithates (with uric acid, oxalates, and sometimes phosphates) generally, tube-casts, both granular and waxy, frequently, blood-corpuscles not seldom, and pus-globules occasionally, are found on examination of what is secreted. The urinary specific gravity mostly averages 1016, and the temperature of the body is, as a rule, from 100.4 to 102 degrees.

d. That the gravity of the prognosis increases in an equal ratio with the quantity of albumen existing in the urine, independently of the amount of throat-affection or kidney disorganisation, and that an early or late discovery of albumen is of serious import.

e. That local mischief attacking the pharynx, larynx, or other structures, and paralysis subsequently occurring, are entirely the result and symptomatic of a morbid poison affecting the general system, just as the sore throat of syphilis is the sequence of a blood-disease previously contracted.

f. That albuminuria, in any quantity, is due to obstruction of circulation through the kidneys, caused by congestion of the Malpighian tufts, this congestion being produced by paralysis of the nerves supplied to them; but that a mere trace only arises, either from pus or else blood-corpuscles, which have casually entered the volume of urine.

g. That the indication of treatment, in albuminuria occurring to an appreciable degree during the progress of a case of diphtheria, is to remove this obstruction by overcoming the paralysis, and that local Faradisation affords the best method of doing so.

h. That, after albumen has disappeared from the urine, it may return, synchronously with a relapse of diphtheria; and that throughout the whole course of this malady little, if any help, is derived from the use of the clinical thermometer.

[Details of seven cases, tending to support the above views, were here read.]

You will observe that *both* the cases which terminated fatally were those in which no Faradisation was employed, and that *five* others, all of very serious nature, recovered after it was resorted to; *all* were marked by unmistakable evidence of blood-poisoning, and albuminuria, with more or less suppression of urine, was noticed in each. The treatment of all was conducted on the same principle, plus or minus the induction coil; the object aimed at being at first, during the premonitory symptoms, to regulate the secretions, and then to support the strength of the system in every possible way. My sheet-anchor was the tincture of perchloride of iron sometimes combined with glycerine, sometimes with chlorate of potash, and sometimes given *per se*. Stimulants and nourishment in every variety were supplied with no sparing hand. Iodide of potassium proved a disappointment. The customary topical

* Abstract of a paper read at Canterbury, May 23rd.