ABSTRACTS FROM CURRENT LITERATURE ON DISEASE OF THE URINARY SYSTEM.


Advice is sought in the great number of instances either because the child is passing a bloody urine, or is puffy and swollen. Occasionally the chief symptom is vomiting, which sometimes precedes an hæmaturia, but more frequently an œdema.

The two types of onset are associated with a group of symptoms which differentiate the cases into two large classes:—(a) Hæmorrhagic; (b) œdematous or hydæmemic. In the hæmorrhagic and larger group, the children show little œdema apart from a slight puffiness, and the hæmaturia frequently follows a tonsilitis. In addition to the naked eye blood the urine shows albumin and casts, but not to the extent seen in the œdematous form. The amount of urine passed varies. Rarely is there an oliguria, but frequently the urine is normal in amount.

In the œdematous group there is seldom a previous history of disease; œdema is the characteristic feature. The urine is usually diminished in amount, rarely shows naked eye blood, though red cells are frequently seen under the microscope. Casts are much more numerous than in the hæmorrhagic form, pus cells frequently present, and albumin present in much larger quantities than in the hæmorrhagic type. The course of these cases is longer, the tendency to recover less marked, but the tendency to become chronic is more marked than in the hæmorrhagic group.

Some cases showed signs of a combination of the two types.

To a further type, which in the course of an illness such as diphtheria, scarlatina, pneumonia, or rheumatism, shows definite albuminuria with or without casts, the name of "febrile albuminuria" is given.

Relying on Cushny's theory of renal secretion, the author endeavours to get a picture of the pathological changes taking place in the kidney during the clinical manifestations. Since it is universally admitted that there is definite secretion of the threshold bodies in the œdematous form, it appears reasonable to assume that the defect in cases which show œdema is a tubular one; and on the other hand, its absence suggests that the tubules are but slightly involved, and that the hæmorrhage is coming from elsewhere in the kidney, e.g., the glomeruli.

Parsons draws up a final classification, retaining the terms "hæmorrhagic" and "œdematous" to emphasise the characteristic symptom of each group, as follows:—

1. Acute hæmorrhagic or glomerular nephritis: (a) mild type (local glomerulitis); (b) severe type (diffuse glomerulitis).

2. Acute œdematous or tubular nephritis: (a) mild type; (b) severe type. This form in many cases passes into a sub-acute form constituting a nephrosis.

3. Mixed forms. A diffuse glomerulo-tubular nephritis, which in many cases goes on to a sub-acute or chronic form (chronic parenchymatous nephritis), and in adult life may develop into the secondary contracted kidney.

As to prognosis the author thinks that the ordinary kidney function tests are useless, and in the acute stages do harm. The best guide to the completeness of the cure is the amount of albumen (Russell's standard haze test) in a urine free from red blood cells and casts.

In treatment, stress is laid on rest, dietary, removal of septic foci. Unless in the very mildest cases rest for three months is advised, at the end of which time, should the urine show an increase of albumen when the patient gets up or is given an increased dietary, the period of rest must be extended. A rigid milk diet is recommended in the form of milk, milk and barley water, and junket, a little sugar being allowed. Purgatives and diaphoretics are of little value, diuretics harmful. Soda bicarb. may be given in cases with a highly acid urine, or where there is much pus; in ketosis occurring in the earliest stages glucose and soda bicarb. may be administered, and in uremia Mag. sulph. intravenously.

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Data are taken from records of the Johns Hopkins Hospital for 1911-1924. Included are all the fatal cases of infants of 2 years or less in whom the diagnosis of pyelitis, pyelonephritis or pyelocystitis was made before death. 29 cases were found in which pyuria was present at the time of death. 21 of these were classed as acute or sub-acute, 4 as chronic, and in the remaining 4 no lesion was found as the cause of the pyuria. Several cases that had recovered from pyuria, to die later of other causes, were investigated. In these no residual lesion was found.

The main findings in the acute and chronic cases were:

1. In one case there was a definite acute inflammation of the pelvis. In this case there was also an acute, multiple, focal, suppurative nephritis.
2. In no other case was there definite pelvic inflammation, though in some there appeared to be some slight increase in the mononuclear cells of the peri-pelvic tissue. In all cases with any urinary tract lesion there was definite interstitial, suppurative nephritis.
3. In one case there was gross cystitis with apparent blocking of one ureter, and also an interstitial nephritis.
4. The lesions were usually bilateral, with a tendency to greater severity on the right side. In unilateral lesions it was more frequently right than left-sided.
5. In 13 cases the urine was cultured, and in all a bacillus of the colonic group recovered.
6. Infection was evident elsewhere in most cases: more frequently parenteral than enteral.
7. Cases were equally distributed between the two sexes.

It is urged that the terms simple pyelitis and pyelonephritis be dropped in the absence of evidence for the existence of such pathological entities in infancy, and that the disease most commonly causing pyuria in infancy, namely, suppurative nephritis be substituted.


The writer sets out the four main functions of the kidneys as follows:

(1) the excretion of certain non-volatile products (urea, uric acid, etc.).
(2) the regulation of the body economy for water and salts.
(3) the maintenance in part of the acid base equilibrium of the body.
(4) the synthesis of hippuric acid and possibly a small part of the urinary ammonia.

Then follows an extensive survey of the various theories of renal physiology. The writer's general conclusion is that mechanical filtration of a protein-free fluid occurs through the glomeruli, and that this filtrate is further elaborated into urine by the action of the tubules through a process either of reabsorption alone or of reabsorption plus active secretion. Reference is made to the usual tests for estimating renal functional efficiency, particular mention being made of the mercury combining power of blood and saliva—a test the writer highly commends. Treatment is along well recognised lines. Milk, however, should be ordered with care if there is urea retention, as it is rich in protein, and if oedema is present it contains too much water and salts. In those cases showing N retention without oedema a low protein diet with plenty of fluid is advised. In chronic nephritis a grave prognosis is indicated by various findings:

(1) a consistently high blood urea or a continued low excretion (5 per cent. or less) of phenolsulphonephthalein in the urine in two hours; (2) the presence of neuro-retinitis; (3) serious myocardial injury; (4) a high blood creatinine. Life expectancy is generally limited to a few months when such findings are present.

Haematuria and Acute Nephritis. (Dreyfus: Gaz. des. Hop., 18th September, 1926.)

Adopting the classification of acute nephritis with haematuria proposed by Hobecourt, the writer studies in detail specified cases of haematuria,