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Bright and Bright's Disease

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Abstract

Richard Bright was born in 1789, the year in which George Washington became first President of the United States of America and France suffered a revolution. His father was a banker living in Bristol, an important city resting on the laurels of five centuries of maritime enterprise. The circumstances of his arrival were but the first of Bright's fortunes for he was a man on whom the sun consistently shone. He had the very best education, locally in Bristol and subsequently at the school of medicine in Edinburgh; he achieved early and continuous success in medicine and lived to enjoy its reflection; he travelled extensively; he had the largest general practice in London and the best facilities for clinical and pathological research; he was a copious author and a delicate artist; he retired young to enjoy his leisure and he died unobtrusively in his 70th year. During his lifetime he was associated with some of the greatest names in medicine; he became himself one of the greatest names in medicine.

As he is indubitably a giant figure in our medical history, I think it is worthwhile to examine his biography more minutely before going on to describe his contributions to medicine, and, more particularly to renal disease.

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Bright and Bright's Disease

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Richard Bright was born in 1789, the year in which George Washington became first President of the United States of America and France suffered a revolution. His father was a banker living in Bristol, an important city resting on the laurels of five centuries of maritime enterprise. The circumstances of his arrival were but the first of Bright's fortunes for he was a man on whom the sun consistently shone. He had the very best education, locally in Bristol and subsequently at the school of medicine in Edinburgh; he achieved early and continuous success in medicine and lived to enjoy its reflection; he travelled extensively; he had the largest general practice in London and the best facilities for clinical and pathological research; he was a copious author and a delicate artist; he retired young to enjoy his leisure and he died unobtrusively in his 70th year. During his lifetime he was associated with some of the greatest names in medicine; he became himself one of the greatest names in medicine.

As he is indubitably a giant figure in our medical history, I think it is worthwhile to examine his biography more minutely before going on to describe his contributions to medicine, and, more particularly, to renal disease.

I can find no evidence that Bright was anything special at school, which he attended in Bristol and Exeter. A portrait of him hitting his mother shows that he was a pretty, if aggressive, child. At any rate, he decided for medicine and arrived in Edinburgh in 1808 to enter the course. While he was here he became a member of the Royal Medical Society delivering two dissertations on "Gangrene" and "Retroversion of the Uterus." These were the first of his contributions to medical literature. In 1811, he continued his studies at Guy's Hospital, but two years later he was back in Edinburgh to graduate M.D. Bright then turned his back on his medical school for ever and, after some years of travel, returned to Guy's—at that time, as now, a Mecca of medicine. With his appointment as Assistant Physician in 1820, his feet were firmly on the great ladder to the top of which they were quick to set out.

From this time until his retirement in 1843, Bright served Guy's continuously. His was just one of the names on the hospital staff of that time which are familiar to us today. A senior member was Sir Astley Cooper, the most popular teacher of Anatomy and Surgery in London. Cooper encouraged and advised Bright prior to the publication of his first and greatest work, and the author accorded him lavish acknowledgement in the preface. But probably more famous still are the names of the two colleagues with whom Bright formed a great triumvirate—one of the finest in the history of any hospital and certainly in that of Guy's. Thomas Addison and Thomas Hodgkin have both left their names in our textbooks. Addison was Bright's close colleague and collaborator for over twenty years, and dominated the medical school for a quarter of a century. He was an eloquent lecturer, a great clinician, and a magnet for the students. But his

fame in his lifetime was local and he never achieved the universal recognition of Richard Bright. Hodgkin performed the autopsies on almost all Bright's cases and described himself "Hodgkin's Disease" which now perpetuates his name. It is easy to see that Guy's at this time claimed a formidable teaching team, and it is a happy reflection that the three greatest components graduated at the University of Edinburgh.

Before coming on to discuss Bright's great contribution to medical history, I must refer briefly to his interests outside medicine. He lived in days when a student with money and recreations had time to self-indulge and there is no doubt that the qualities of minute and accurate observation which he later brought to his medical writing, had been bred far from the hospital ward and post-mortem room. He was a biologist, a linguist, a traveller of enthusiasm and an author of some distinction. While at this University, he sailed to Iceland with Sir George Stewart MacKenzie, a noted geologist, and amassed botanical and zoological material for a contribution to MacKenzie's published account of the expedition. On his way home he was fortunate enough to be blown ashore on the Orkneys, from which islands, however, he quickly sailed for Leith. In 1814 he was abroad again, in Berlin, in Vienna during the famous Congress, and then travelling widely in Hungary. While in Vienna he had an audience of the King of Rome, an infant whom the doctor did not impress, and on his way home he stood on the field of Waterloo a fortnight after the defeat of the King of Rome's father, Napoleon Buonaparte. In London again, Bright published a graphic travelogue, which loses little by comparison with his subsequent medical literature.

I have now outlined the background to the life's work of Richard Bright. The man I have described was, in everything he did, a keen and thorough student, an accurate observer, a rigid recorder, above all an ardent enthusiast. To medicine he was a devotee: let us now see what medicine got from him.

The first published account of Bright's work appeared in 1827 in three volumes entitled "Reports of Medical Cases selected with a view of illustrating the Symptoms and Cure of Diseases by a reference to Morbid Anatomy." The first part of Volume 1 deals with diseases of the kidney and it is now a landmark in the history of medicine. Twenty-three cases are described which had been under his care, and, in all, the emphasis is on the association of albuminous urine with dropsical effusions or anasarca. Each case is followed to the post-mortem room, where, with the aid of Hodgkin, Bright consistently demonstrated structural derangement of the kidney visible to the naked eye. There are five delicately executed plates which cover a wide range of renal pathology.

In the preface to this volume, Bright gives an explanation of the significance of these cases. Dropsy, by which he meant effusions into the body cavities and tissues, was clinically evident in diseases of the heart, the liver or the kidneys, but only in the latter case—renal disease—was it associated with albumen in the urine. He began to feel that the association of dropsy and albuminous urine in any patient indicated kidney pathology, and the 23 cases he presented in this volume illustrate how his clinical suspicions were confirmed at autopsy. He also remarked that he had in almost all instances found some degree of hæmaturia.

The clinical and pathological details are very comprehensively presented, and in the course of his cases he describes perfectly the conditions we now know as Nephrosis and Chronic Nephritis. He suggests also that there are three possible morbid appearances of the kidney, and his descriptions coincide exactly with the contemporary nephrotic, chronic nephritic and

arteriosclerotic kidneys. It will be of value here to give two examples of the extraordinary precision with which Bright described conditions identical to those we come across today—130 years later.

John King, aged 34, had for three weeks had swollen limbs and oedematous hands and face. On admission he had scanty urine, about one pint in 24 hours. Shortly afterwards the urine became copious, dingy brown in colour and coagulable by heat. While in hospital he first of all suffered from Herpes Labialis, then Pneumonia and all the time from symptomatic treatment without hope. He eventually succumbed to pneumonia and was found at post-mortem to have considerable fluid in the pleura, pericardium, and abdominal cavity, a healthy liver, and kidneys—to quote Bright—“completely granulated throughout.”

Henry Izod, aged 25, suffered a slight attack of dropsy after which he was well for a year before he became swollen all over and died seven weeks later. His urine was not examined but after death his kidneys were found to be “almost white in external appearance, rather large and lobulated.” His heart was also slightly enlarged.

These two cases I have only outlined, Bright having given minute clinical and pathological detail. They serve, however, to demonstrate what an enormous alteration his observations must have made to contemporary thought on renal disease, and he experienced probably a unique credit when, with hardly a word of dissension, the medical world immediately began to talk about “Morbus Brightii”—Richard Bright’s disease—a title that selected itself for a condition which had not experienced material advance since the days of Rhoupfos in Greece, 1700 years before.

But Bright did not rest on his laurels. Several times in his publication of 1827 he had referred to an enlarged heart post-mortem—as, for example, in both the cases I have quoted above. In the Guy’s Hospital Reports of 1836, he refers to it again and also to a number of deaths from apoplexy in people suffering from renal disease. We now, of course, know how accurate his observations were for the subsequent development of techniques for measuring blood-pressure has led us to recognise hypertensive encephalopathy and left heart failure as common terminal events in Chronic Nephritis.

This article of 1836 also contains a classical description of Acute Nephritis. The frequency of Scarletina in the aetiology is pointed out and we are assured that “nice analysis of the blood will frequently detect a great deficiency of albumen, and sometimes manifest indications of the presence of urea.” We know very little more in 1960 about Types I and II Nephritis and Chronic Nephritis than was pointed out by Bright in these two papers. Only the arrival of the microscope and the sphygmomanometer have at all improved our knowledge without, regrettably, a parallel advance in treatment.

Only one proposal of Bright’s do we challenge today and that is his effort to persuade us of the importance of drink in the aetiology of nephrosis. Looking back from our age of temperance, we label it coincidence that so many of his patients were also alcoholics and chronic dissipants.

In the Guy’s Hospital Reports of 1836, he further presented a tabular view of the morbid appearances of 100 cases who had died with albuminous urine. All were found to have either hard, contracted kidneys or large, soft kidneys, and the most frequent causes of death were cerebral derangements and intercurrent infections. Many were found to have hypertrophied hearts and oedematous lungs, but overall the other organs were healthy. These observations are all very basic in our present knowledge of the outcome of subacute and chronic Bright’s Disease.

His fourth publication on renal disease—the Gulstonian lecture of 1833—presented a summary of the signs he had come to associate with albuminous urine—anasarca, uræmia, absence of urea from the urine, infections, cardiac hypertrophy, cerebral symptoms and pathology of the kidneys.

Bright died in 1838 at the height of a widespread and just reputation. His contribution to all branches of clinical research had been prolific—the heart, the liver, the spleen, the pancreas, the Gastro-intestinal tract, and the Central Nervous System—all had been faithfully observed in their pathological behaviour in life and appearance after death. He was a cheerful and attractive personality always careful to acknowledge the assistance of his juniors. In his heyday he had had no competition in research into kidney disease: the scope and detail of his own findings hardly encouraged it. But at the time of his death he had been retired from Guy's for 15 years, and would-be competitors, having sat back to take stock of the whole new concept, had in the meantime set the ball rolling again. So nephritis left Bright behind and emerged into the present century, where the successive efforts of Volhard and Fahr, Dorothy Russell and Ellis to classify his disease have detracted nothing from, and added many complications to, the original simple account of Richard Bright himself.

REFERENCES

1. Osman: Original Papers of Richard Bright on Bright's Disease.
2. Major: Bulletin of Medical History, 23, 453-460.
3. Guy's Hospital Reports. 1927.
4. Guy's Hospital Reports. 1958.
5. Hale-White: Great Doctors of the 19th century. 1935.
6. Nixon: Bright's Disease, 1954.

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REFERENCES

1. Macpherson. A.I.S., Postgraduate Medical Journal, 1956, 32, 474.
2. Daniell and Pritchard, "J. Physiol." 1951, 114, 521.
3. Riddell *et al.* Clin. Sci., 1957, 16, 315.
4. Atkinson and Sherlock, "Lancet," 1954, 1, 1325.
5. Paton, Reynolds and Sherlock, "Lancet," 1953, 1, 918.
6. Cameron and De Saram, "J. Path. Bact.," 1939, 48, 41.
7. R. Milnes Walker, "Portal Hypertension," Edward Arnold. 1959.
8. Sherlock, "Diseases of the Liver," Livingstone, 1955.
9. Shobinger, Cooper and Rousselot, "Ann. Surg." 1959, 150, 188.
10. R. M. Walker, Proc. R. Soc. Med., 1943, 36, 445.
11. Atkinson, "Lancet," 1959, 2, 819.
12. Macpherson, Postgraduate Medical Journal, 1954, 30, 184.
13. R. M. Walker, "J. Fac. Radiol." 1957, 8, 178.
14. Hunt, Jacksonian Prize Essay, Royal College Surgeons. 1956.
15. Macbeth, "Brit. Med. J.," 1955, 2, 877.
16. Tanner, Ann. R. Coll. Surg., 1958, 22, 30.
17. Linton and Elliss, J. Amer. Med. Ass., 1956, 160, 1017.
18. Madden. Surg. Gynaec. Obstet., 1954, 99, 385.
19. Ryss, S. M., and Smagin, V. G., Terap, Arkh., 1958, 2, 37.
20. Dawson, McLaren, Sherlock, "Lancet," 1957, 2, 1263.
21. Macpherson, A.I.S., *et al.*, "Lancet," 1954, 2, 353.
22. Large, *et al.* "Ann. Surg.," 1952, 135, 22.
23. Patek *et al.* J. Amer. Med. Ass., 1948, 138, 543.