attacks and for the development of left ventricular insufficiency.

The occurrence of complication markedly affects the prognosis of the patients. The acute complication might be hypertensive crises – being a sudden raise of the blood pressure over the values 220/130 Hg and a fast development of the signs and symptoms of hypertensive encephalopathy: severe headache, nausea and vomiting, epileptic convulsions, aphasia, disorientation, somnolence, and finally coma. The opthalmoscopic examination reveals papillary oedema and retinal hemorrhages and exudates. The morphological substrate of this syndrome are multiple thrombi in the small cerebral arteries and brain oedema. The main reason for the occurrence of hypertensive encephalopathy is the failure of the normal autoregulatory mechanism of the cerebral blood flow. It was found out that as a result of the high blood pressure in the systemic circulation in the hypertensive patients there will not be an autoregulatory conditioned drop of the cerebral blood flow, but on the contrary, the cerebral blood flow will be increased.

This will increase the capillary hydrostatic pressure and as a consequence there will be an increase of transudation of fluid from the intravascular space into the interstitium and by this mechanism cerebral oedema occurs. Since the brain is enclosed in an unexpandable skull, the capacitance blood vessels are compressed as a result of additional transudation of fluid into the interstitium. These changes in interstitial space will compress the cerebral capillaries and therefore the blood flow via the brain decreases. The wall of the cerebral arterioles that are affected by the atherosclerotic changes and thickened become poorly adaptable to the changes of the systemic blood pressure. At the physiological conditions an increase of the systemic blood pressure induces vasoconstriction, whereas the decrease of the systemic blood pressure causes vasodilatation of the cerebral blood vessels. Brain hypoxia occurs in he normotonic people when the mean arterial blood pressure is 40 mm Hg (5,5 kPa), and in hypertonics when the pressure is 70 mm Hg (9,3 kPa). The hypertensive patient is not able to respond to the hypercapnia and acideamia in the cerebral vessels by vasodilatation and can not respond to hypocapnia and alkalosis by vasoconstriction as well, as it is in physiological conditions. The relative hypercapnia and cerebral hypoxia with the corresponding changes of the brain haemodynamic can explain the characteristic morning headaches in the hypertensive people. The serious life threatening complications of the essential hypertension can be:

- left ventricular failure leading to pulmonary oedema
- acute myocardial infarction
- cerebral hemorrhage
- dissection aneurysm of the aorta
- progressing renal insufficiency.

## 3.15 Secondary arterial hypertension

The secondary hypertension usually accompanies diseases of the kidneys, cardiovascular system, endocrine and nervous systems.

### 3.15.1 Renal hypertension

It occurs as a result of the primary injury of the functional renal parenchyma – nephrogennic hypertension, or it is due to some anomalies and changes of the renal vasculature – renovascular (vasorenal) hypertension. The pathogenesis of the arterial hypertension is in those patients explained by two mechanisms: volumetric or resistance.

#### 3.15.1.1 Injury of the renal parenchyma

Hypertension during parenchyma injury is one of the most common types of the secondary hypertension. An increased blood pressure is mostly the first sign of the original renal disease.

One of the pathogenic factors to which is related the cause of the occurrence and the maintenance of the hypertension is a drop in the $Na^+$ excretion. The increased volume of the circulating blood will lead to an increased venous return into the heart. That is why the central venous pressure is increasing and the ventricular filling is increased as well. As a result of

these changes there will be an increment of the cardiac output and the systemic arterial blood pressure – this is known as a volume hypertension.

The volume hypertension occurs in acute glomerulonephritis. It is usually transient – and after the acute stage the blood pressure returns to normal values. But hypertension may manifest later, after several years when additionally to the glomerular lesions some changes in the arteries and the arterioles arrive. In such cases there is already a combination of the volume and the resistance hypertension.

In patients with the tubulointerstitial diseases of the kidneys, the hypertension is less common as that in the glomerulonephritis. The cause lies in the lower ability of the reabsorption of water based on the primary injury of the tubules.

Another mechanism, by which the renal disease can result in an increased blood pressure is the resistance mechanism. The mechanism of the resistance hypertension is explained as the result of the effect of the pressor and depressor substances that are formed in the kidneys. One of the pressor substances that is incorporated in the regulation of the blood pressure in the physiological but mainly in the pathological conditions is the renin–angiotensin–aldosteron system.

The decisive role of the renin–angiotensin system (RAS) in the pathogenesis of hypertension is assumed in renal infarction, and in unilateral renal affection, and it is absolute and very aggressive in cases of renal tumor that secretes rennin. In cases of bilateral parenchymal diseases of the kidneys, the level of the plasma rennin activity (PRA) is only slightly increased, or it might even be normal, what on the other hand does not exclude the role of this factor in the pathogenesis, mainly when we are dealing with a consequent increase of the plasma volume. In 5–10 % patients with chronic renal failure that are treated by dialysis the hypertension does not subside after the normalization of the content of $Na^+$ and fluids in the organism. The finding increased PRA as well as the normalization of blood pressure after nephroctomy prove that the RAS is the determining factor in the pathogenesis of hypertension (known as renin-dependent hypertension). RAS also plays an important role in the maintenance of vasoconstriction and of a high blood pressure in malignant hypertension. A certain role in the pathogenesis of some types of the renal hypertension can play the substances with vasopressor effect and natrium uretic effect, i.e. prostaglandins and kalikrein-kinin system.

Lowering the vasopressory function of the kidney explains the hypertension that accompanies renal diseases, which are presented with prominent destruction of the renal medulla, which produces the depressor substances (prostaglandins): chronic interstitial nephritis, pyelonephritis, obstructive uropathies, and polycystic kidneys.

#### 3.15.1.2 Renovascular hypertension (vasorenal hypertension)

Renovascular hypertension results from a narrowing of the renal artery. The cause of its narrowing lays in many pathological processes, most commonly they are atherosclerosis and fibromuscular hyperplasia.

As a result of the classical experiments with hypertension in animals it was considered sure till recent time that the cause of hypertension in those animals was due to renin – angiotensin system, that is activated by renal ischemia (i.e. by the lower pressure amplitude in the renal vascular area) caused by the drop in the kidneys perfusion. However, later on there appeared many pieces of evidence which deny a direct relationship between this system and a chronic clinical or experimental hypertension.

The course of renovascular hypertension is similar to the course of the essential hypertension, however it is usually more serious and a malignant deterioration is frequent. What is specific for this type of hypertension is a progressive ischemic post stenosis renal atrophy. From the mentioned above it is clear that two mechanisms share the pathogenesis of the renal hypertension.

The volume (hyporenin) mechanism and the resistance (hyperrenin) mechanism. According to the type of renal disease, the anatomical and functional renal injury, and the stage of this disease both those mechanisms can alternate with each other or interact with each other. In certain cases one of them has the primary role, that will promote the whole mechanism, in other cases it is the factor that maintains the hypertension.

Apart from the mentioned factors the haemodynamic changes caused by anemia play an important role in the pathogenesis of renal hypertension that accompanies chronic renal insufficiency.

For maintaining of hypertension an important role

is played by the adapting mechanisms of neural regulation of the circulation and also by the secondary morphological changes of the cardiovascular apparatus (atherosclerosis and mainly its renal form-nephroangiosclerosis).

### 3.15.2 Endocrinal hypertension

It is a hypertension caused by an absolute or a relative abundance of hormones with pressor effect or by an abnormality of the hormonal balance, that affects some components of the regulatory mechanisms which influence the blood pressure.

**PHEOCHROMOCYTOMA** (chemodektom). It is a disease caused by an autonomic overproduction of catecholamines by the chromaphin cells tumor of the sympatoadrenal system. These are usually benign tumors.

Most of the pheochromocytomas produce noradrenalin and adrenaline. In nearly 90 % of cases the tumor is situated in the adrenal medulla; in the remaining 10 % it is situated in the area of the abdominal aorta, and less common in other places. Adrenaline stimulates mostly the beta-receptors and increases the minute cardiac output. Noradrenalin via the alpha receptors increases the peripheral vascular resistance.

With regards to the fact that in most patients with pheochromocytoma the overproduction of both hormones is mixed in various quantitative relations, the circuatory changes are markedly variable. Hypertension can be manifested in three different forms:

1. As a permanent hypertension (in nearly half the patients)
2. As a permanent hypertension with paroxysms of increasing values of the blood pressure
3. As paroxysmal hypertension (with otherwise normal blood pressure).

The course of permanent hypertension is very much similar to the course of essential hypertension. The paroxysmal forms of the disease threaten the patients with the corebrovascular accidents, myocardial infarction, and heart failure.

**ADRENOCORTICAL DYSFUNCTION**. It is a case of hypertension with an absolute or a relative overproduction (or inversely – an insufficiency) of some hormones of the adrenal cortex.

There are three types of hypertension that are caused by an adrenocortical dysfunction:

1. **Primary hyperaldosteronism**. It is a disease caused by an primary overproduction of aldosteron in the adrenal gland (benign adenoma, malignant tumors, bilateral hyperplasia), which differs from the secondary hyperaldosteronism, that occurs as a result of an overstimulation of the renal cortex by the system renin – angiotensin and by other primary diseases (e.g. nephrotic syndrome, liver cirrhosis, cardiac insufficiency, an advanced essential and renal hypertension). An overproduction of aldosteron in primary hyperaldosteronism is responsible for all the clinical and the laboratory characteristics of the disease, which become normal after its removal. A long lasting overproduction of aldosteron can be manifested by:

   - Arterial hypertension, and by increase of the extracellular $Na^+$ content with a predisposition for hypernatriemia
   - Hypokaliemia with alkalosis (a result of a long lasting kalium depletion are some neuromuscular changes such as spasm, and the development of kaliopenic nephropathy).

     The raise of $Na^+$ content and of the extracellular volume leads to (by a feed back mechanism) the lowering of the plasma renin activity, however not to a point that causes a clinically manifested oedema. And so in arterial hypertension caused by the primary aldosteronism there are:

     – increased of $Na^+$ content and of the extracellular volume
     – high level of aldosteron in the plasma and urine
     – suppression of the plasma renin activity

   Since the description of primary hyperaldosteronism in 1955 by Conn, it was shown that it is not a unified syndrome, but this disease has at least three subtypes:

   - The classical Conn's syndrome, when the aldosteron overproduction is caused by an

aldosteron producing adenoma, less common by a carcinoma affecting one adrenal gland.

- Idiopathic hyperaldosteronism – in cases of micro- or macronodular hyperplasia of both adrenals.
- Dexamethazon – suppressible hyperaldosteronism (Murlow's). It is a rare overproduction of aldosteron by hyperplastic or normal adrenals in children. Hypertension in this case can be stabilized only by suplementation of Dexamethazon, that will inhibit the overproduction of aldosteron by a feed backmechanism.

2. Cushing syndrome. Hypertension results from the overproduction of glucocorticoids and sometimes also the mineralocorticoids. They could be produced by tumors or hyperplasia of the adrenal cortex, and also by adenoma of the adenohypophysis. The clinical and the laboratory picture is given by the ratio of overproduction of the glucocorticoids and mineralocorticoids. In other words the hypertension is a result of the overproduction of glucocorticoids, that increase the sensitivity of the vascular wall to endogenous pressor factors and most probably they also change the contractility of the myocardium and so increase of the cardiac output.

   Cortizol also stimulates the formation of angiotensinogen in the liver and that is the reason why there is an increase of the plasma angiotensin concentration. Apart from this it increase the vascular wall sensitive to the pressor substances. The overproduction of mineralocorticoids promotes its effect via the $Na^+$ retention.

3. **Adrenogenital syndrome with hypertension**. They are rare congenital abnormalities that occur as a result of a disturbed formation and hence production of steroids. Hypertension is accompanied by hypokaliemic alkalosis, by a suppressed plasma renin activity and by a disturbed sexual maturation and development. The inborn insufficiency of the enzymes needed for normal steroidogenesis causes stimulation of the formation of deoxycorticosteron (DOC) and hence deoxycortizol and androgens. That is why a mineralocorticoids hypertension is accompanied by the increased virilization. In case of a disturbed biosynthesis of cortizol, but also androgens and estrogens, the mineralocorticoid hypertension is usually accompanied by hypogonadism.

**PRIMARY HYPERRENINISM**. It is a rare disease, caused by renin overproduction by renal or extrarenal tumors. These are most commonly originating from the juxtaglomerular apparatus of the kidneys. Hypertension is caused by renin overproduction, that leads to the formation of large quantities of angiotensin II and so to a secondary hyperaldosteronism.

**HYPERTENSION THAT ACCOMPANIES OTHER ENDOCRINOPATHIES**

1. Acromegaly. It is due to the overproduction of growth hormone most commonly in cases of adenoma of the adenohypophysis. Apart from the morphological and biochemical changes which are characteristic for the acromegaly, this disease is accompanied by the hypertension which does not differ from the essential hypertension. The cardiomegally corresponds with the level of hypertension.

2. Hyperparathyreoidism. An increased production of parathormone can be most commonly seen in cases of the parathyroid glands adenoma. The early renal complications that occur in hyperparathyreoidism can play an important role in the development of hypertension.

3. Hyperthyroidism. An overfunction of the thyroid gland can also be accompanied by hypertension. In cases of hyperthyroidism it is usually a systolic hypertension with a high cardiac output (volume hypertension). Hypertension as well as other common cardiac complications are caused by the direct effect of the thyroid hormones on the myocardium.

4. Diabetes mellitus. The relation between hypertension and diabetes is well known for over 60 years. The presence of hypertension is more common in cases of non insulin dependent diabetes (type II) where it correlates with age, obesity, and with the drop in the renal function. Pathogenesis of this hypertension is quite. Many factors play a role here:

e.g. renal factors, macro- and microangiopathy, renin- angiotensin-aldosteron system and catecholamines.

### 3.15.3 Hypertension in cardiovascular diseases

1. Coarctation of aorta. It is an inborn narrowing of the aortic isthmus, i.e. of the section between the ostia of the subclavian artery and the attachment of the ductus arteriosus. In broader sense of the coarctationof aorta its stenosis might be localized along the whole aorta, proximal and distal from the isthmus itself.In aortic coarctation the adequate blood flow and pressure in the lower half of the body is obtained via three mechanisms:

   - by increasing the systolic blood pressure in the proximal segment of the aorta
   - by arteriolar vasoconstriction that maintains a high diastolic blood pressure
   - by a collateral circulation and possibly by the opening of ductus arteriosus.

   The blood pressure measured in the upper limbs shows an increased systolic, diastolic, and mean arterial blood pressure, while the systolic blood pressure is measured in the lower limbs it is in all cases low, and the diastolic pressure is increased. The mean arterial blood pressure in the lower limbs is always low, however it is kept at the value arround 50 mm Hg (6,6 kPa), what is the minimal pressure needed for adequate function of the kidneys.

2. Aortic valve insufficiency. It will cause a raise in the systolic blood pressure, by the fact that a part of the systolic cardiac output flows back into the left ventricle at the beginning of the systole and hence increases its diastolic filling. The left ventricular systolic volume gets larger as the regurgitation volume increases. The diastolic blood pressure becomes lower due to the faster blood flow from the aorta. The total peripheral resistance is lower as a result of the adaptation to the increased cardiac output.

3. Large arteriovenous fistulae (congenital or acquired). They cause the increase of the systolic blood pressure what is the result of the increased cardiac output. The blood flow passes by the arterioles and the capillaries and that is why the diastolic blood pressure decreases. As an example of this is the systolic hypertension in cases of multiple arterio-venous aneurysms in the skeleton as it is in Pagets disease (chronic osteitis deformans – processes of new formation as well as bone resorbtion are faster and the blood supply is increased).

4. Increase of the minute cardiac output and tachycardia at hyperkinetic syndrome. It occurs at disfunction of the vegetative nervous system which is due to an increased sensitivity of the cardiac or blood vessels beta adrenoreceptors. The positive effect of beta blockers strengthens this hypothesis. We consider a hyperkinetic circulation also states with an increased cardiac output at rest and they are accompanied by a normal blood pressure (e.g. anemia). In cases of juvenile hypertension and also in early stages of the essential hypertension the peripheral vascular resistance is not able to adapt itself to this high blood flow and so the blood pressure increases. So the hypertension here does not start as a generalized vasoconstriction, rather as an inadequate ability of the peripheral vascular bed produce effective dilatation. This is considered to be so called relatively increased peripheral vascular resistance.

5. Decrease of elasticity of the large arteries with a restriction of aortic elasticity. It is usually an accompanying feature of a generalized atherosclerosis. The systolic blood pressure is usually slightly increased. The rigid arteries do not expand during systole. The diastolic blood pressure is normal or lower that normal, because the impedance strength which pushes the blood to the periphery during diastole cannot be applied. This type of hypertension is called elasticity hypertension.

### 3.15.4 Hypertension in diseases of the nervous system

The hypertension might occur in patients with brain tumors, cerebral hemorrhage, encephalitis, or menin-

gitis and also after cerebral accidents. Its occurrence is explained by:

- increase of the intracranial pressure
- cerebral ischemia
- lesion of the vasomotor centers.

In cases of acute cerebral ischemia (e.g.: stenosis of the carotid artery) the blood pressure is promptly increasing by so called Cushing's reflex. The brain ischemia is an enormously strong stimulus for the activation of the central components of the sympathetic nervous system and hence a factor leading to the raise of the systemic arterial blood pressure. The reaction of the cerebral blood vessels to this increment of systemic blood pressure is vasoconstriction, that will lead to the closure of a vicious circle. Hypertension often accompanies the atherosclerosis of main vessels of the brain.

Another mechanism that can play a role in the occurrence of the hypertension is the disinhibition of higher centers of the brain as a result of a mechanic, inflammatory, degenerative or toxic affection of the peripheral neurons in polyneuropathies (e.g.: alcoholic neuropathy), in diphtheria, porphyria, thallium intoxication, lead intoxication, phosphorus intoxication, etc.. Hypoxia and hypercapnia have a share in the pathogenesis of hypertension.

Low sensitivity of baroreceptors can be the cause of the systemic blood hypertension when there is a low pressure in the bulbus caroticus and the aorta which is characteristic for Takayasa disease (syndrome of the aortic arch known as an inversed stenosis of the aortic isthmus). What predisposes to hypertension here is also brain hypoxia and a lower aortic elasticity.

### 3.15.5 Hypertension in gravidity

Hypertension together with oedema and proteinuria form the basic trias of the late pregnancy eclampsia. During the gravidity the blood pressure is slightly lower than normal and this is why its increase by 30/15 mm Hg (4/2 kPa) or over 140/90 mm Hg (19/12 kPa) is considered as hypertension. Vasodilatation in pregnancy is caused most probably by the higher secretion of progesterone and prostaglandins. During the first months of gravidity the renal blood flow increases by about 50 % and also increases glomerular filtration. When approaching the end of the gravidity both those values return to normal.

Pathogenesis of the hypertension in gravidity is complicated. It is most probable that its pathogenesis is shared between the volume and resistant factors. One of the hypothesis provides us with the following explanation: During pregnancy the total body fluid volume is increased by at least 8,5 l. From this total volume the plasma volume during the gravidity increases by nearly 1,5 l above the normal. By this the appropriate conditions for the congestive circulation and for the occurrence of volume hypertension are created. Apart from this the postural characteristics of the pregnant women also have a certain rolein the pathogenesis of this hypertension. In the late stages of pregnancy the pressure of the gravid uterus on the inferior vena cava in the horizontal position decreases the venous return from the lower limbs, what leads to a marked drop of the cardiac filling during diastole. As a result of this lowering of the cardiac filling the cardiac output decreases what is compensated by vasoconstriction in the splanchnic vascular bed and in the skin blood vessels. The lower blood flow via the kidneys is a very important factor which leads to the activation of the renin – angiotensin – aldosteron system.

Recently many hypothesis which tried to explain the pathogenesis of hypertensionin gravidity come out of the fact that in some gravid women the renin – angiotensin – aldosteron systemis activated. In the first gravidity preeclampsia state usually occures after 20th week of the gravidity. It is characterized by a rapid gain of weight, the formation of oedema, proteinuria and by the hypertension. The opthalmoscopic examination of the retina reveal vascular spasms and sometimes papillary oedema. The renal blood flow as well as the glomerular filtration are being worsened. In the inadequately perfused uterus renin is formed. Angiotensin then causes vasodilatation in the placenta by its effect on prostaglandin E release, but in other places of vascular bed angiotensin causes vasoconstriction. The glomerular filtration decreases and thesefore the natrium retention and the extracellular fluid volume increase. Those changes together with vasoconstriction lead to the increase of the systemic blood pressure.

### 3.15.6 Hypertension caused by drugs and toxic substances

The arterial systemic hypertension can occur as an unwanted side effect that accompanies treatment with some drugs. From the etiopathogenic point of view drugs may lead to hypertension via three mechanisms:

- due to their natrium retention effect – similar to mineralocorticoids
- due to their effect on the biosynthesis, secretion or metabolism of the pressoric humoral substances
- by their direct vasoconstrictive effect

**1.** Hypertension resulted from the use of steroid peroral contraception.The steroid peroral contraceptive agents increase blood pressure in long term use even in otherwise healthy women. Yet chronic hypertension develops only in some women. We usually find hypertension in those females with a positive family history of hypertension in gravidity, in obese women, and in woman with a higher predisposition for $Na^+$ retention and the formation of edema, as well as in women with chronic pyelonephritis.

The mechanism of the peroral contraceptive agents that leads to hypertension is complex and depends on estrogen content. Estrogens have a direct natrium retention effect and they activate renin – angiotensin – aldosteron system via a higher angiotensinogen synthesis in the liver.

The result of all this will be:

- a higher plasma renin activity till three folds more than normal
- more angiotensin II that via
- the stimulation of aldosteron leads into a secondary hyperaldosteronism and hence into
- an increased $Na^+$ retention

The synthetic progestrones in contrast with the natural progesterones have also a mineralocorticoid natrium retention effect.

**2.** Other substances with estrogen and progesterone activity A relatively common occurrence of hypertension was described in those patients who were on a long lasting treatment with conjugated and synthetic estrogens, or synthetic gestagens. Natural progesterones inversely have an antihypertensive effect.

The mechanism of the development of hypertension here is similar to that occurring post the use of oral steroid contraceptive agents. There will be retention of $Na^+$ via either direct natrium retention mechanism (estrogens or synthetic progesterones) or via the activation of rennin – angiotensin – aldosteron system(estrogens).

**3.** Glucocorticoids and mineralocorticoids.

During the therapy with adrenocortical steroids previously some coarse extracts of the adrenal cortex were used and so hypertension was a common side effect of the treatment with corticosteroids. Today it is a rare complication during the substitution therapy of hypofunction syndromes.

During the use of mineralocortid substances, hypertension is accompanied by hypokaliemia, alkalosis, and a suppressed plasma renin activity

**4.** Monoaminooxidase inhibitors. The treatment of depressive states by the use of monoaminooxidase inhibitors (MAOI) lead into often hypertensive crises in these patients after the uses of fermented drinks and food. MAOI causes hypertension via two mechanisms:

- by inhibiting the intracellular MAO and hence an increase in the level of monoamines due to their low turnover (break down)
- by interfering with tyramine deamination. In normal cases tyramine is quickly oxidized, yet with the presence of MAOI it cumulates in the tissues. Tyramine then releases adrenaline and noradrenalin from their reservoir in the nerve endings. Apart from this it has a direct pressor effect. A higher release of catecholamines and their lower break down is observed after meals which contain the tyramine (some cheese, alcohol drink, meat extract from cans, chocolate.In patients treated with MAOI (or $\alpha$ methyldopa) these meals causes a progressive increase in the plasma concentration of catecholamines and a prominent hypertensive crises.

**5.** Lecorice – Na carbenoxolon.

A chronic use of lecorice (like in some expectorants or sweets) can cause hypertension that is combined with hypokaliemia, alkalosis, and suppressed renin. This is caused by a natrium retention effect of the ammonia salts of the glycyrhinic acid, contained in the lecorice extracts. In other words they act similarly to mineralocorticoids.

**6.** From the toxic effects hypertension can be caused by acute porphyria, lead, thallium, carbon monoxide, mercury poisoning, and experimentally cadmium poisoning. The pathogenic mechanism is probably a centrally conditioned stimulation of adrenergic activity.

**7.** Postradiation hypertension. Occur in those patients treated by radiotherapy. Those are patients with abdominal tumors probably due to the occurrence of so known radiation nephritis. This hypertension might inquire a malignant character.

## 3.16 Systemic arterial hypotension

A permanently low systolic blood pressure below 13,3 kPa (100 mm Hg) and a diastolic blood pressure below 8,0 kPa (60 mm Hg) in different positions (lying, sitting, standing) is marked as chronic arterial hypotension. It is not considered as a disease condition, the hypotensive patients usually reach older ages than the normotonics. The cause of the continuously low blood pressure is not exactly understood. From the theoretical point of view the hypotonics are on the opposite side than the hypertonics in the curve that determines the role of hereditary factors on a level of blood pressure. It might be a sign of a generalized asthenia in vagotonics. It course is usually asymptomatic with the exception of the frequent sleeping or an increased tiredness. Cases of a transient hypotension (syncope and shock) are described elsewhere.

Arterial hypotension might occur in many diseases. It is the accompanying sign of inadequate adrenal function (Addison's syndrome), malabsorbtion syndrome, heart failure, aortic stenosis and constrictive pericarditis.

### 3.16.1 Orthostatic (postural) hypotension

The blood vessels are relatively elastic and permeable pipes. Upon taking the upright position, and due to the effect of gravity there is a tendency for the blood to cumulate in the distendable veins below the heart level and also to the escape of plasma into the interstitium. This transient blood distribution would be the cause of a sudden drop of the venous return, leading to a decrease of the arterial blood pressure with the consequent lower cerebral perfusion. In normal conditions when taking the upright posture there are reflex compensatory mechanisms keeping the arterial blood pressure within a certain limit of range. In the human organism the most important are the baroreceptor areas mainly those situated in the vessels above the heart level. Upon standing up the blood pressure above the heart level drops. This change is registered by the baroreceptors where the final effect is the reflexly increased sympathetic tonus of the vasomotor fibers that will provide vasoconstriction in the resistant vessels (a compensation of the lower systolic output) and even in the capacitance blood vessels (lower blood acummulation and hence a larger venous return). Despite the narrowing of the resistant vessels, the hydrostatic pressure in the lower limb capillaries in orthostasis would be over 13 kPa (100 mm Hg), what considerably exceeds the colloidal osmotic pressure of blood protein. There would be a prompt plasma diffusion into the interstitium. The protective mechanism here is represented by the contraction of the precapillary sphincters. An abnormal vessel dilatation will, at the same time, be prevented by the muscular contractions that push the venous blood towards the heart.

The muscle contraction and muscle massage of the vessels in the lower limbs can be out of function in case of the passive tilting. During this examination the patient's position is changing on a mobile bed, to which the patient is tightened. According to the level of blood pressure during the passive changing of the patients position, we might evaluate the activity of the sympathetic vasomotor reflexes with-