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Etiology and Symptomatology of Acute and Chronic Renal Failure

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Incidence

There are only few data of the incidence of renal failure in children. According to a memorandum of the British Paediatric Association (1969) the death-rate from renal failure in children aged 0-14 years is estimated to be 3-5/106 total population per annum (Barratt 1971). It is possible that this figure means underestimation of the problem because the incidence of bilateral renal agenesis alone is estimated to be between 2/106 (Butler et al. 1969) and 6/106 (Potter 1946) total population per annum. Of these more than a half are live born.

According to Gautier anuric nephropathies are most common in early infancy. The incidence is still higher than later during the second and third year of life. Congenital malformations of the urinary tract and tendency to water and electrolyte disturbances play a considerable role in these figures.

Practical importance of different etiological factors causing renal failure have many regional and constitutional variations: the series of Lloyd-Still and Atwell (1966) from London for instance was dominated by congenital anomalies, whereas in the Argentine the major problem is the hemolytic-uremic syndrome (Gianantonio et al. 1964) and in Mexico acute tubular necrosis complicating gastroenteritis (Gordillo-Panigue 1967).

Etiology

In practical work, it is important to distinguish three categories of the etiology of renal failure.

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1. *Postrenal causes* = obstruction in urinary ways (Table I)
2. *Renal causes* = failure due to kidney lesion (Table II)
3. *Prerenal or functional* = failure (Table III)

Functional acute renal failure is seen after surgical operations, hemorrhagies, burns, shock, dehydration etc. and is mainly due to contraction of extracellular space. The water and electrolyte compo-

TABLE I

POSTRENAL CAUSES OF RENAL FAILURE

Uremia due to Block of Urine Flow
– Obstructive Uropathies
– Retroperitoneal Hematoma
Or Tumour

TABLE II

RENAL CAUSES OF RENAL FAILURE

	Glomerular	Tubular and Interstitial	
Principal Types	Acute Glomerulonephritis	Acute Toxic Tubular Necrosis	Kidney in Shock States
Usual Etiology	Focal Infection	Poisoning (Hg, CCC_{14}, etc.)	Operative Shock, Traumatic Shock, Incompatible Transfusions, etc.
Usual Clinical Manifestations	Proteinuria, Edema, Hematuria, Renal Failure, Hypertens'ion	Anuria	Anuria
Usual Course	May Become Chronic	Cure Usually Complete if the Critical Phase of Acute Renal Failure is Weathered	

TABLE III

PRERENAL CAUSES OF RENAL FAILURE

Usual Etiology	Usual Clinical Manifestations	Usual Course
Acute Hypotension, Cardiac Failure, Water and Electrolyte Disturbances	Acute Renal Failure	Rapidly Reversible if Cause is Corrected

sition of the body and relatively frequent acute changes in it makes it very important as an etiological factor of acute renal failure especially in early infancy. In functional renal failure the relation: urine osmolality/serum osmolality tends to be = 1.3 and urine urea/serum urea = 5. This provides some diagnostic help.

In all categories the renal failure can be acute or chronic. The urologic examination is especially important in chronic renal failure.

At birth plasma electrolyte and urea levels are always normal because the placenta is an efficient kidney. About 99% of healthy infants have micturated by 48 hours after birth and 92% already 24 hours. Failure to micturate suggests complete urinary tract obstruction, bilateral renal agenesis or severe dysplasia or a renovascular accident associated with perinatal asphyxia. Failure of intrauterine micturition results in oligohydramnion, amnion nodosum and compression deformities, particularly talipes and abnormal facies (Bain and Scott 1960, Potter 1965). In many cases (50 %?) there are other anomalies in association of congenital malformation of kidneys and the cause of death may be, e.g., asphyxia due to hypoplastic lungs.

In the neonatal period kidney anomalies and other, mainly obstructive urinary tract anomalies, septic infections - often due to gram-negative bacteria - and dehydration, renal artery and venous thrombosis with or without infection are the most common causes of renal failure.

In infancy after neonatal period the following possibilities must be kept in mind: dehydration, infections, obstructive uropathies, acute tubular necrosis, acute cortical necrosis with or without thrombocytopenia, hemolytic-uremic syndrome, thrombotic thrombocytopenic purpura and nephrotic syndrome. Nowadays renal biopsy is a valuable tool in distinguishing these different lesions.

In later childhood renal failure is often due to acute glomerulonephritis, anaphylactoid purpura nephritis, acute interstitial nephritis or pyelonephritis and poisonings.

Symptoms

In acute renal failure oliguria or anuria is the main symptom. It may, however, be overlooked, as in chronic failure, and so the benefit of early diagnosis will be lost. That is why the limits of normal urinary output in different age groups has to be kept in mind (Table IV). Early measurement of urinary out-put (per/hour) is urgently important in all cases where a suspicion of functional renal disturbances exists.

TABLE IV

FLUID INTAKE AND NORMAL DAILY AND HOURLY URINE EXCRETION

Age	Fluid Intake 24 Hours	Urinary Excretion Per Hour
0 – 12 Months	200 – 500 ml	8 – 20 ml
1 – 4 Years	500 – 575 ml	20 – 24 ml
4 – 7 Years	575 – 650 ml	24 – 28 ml
7 – 10 Years	650 – 725 ml	28 – 30 ml
10 – 12 Years	725 – 800 ml	30 – 33 ml
Adult	1500 –2000 ml	50 ml

by Charles C. Wolferth, Jr. et al.: Pediatr. Clin. North Am. 11: 1029, 1964.

The trias oliguria, acidosis and high blood urea nitrogen are the main characteristics of acute renal failure, uremia. The serum osmolality is usually higher than normal in spite of low serum sodium, often found especially in cases of acute tubular necrosis. It is assumed that in addition to urea a lot of other osmotic active (toxic) substances accumulate in the organism. Many of these susbtances are still unknown.

The *metabolic acidosis* in uremic syndrome is assumed to be the result of two components:

a. continuous production of hydrogen ions as an end-product of tissue metabolism and

b. impaired ability to excrete the accumulated acids.

Hyperpotassemia is a frequent finding in uremic syndrome and may cause fatal irregularities of heart function. The release of intracellular potassium in response to acidosis is a reason to the rise of S-K+. The catabolic state of the uremic patient and the simultaneous inability to excrete potassium through kidneys are co-working factors, e.g., the catabolism of 6-7 g of tissue protein releases 3 mEq K+ into the extracellular space.

Neurological symptoms, especially *convulsions*, often complicate the uremic syndrome and may sometimes be the first found symptom. Their etiological background is not quite clear but low serum sodium, hypocalcemia, usually associated with hyperphosphatemia, hypertension etc. are assumed to be responsible for this symptom.

Hypertension is often associated to uremic syndrome, e.g., in acute and chronic glomerulonephritis. Poor renal perfusion and increased excretion of renin in a closed overexpanded system are assumed to be behind this symptom.

Cardiac failure and pulmonary edema sometimes seen in renal failure are thought to be due to the overload of extracellular volume. In addition to this, the accumulation of different metabolites may have some kind of toxic effect directly upon the heart.

Anemia is a rule in hemolytic-uremic syndrome. It often complicates other types of acute uremia partly due to the bleeding tendency. Its coexistence in chronic renal failure points partly to the role of kidneys in erythrocyte formation.

The clinical symptoms of acute renal failure are listed in Table V. They do not need more clarification.

TABLE V

CLINICAL SYMPTOMS OF ACUTE RENAL FAILURE

Oliguria (400 ml/kg/24 h or 15 ml/kg/1 h)
Dehydration or/and Edema
Urea-Nitrogen Retention
Neurological Symptoms, convulsions
Hypertension
Cardiac Failure
Pulmonary Edema
Proteinuria, Hematuria
Metabolic Acidosis
Anemia

In chronic renal failure there may be a great variety of symptoms, not necessarily pointing to renal disease. In different age groups these symptoms, giving reason to suspect a chronic renal disease, may be very different. In the newborn period one has to keep this possibility constantly in mind when the child is ill (Table VI). In later infancy and childhood the symptoms are also often atypical (Table VII).

Symptoms, always to be remembered, are polyuria and polydipsia - due to a failure of urine concentration - as well as the retar-

dation of growth. The last phenomenon is seen in the attached (Fig. 1) growth curve of a 14-year old boy whose nephronophthisis was diagnosed obviously years after the disease had begun.

TABLE VI

CLINICAL SIGNS OF KIDNEY DISORDERS OF THE NEWBORN

– Asphyxia
– R D S
– Signs of Cerebral Lesions
– Icterus
– Dehydration
– Signs of Infection
– Urine Findings
– Enlarged Kidneys

TABLE VII

SYMPTOMS OF CHRONIC RENAL FAILURE IN CHILDHOOD

1. Symptoms of Urinary Ways
2. Edema
3. Anemia, Pallor
4. Tiredness
5. Hypertension
6. Retardation of Growth
7. Osteodystrophy
8. Changes in Serum Chemistry
9. Polyuria
10. Polydipsia

This short review shows the large etiological differencies in renal failure and also the importance of the age factor.

The prognosis of both acute and chronic renal failure is very often dependent not only on skilled treatment, but also on early diagnosis. Therefore it is extremely important to know and understand the early symptoms and also establish an adequate treatment (Oken 1970).

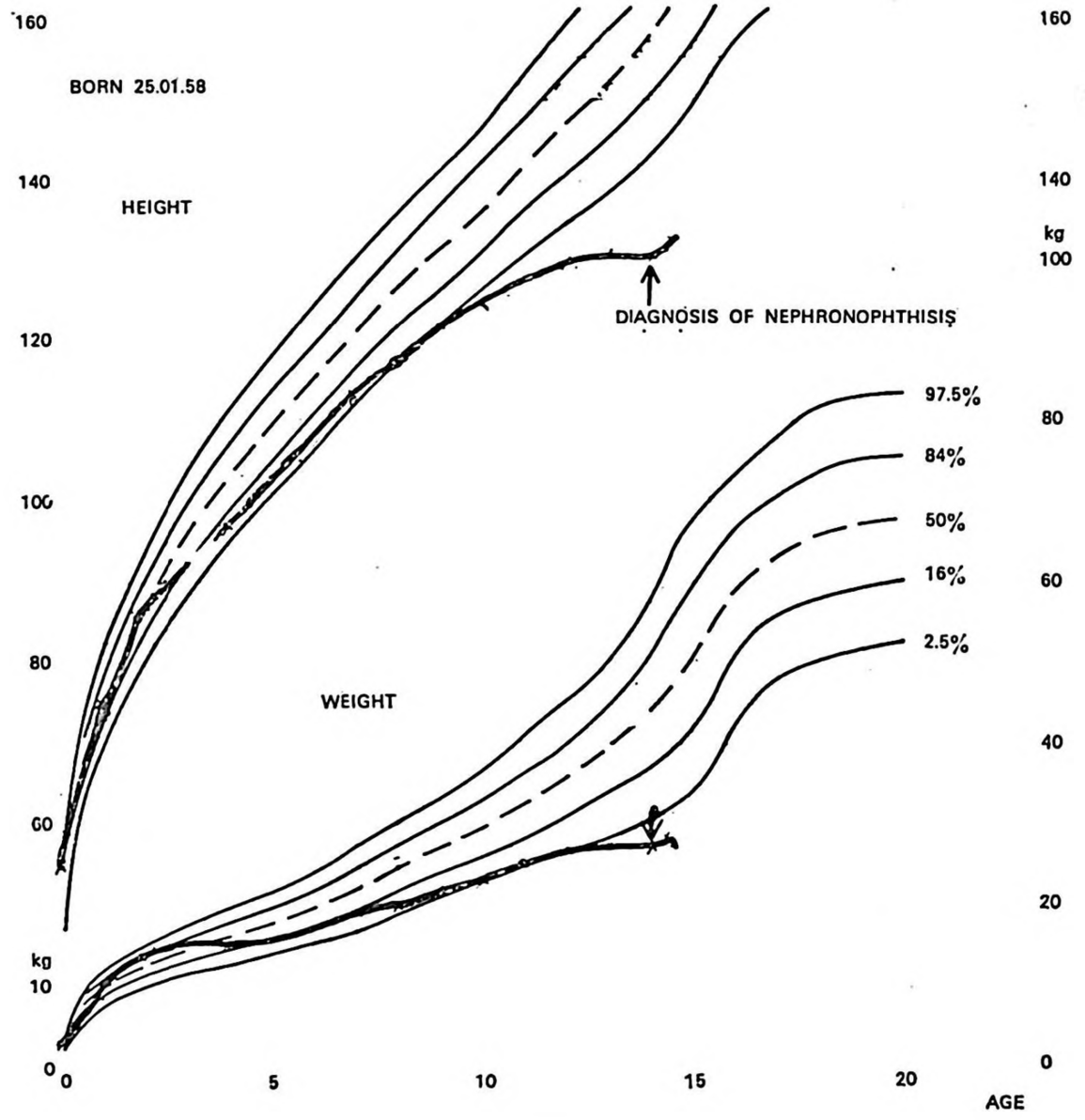

Figure 1

Growth Curve of a 14-Year Old Boy With Chronic Renal Failure

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