ever not well understood to which extend the manifested pyelonephritis might participate in these alterations. Therapeutic management of hypokalaemia and the best adjustment of potassium levels in serum leads within several months to functional and morphologic renal improvement.

4.11.1.6 Neoplastic processes

In malignant processes renal alterations with various manifestation are observed. Postmortal studies revealed histological renal alterations in about 50 per cent of patients. Not only the metastatic process by itself, but also metabolic disturbances due to the basic primary disease cause renal damage.

4.11.1.7 Tubulointerstitial reaction due to drugs

Many drugs may be the cause of tubulointerstitial renal damage. In addition to sulphonamides, the antibiotics and diuretics may be nephrotoxic. The renal lesions are observed interstitium. Oedema and polymorphonuclear leucocytic infiltration of interstitium is frequently found. In extensive damage the necrosis of tubular cells is observed.

In addition the drugs attach to the basement membrane whereby it becomes antigenic. If this occurs the disturbance will present clinically two weeks after the drug administration in form of acute glomerunephritis.

4.11.1.8 Sjögren's syndrome

Dry keratoconjunctivitis and lesions of mucous membranes of salivary and lacrimal glands are due to immunological disturbance, accompanied sometimes with rheumatic arthritis. Renal impairment may be found. Histologic examination shows lymphocytic infiltration in tubulointerstitial region of kidneys.

4.11.1.9 Irradiation nephropathy

Nephropathy due to irradiation is developing several weeks following exposition to radiation. Glomerular hyalinization, tubular atrophy, interstitial fibrosis and hyalinization of media of renal arteries are found. The underlying cause of these alterations is ischaemia due to irradiation damage. Azotaemia, systemic arterial hypertension, anaemia and proteinuria are rapidly developing.

4.11.1.10 Balkan nephropathy

This condition occurs in the watershed of Danube. Its occurence is endemic and the underlying cause unknown. The histological examination shows tubular atrophy, interstitial oedema and diffuse interstitial fibrosis. Disease can result in renal failure.

4.12 Renovascular diseases

This group of diseases comprises renal disturbances related to renal blood supply. The primary disorder is in fact of non-renal origin. Disturbances occuring during stenosis and occlusion of renal arteries, nephrosclerosis, polyarteritis nodosa, haemolytic uraeamic syndrome, scleroderma, and prae- eclampsia may be included. Renal damage depends on the speed of occlusion development determining the extent of ischaemia.

As already mentioned, the renal blood flow through kidney, calculated per gram of tissue is the highest in the whole organism.It exceeds many times the heart, liver and brain blood flow. The purpose of this arrangement is not only the oxygen supply but, above all, the glomerular plasma filtration. Kidneys have at disposal several mechanisms to ensure appropriate filtration of plasma and a proper sodium economy. Nevertheless, these mechanisms may influence other systems, mainly the circulation and act at their expense. Extremely reduced glomerular filtration can result in renal failure.

4.12.1 Diseases related to the blood supply

4.12.1.1 Acute renal artery occlusion

This term includes acute occlusion of the renal artery, or occlusion of its main intrarenal branch. The acute occlusion may be caused by trauma or embolia. Embolus can occur in stenosis of mitral valve, in bacterial endocarditis, myocardial infarction or atherosclerosis of aorta. The embolus might be, of course, of various size and according to it, it can occlude small or larger branches of renal artery. The

resulting coagulation necrosis is located in the region supplied by occluded artery. The clinical manifestations depend on the necrosis extent. A restricted infarction located in renal cortex might not be clinically apparent. A large infarction induces an immediate, intense and permanent lumbal pain accompanied with fever leucocytosis and haematuria. It is to realize, that the occlusion of renal artery main branch need not lead to evident alterations in blood. If the functions of the other kidney are preserved the urea and creatinine plasmatic levels are not elevated.

4.12.1.2 Renal artery stenosis

Partial occlusion (stenosis) of renal artery or of its main branch is commonly due to atherosclerotic narrowing or to fibromuscular dysplasia of the vessel. The main consequence of stenosis is the hypertension. Intrarenal pyelography and radionuclid renography may reveal the cause of hypertension. The affected kidney is notably smaller. Arteriography provides the definitive distinction.

4.12.1.3 Arteriolar nephrosclerosis

Alterations of small renal vessels are considered to be in relation to systemic arterial hypertension. This conception could be accepted with some reservations. Alterations of small renal vessels have been found in subjects with normal values of blood pressure ,deseased in age more than 60 years. Signs of ischaemic atrophy and glomerular sclerosis associated with alterations of small vessels are present in patients with hypertension.

The alterations in small vessels are sometimes thought to be a concomitant sign of aging. They explain the reduced glomerular filtration rate occuring in elderly.

In essential hypertension the acceleration of nephrosclerosis may appear, leading to malignant hypertension. This reversal can arise also during secondary hypertension including hypertension in glomerulonephritis, pyelonephritis, pheochromocytoma and in other diseases. In kidneys petechiae, fibrinoid necrosis of afferent arterioles and the hyperplastic endarteritis occur.

The clinical picture is completed by hypertensive encephalopathy, progressive uraemia and heart failure. Proteinuria and haematuria are present. Plasmatic levels of urea and creatinine are elevated.

4.12.1.4 Sclerodermia

Is an uncommon disease caused by autoimmune lesions of blood vessels and of connective tissue. The alterations are usually present in the skin, lungs, and especially in kidneys and oesophagus. The intralobular arteries in kidneys are notably narrowed, sometimes completly occluded by fibrinoid alterations and deposits of fibrin and mucopolysaccharides. In other cases fibrin thrombi and fibrinoid necrosis are observed. The condition may lead to systemic arterial hypertension. When concomitant oliguria occurs, sclerodermia progresses rapidly to the fatal end.

4.12.1.5 Nephropathies in condition with abnormal haemoglobin types

Presence of abnormal haemoglobins (e.g. of haemoglobin S) is relatively frequently accompanied with morphologic and functional renal disorders. The best known disease of this group is the sickle cell anaemia. Sudden, unexpected haematuria appears without preceding trauma or other overt cause. Renal urinary concentrating ability use to be impaired. Vascular lesions are most notable in vasa recta. If the proteinuria is severe, this condition may develop like nephrotic syndrome. The underlying cause are the microscopic infarctions in kidneys,commonly located in papillae and in renal cortex. Infarctions are due to hypoxia and elevated osmolality of blood of these patients.

4.12.1.6 Haemolytic-uraemic syndrome

The most important symptomes are: the haemolysis, decreased numbre of thromocytes and intravascular coagulation. Combination of acute renal failure with haemolytic anaemia and thrombocytopenia occurs commonly in children. The prothrombin time is usually prolonged and fibrin degradation products appear in serum. It is not well understood how are the kidneys involved in this complex disturbance.

4.12.1.7 Thrombosis of renal veins

Commonly occurs during membranous glomerulonephritis. It may apper during the vena cava thrombosis, trauma, or hypernephroma and extreme dehydration. Morphologic alterations and the clinical picture will depend on the occluded vein calibre and on the collateral blood flow. Complete

thrombotic occlusion is manifested by intensive pain, haematuria, oliguria and renal failure. If the renal functions recover a massive proteinuria appears.

4.13 Prae-eclampsia and eclampsia

In the last trimester of pregnancy sometimes abnormalities occur chacterised by trias of symptomes: systemic arterial hypertension, oedemas and proteinuria. This condition is termed prae-eclampsia. In some cases unconsciousness and convulsions with impaired hypertension may occur. This condition is termed eclampsia.

In kidneys a generalized oedema is present. The thickened glomerular capillary basement membrane and narrowed capillary lumen due to enlargement of endothelial cells occur. This finding is sometimes termed glomerular endotheliosis. In addition, between and under the endothelial cells fibrinoid deposits are observed. In patients deceased in this stage of disease necroses in renal tubules, liver cells, petechial haemorrhage in brain accompanied with signs of disseminated intravascular coagulation are found. The pathogenesis of this condition is not clear. Attention is focused mainly on renin-angiotensine-aldosterone system and sodium balance. It is generally accepted,that during the pregnancy sodium is retained expanding the volume of circulating blood. When the symptoms of prae-eclampsia appear the volume of the plasma is lower in comparison with nornal pregnancy. It is not well understood why the plasmatic volume is reduced. In this condition the signs of utero-placental ischeamia are stated. It is generally known that in uterus the renin and prostaglandins are synthesized. Thus, it could be supposed,that the renin production in uterus might be responsible for the hypertension. The hypertension is accompanied with oedemas and later with proteinuria.

If seizures appear induced termination of gravidity becomes inevitable. This is the most effective therapeutic intervention. All disorders disappear in some weeks. It is not univocally clear if the women with prae-eclampsia or eclampsia are the candidates for later developing of hypertension.

4.14 Hereditary renal diseases

4.14.1 Polycystic kidney disease

Is inherited as an autosomal dominant trait. It is relatively uncommon, occuring in 5 per cent of all terminal renal conditions.

Globular cysts of various dimmensions, with diameter measuring from 1 mm to some cm, occur in kidneys. Among the cysts are islets of normal tissue. The cysts are pressing the nephrons and cause so intrarenal obstruction. The cysts may occur also in other close, or distant organs.

The disease becomes clinically manifest in adults by lumbosacral pains or haematuria appearing following trauma or physical exertion. The condition is in many cases associated with hypertension and leads to the progression of renal failure. Proteinuria is present very frequently. Acute renal failure may be due to the obstruction, resp. compression of ureters by large cysts.

One form of polycystic kidney disease is combined with congenital hepatic fibrosis and portal hypertension; it is a relatively severe hepatic disturbance, therefore the hepatic symptoms prevail.

4.14.2 Disturbances of tubular functions

4.14.2.1 Bartter's syndrome

This condition is characterised by hypokalaemia due to potassium loss induced by renal disturbances. Renin plasmatic acitivy and aldosterone secretion are elevated. The pressor reaction following angiotensin II administration and the cellular hyperplasia in juxta-glomerular apparatus are evident. Weakness and polyuria appear in patients. Histological examination reveals hyperplasia of interstitial cells producing prostaglandins E and F in renal medulla.