

Past and present : the inaugural address of the Guy's Hospital Physical Society, delivered October, 4th, 1907 / by G. A. Gibson.

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PAST AND PRESENT.

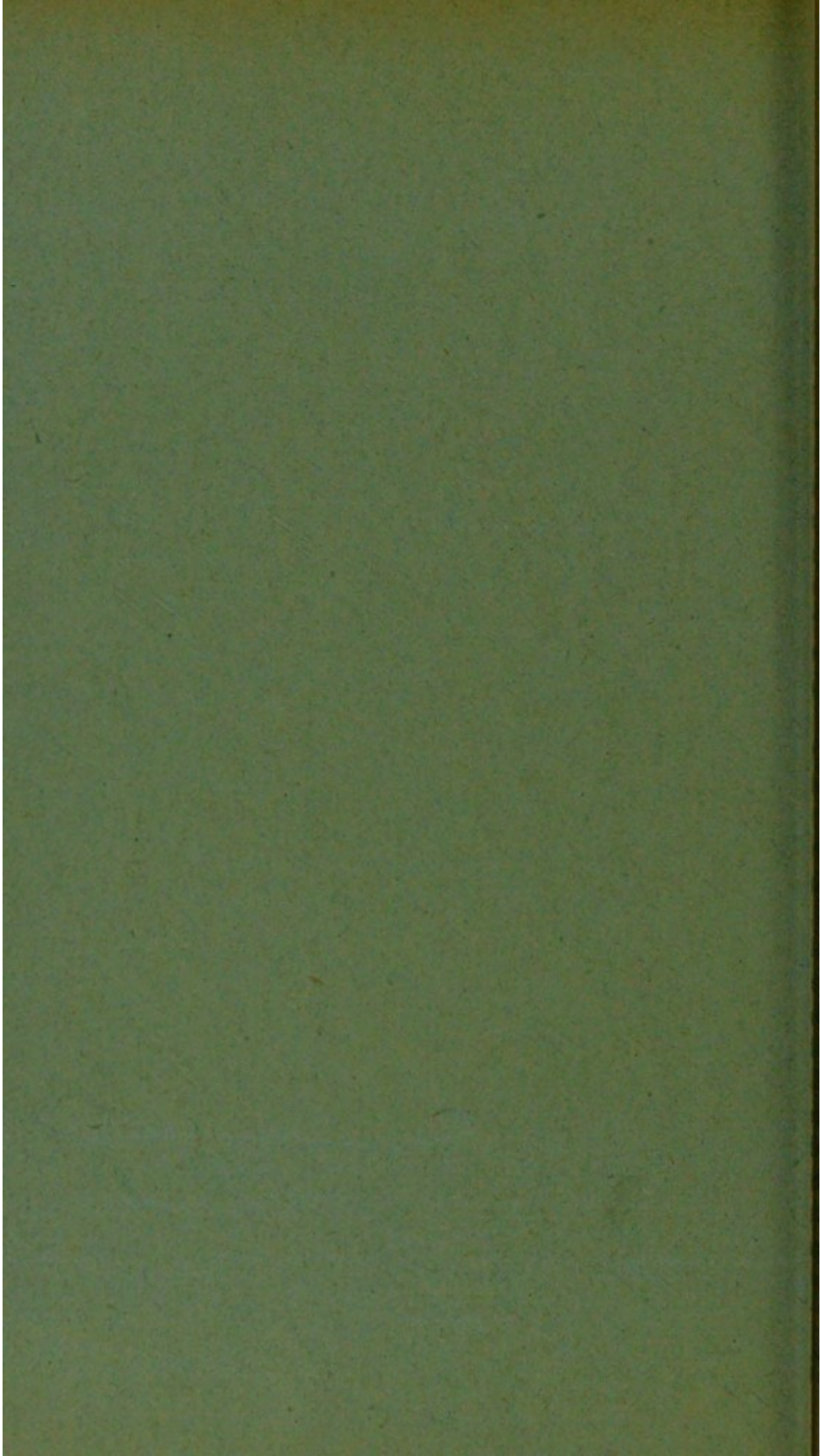
*The Inaugural Address of the Guy's Hospital
Physical Society, delivered October, 4th, 1907.*

BY

G. A. GIBSON,

M.D., Sc.D. EDIN., LL.D. ST. ANDREWS.

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Past and Present.

GENTLEMEN,—Among the many reasons which led me, very gladly, to accept your cordial invitation to be present this evening, three stand out most prominently. In the first place, the general sympathy which exists amongst men who have the same aims in common naturally leads each of us to rejoice in meeting with his fellows. Within the realm of science, in which medicine plays an important part, there ought to be no narrow jealousies. When one school flourishes, it offers an incentive to others to strive to emulate it in generous rivalry. In a word, the solidarity of modern science necessarily brings with it a spirit of comradeship throughout the civilised world.

In the next place, it is historically interesting to observe that the foundation of Guy's Hospital, in London, and the Royal Infirmary, in Edinburgh, took their origin in the same epoch. The Will of Thomas Guy was signed September 4th, 1724, and was proved on January 4th, 1795. Guy's funeral took place three days later, but even before his obsequies were conducted the hospital had been opened, and, as we learn from the interesting "History" of Sir Samuel Wilks and Mr. Stantony, sixty patients were in residence at that time.¹ In the same year, the Royal College of Physicians of Edinburgh, according to Sir Thomas Grainger Stewart,² made a proposal that a hospital should be erected for the benefit of the sick poor and the furtherance of medical studies, and initiated a public subscription with this aim in view. Four years later a "small hired house" was rented for the reception of six patients, very near the spot where the tragedy of Kirk o' Fields was enacted.

¹ A Biographical History of Guy's Hospital, London, 1892, p. 73, p. 75.

² Edinburgh Hospital Reports, 1893, vol. i., p. 1.

From such small beginnings the Edinburgh Royal Infirmary took its origin. It obtained a Royal Charter in 1736, the foundation stone was laid in 1738, and the old building, which was left twenty-eight years ago, was opened for the reception of patients in 1741. Our hospitals, therefore, date from the same period, and have the same length of tradition to look upon.

And, in the last place, above and beyond these reasons for a brotherly feeling, there exists between the southern and northern schools the strong tie formed by personal attachments. Throughout the last two centuries many distinguished graduates of Edinburgh continued their studies afterwards at Guy's, while, on the other hand, many of the most brilliant Guy's men came north to Edinburgh to study and graduate there. Time would fail me in any attempt to recite a list of such interchanges, but you will, perhaps, permit me to refer to two of the most distinguished ornaments of Guy's Hospital—in fact, two of the most renowned physicians who have ever adorned the annals of English medicine—I mean Bright and Addison. Both these great men studied in Edinburgh where they had notable undergraduate careers, and both of them shed the lustre of their genius upon the debates of the Royal Medical Society. When the Society, a few years ago, published a series of "Dissertations by Eminent Members of the Royal Medical Society," that of Richard Bright on Gangrene³ was gladly included in the volume. In the Preface, which was written by the late Sir Douglas Maclagan, the statement occurs:⁴—"It is to be noted, however, that there are several names, and some of them among the most illustrious, of whom there are no dissertations extant. Of these may be mentioned William Cullen, one of the founders of the Society, Joseph Black, Haller, the Monros, Oliver Goldsmith, Mark Akenside, Mungo Park, Sir Charles Hastings, Thomas Addison, and Charles Darwin." Sincere regret has been expressed on all hands in the Edinburgh School for the absence of any trace of Addison's dissertation

³ Dissertations by Eminent Members of the Royal Medical Society, Edinburgh, 1892, p. 64.

⁴ *Ibid.*, p. vii.

For these various reasons it has afforded me more than usual gratification to stand in this place this evening, and the pleasure is enhanced by the presence of Sir Samuel Wilks in the chair, in whose person there is a bond between Guy's and Edinburgh, seeing that he is an honorary graduate of my own University. Will you allow me, in this connection, to mention how frequently the friends of Guy's Hospital have had to mourn over the premature losses which it has sustained. Crabington and Fagge died long before their natural term of office had expired, while Bird, Mahomed, Carrington, and Washbourn were cut off while still assistant physicians, and King and Hodgkin did not even attain that position. Modern medicine has lost much through these unfortunate and lamented disasters.

It did not occur to me, in suggesting the title which has been used for this address, that it savoured of imitation, inasmuch as it is that borne by Carlyle's most charming work, until it was too late to effect an alteration. Before plunging into the subject-matter, let me express the hope that you will absolve me from any attempt at competition with my great countryman, the "Seer of Chelsea." The current of thought, in truth, leading to the use of such a title, was very largely directed by pondering over the great obligations to Guy's Hospital under which modern medicine lies. It would not become me—it would, in fact, be beyond my powers—to say anything with regard to surgery, obstetrics, or any special province of the medical sciences; but, confining our attention solely to internal medicine, we owe much to the physicians of this hospital. Here let me observe how much we gain by keeping ourselves acquainted with the labours of the past. It is interesting indeed to survey the methods by which advances in knowledge are gained. At one time the laborious efforts of many bring about the acquisition of new facts. At another, the brilliant generalisation of one throws a flood of light upon a subject. It must be encouraging to all of us to observe at the origin, even of the most wonderful discoveries or transcendent speculations, how a small germ may be

dimly discerned which in time shows the most remarkable growth. Nowhere, perhaps, does this strike one so forcibly as in observing the development of Charles Darwin. In the Journal of the Voyage of the "Beagle," written in his early years, when speaking of a certain snake, Darwin says³—"Cuvier, in opposition to some other naturalists, makes this a sub-genus of the rattlesnake, and intermediate between it and the viper. In confirmation of this opinion, I observed a fact, which appears to me very curious and instructive, as showing how every character, even though it may be in some degree independent of structure, has a tendency to vary by slow degrees. The extremity of the tail of this snake is terminated by a point, which is very slightly enlarged and as the animal glided along, it constantly vibrates the last inch; and this part striking against the dry grass and brushwood, produced a rattling noise, which could be distinctly heard at the distance of six feet. As often as the animal was irritated or surprised, its tail was shaken, and the vibrations were extremely rapid. Even as long as the body retained its irritability, this tendency to this habitual movement was evident. The *Trigonocephalus* has, therefore, in some respects, the structure of *Vipera*, with the habits of a *Crotalus*, the noise, however, being produced by a simpler device. Here we have the earliest intimation of one of the most important pieces of evidence which led the genius of Darwin to those theories embodied in his immortal works. Truly the teaching of scientific history should lead us to dwell much on the labours of those who have gone before us, to ponder over the lessons which they have taught us, and to do the work of the present in the light of the past. In a busy age we are undoubtedly prone to make light of the results of former times. In this connection you will perhaps allow me to recall to you the burning words of Ruskin upon a somewhat analogous subject⁴:—

"There is a curious type of us given in one of the

³ Journal of Researches into the Geology and Natural History of the various Countries visited by H.M.S. "Beagle," London, 1839, p. 111.

⁴ *Sesame and Lilies*, London, 1865, p. 103.

lovely neglected works of the last of our great painters. This is a drawing of Kirkby Lonsdale churchyard, and of its brook, and valley, and hills, and folded morning sky beyond. And unmindful alike of these, and of the dead who have left these for other valleys and for other skies, a group of schoolboys have piled their little books upon a grave, to strike them off with stones. So, also, we play with the words of the dead that would teach us, and strike them far from us with our bitter, reckless will, little thinking that those leaves which the wind scatters had been piled not only upon a gravestone, but upon the seal of an enchanted vault—nay, the gate of a great city of sleeping kings, who would awake for us, if we knew but how to call them by their names. How often, even if we lift the marble entrance gate, do we not wander among those old kings in their repose, and ringer the robes they lie in, and stir the crowns on their foreheads; and still they are silent to us, and seem but dusty imagery; because we know not the incantation in the heart that would wake them—which, if they once heard, they would start up to meet us in their power of long ago, narrowly to look upon us, and consider us; and, as the fallen kings of Hades meet the newly-fallen, saying, 'Art thou also become weak as we—art thou also become one of us?' so would these kings, with their undimmed, unshaken diadems, meet us, saying, 'Art thou also become pure and mighty of heart as we? Art thou also become one of us?''

Here the *genius loci* almost inevitably leads me to think specially of some of the advances which we owe, in the first place, entirely to Guy's Hospital, and you will undoubtedly extend your indulgence to me for a few minutes in an attempt to estimate the importance of Edison's discovery, and the recent developments to which it has led. No better illustration could be obtained of my theme. It is obvious that Addison was in no hurry to bring forward his discovery. After long observation and full reflection, he brought his views before the South London Medical Society in 1849.⁵ His

⁵ Lond. Med. Gaz., New Series, 1849, Vol. viii., p. 517.

observations speedily bore fruit, and it will be of interest to submit to you a piece of unrecorded contemporary biography. A few years ago Sir William Turner, Principal of our University, came to my wards in the Royal Infirmary, in order to see a beautiful case of acromegaly then under my care. The consideration of the subject led us to speak of the discovery of certain affections, and he mentioned that, when he was a clinical clerk in the medical wards of St. Bartholomew's Hospital, Sir George Burrows brought before his clinique the discovery recently made by Addison. Sir William informed me that he thought the date was in the end of 1853 or early in 1854. When thinking over the subject of this address, it occurred to me that it would be well to be on certain ground in this respect, and accordingly the Principal was good enough to have a talk with me upon the subject. The result of this conference may best be embodied in the following letter:—

5, Eton Terrace,
Edinburgh,

My Dear Gibson,

12th Sept., 1907.

Since our conversation yesterday I have examined the notes which I took when Clinical Clerk to Sir George Burrows, and I find in them a detailed account of the case of suprarenal disease.

Patient, a man *æt.* 26, was admitted into John Ward, April 7th, 1854. I need not give you the notes but the skin colouration was distinctive. He was ill-nourished, and had led a vagrant life. He had, two years previously, had an abscess over the fifth lumbar vertebra, which discharged for nearly four months. He stated that he had been gradually wasting before admission, and he collapsed and died eight days afterwards. The *post-mortem* examination showed tubercular disease of both suprarenal bodies, and the right contained purulent-looking fluid, apparently disintegrated tubercle. The case fixes the date of Sir George Burrows' remarks to his clerks.

Very truly yours,

WM. TURNER.

Let me remark in parenthesis how valuable a habit that of keeping notes is.

Addison's discovery was fully published in 1855.⁶ It contained a description of eleven cases, and was accompanied by eleven plates, and was so thorough that very little was left from the clinical point of view for subsequent observers. The cutaneous, circulatory and elementary symptoms, together with the profound anæmia, were most fully described. There can be no doubt, as Rolleston says,⁷ that the general recognition of the disease is very largely, if not entirely, due to the loyal efforts of Sir Samuel Wilks. Undoubtedly Addison was of opinion that any morbid lesion of the suprarenal glands might produce the characteristic symptoms of the disease, and that its features resulted not so much from the nature of the pathological process as from the disturbance of the particular function of the capsules. It appears probable, nevertheless, that Addison was inclined to modify some of his views as to the multiplicity of causes of the affection. In the Prefatory Remarks by Wilks and Daldy to the New Sydenham Society's collection of Addison's Works, the following remarks occur:—"There is another important point to which the attention of the reader should be attracted, that in transcribing, as we are bound to do, all the pages of Addison's Treatise, we necessarily include some paragraphs which the author, had a more extended observation been accorded to him, would have been the first to expunge, under the belief that he was supplying an impulse to the further investigation of the disease. Addison unfortunately inserted some paragraphs implying that, if the suprarenal organs were involved in cancer or tubercle, symptoms corresponding to those which he had described would result. All subsequent experience has shown that on this point he was unquestionably in error."⁸ Sir Samuel Wilks⁹ and Greenhow¹⁰ believed

⁶ "On the Constitutional and Local Effects of Disease of the suprarenal Capsules." Lond., 1855. Pp. viii., 43.

⁷ Brit. Med. Journ., 1895, vol. i., p. 687.

⁸ "A Collection of the Published Writings of the late Thomas Addison, M.D. Lond., 1868, p. ii.

⁹ Lectures on Pathological Anatomy, Lond., 1859, p. 351.

¹⁰ "On Addison's Disease," Lond., 1866, p. 21.

all real cases of Addison's disease to be produced by fibro-caseous changes in the suprarenal glands. It is necessary to mention, before going further, that the changes in the suprarenal bodies recorded by these authors as a primary inflammation and ending in the fibro-caseous change sometimes terminating in an atrophic condition, are now universally regarded as tuberculous. Habershon,¹¹ while a member of this school, was the first to show that inflammation beginning in the suprarenal bodies may extend to the semi-lunar ganglia and embrace them in fibrous tissue, and Greenhow¹² elaborated two distinct phases in the development of the process. But changes in the semi-lunar ganglia and their nervous connections must still be regarded as extremely doubtful, and the important observations of Hale White¹³ have taught us how variable are the appearances presented by these structures, even in conditions which would be regarded as healthy. It must be admitted that, in addition to tuberculous disease of the suprarenal bodies, other changes in these organs produce symptoms closely similar to, if not identical with, Addison's disease. Chronic interstitial changes leading to atrophy, and comparable to atrophic cirrhosis of the liver, were shown by Hadden.¹⁴ Hæmorrhages into the suprarenal bodies have been found with symptoms of Addison's disease by Carrington¹⁵ and others. Simple atrophy, without any increased fibrous tissue or fibrous adhesions, has been described by Spender¹⁶ and Goodhart.¹⁷ It must be added that undoubtedly the complete clinical picture of Addison's disease has been found associated with malignant changes in the suprarenal capsules. In Rolleston's Goulstonian Lectures¹⁸ the following summary regarding the conditions of the suprarenal bodies

11 Guy's Hospital Reports, 1864, 3rd Series, vol. x., p. 78.

12 "On Addison's Disease," Lond., 1875, p. 73.

13 Journal of Physiology, 1889, vol. x., p. 341.

14 Trans. Path. Soc., 1885, vol. xxxvi., p. 436.

15 Ibid., 1885, vol. xxxvi., p. 454.

16 Brit. Med. Journ., 1858, p. 768.

17 Trans. Path. Soc., 1882, vol. xxxii., p. 340.

18 Brit. Med. Journ., 1895, vol. i., p. 690.

given: "To summarise the conditions of the suprarenal bodies recorded in Addison's disease, there are the following:—

1. The fibro-caseous lesion due to tuberculosis, far the commonest condition found.
2. Simple atrophy.
3. Chronic interstitial inflammation leading to atrophy.
4. Malignant disease invading the capsules, including Addison's case of a malignant nodule compressing the suprarenal vein.
5. Blood extravasated into the suprarenal bodies.
6. No lesion of the suprarenal bodies themselves, but pressure or inflammation involving the semi-lunar ganglia.

The first is the only common cause of Addison's disease; the others, with the exception of simple atrophy, may be considered as very rare."

Undoubtedly many of the symptoms of Addison's disease may be found without any distinctive lesion in the suprarenal bodies, as in a case recorded by Conder from my own wards,¹⁹ while, on the other hand, variations of different kinds have been described in the suprarenal bodies without any resulting clinical symptoms. There can be no doubt that our conceptions of Addison's disease are passing through a period of transition. In the modern literature of the affection there are many cases described in which the diagnosis of the disease has been attained in the total absence of pigmentation. A recent paper by Stursberg²⁰ may be cited in proof of this statement. Cases showing general weakness and feeble circulation, along with gastric and intestinal symptoms, are, now-a-days, not infrequently found to be associated with characteristic changes in the suprarenal bodies. This leads me to the only point on which it is possible for me to add any original observations of my own. Throughout the whole literature of Addison's disease the observation has been constantly repeated that the pulse has been

¹⁹ Edin. Med. Journ., 1905, vol. xvii., p. 275.

²⁰ Münch. med. Wochschr., 1907, LIV. Band, S. 773.

weak or the blood pressure low, and yet, until quite recently, very few real pressure estimations have been made. Even these have resulted in discrepant statements. Janeway,²¹ in his exhaustive work, only gives two observations. One was in the case of a woman still under observation, with the typical features of Addison's disease, who showed, by means of Hill and Barnard's instrument, a diastolic pressure of 90 mm. Hg. "The other, a man in my service at the City Hospital was in the last stages of emaciation and asthenia, and showed at autopsy typical fibrosis and caseation of both suprarenals. A number of determinations, up to within about two weeks of his death, showed a systolic pressure of 140 mm. in one arm. In the other the radial was exceedingly small, and systolic pressure was about 100 mm." Here we have, by an observer of unimpeachable accuracy, an interesting observation showing that the pressure in this disease may sometimes be quite up to the normal. On the other hand, in two cases described by Stursberg, the pressure was remarkably low, being in the one case only 70 and in the other 65, of systolic pressure. My own observations, until the last two or three years, were made with Hill & Barnard's apparatus; but, as their sphygmomanometer only yields readings which may be regarded as an approximation to diastolic pressure, they are not of much value. During the last two or three years, when various forms of mercurial manometers have been in use in my wards, opportunities of observing cases of Addison's disease have been somewhat scanty. Two cases, however, may be referred to. One is that of a Russian Jew, aged 45, at present under treatment, admitted with profound asthenia, deep pigmentation, and serious gastro-enteric symptoms, who yielded a maximum systolic pressure of 95 mm., and a minimum diastolic pressure of 72. Another patient, who has been long under the care of my friend and colleague, Dr. Bramwell

²¹ "The Clinical Study of Blood Pressure." New York and London, 1904, p. 237.

²² Loc. cit.

held a maximum systolic pressure of 150 mm., and a minimum diastolic pressure of 96. This patient was diagnosed some eight or nine years ago as a case of chronic Addison's disease. Undoubtedly he is profoundly anæmic, and has occasionally gastro-enteric attacks, while the pigmentation of his skin varies considerably from time to time. Whether he can be regarded as a typical instance of this affection must be allowed to be doubtful. My friend, Dr. J. M. Cowan, Physician to the Royal Infirmary, Glasgow, has put at my disposal some notes of an interesting case of four years' standing, with very marked general pigmentation, in which the systolic pressure is 90 mm. Further observations upon the blood pressure are urgently required.

Taking a broad general view of these facts, which manifest an undoubted variability as regards the circulatory appearances, and considering them in the light of the fluctuations of the other clinical phenomena, it seems probable that ere long it may be possible to attain some classification of different types of this interesting affection. It must be regarded as at least possible that in cases of profound pigmentary changes in the Malpighian layer, without obvious alteration in the suprarenal bodies, there may be glandular inadequacy; the converse may be explained by the supposition that even when extensive changes are present in the glands there may be enough tissue still in possession of sufficient activity to avert the usual results.

So much from the clinical point of view. We cannot linger over the subject, and must pass on to consider certain of the pathological problems which arise in connection with the affection. The views originally advocated by Wilks²³ and Greenhow²⁴ are that a primary affection of the suprarenal bodies which is uniform in its nature leads to secondary changes in the neighbouring nervous structures. The changes in the nervous textures were considered to be the factors by which the various clinical appearances were brought about. These

23 Loc. cit.

24 Loc. cit.

opinions have had the support of many eminent authorities, whom time forbids me to mention. The hypothesis fails to explain the cases—of which many have been recorded—in which the semi-lunar ganglion and sympathetic nerve have been found absolutely healthy. And it further does not help us in those cases in which the disease has been found to result from simple atrophy of the capsules. Such considerations lead to the rejection of the nervous theory of Addison's disease.

The hypothesis has been advanced that the suprarenal bodies remove pigmentary substances and various poisons from the blood. MacMunn²⁵ holds that the suprarenal bodies act in this way, and that when they are destroyed an accumulation of such waste substances takes place in the circulation; but no solid basis has ever been adduced in favour of such a view. Not only is it a fact that no pigment granules have ever been found in the blood of patients suffering from Addison's disease, but further, no increased pigment has been discovered in the renal secretion. This has not only been shown by Dixon Mann,²⁶ but also by Garrod, as reported by Rolleston. 'The same is true as regards the idea that the suprarenal bodies remove poisonous substances, or at least render them innocuous. It is undoubtedly true that the removal of the glands experimentally is followed by death, but the fact may be explained in another way than that of failure of excretion. Tizzoni²⁸ and Nothnagel²⁹ found that when the suprarenal bodies were crushed and allowed to be absorbed, no signs of poisoning occurred, at any rate within a considerable period of time from the date of the experiment. Abelous³⁰ and Langlois found that, after the removal of the suprarenal capsules, followed by the administration of suprarenal extract, no toxæmia resulted. The former experimen-

25 Brit. Med. Journ., 1888, vol. i., p. 233.

26 Lancet, 1891, vol. i., p. 764.

27 Brit. Med. Journ., 1895, vol. i., p. 745.

28 Ziegler's Beiträge, 1889, vi. Band, s. i.

29 Zeit. f. klin. Med., 1879, Band i., s. 77.

30 Arch. de Physiol., 1892, v. Sér., Tome iv., p. 269 and 465.

It is clear that the organs do not contain any toxic substances which they have been supposed to remove, while the latter show distinctly that if the bodies did excrete a poison, the administration of suprarenal extract could increase the evil effects. It must, therefore, be absolutely clear that the suprarenal bodies do not remove waste substances, whether pigmentary or toxic, from the blood.

The last theory is that Addison's disease is caused by inadequate internal secretion of the suprarenal bodies. This view was founded on the investigations of Abelous and Langlois,³¹ as well as of Oliver and Schäfer.³² It has been found that normal suprarenal bodies yield a powerful extract which acts as a pressor agent, and it has been discovered by Schäfer and Oliver³³ that from suprarenal bodies obtained from cases of Addison's disease, no active extract can be obtained. The result of these investigations has been to render the theory that Addison's disease is due to a lack of internal secretion almost certain.

The observations which have just been quoted serve as the starting-point of a number of most interesting investigations. It has been found, for example, that the cortex does not yield adrenalin, the principle which acts on sympathetic nerve endings, and that this is furnished entirely by the cells of the medulla. This portion of the organs is, accordingly, one of the most important parts of the glandular system leading to vascular contraction. The cortical substance has apparently some association with growth and development, according to the observations of Wooley,³⁴ as well as of Bulloch and Queira.³⁵ Precocious development has been seen in a considerable number of cases. Undoubtedly these facts are analogous to certain of the changes associated with involution in the anterior lobe of the pituitary body. Schäfer and Herring,³⁶ indeed, have shown that the

Loc. cit.

Journ. Physiol., 1895, vol. xviii., p. 230.

Loc. cit.

Trans. Assoc. Amer. Phys., 1902, vol. xvii., p. 627.

Trans. Path. Soc., Lond., 1905, vol. lvi., p. 189.

Phil. Trans. Roy. Soc., Lond., 1906, vol. cxciv., p. 27.

anterior lobe of the pituitary body presents a remarkable resemblance in many respects to the cortex of the suprarenal gland.

It may be supposed that Addison's disease may result in part from inadequacy of the medullary portion leading to asthenia and hypopiesis³⁷ by loss of adrenalin and by failure of the cortex to maintain certain as yet unknown, but probably important, functions particularly associated with pigmentary excretion and toxine destruction. On this view there may be a most variable assemblage of symptoms, according as the one or the other portion of the suprarenal body is mainly affected.

It would be of the greatest interest to follow the recent observations which have been made upon the action of suprarenal extract and its active principle. A chronic deficiency of suprarenal activity has been described, to which Boinet³⁸ has given the name of Addisonism. The existence of an acute failure of suprarenal activity is undoubted, especially as seen in certain acute infections *e.g.*, diphtheria, where the suprarenal bodies are found to be in a state of such acute changes as cloudy swelling and leucocytic infiltration. The observations of Rivière and Dudgeon⁴⁰ are of great interest in regard to this matter. Mott and Haliburton⁴¹ have shown that the amount of adrenalin may be greatly diminished by acute disease. On the other hand, it is probable that an excessive secretion takes place on the part of the suprarenal glands. A case was recently described by Vaquer and Aubertin⁴² in which parenchymatous nephritis and great cardiac hypertrophy were associated with marked increase of the medulla of the suprarenal bodies, and Wiesel⁴³ has described some instances of granular kidney with arterial degeneration and high blood pressure in which there was enlargement of the medulla.

³⁷ As is well known, Clifford Allbutt has introduced the term hyperpiesis (*Bristol Med. Chir. Rev.*, 1905, p. 7), and we may well use its antithesis.

³⁸ *Arch. gen. de Méd.*, 1904, vol. cxciv., pp. 2324 and 2525.

³⁹ *Trans. Path. Soc. Lond.*, 1902, vol. lii., p. 368.

⁴⁰ *Amer. Journ. Med. Sc.*, 1904, vol. cxxvii., p. 134.

⁴¹ *Arch. of Neur.*, 1907, vol. iii., p. 123.

⁴² *Comptes rend. Soc. de Biol.*, 1907, Tome lxii., p. 967.

⁴³ *Wien med. Woch.*, 1907, Band. lvii., s. 674.

the suprarenal bodies. These and many other developments of our modern knowledge are most tempting matters for consideration and speculation, but time forbids me further to dwell upon them. When in the act of finishing up these remarks, the admirable address by Rolleston⁴⁴ to the Canadian Medical Association, "On Some Problems in Connection with the Suprarenals," made its appearance. It deals very fully and ably with many of the more recent facts which have been briefly referred to.

The consideration of the facts and views thus briefly referred to furnishes us with an instructive picture of the ebb and flow of scientific thought. The conception of disease, as originally described by the great man in whose honour it has well been named, was from the medical standpoint almost complete, while the pathological explanation for long seemed adequate, and was regarded as having attained finality. Even in the early stages of its recognition, however, as we have seen, there were currents of thought advancing and receding, like the waves of the rising tide, but ever with an onward movement. It is so, it must be so, in every branch of knowledge. The fact is constant, the explanation is changing; subsequent generations modify their views according to their gifts and lights. Similar instances of changing views meet us everywhere on our way through the world. When in Brittany lately, this thought was strongly borne upon me. Passing through Finistère, the great menhir of Trégunc caught my eye, one day, and led me over the ether and through the furze to have a nearer view. Closer inspection revealed a majestic monolith, erected by the early inhabitants of that fair region—whose descendants undoubtedly are the Brythonic Celts of to-day, still in occupation of their ancestral home—for the celebration of some of their religious ceremonials. But what rivetted my eye, and arrested my attention, was the presence, on the summit of the megalithic monument, of a small stone crucifix. What had arisen as a Pagan symbol, had been by this simple device changed into a Christian emblem.

44 Lancet, 1907, vol. ii., p. 875.

And looking upon this interesting illustration of "Past and Present," the charming words of my friend, the Rev. Dr. Walter Smith,⁴⁵ came into my mind:—

"And on the spit of land, a stone,
 With lichen tinted and with moss,
 Stands on the tufted grass alone,
 Its face graven with a simple Cross;
 There is no word of pious lore,
 Nor wreath, nor ring, nor ornament,
 Nor sacred letters nicely blent—
 A simple Cross, and nothing more.

"Not other is the stone from those
 That in the mystic circle stand;
 An unhewn slab, and yet it shows
 New light risen on a darkling land;
 In monumental speech, it tells
 The story of the ages gone,
 The story of the Pagan stone
 New-charmed with sacred Christian spells."

⁴⁵ The Poetical Works of Walter C. Smith, Collected Edition
 London, 1906, p. 236.

