aortic segment. The real aneurysm is a pathological dilation of all three aortic layers. In a pseudoaneurysm there is a tear in intima and media, while adventia is dilated. In a fusiformed aneurysm the aorta is dilated all around its circumference. In a saccular aneurysm only a part of the circuit is affected. Aneurysms of the aorta are usually caused by atherosclerosis. The most common location is the abdominal aorta. The ascending aorta is affected by cystic medial necrosis. Aortic aneurysms are most often clinically silent. Sometimes pain occurs. Troubles may be caused also by compression or by erosion of the surrounding tissues. In the dilated area thrombi may occur and may cause a peripheral embolisation. Aneurysms may get perforated. A perivascular bleeding is accompanied with a pain and tension feeling.

3.20.2.1   Atherosclerotic aneurysms

are usually located in the abdominal aorta below the origin of the renal arteries. Most often they are asymptomatic. During abdominal palpation a pulsation may be discovered. The ultrasound examination will establish the diagnosis. The prognosis depends on the extent of the aneurysm. One half of the very big aneurysms end-up lethally in two years when not treated surgically.

Cystic medial necrosis is a degenerative process when collagenous and elastic fibres of the aorta change into a mucoid matherial. Affected is the proximal aorta. A fusiformed aneurysm develops. It often occurs in Marfan's syndrome but also in pregnacy and with hypertension.

Intimal disruption of the aorta. It's origin is not exactly known but basicaly it occurs in places of the greatest mechanical load. Most often the external part of the aortic arch is affected. The disruption continues to the descending aorta. Under the disrupted intima a false duct can be found. The predisposing factors are hypertension, cystic medial necrosis and congenital defects of the aortic valve. In the latter case the disruption begins near the valve. The clinical sign is pain between the scapulae. With a dramatic onset fatigue, dyspnea and syncope may occur. Aortic regurgitation may result in pulmonary edema. The prognosis is very bad.

Atherosclerotic occlusion of the aorta begins most often in the distal part of the abdominal aorta below the origin of the renal arteries. The occlusion is dis-

tending slowly in both directions. The presence of symptoms depends upon the development of collaterals.

Syphilitic aortitis is the best known inflammatory process of the aorta. The ascending aorta and the aortic arch are affected most often. An aneurysm often occurs. Sometimes a rheumatic aortitis may develop.

3.21   Coronary circulation disturbances

The heart has the action of a valvular pump. This means that the *pumping of blood* takes place by filling and expulsion in the direction determined by its anatomic structure and the functioning valves. The physiological outcome of the heart function is the cardiac output. Both the ventricles have the same cardiac output otherwise there will be an accumulation of blood ahead from the ventricle that expels less blood. The cardiac function is both pressor and volumetric work. This work is provided when the heart changes the speed of blood flow, which takes place from the heart to vessels. The cardiac output is expression of the heart function, and its effectiveness. We may have an idea about the cardiac function by measuring its energetic expenditure. The requirement of O_2 in the myocardium per minute equals three times the O_2 needed by the brain and ten times the O_2 needed by the skeletal muscles. As blood flows through the myocardium the oxygen is maximally extracted from it. That is the reason why the difference between the arterial and the venous oxygen is the largest in the body. The exchange of substances in the heart takes place in aerobic conditions. The heart cannot work with oxygen deficit (debt.), and that is why it needs a very effective arterial blood supply.

The myocardial blood supply is provided by the coronary arteries. The coronary circulation actually represents a junction between the aorta and the right atrium. It is hence the shortest circulation in the body with the highest pressure gradient. The coro-

nary circulation is very variable. Yet originally we have to agree with the opinion that says that the left coronary artery supplies the left ventricle and the right coronary artery supplies the right ventricle.

The coronary arteries run on the epicardial surface of the heart. The branches to the myocardium branch from the coronary arteries at a right angle. Under the endocardium (subendocardially) they form an interconnected network. The myocardium possesses a rich capillary supply. There is between $2\,000$ till $4\,000$ capillaries per $1\,mm^2$ of the myocardium, value which is ten times more than in the skeletal muscles.

The venous blood collected from the areas supplied by the left coronary artery is then directed to the coronary sinus. A small amount of blood collected by some veins flows directly to the right ventricle. From the area supplied by the right coronary artery blood flows directly to the right atrium.

Around 200–250 ml of blood is flowing via the coronary field per 1 minute. This represents about 80 ml/min/100 g myocardium. In case of a heavy load on the heart the blood supply may increase 5 to 7 times. In normal conditions the coronary field has a very high pressure gradient. The blood flow varies rhythmically with the pulse pressure changes. Apart from this it changes accordingly with the contraction cycle of the heart. The flow of blood across the coronaries is affected by the coronary vascular resistance to the blood flow, this is regulated by the cardiac neuronal mediators. It is as well regulated by some direct myocardial factors locally and the amount of O_2 supply of the blood.

The flow of blood across the coronary arteries is not continuous. Along the isometric phase and due to the high myocardial tension the flow of blood becomes slow. As the ventricular contraction during systole is stronger, the flow of blood via the coronaries becomes less. Sometimes it may stop completely. By the end of the ventricular ejection it becomes markedly faster. And during the fast ventricular filling phase the coronary blood flow is at its maximal level. Here the conditions are most favorable for myocardial blood flow. The amount of blood flowing across the coronary field depends upon the diastolic pressure in the aorta and the length of the diastole. The shortening of diastole in case of tachycardia or a drop in diastolic pressure in aortic regurgitation re-

sult in a marked reduction of the coronary flow, that might even lead to ischemia of the myocardium.

The coronary field, and mainly the intramural resistant arterioles has a great ability to provide all the needed blood and oxygen in every and even extreme situation (a tiring task for the human). It is very important to regulate the coronary blood flow according to the metabolic needs. Apart from this the coronary flow autoregulation renders the coronary flow suitable for other physiological or pathological conditions. The epicardial arteries as well has the ability to contract and relax. They are considered to be distributing vessels. The intra mural arteries have the ability to change their wall tension fast, and this is why they are considered to be resistant vessels.

In case of **coronary artery narrowing**, the blood flow depends on the pressure gradient. The flow becomes limited when the stenosis exceeds $75\,\%$ of the vascular lumen. The vascular field distal to the stenosis becomes dilated. This leads to a raise in the pressure gradient. If stenosis continues in progression, the flow might only be increased by increasing the pressure of blood in the pre stenozed area. If the cardiac need is high the blood flow becomes insufficient. **The myocardial need of oxygen** is increasing linearly with the increment of the cardiac action. The oxygen extraction is at its maximum in a resting stage. That is why a higher oxygen need by the myocardium can only be obtained by a higher blood flow through the myocardium. For the blood supply to be adequate all the time its regulated according to the actual needs. When the metabolic needs of the heart are increasing, the coronary artery branches react by vasodilatation, that ensures a high blood flow. The main regulating factor is adenosine. In cases of a relative hypoxia, that results from the increasing myocardial demand, in the endothelial cell membrane is adenosine produced. Adenosine reaches the interestitium, where it acts as a vasodilatatory agent by its direct action on the adenosine receptors in the smooth muscle cells of the vascular wall. The high blood flow washes away adenosine. Using of this mechanism may increase up blood flow to five times. The process of auto regulation is very complicated and it needs many other factors.

The ideal function of the pump – namely the circulatory flow of blood depends upon the circulating blood in the pump itself – namely the coronary field.