no fundamental objection to regarding pregnancy as the most delicate test of renal function that we possess.

The value of this view of the etiology of albuminuria is twofold. In the first place it gives us a working basis for trying to improve our results. If the recurrent albuminuria is really caused by an occult nephritis due to the first toxæmic pregnancy, plus a subliminal toxæmia due to the present pregnancy, it is by paying more attention to the first toxæmia that we may hope to prevent the occult nephritis, and so avoid at least one important factor tending to cause recurrence subsequently. It has been shown that the length of time the initial toxæmia lasts is of considerable importance in the causation of this occult nephritis; but that this is not the whole question is obvious from the fact that, even when it lasts for only three weeks or less, the recurrence rate is still 40 per cent. We must study carefully these initial attacks of toxæmia to try and discover other factors which tend to cause occult nephritis if we hope further to reduce the incidence of recurrent albuminuria. In the second place this view enables us to explain the difficulty that is so frequently encountered of placing certain cases of albuminuria into one of the two pathological groups, either by clinical or by chemical tests. Many cases belong to both groups at the same time, and we should not be surprised, therefore, when we are unable to class a particular case as either "nephritic" or "toxæmic."

A chronic nephritic who becomes pregnant is suffering from albuminuria which is almost entirely "nephritic" in origin, only a very small element being "toxæmic." At the other extreme is the healthy patient who develops albuminuria during pregnancy. She is suffering from "toxæmia" with no primary "nephritic" element. Between these two extremes come all shades of combinations of the two elements, and many of the recurrent albuminurias can be regarded as a fairly equal mixture of the two pathological processes.

CONCLUSIONS.

Toxæmia of pregnancy can be regarded as the cause of all first attacks of pregnancy albuminuria in previously healthy patients. The renal damage in these cases is purely secondary to the toxæmia.

As a result of this toxæmia a certain proportion (about 10 per cent.) develop frank chronic nephritis, and bear the stigmata of this disease, whether or not they become pregnant again.

A further proportion (about 40 per cent.) recover their kidney function completely after delivery, and subsequently have normal pregnancies without albuminuria.

The remainder (about 50 per cent.) recover their kidney function only so far as to be free from signs of renal disease in the intervals between pregnancies. If they subsequently become pregnant they suffer with recurrent albuminuria.

Recurrent albuminuria may be looked upon as due partly to a subliminal pregnancy toxæmia, and partly to a pre-existing occult nephritis caused by the first toxæmic pregnancy.

Pregnancy itself is the most delicate test of renal function that we possess.

If this view is accepted the difficulties of placing certain cases of albuminuria into one of two pathological groups are easily understood. Many cases of albuminuria of pregnancy are due to a mixture of both the "nephritic" and the "toxæmic" elements.

Although the incidence of chronic nephritis and of recurrent albuminuria can be favourably influenced by earlier induction of labour in the first attack of toxæmia, this measure by no means solves all the difficulties. We have still to find out the other important factors in the first attack of toxæmia which tend to produce permanent renal damage.

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CAUSES OF SPINAL COMPRESSION.

BY

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THE following four cases of spinal compression seem worthy of record in view of their etiology and symptomatology; thus in the third and fourth cases tuberculosis and syphilis gave rise to the condition—a comparatively infrequent occurrence. The clinical histories of the patients are given below.

CASE I.

A boy, aged 12, first began to complain of weakness of the legs and difficulty in walking in March, 1927. This was followed in April by increasing stiffness of the legs, until he was confined to bed and was unable to walk. The patient had also noticed that for eight months he had been slowly losing the sense of feeling in the legs. The previous medical and family histories were uneventful.

On examination (January 7th, 1928) the patient was found to be quite normal mentally, and no gross abnormality was noticed as affecting the cranial nerves, or the motor or sensory systems in regard to the arms. The lower limbs showed a tendency to involuntary flexion when handled; there was weakness, increase of tone, and very marked loss of power in both legs. The knee and tendo Achillis jerks were markedly exaggerated; the plantar responses were extensor and Oppenheim's reflex was present; the superficial abdominal reflexes were absent; and ankle and patella clonus were present. Sensation to light touch was impaired to the level of the fourth rib, and to a three-inch brush to the level of the umbilicus; heat and cold perception was affected over a similar area. The joint and vibration senses were impaired over the lower limbs. The fundi were normal. No changes were noticed in the other systems.

The Wassermann reactions of the blood and cerebro-spinal fluid were negative, as was also an x-ray examination of the spine. The Queckenstedt test showed a complete block, while the fluid removed from the cisterna magna and the lumbar region gave evidence of stasis of the cerebro-spinal fluid when examined chemically.

Lipiodol injected into the cisterna magna came to rest at the level of the second dorsal vertebra, and was crescentic in shape. A laminectomy revealed the presence of an extradural tumour at the level of the third dorsal vertebra. This was removed; the patient made an uneventful recovery, and is now able to walk about in a normal fashion. The tumour on section proved to be an endothelioma.

CASE II.

A woman, aged 53, first began to be ill with a dull aching pain at the lower part of the spine six years ago. Two years ago this pain grew worse, and tended to pass round the lower part of the abdomen, and down the legs to the knees. Six months ago the legs grew weak, and tactile sensation began to diminish. The patient became unable to walk.

On examination on January 24th, 1928, the patient was found to be quite normal mentally; there was no gross abnormality affecting the cranial nerves, and the motor and sensory systems in the arms were not involved. The legs showed loss of power and a slight increase of tone, but no wasting; the knee and tendo Achillis jerks were sluggish, there was no ankle clonus, and the plantar response was extensor on both sides. There was loss of sensation to pain, touch, heat, and cold to the level of the umbilicus. The fundi were normal, and the other systems showed no changes. The blood count was quite normal, and the Wassermann reactions of the blood and the cerebro-spinal fluid were negative. An x-ray examination showed some osteo-arthritis of the dorsal spine. The Queckenstedt test revealed a partial block, while examination of the fluid removed from the cisterna magna and lumbar regions gave evidence of stasis.

Lipiodol injected at the cisterna magna came to rest at the level of the eleventh dorsal vertebra. A laminectomy revealed the presence of an intramedullary tumour at the level of the eleventh dorsal vertebra. Removal was impracticable, so the dura was incised to effect a spinal decompression. The patient is now having x-ray treatment, and shows slight improvement.

CASE III.

A man, aged 27, first complained in May, 1928, of loss of the use of the right leg, followed in two or three days by loss of the use of the left leg. About fourteen days later the paralysis became complete, with total anaesthesia to the level of the umbilicus. The patient had suffered from Pott's disease since the age of 5.

On examination (June 16th, 1928) he was found to be thin, and to have clubbing of the fingers; the spine showed marked kyphosis and lordosis, but there was no tenderness on pressure on any part. The nervous system showed the following changes. The legs were flaccidly paralysed, with loss of power and tone; the knee and tendo Achillis jerks were just present; the plantar response was extensor on both sides; there was no ankle clonus, and the superficial abdominal reflexes were absent. There was loss of sensation to pin and brush to eight inches above the umbilicus, with a tendency to harlequin distribution. No gross changes were noticed in any other system, apart from the deformity of the chest due to the spinal changes.

An x-ray examination of the spine showed old Pott's disease, with angular curvature in the mid-dorsal region, as a result of which the thoracic cavity was shortened and distorted. The Wassermann reactions of the blood and cerebro-spinal fluid were negative. The cerebro-spinal fluid taken from the lumbar region showed xanthochromia; the cells numbered 2.3 per c.mm., all being lymphocytes. Protein was present to the amount of 880 mg. per 100 c.cm.; the chloride figure was 720 mg. per 100 c.cm., and the sugar 0.05 per cent. No tubercle bacilli or other organisms were seen.

A laminectomy was performed. This revealed at the level of the fourth to the seventh dorsal vertebrae a reddish-blue, thickened, nodular dura, which enclosed the cord in a collar. The cord was non-pulsatile, and could not be isolated. Some thick, greyish pus exuded from this tissue, and a section of it showed tuberculous granulation tissue. Tubercle bacilli could not be demonstrated in the pus or in the tissue. The patient was discharged unrelieved.

CASE IV.

A man, aged 64, first noticed about eighteen months ago that his right leg was becoming weak. This change slowly developed in the left leg, and four months afterwards stiffness slowly came on; the patient states that his legs tended to jerk up. He added that six months previously he had a girdle pain at the level of the umbilicus, and that he was unable to appreciate heat and cold in the legs.

On examination, on April 30th, 1928, the nervous system showed the following changes. The cranial nerves were unaffected, but there was marked wasting of the arms and weakness of the small muscles of the hands, with some slight inco-ordination in the finger-nose tests; the reflexes were sluggish. The trapezius muscles and the sterno-mastoids were very wasted. The legs showed a tendency to a "mass reflex"; the feet exhibited pes cavus, and there was loss of power, marked wasting, and increase of tone. The knee and tendo Achillis jerks were sluggish; there was no ankle clonus; the plantar response was extensor; and Oppenheim's reflex was present. The superficial abdominal reflexes were absent. Sensation to pin and brush was impaired over an area extending from six inches above the nipple and down the inner side of both arms, involving all the fingers, but sparing the thumbs. The Wassermann reaction of the blood was positive in a 1 in 45 dilution, and weakly positive in the case of the cerebro-spinal fluid; this fluid contained 6.6 cells per c.mm.—all lymphocytes; chemical examination of the fluid obtained from the cisterna magna and lumbar regions gave evidence of stasis.

Lipiodol injected at the cisterna magna lodged chiefly in the cervical region. The patient was treated with antisyphilitic measures, but died in about three weeks.

A necropsy revealed the presence of syphilitic aortitis and an extradural cyst at the level of the third to the fifth cervical vertebrae. The cyst was about three-quarters of an inch in diameter, and contained brownish material; the corresponding area of the cord was softened. A section of the cyst showed that it was a gumma undergoing degeneration. The spinal cord below this level showed degeneration due to compression by the cyst.

The points of interest in these cases are the following:

1. In three of the cases (Nos. I, III, and IV) the first abnormality noticed was impairment of the motor system occurring much earlier than sensory involvement.

2. In cases Nos. I and III pain was never reported, while in Case II the pain complained of for six years was probably due to the osteo-arthritis, and only latterly was attributable to the tumour.

3. It is also of interest to note that when pain was complained of it was not felt at the level of the tumour, and was probably due to stretching of the sensory tracts in the cord.

4. Another point of interest was the occurrence of pain in Case II, which was an intramedullary tumour.

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CONGENITAL RETROPERITONEAL HERNIA.

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RETROPERITONEAL hernia is an uncommon condition. Moynihan and Dobson have dealt with the various forms and their method of origin in their valuable and exhaustive work *Retroperitoneal Hernia*. The case here described does not conform to any of the types noted therein. It was obviously a congenital hernia, comparable in many ways to a left duodenal hernia as described by them.

A farm labourer, aged 66, had suffered for years from occasional attacks of abdominal discomfort, which he described as "indigestion," and could usually attribute it to certain foods, especially pastry, after the ingestion of which he would get epigastric discomfort, lasting from a few hours to one or two days.

In January, 1921, he was admitted into the Dorset County Hospital with, apparently, symptoms pointing to acute appendicitis; the surgeon's notes state that the appendix was inflamed, and was removed; there is no mention of any other abnormal condition in the abdominal cavity having been noticed.

On January 29th, 1926, at 10 p.m., he was admitted into the same hospital under my care, suffering from intestinal obstruction; he gave the following history. On January 27th he awoke quite fit, had his usual breakfast, enjoyed the usual action of his bowels, went as usual to his work in the fields, returned home for his midday meal, at which he ate peas, returned to his work, got "indigestion," which he attributed to the peas, and was so uncomfortable that he returned home and went to bed. On January 28th, at 2 a.m., he vomited; this relieved him somewhat. His medical attendant saw him and treated him. The discomfort continued; notwithstanding the use of aperients, his bowels did not act. The condition remained the same on the 29th; at 7.30 p.m. he vomited some "brown, nasty-smelling stuff"; he was sent into hospital.

On admission his temperature was 97.5° F., pulse 104, respirations 24; he had little abdominal pain, but was uncomfortable. His face was quite placid, and even cheerful during conversation; his heart and lungs were normal; his abdomen was a little "en garde"; although slight, it was quite definitely so; the abdomen was a little prominent, chiefly in the umbilical and hypogastric region; it did not move with respiration; there was no peristaltic movement to be seen or felt, nor was anything to be seen or felt at any of the hernial orifices; the rectum was, on examination, found to be empty, and the prostate was enlarged. An enema was given, and was returned clear.

In spite of his slight symptoms an immediate laparotomy was performed at 11 p.m. He was given ether by the open method. A catheter drew off 20 oz. of normal urine. The abdominal cavity was opened by a left paramedian incision; the edges of the incision were well retracted; no bowel came into view, but a large, flat, tense, elastic swelling, with the great omentum spread over it, was in contact with the anterior abdominal wall. On pushing upwards the great omentum there was exposed to view a large, smooth, shiny sac wall, in which distended coils of intestine could be seen tightly packed. On exploring the lower portion of this it was found to extend into the pelvis, reaching to the level of the promontory of the sacrum. The fingers could be passed behind the sac round its lower convex surface; following the sac upwards anteriorly it was found that the transverse mesocolon was arching over it and deeply indenting it. There was only a small portion of the terminal ileum free in the general peritoneal cavity, and this, together with the caecum and the ascending and descending colons, was collapsed; along the large bowel were small scybala at intervals. Coursing in the front wall of the sac from below upwards, and slightly to the right, were three small long blood vessels. Evidently the whole, or practically the whole, of the small intestines and the mesentery were packed away in the sac.

A hole was made in the front wall of the sac, in a bloodless area, and the intestines popped out; these were not greatly distended, and were only slightly congested. Five different portions of gut, which had evidently been nipped by the transverse mesocolon, acting as a constricting band, showed marks of this pressure in linear chocolate-coloured discolorations; each mark was about an eighth to a quarter of an inch wide, and varied from one to three inches in length; in no case was the viability of the bowel impaired. The constricting mesocolon could now be felt to be quite free from the sac wall, it being possible to sweep the finger under it from one side to the other of the three-inch arch. The constricting band having been loosened and the bowel released, the toilet of the wound was completed, and the patient returned to his bed.

On recovering from the anaesthetic he began to complain of increasing pain in his right chest. Notwithstanding appropriate treatment, his breathing became more and more laboured and