an olive, the greatest diameter of which was two centimetres." In Murchison's very interesting case above mentioned there were grave doubts entertained as to the diagnosis, although thirteen years previously regular attacks of biliary colic, followed by jaundice, appear to have existed. Cancerous disease is apt to be suspected in such cases, and was so in this case by some. But the post-mortem examination showed that a large gall-stone, probably long impacted in the cystic duct, had made its way latterly into the common duct, and was there retained, and had been ulcerating through the mucous membrane of the duodenum, so as to cause hæmorrhage and other unusual symptoms, among which was, for a time, "almost constant diarrhœa."

But these cases, illustrating the long-continued absence of jaundice when the calculus is so large as to be detained in the cystic duct, can hardly be considered as throwing any light on the total absence of jaundice in our case, where the stones were so numerous and so small, and the biliary passages so much dilated throughout as to give them comparatively an easy and rapid course to the duodenum.⁶ Assuming that the attacks of epigastric pain suffered at intervals for more than two years by our patient are to be regarded as similar in origin to the fatal attack, we can only suppose that the comparatively small size and faceted character of the individual gall-stones passed during this period incapacitated them from completely obstructing the duct, or that their very rapid passage gave no time for jaundice to take place in each separate attack. "If the stone finds its way into the bowels within twenty-four hours," says Murchison, following in the main Trousseau, "or if it does not get beyond the cystic duct, or if its form is so angular as not entirely to block the flow of bile, it is quite possible to have biliary colic without jaundice. There is good authority for the statement that small biliary calculi have been found in the stools of individuals who have never had jaundice, but such cases are certainly exceptional; and I am unable to understand the statement of Wolff,⁷ that twenty-five out of forty-five patients observed by him passed through the whole train of symptoms of biliary colic without jaundice, the concretions being found in the evacuations."⁸

In Von Schüppel's elaborate article⁹ the following closely corresponding passage is found: "If the stone makes its way into the intestine after having been arrested in the common duct less than from twelve to twenty hours, or if it does not advance beyond the cystic duct, or if it is of angular form and the bile can flow alongside of it, no jaundice will occur, although hepatic colic is present. By bearing this in mind we shall understand those cases in which people suffer repeatedly from attacks of colic, which are regarded as gastric colic, because no jaundice ever appears, and in which the subsequent course of the disease leaves no doubt that the paroxysms of pain were really due to the presence of gall-stones. In such cases the diagnosis is sometimes confirmed by observing that the attack of pain is associated with a very slight yellow colouring of the conjunctivæ at least, or that the urine shows small quantities of biliary colouring matter."

These citations will serve to show that the case now brought before you is not, perhaps, altogether unexampled,

⁶ In this fact, too, is probably to be found the reason why the pain, though often no doubt considerable, never assumed under observation the exquisitely agonising character attributed to that of gall-stone; nor had it, I think, the extremely sudden invasion usually observed in the case of a large calculus, or even of a smaller calculus passing for the first time through the bile-duct.

⁷ Virchow's Archiv, vol. xx., 1861: Beiträge zur Symptomatologie und Diagnostik der Gallensteine; von W. Carl. Wolff, Pract. Ärzte in Bonn. After a careful perusal of this interesting and valuable paper, I am disposed to attach more importance to its conclusions in this respect than Dr. Murchison appears to have done. But, as Dr. Wolff gives no details of his cases, it can only be here stated in general terms that they were mostly uncomplicated—i.e., without organic disease or serious illness of any kind, apart from the symptoms specially described. As only such cases are included as were actually found to have passed gall-stones with the evacuations, it is plain that a more or less considerable number of doubtful cases must have been eliminated. On the other hand, this fact gives to the study of the symptoms of the cases referred to an unusual measure of importance. Among these symptoms, next to pain and jaundice, the author has been led to give the first place to a slowing of the pulse, which takes place usually to the extent of from five to ten beats, and sometimes more, in the minute during the urgency of each paroxysm. This he observed in one case in which the attacks returned almost daily, and were watched for many months, with all precautions against fallacy. In uncomplicated cases, whether with or without jaundice, he found this symptom so constant that in doubtful cases of the latter description he considers this the leading element in the diagnosis.

⁸ Murchison, ut supra, pp. 339, 340.

⁹ Von Ziemssen's Cyclopædia. English translation, vol. iv., p. 748.

although I confess that I had never before either seen or heard of one in which such a succession of attacks, extending over so long a time, and involving in all probability the passage of quite a multitude of gall-stones, had occurred without either yellowness of the skin and conjunctivæ, or the detection of bile pigment in excess in the urine. I have therefore thought it my duty as a clinical teacher to publish the case.

ADDISON'S DISEASE WITHOUT TUBERCULAR DEGENERATION OF THE SUPRARENAL CAPSULES.

By J. WICKHAM LEGG, F.R.C.P. LOND.

It would seem that the theory which makes tubercular disease of the suprarenal capsules an invariable accompaniment of the clinical symptoms of Addison's disease will have sooner or later to be abandoned. Eleven years ago there came under my notice the first of a series of cases in which undoubted Addison's disease has existed during life, and yet after death no tubercular disease of the suprarenal capsules has been found. A woman, thirty-seven years of age, about eighteen months before her death noticed a black line on her lips, and two months before this she had begun to be very weak, with shortness of breath; she was also subject to vomiting, which the patient herself called being bilious. On admission the face was noticed to be pigmented, while the sclerotics were a clear white; the buccal mucous membrane was also pigmented, the lips and the gums being marked with bluish-black lines, and the palate spotted with bluish spots. Further, on the arms were the little black pigmented spots which are considered so characteristic of Addison's disease. The legs, arms, and trunk were generally of a brownish colour, which was not more marked around the umbilicus or the mammae. She died about a month after her admission into St. Bartholomew's Hospital, with symptoms of extreme weakness and vomiting.

The examination showed well-marked pigmentation of the knees, the inside of the thighs, the outer genitals, the abdomen, and the lips. Under the skin there was a fair amount of fat. There was no phthisis, and the only noteworthy appearances found in the body were connected with the suprarenal bodies. A very careful dissection failed to detect any right suprarenal body. First of all, the inferior cava was drawn down and the sympathetic nerves behind it dissected; no naked-eye appearances of disease could be made out; the place of the right suprarenal body was taken by a small lump of fat, not so big as a hazel nut, into which an artery from the junction of the right renal with the aorta could be traced. This little artery was about as thick as a common pin. On the left side there was found a black body in the place of the suprarenal capsule, and of its shape, but not of its thickness. It had vessels like those of the right capsule, but it was a mere shell; when cut into, one wall could at once be raised from the other, and there appeared to be nothing between them. This body under the microscope showed well-marked connective tissue fibres with numerous fat drops in the meshes, but no remains of the ordinary texture of the suprarenal body.

This case was noticed by Dr. Greenhow in the second Croonian lecture read before the College of Physicians in 1875, but he did not admit that it was a case of Addison's disease. And on these grounds—First, that there was a "bilious tendency to explain the vomiting." Now, it is true that the patient did say that she was often bilious; but those who have had much to do with hospital, or indeed other patients, will know that with them an onset of vomiting is usually called being "bilious." Though I thought it right to record the patient's own words, yet one is not bound thereby to an approval of what is said or of the theories of disease which are connoted in such expressions. Secondly, "the patient had lost four stone in weight during her illness." Now, these are again merely the patient's own words, and they possess only the usual worth of such statements. After death there was found a fair amount of fat under the skin. Last of all, it was objected that the temperature was above the normal. It was once 99°. I hardly think much stress can be laid upon a single observation,

only 0·4 or 0·6 of a degree above what is natural. Besides, it will hardly be allowed that the temperature in Addison's disease is never raised. I have myself had under my care a very marked case of Addison's disease in a little girl of twelve, in whom the suprarenal capsules proved after death typical specimens of tubercular degeneration, and yet during her stay in the hospital the temperature was seldom down to 98·4°. This elevation of temperature seems to be now an admitted clinical fact, for I find in the last edition of Dr. Bristowe's "Theory and Practice of Medicine"¹ that the temperature sometimes "rises one, two, or three degrees." So that a temperature of 99° can hardly be considered a point sufficient altogether to exclude the diagnosis of Addison's disease; and this rise of temperature seems to be as little worthy of consideration as the thirst, swollen gums, and red, fissured tongue also pointed out as inimical to the diagnosis. On the other hand, the symptoms declared in the Croonian lectures to be so important were very marked—to wit, the shortness of breath, the weakness, the vomiting, and pigmentation of the skin and mucous membrane. With this last there was an appearance, "one of the most decisive external signs of Addison's disease," the small black patches of pigment on the skin of the arms.

While recording this case in 1874, in the St. Bartholomew's Hospital Reports for that year, I ventured to remark that if the facts were good, as there seemed no reason for doubting, some of the then prevailing theories as to the nature of Addison's disease would have to be greatly modified, or even altogether abandoned. And the progress of events has been somewhat in favour of what was then foretold. On the 7th February, 1882, two cases of atrophy of the suprarenal capsules with Addison's disease were laid before the Pathological Society by Dr. Goodhart (for Mr. George Eastes) and by Dr. Henry Davy; and more lately, on February 3rd last, several other cases were brought forward by such excellent observers as Dr. Coupland and Dr. Thomas Barlow, and Dr. Coupland gives an account of all the cases of atrophy of the suprarenal capsules and of Addison's disease, which have yet been published.

There is thus a considerable and increasing number of cases of Addison's disease in which all the symptoms of this malady have been present during life, but in which after death the tubercular degeneration of the capsules has been absent, and only a wasted and atrophied state of these organs has been discovered. These cases are quite different from those which were formerly brought forward as Addison's disease without tubercular degeneration—cases which were so imperfectly described or so imperfectly examined that the account of many of them need only be looked at to convince the reader of its worthlessness to prove anything. Cases in which only the kidneys have been examined, or cases in which disease of the suprarenal capsule has been found without any symptoms of Addison's disease during life, or cases in which no description is given of the disease found in the suprarenal body, may all be put aside at once. It may be that the ease with which such cases have been hitherto answered has led some of the defenders of the tubercular nature of Addison's disease to fancy that the more modern instances may be as lightly passed over. But these later cases are far more difficult to overthrow. They have been seen for the most part in the practice of experienced hospital physicians familiar with Addison's disease, and the examinations after death have been made by experienced morbid anatomists who were not likely to pass by any important and gross changes in other organs which could cause death. These can hardly be set aside by any amount of special pleading; such carefully worked-out cases would be almost enough in themselves to destroy any theory of disease to which they are opposed.

It may interest some to hear of another case in which the same set of appearances was noticed. My colleague, Dr. Church, drew my attention to the patient soon after her admission as a well-marked case of Addison's disease; yet, after death, hardly any suprarenal capsule on the right side, and only a wasted remnant of the organ on the left, could be discovered by Dr. Norman Moore.

A woman, aged twenty-nine, was quite well up to December, 1883, when she began to be short of breath, to be giddy, and to vomit. A month later the discolouration of skin began. On admission into St. Bartholomew's Hospital, in May, 1884, her skin generally was pigmented of a tawny-brown colour; two deeper patches of pigment were seen

over the right eye and the xiphoid cartilage; the axillæ were deeply pigmented, but there was no excess of pigment over the breasts. The lips were slightly pigmented. The whole body was rather wasted, weight 6 st. 11 lb. There were no physical signs in the lungs, but there was a short harsh systolic murmur at the apex of the heart, which was not audible at the base. The glands under the jaw were considerably enlarged, especially on the left side. The urine contained no albumen or sugar. She continued to grow much weaker, and had many attacks of vomiting. On May 27th, coinciding with an attack of fever, it was noted that the colour had become much darker. She died on June 11th. This patient had paroxysms of fever. From May 16th to 18th the temperature was above natural, reaching on the evening of the 16th to 101·8°. It was also above natural from May 24th to May 31st, the highest point being 102·6°; and again four days before death, the highest point being 102·8°.

The examination was made by Dr. Norman Moore, about thirty-three hours after death. The body was found wasted, and there was much dark-brown pigment in the skin. This appearance was more marked on the abdomen than on the chest, and was very distinct around the nipples. The whole of the back was much pigmented. Nothing unnatural was discovered in the spinal cord, brain, or ears. The lungs were quite natural; the heart was small, the muscular tissue brownish, the valves natural; weight, 6½ oz. The contents of the abdomen were natural, with the following exceptions: there were three large gall-stones with facets in the gall-bladder, but the ducts were quite free, and the right suprarenal capsule was represented by a small shred of tissue, quadrangular in shape, about half an inch long by a quarter broad. The left was black, of natural outline, but completely wasted, and as thin as paper. The solar plexus to the naked eye was quite natural.

If, then, as seems likely, we shall be forced to give up the theory of the necessity of tubercular degeneration of the suprarenal capsules to Addison's disease, have we any other theory to put in its place? I think at the present moment we must answer "No," and in the very great obscurity in which the subject lies it will be rather an advantage if we abstain from generalisations and inductions for awhile, and devote ourselves to the more elementary processes of the gathering and sifting of facts. After all, this latter occupation is really harder than the weaving and spinning of theories, and it was not without some justice that Cullen could say in the introduction to his "First Lines" that "there are more false facts than false opinions in physic."

Green-street, W.

ON CERTAIN CASES OF DISEASE OF THE CENTRAL NERVOUS SYSTEM IN WHICH NO NAKED-EYE CHANGES ARE FOUND AT THE POST- MORTEM EXAMINATION.

By SEYMOUR J. SHARKEY, M.B., F.R.C.P.,

ASSISTANT-PHYSICIAN AND JOINT LECTURER ON PATHOLOGY AT
ST. THOMAS'S HOSPITAL.

F. B—, aged six years, was admitted into St. Thomas's Hospital under the care of Dr. Payne on August 26th, and died on August 31st, 1882. The boy was an only child, and his mother was suffering from rheumatism at the time of his birth. His family and personal history was otherwise unimportant. He had never been ill in any way until six weeks before admission to the hospital, when he had a fall and bruised his forehead. His nose bled profusely at the time and at intervals for some days afterwards. The accident appeared, however, to have no effect upon his health. About a week later he was bitten by a dog on the left arm; the wound was cauterised although the animal was not mad. He was said to have been irritable and fretful after this, but otherwise well, and nothing of any importance was noticed until a week before he came to the hospital, when his legs gave way under him, his manner was somewhat foolish, his speech became affected, and he lost control over his evacuations.

On admission he was found to be a well-nourished boy, totally unable to stand or walk, or even to remain sitting

¹ Fifth edition, 1884, p. 577.