Attention was drawn to these facts by Jordan Lloyd, in a valuable paper published in 1892,⁶ in which he relates two cases of accumulation of fluid in the lesser peritoneal cavity, in one of which the middle two-thirds of a gangrenous pancreas was detached and lay free in the fluid. A similar case to this last one of Lloyd's was reported by W. W. Ashhurst, of Mexico, in 1894,⁷ in which a piece of the pancreas lay at the bottom of the cyst cavity, whose contents were seropurulent. The case is recorded as a cyst of the pancreas. Pancreatic cysts always result from obstruction of the duct, with retention of secretion and subsequent dilatation of the duct; or, in case of a laceration of the gland, from escape of the secretion into its parenchyma, and the subsequent dilatation of the capsule into a cyst. Neither of these cases is consistent with such a pathology.

The close resemblance as well as the points of contrast of pancreatic cyst to effusion into the omental bursa are well brought out in the history I have detailed. Both cysts occupied the same area, but differed in shape, the peritoneal effusion extending less to the right and further downward, being somewhat irregular in shape, with its longest diameter nearly vertical, while the pancreatic cyst was somewhat quadrilateral in shape, with the long diameter transversely. The peritoneal effusion took place within a few days, more rapidly than a cyst could form in so firm and friable a structure as the pancreas; while the pancreatic cyst formed comparatively slowly, increasing steadily in dimension.

The constitutional disturbances attending the development of both kinds of cyst bear a very close resemblance, as indeed they should, because they would press upon and injure the same nerve structures—the solar plexus and semilunar ganglia. In the peritoneal effusion, as well as in the pancreatic disease, there may be pain, vomiting, and prostration; emaciation and anæmia may be much more marked than these symptoms would account for. The temperature is usually little disturbed in either affection, and may be subnormal throughout in both. In a case of supposed tuberculous disease of the lesser peritoneal cavity closely resembling pancreatic cyst, under observation a few years ago all these symptoms were very marked.⁷ After the cyst was incised and drained recovery was very rapid, the sinus being completely closed in less than a month, with much retraction of the epigastrium. It is probable that herein lies a distinction between the true and false pancreatic cysts, the latter becoming closed in a much shorter time than required by true cysts of the pancreas, especially if some of the gland tissue remains communicating with the cyst cavity.

Some years ago Senn⁸ pointed out that complete obstruction of the pancreatic duct uncomplicated by morbid changes in the parenchyma of the gland never resulted in the formation of a cyst, the pancreatic juice accumulating in the duct being removed by absorption. If, however, pathological changes have occurred in the gland, absorption of the fluid is interfered with, and gradual distension follows, it may be, to enormous proportions. In this case repeated attacks of inflammation of the organ probably took place, the most severe being in September, 1895, and a second one before the sudden distension of the omental bursa with serum.

It has been thought that true, as well as false, pancreatic cysts are due to hæmorrhage, and that the hæmorrhage results from a lesion of the sympathetic system.⁹ The first proposition is doubtless generally true of cysts following traumatism, but of those originating apart from traumatic causes, in many—perhaps the majority—no hæmorrhage takes place. In the case here detailed is an instance of a true pancreatic cyst without hæmorrhage.

There is ample evidence in this case in the preservation of a good state of nutrition and general vigour, together with the absence of excess of fats and of undigested proteids in the fecal discharges, and of any symptoms of diabetes, that a part of the gland remains intact, discharging its secretion into the intestinal canal, as well as furnishing to the blood the "internal secretion" peculiar to it, without which general metabolism would be deranged. At no time during his protracted illness did he apparently suffer from derangement of the function of the pancreas.

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CLINICAL LECTURE

ON

HÆMATURIA.

By F. R. ECCLES, M.D., M.R.C.S.ENG., F.R.C.S.EDIN.,
Professor of Gynæcology in the Western University, London, Ontario.

I PURPOSE giving short clinical reports of a few cases of hæmaturia that have come under my observation, adding afterwards some general and individual remarks.

CASE I.—Mrs. M., aged 62, mother of four children, passed the climacteric with only the ordinary disturbances, and had fairly good health up to a few weeks ago. On October 27th, 1887, she passed a large quantity of blood. This has since continued intermittently for some days, but with no inconvenience; then, when seen, she had fever, temperature 102°, and quite a number of long rod-like clots (ureteral). The quantity of urine at this time was from 2 to 3 pints, and there was tenderness and enlargement of the kidney. The cardinal symptoms were here present—tumour, hæmaturia, pain (although the pain was not marked); the bleeding occurred from time to time. The bleedings did not seem to be dependent on any exercise or sudden movements; it would come on when she was lying quietly in bed. When blood was not present in the urine there was no albumen, or at times only a trace. On March 24th, 1888, ureteral clots again passed, but only occasionally after that. She gradually lost flesh, and presented the usual cachexia, but lived until April 24th, 1890—that is, for over two and a half years. As a rule, this patient during the greater part of her illness passed more than the average quantity of urine.

CASE II.—Mrs. B., aged 60, admitted August 6th, 1896, for ulcer of the leg, which was treated in the usual manner. On September 2nd, 1896, she passed considerable blood, which was more or less mixed with the urine, and which partly clotted at the bottom of the vessel. The patient was then transferred to me. Upon examination, the kidney was found tender and enlarged—quite a tumour—and displaced forward and internal to its normal position. It was not nodulated. On September 7th, 8th, and 9th she passed blood each day; on the 9th the clots were large, and the temperature went up to 102°. On the 11th and 15th she passed dark blood and clots; temperature 101°. On October 1st chill, temperature 103.4°, and ureteral clots like fish worms. The patient was passing at this time from 40 to 60 ounces of urine in twenty-four hours. On October 2nd, 3rd, and 4th blood in large quantities appeared. On October 5th she had a chill lasting thirty minutes, followed by elevation of temperature to 104.4°. Patient gradually sank and died on the 7th.

CASE III.—W. M., aged 42, and of tuberculous history, and somewhat below the normal weight, complained of pain and tenderness in the right renal region, which became aggravated at times. In addition he had slight hæmorrhages and several attacks of renal colic. The urine contained albumen and pus. He was driving with me at one time when an attack came on. It did not come on very suddenly, but gradually got worse, requiring anodynes and stimulants to enable him to continue his journey. The symptoms were those of stone, and my opinion was thus given; but more advice was sought, and it was thought advisable to determine whether bacilli were present or not. None were found in any examination of the urinary sediments. Inoculation was then advised. After cleansing of the meatus the urine was passed into a sterilised bottle and a portion of the sediment injected into the anterior chamber of the eye of a rabbit; when in four weeks the iris was clearly and plainly packed with tubercles, and a large deposit on the lens capsule. The diagnosis was thus established. Dr. Howard Kelly frequently detected in women which kidney was tuberculous by passing the ureteral catheter into both ureters, and then examining the urine of each kidney, when there were no indications by tumour or otherwise which kidney was affected.

CASE IV.—M. M., aged 19, of good family and personal history. On March 13th, 1891, he had pretty free hæmaturia, no clots, and blood intimately mixed with urine. Temperature 102°, and pulse 140. Aconite, citrate of potash, and buchu were prescribed. On my second visit the temperature was still 102°, but the pulse had fallen to 118. Quinine was then prescribed, 12 grains daily. The bleeding continued for three or four days, and the temperature then returned to the normal. The patient was anæmic from loss of blood, but in the course of six or eight weeks regained her usual health. In June, 1889, this patient also had an attack of hæmaturia, but I made no notes, and do not now remember whether she had an elevated temperature or not. Neither do I know what the treatment then was. She has never had any hæmaturia since.

CASE V.—Mrs. C., aged 27, mother of two children; no miscarriages; no uterine trouble except dysmenorrhœa. She had some bad headaches, suffered at times from rheumatoid arthritis, and had slight lateral curvature. Family history: One brother died of heart trouble; father died of angular curvature of the spine at 33 years; mother alive and well, age 77; one sister in good health. September 16th, 1896, patient had some chills and fever. I first visited her on September 18th, and found a considerable amount of blood in the urine, uniformly mixed. Her temperature was then 102°. I immediately put her on Warburg's tincture, and in four or five days the temperature was reduced to normal, and the hæmorrhage had ceased. She has had no attack since.

CASE VI.—Mrs. J., aged 30, married; one child aged 6 years. She had complete laceration of the perineum, on which I operated June 13th, 1892, with good result. December 29th, 1894, I again saw her, and she com-

plained of irritable bladder, which had lasted for some ten or twelve months. She passed blood December 1st, 1893, small quantity. April 6th, 1896, again passed blood, and as far as I could find out at the end of micturition and accompanied with burning, smarting pain. She also complained of pain in the right renal region, and upon examination of the kidney it was movable and enlarged. She had chills, and temperature 102.5°. On April 18th it dropped to 100°. The treatment was quinine in full doses. On April 28th I gave her a belt to wear to keep the kidney up. October 1st, 1896, no blood since, but passed two little substances (not hard) like grains of wheat. There was pain in the right kidney, passing down to the groin, but the kidney was smaller and less mobile. February 17th, 1897. She has had pain in the right kidney when she drives, especially when the roads are rough. When she lies down and rests it gets better. The urine was examined several times—result, no albumen. I do not know what was the cause of the hæmorrhage in this case; thought it was malaria, but the pain in the kidney and down the groin negated it, especially as these symptoms continued from time to time.

CASE VII.—J. L., aged 48. Father well at 83, mother died in confinement; five brothers and five sisters in good health, no tuberculosis on either side. On January 6th, 1897, he called upon me, looked pale, complained of frequency and scalding, water abundant and clear. It was not examined and I did not take the temperature. I gave triticum repens and alkalies. In the evening of the same day I visited him and there was considerable hæmaturia, the blood being intimately mixed. On the 7th there was blood again and pain at the end of micturition; temperature 102°. The right kidney was tender, and there was pain running round to front. I gave quinine in full doses. There was albumen in the urine and some pus. On the 10th temperature was 103° and patient somewhat delirious. Warburg's tincture and alkalies were then given. On the 11th, high temperature continuing, quinine and Warburg's tincture were both given. On 13th Dr. McCallum visited him with me. We continued the tincture and the blood disappeared.

CASE VIII.—Dr. W., about 50 years of age, with good personal and family history. He was taken suddenly ill March 10th, 1894, with hepatic colic, followed by jaundice, which lasted two or three weeks. He seemed to be recovering satisfactorily when hæmaturia with frequency and pain occurred, accompanied by chills and fever, the temperature at times rising to 104°, followed by profuse sweats. Quinine in large doses was very properly given by his attending physician but with only partial relief. On April 13th I saw the patient in consultation, and he was much enfeebled from the loss of blood and the prolonged chills and high temperature. As quinine had been faithfully and perseveringly tried I advised large doses of Warburg's tincture, and the patient gradually and steadily improved. To use the doctor's own words, "As soon as I commenced taking the Warburg's tincture I improved rapidly in strength and weight; I lost 50 lbs. while ill and gained half of that."

Hæmaturia other than accidental as a symptom is of more than ordinary importance. Roughly speaking, it means blood from any portion of the urinary tract—kidney, ureter, bladder, or urethra. It is nearly always possible to know from what portion of the tract the blood comes: (a) If from the urethra it may ooze between the times of micturition; (b) if from the neck of the bladder it is more particularly noticed at the end of micturition; (c) if from the bladder, it is usually abundant, more persistent, more likely to clot, and is accompanied by bladder irritability. It is well to remember that it occasionally produces obstinate retention.

Having determined with great care the point from which the blood comes, the more difficult problem lies before the inquirer—the determination of the cause of the blood flow.

In bleeding from the kidney the blood is generally intimately mixed with the urine. When, however, the quantity is large, we have ureteral clots, which I look upon as being highly diagnostic. They are occasionally productive of the same symptoms as stone in the ureter, and the colic continues until the blood is forced into the bladder.

There are cases of bleeding from the kidney, the causes of which are difficult to determine. The symptoms at a certain stage of tuberculous kidney and stone in the kidney have so much in common, that one is wholly unable to determine to which this symptom belongs. In both the hæmorrhage is usually small. The character of the pain is similar, and the quantity of albumen much the same. Repeated examinations of the urine may fail to discover the bacilli.

In the report of these few cases it will be noticed how this point was determined, and the diagnosis of a difficult case made plain, and the treatment properly directed. It is impossible to over-estimate this precise knowledge.

If tuberculous kidney is discovered in the early stage the operation ought to be as successful here as in local tuberculous disease in other parts, an operation ought not to be delayed long. Whereas if it be a case of stone in the kidney the operation could in most cases be delayed without endangering the case, and the operation is more frequently nephrotomy than nephrectomy.

In considering the report of these cases I have grouped them. Nos. 1 and 2 were, in my opinion, undoubtedly malignant, although no *post-mortem* examination was made. When tumour (kidney enlarged), pain, and hæmaturia co-exist, we may feel pretty certain of the diagnosis. Pain was

present, but was not a marked symptom in either, although No. 2 had considerable tenderness over the kidney from the time of my first examination. Emaciation and cachexia towards the end of the disease were well marked in both. Both passed ureteral clots. Several of them rolled up together, looked much like a bunch of earth worms; this alone is fairly diagnostic. Chills and fever were present in both cases, and both passed more than the normal amount of urine in 24 hours. In No. 2 the temperature three days before death ran up to 104.4° F., this condition was probably due to local septic causes.

No. 3 was tuberculous, and the report of the manner in which the diagnosis was established requires no comment from me.

The report of the inoculation and the cautions necessary in making it I have given in the words of the doctor who made the inoculation.

Causes 4, 5, 6 (?), 7, and 8 were in my opinion malarial. I now regret that the blood was not examined for the malarial "parasite."

But the fact that quinine or Warburg's tincture produced a termination of the hæmaturia, and a speedy return to health is important. Some authors speak of transient hæmaturia—not depending on any cause (perhaps it would be better to say any discoverable cause), and might assign these cases to this class, but the manifest improvement in these cases under the influence of drugs which are designated as specifics in the treatment of malaria must have considerable weight in the determination of the cause.

Case No. 6 I marked with a note of interrogation, as she had movable kidney and symptoms continuing after the attack which might point to stone—notably the tendency to pain in the kidney when driving over rough roads, and the relief of these symptoms when a recumbent posture was assumed. As she lives a long distance from town I see her only occasionally, but at no time after recovery from the attack of hæmaturia has albumen been detected in the urine.

I would suggest, therefore, in all cases of recent hæmaturia where the diagnosis is doubtful, and where there have been chills and fever, or where there has been a simple elevation of temperature, that Warburg's tincture should be tried.

A CLINICAL LECTURE

ON

THE TREATMENT OF DIPHTHERIA.

By JOHN HERALD, M.A., M.D.,

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THE etiology of diphtheria I do not intend to discuss, merely stating that I concur in the now almost universally accepted opinion that it is primarily a local disease due to the presence of a specific germ, and secondarily a constitutional disease, the constitutional disturbances being occasioned by the absorption from the local focus of a chemical poison or toxin generated at the seat of infection by the bacilli diphtheriæ. This toxin may, and frequently does, affect distant organs, and so we may have during the course of diphtheria intercurrent diseases or complications. In this way the heart, the lungs, the kidneys, the nervous system, may be seriously affected. The treatment, therefore, has three objects in view: the destruction of the bacilli at the point of infection; the neutralisation of the absorbed toxin; the combating the complications as they arise.

Such has been the mode of treatment adopted by myself and other physicians here. To illustrate I will give a synopsis of 100 successive cases treated in the Kingston General Hospital from October 1st, 1896, to March 31st, 1897. These cases were not all attended by myself, but as I have the records of all these cases before me, I will make use of them. In this hospital a patient who can afford to pay for a private ward is allowed to employ his own physician, while all who go into the public wards are placed under the care of the attending physician. For this reason these 100 cases of diphtheria were attended by different physicians, 36 coming under my care as