in pathological states, others may be added. We may be reminded that uric acid has almost certainly several sources, and more than one import. I cannot say at present that these observations go far to unravel the mystery of the presence of indigo in the calculus. Dr. Bloxam tells me that the patient took creasote, for some days before death, for vomiting; and this may be one source. Dr. Thudichum's suggestion is, that the indigogenous material was found in the suppurating left kidney in the presence of an alkali, was absorbed thence, and carried in the alkaline blood to the healthy right kidney, and then deposited as indigo-blue on contact with acid urine. This is most ingenious and plausible. There is yet another possible source in the cachexia of the patient. To me the value of the calculus will not be in my being able to explain its formation as an isolated phenomenon, but in the impulse which it will give me to work at the meanings lying beneath the appearance of indigo in urine. On this subject, Mr. Seymour Taylor and I have already set to work, and I hope some day to tell you more than I know now.

The calculus from the left kidney may now be examined. It is, as you see, a long branched calculus, corresponding roughly to the form of the pelvis and infundibulum of the kidney. In the first place, it must be stated that it contains no indigo, or indigo-yielding material. Its composition is very different in different parts, and a short review will show you how a calculus may be made to tell its own history. The central part is very friable, and of a very faint pinkish-white colour. The constituents are phosphate of lime, a very little carbonate, and uric acid. The latter occurs in beautiful bunches of prisms, and is apparently combined with lime. The phosphate is in spherules of small size. The crust contains in some layers no uric acid at all. These are the outer-layers, and they are composed very largely of carbonate of lime. It is worthy of note that the carbonate of lime here fails to give one ordinary reaction. It does not effervesce with slightly diluted acetic acid, although the effervescence with hydrochloric acid is very active. This is probably due to the organic matter with which the carbonate is intimately mixed. The centre of the calculus is evidently an urinary product. But the presence of carbonate of lime in large proportion, and the absence of uric acid in the outer layers, makes it probable that these were deposited after the kidney had, by the occlusion of the ureter, been shut off from the bladder, and ultimately reduced to a cyst. There is generally reason to suppose that, in man, carbonate of lime is very rarely deposited from urine, being formed only in the glandular diverticula of the urinary tracts. In herbivora, however, this is not the case. The matrix of the calculus is structureless, and gives us no information.

CASE OF GENERAL ATROPHY OF STOMACH, WITH ABSENCE OF ORGANIC DISEASE.

By A. B. BRABAZON, M.D.,
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ON September 15th, 1877, at 7 P.M., I was requested to visit a lady in my immediate neighbourhood, about whose present condition her friends had become suddenly alarmed. I immediately attended; and there was presented to my notice a lady whom I knew well personally, and whom for years I had seen almost daily in her usual walks and drives. I had not had an opportunity of seeing her for about six weeks before the present occasion, as she had been absent from home.

I was almost horrified to see the change which had taken place in her personal appearance and aspect. When I had last seen her, she then presented the appearance of, I may say, "rude health": stout, but not inordinately so; active in her habits, cheerful in her disposition; her face almost ruddy with the glow of health; in fact, a "comely English matron". Now I saw a perfectly colourless face; a countenance worn and pinched; gums and lips blanched, and conjunctivæ yellowish white. The extremities were cold; pulse 128, small and feeble; sounds of heart indistinct; first sound masked by anæmic *bruit*; respiration natural. She complained of no pain in any part of her body. There was a total absence of subjective symptoms. She complained only of an indescribable feeling of utter prostration and complete inability, or rather dislike, to the slightest exertion.

I at once ordered her to bed and advised the usual restoratives; and, finding that my friend Mr. Biggs had been in attendance for some time, I advised that they should send for him as early as possible, and explained that, except in conjunction with him, I could not see the patient again.

According to the wishes of the lady's friends, we met on the following day, and I had the advantage of Mr. Biggs's previous experience of the case. As to medical history, there was really none. This lady was sixty-three years of age, and had gone to Clevedon, apparently in

good health, for her usual summer sojourn. While there, she had an attack of diarrhoea—not an unusual occurrence with her; and her appetite declined. She complained of feeling weak and depressed, and her colour gradually disappeared. There was no apparent loss of flesh; no sickness; no pain anywhere complained of; no great thirst. The diarrhoea ceased very soon. Mr. Biggs saw her on her return home, and was struck by the change in her appearance. He carefully examined her, and found nothing to account for the change.

We made a most careful examination on this occasion. To be brief, there was not a single objective symptom of organic disease of head, chest, or abdomen. The subjective symptoms, with the exception of those already stated, were equally negative: no pain; no nausea; no oppressive thirst; no diarrhoea, or the reverse. The urinary secretion, carefully tested by Mr. Biggs, was perfectly normal in quantity and quality. The temperature was normal. It was certainly a puzzling case. The general aspect of the patient suggested the possibility of malignant disease. Her age suggested the possibility of cancer uteri; but inquiries negatived the idea. The only diagnosis we could arrive at was that assimilation and nutrition, as far as sanguification was concerned, were in almost complete abeyance. Time alone could possibly reveal the cause of these conditions. The prognosis was most unfavourable.

Matters went on thus for a week. There was no sign of improvement; strength was gradually decreasing. The patient gave the same answer day after day: "No pain whatever". I should mention that, with all this, there was no corresponding emaciation. As far as external appearances went, the body was well nourished.

Considering the peculiar nature of the case, its rare character, and the certain fatal termination, Mr. Biggs and I suggested to the friends the advisability of having a third and perfectly independent opinion, for their future satisfaction. This they agreed to, and accordingly on Saturday, September 29th, we met Dr. Goodridge in consultation. Dr. Goodridge examined the patient with all the attention to detail of symptoms, objective and subjective, for which he has acquired a deserved reputation. His opinion was that there was no evidence of organic disease, malignant or non-malignant; that there was serious interference with the processes of assimilation and nutrition, and consequently blood-making; and that the only cause of this condition which occurred to him was atrophy of the glandular apparatus of the stomach, as the case brought to his mind some similar case or cases recorded by Dr. Fenwick, in which such a condition existed. He fully concurred in a most unfavourable prognosis. The symptoms remained the same, if nothing but progressive weakness can be called a symptom; and the patient gradually became unconscious, and died on October 5th.

We were fortunate in being permitted to make a *post mortem* examination with certain restrictions. On October 6th, Mr. Biggs and I made an examination of the contents of the abdomen. We found the body fairly nourished; subcutaneous fat in abundance. The omentum also was well supplied with fat. The solid viscera were all normal as regarded size and position. The liver was healthy, but infiltrated with bile. There was a gall-stone in the neck of the gall-bladder. The spleen was natural in size and structure. The kidneys were small, but perfectly healthy; so also was the pancreas. The condition of the stomach attracted our particular attention. As a demonstrator and lecturer on anatomy for many years, I have seen and examined hundreds of stomachs. I have never seen so small a stomach relatively to the size of the patient, and I have never seen such complete atrophy of any organ constantly in use. The condition of the coats of the stomach was more like that of brown paper saturated in water than anything else to which I could compare it. The muscular and fibrous coats were alike in a state of complete atrophy. A ligature placed around the œsophageal extremity cut through it like wet blotting-paper. I need scarcely say this was no *post mortem* softening. A portion was removed for microscopical examination, but unfortunately an accident prevented the intention from being carried out. However, I have no doubt that examination would have revealed the conditions presupposed.

REMARKS.—I have ventured to place this case before my professional brethren: first, because, as far as I know, it is one of rare occurrence; secondly, as a reminder that absence of positive symptoms does not always indicate absence of structural disease; thirdly, as a proof of the rapidity with which the abeyance of the primary function of nutrition will cause death. There are one or two peculiarities about this case to which I beg to draw attention. 1. The case was eminently one of impaired nutrition; yet there was no emaciation; on the contrary, there was abundance of adipose tissue over the body. 2. Although the process of fat-making went on, the process of sanguification, as far as red corpuscles were concerned, did not. If I may venture an explanation

of these apparently contradictory conditions, I would say that the condition of the stomach, particularly of its glandular apparatus, causing a deficiency of gastric juice, prevented the conversion of the albuminous portion of the food into the condition requisite for sanguification; whereas the functions of the liver and pancreas, not being interfered with, the conversion of the starchy and saccharine elements of the food into fat, by admixture with the bile and pancreatic fluid, went on as usual in the duodenum. Again, there was no great waste of fat for purposes of combustion and keeping up animal heat.

As far as I know, pathology does not throw much light on the case. Rokitsansky refers to gelatinous softening, described by Cruveilhier, which presents appearances somewhat such as those described above, but there are characteristic differences in the symptoms. Cruveilhier describes it as a "lesion of innervation" from cerebral causes, none of which existed in this. Dr. Brinton says that "atrophy of the stomach, except as incidental to other maladies of the stomach and in a subordinate form, can scarcely be accorded the nosological rank of a substantial and independent malady". He says: "A marked degree of atrophy, chiefly or exclusively involving this organ, is one of the *very rarest lesions* met with in the human body."

Drs. Jones and Sieveking remark "that a not uncommon change in the condition of the stomach is infiltration of low fibrous tissue loaded with nuclei among tubes which themselves undergo atrophy, so that the tubular character is lost. Dr. Bristowe admits that degenerative changes play an important part in the various chronic affections of the stomach; but that at present the symptoms referable to these forms do not admit of identification.

I hope I may be excused for giving at length the details of a case somewhat without history, and certainly without definite or tangible symptoms; but the absence of any such symptoms, and the fact of any attempt at diagnosis being necessarily worked out by a process of exclusion, I think, contribute to its interest, while its rarity is evident by the absence of literary information on the subject.

ON THE MORBID HISTOLOGY OF THE SPINAL CORD IN FIVE CASES OF INSANITY.*

By RINGROSE ATKINS, M.A., M.D.,

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[Continued from page 99 of last number.]

CASE IV.—W. J., male, aged 38, was admitted on December 3rd, 1876, suffering from general paretic dementia. The patient, a Welshman and a pensioner, had served ten years in the army, and subsequently acted as servant to a gentleman. He had never been in the tropics, and had been last employed as a "pointsman" on the Great Southern and Western Railway at Mallow. His present illness, stated to be of eight months' duration, was attributed to a fright he received while on the line, an accident causing the death of an official, which he witnessed. His wife stated that he was always a steady temperate man, but of a silent nervous temperament. He had several healthy children. Some time after receiving the mental shock, he became moody and sullen, and then suspicious of his wife's faithfulness, followed by imaginary fears, but never developing any extravagant or optimistic delusions. For several months past, he had gradually become worse, and as suicide, it was stated, was feared, he was sent to the asylum. On admission, there was no mental exaltation, the reverse rather being the case. He was confused and undecided in his manner and actions, and very feeble-minded, with an expression of abstraction and indecision. There was considerable loss of memory, and he could give no coherent account of himself. At times, there was considerable excitement and general restlessness, with visual hallucinations. When placed opposite the windows, he used to fancy he saw the train coming, and would then loudly declare that he had to go away in it, and tried to act accordingly. The diagnosis of paretic dementia was based on the physical condition. There was marked impairment of the power of articulation; he spoke in an uncertain hesitating manner, dropping some syllables and sometimes pronouncing the word entirely, and there was a twitching of the lips and zygomatic muscles. The tongue was protruded in a spasmodic manner, its movement being apparently with difficulty controlled, and it was marked by fibrillary twitchings. The pupils are irregular, the left being considerably larger than the right; both were equally motile. His gait betrayed no paralysis; but there was a noticeable degree of unsteadiness and irregularity. The manual grasp

was fairly powerful, and equal in both hands. The tactile sensibility was not at all acute, but it was difficult to ascertain anything accurately as regarded the condition of the sensory system. His muscular development was poor; skin sallow; hair and eyes dark.

Abbreviating the history, it will be sufficient to say that, during his residence in the asylum, the mental and physical symptoms became progressively worse. Every function gradually deteriorated; his memory failed, so that he did not recognise his wife, and the visual hallucination faded. The power of articulation became more and more impaired, and the muscular unsteadiness and twitchings increased. He was now quite helpless, and unable to move without assistance. When lying down, however, he had still the power of flexing and extending the lower limbs and of using the arms and hands. He was dirty in his habits, passing urine and faeces involuntarily. During the night of April 28th, he became unconscious, though there was no perceptible fit. He lay with the eyelids closed, the eyes fixed and insensible to the touch; pupils equal and fixed, and diminished in size. The arms were flexed across the chest, and, when raised, were rigid and difficult to extend. The lower limbs were, to a less extent, in the same condition. Tactile sensibility was almost lost; he made no outward manifestation when sharply pricked with a pointed probe. Reflex excitability appeared to be increased; when the soles were tickled, the feet moved convulsively. Breathing was unaffected; but, when a deep inspiration was taken, there was some puffing of the cheeks on expiration. On the following day, the comatose condition had passed away, and, with the exception of being now quite incapable of articulating and more sunken in appearance, he was much as before the attack. On the next day (May 2nd), the right hand and arm, while in a state of tonic rigidity, were perfectly paretic, falling helpless when released from the grasp. The fingers were rigidly flexed, being difficult to straighten, returning to the same position when again let go. On the back of the ball of the thumb, and on the posterior aspect of the second phalanges of the fore-middle and third fingers, were large blebs of brownish serum, which formed in twenty-four hours. When the mouth was forcibly opened, the tongue could be seen lying in the buccal cavity, occasionally being moved about, the organ being atrophied, red, and glazed. Reflex excitability was now very greatly diminished, especially in the right leg. Faradomuscular contractility was weakened in the lower limbs; it was still persistent in the upper limbs and face, where its sensibility was still felt. Across the junction of the sacrum and buttocks, a large eschar was forming, surrounded by an erythematous zone. He could only swallow a little liquid food. No further symptoms presented themselves, and he died on May 5th.

Necropsy thirty hours after death.—Rigor mortis marked; body generally emaciated. Over the lower portion of the sacrum, and extending across the left buttock, was a black eschar about five inches long, surrounded by a zone of congestion; the remains of the bullæ existed on the backs of the fingers and the ball of the thumb. The scalp was thin, and considerable force had to be exercised in stripping it from the bone. The cranial outline was asymmetrical, the right hemisphere being prominent and bulging, the fronto-parietal region of the left flattened and sloping. The bone was of normal consistence, its inner surface being of a slaty-blue colour. On the external surface of the left frontal eminence was a scar, like the remains of an old fracture; the scalp was firmly adherent to this; there was no corresponding mark on the inner surface. On opening into the cavity of the skull, a quantity of non-sanguinolent sero-purulent fluid escaped. The dura mater was covered laterally by a thin layer of gelatinous material, easily scraped off with a scalpel, and it was adherent to the arachnoid along the great vertical cleft. On removing the membrane, its visceral surface and the corresponding surface of the arachnoid over the left hemisphere only were found covered by clear gelatinous material easily removed. It was from this region the fluid above mentioned came. This coating extended over the three tiers of the frontal convolutions, and reached posteriorly, covering the ascending frontal and parietal as far as the anterior borders of the superior and inferior parietal lobules, involving also the three tiers of temporo-sphenoidal convolutions. The pia mater was thickened over the regions specified and adherent to the cortex; the hemisphere was subsequently prepared in nitric acid solution, and the exact localisation of these adhesions determined the result I have described in another paper on that subject (BRITISH MEDICAL JOURNAL, May 11th, 1878). The convolutions were wasted and flattened. The arteries at the base of the brain were delicate-looking and nowhere atheromatous. The basal ganglia were small, but, with the exception of some diminution in consistence of the crura cerebri, were unaffected. The entire brain, previously to draining, weighed 1,555.209 grammes; the cerebellum, pons, and medulla 138.8 grammes. The hemispheres were carefully

* Read in the Section of Psychology at the Annual Meeting of the British Medical Association in Manchester, August 1877. Drawings of the appearances described were exhibited.