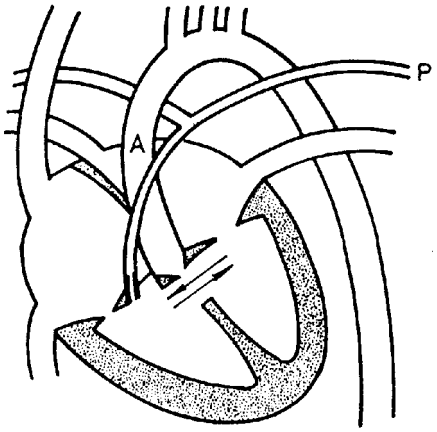

Figure 3.17: Fallot's tetralogy

shortly after birth, it usually appears before the first year of life. The explanation of this phenomena is that ductus arteriosus remains patent for few months after birth (its closure is opposed by the pressure gradient). The limiting factors of the proper haemodynamics are the pulmonary stenosis in the first place and the size of ventricular septal defect in the second place. The blood flow across the pulmonary artery can represent only Y3 the blood flow in the aorta.

When Fallot tetralogy is associated with an atrial septal defect we are dealing with a case of pentalogy.

The child becomes cyanosed during feeding and when crying. The child will be become bluish, dyspnoic, spasms may occur, and there might be loss of consciousness. Later in the child development we notice a marked loss of performance and frequent resting. There is a retardation of growth. There will be what is know as cardiac nazism with infantilism. An increased blood viscosity and hypoxia are helping factors in the occurrence of thrombi, especially in the infective endocarditis.

In those children the precordium is usually prominent and we can palpate a right ventricular heave. We can hear a harsh systolic murmur in the left parasternal area in the second and forth intercostal spaces. The second heart sound above the pulmonary artery can not be heard.

3.12 Infective endocarditis

When the endocardium is colonized by microbes the resulting disease is known as infective endocarditis. The most significant pathological changes are taking place on the edges of valves. There is some distraction which is mainly shown as changes in the shape of the valves and formation of vegetation. Infective endocarditis is the new term of what is previously known as bacterial endocarditis. the main reason of the new nomenclature is that this disease can be caused by fungi or chlamidia. From the clinical point of view we are usually talking about acute and sub acute bacterial endocarditis, valvular endocarditis, and valvular endocarditis occurring after valve transplantation. **Non bacterial thrombotic endocarditis** is an individual clinical unit.

Infective endocarditis is most commonly (80 %) caused by streptococci, staphylococci. Streptococcus a-haemoliticus viridians is the dominant bacteria causing infective endocarditis. It forms 65 % of cases. Infective endocarditis can occur following many various infective diseases. The can even form complexes of diseases such as pneumococus pneumonia, meningitis, and endocarditis, this triad is called Austrian syndrom.

Valvular defects were the previously dominating problem in the developed countries, those defects were the results of a chronic rheumatic process. Meanwhile these causes are in regression. The problem now is infective endocarditis which result from some **predisposing factors** like some congenital diseases and mainly some discrete changes being for example mitral valve prolapse. Another predisposing factors are some cardiosurgical procedures. Of course every overcomed or treated case of infectove endocarditis carries a potentional danger of reoccurrence. Many factors predispose to the occurrence of infective endocarditis. Bacteriemia as such being (the presence of pathological microbial agents in the blood) is not enough to cause infective endocarditis. The first condition for the occurrence of this

disease is that the endocardium has to be *prepared* for microbial colonization. We mean by this endocardial injury. The second condition is the ability of the microorganisms to adhere to the endocardial surface. The third condition is the replication of the microorganisms and the infecting vegetation on the endocardium.

Endocardial injury can be caused by many factors. A direct injury of the endocardium can be for example caused even in cases when the patient is exposed to a severe cold or a general hypoxia. A turbulent blood flow that accompanies valvular defects is in some conditions also a factor causing endocardial injury. The endocardium is many times a proper culture media for many microorganisms, where immunocomplexes are embedded this occurs commonly in cases of systemic lupus erythomatosus and in rheumatic disease of the heart. Stress as well do represent an evident cause of endocardial injury.

Endocardial injury is manifested by sloughing of the endothelial cells and **the exposure of the endothelial basal membrane**. The basal membrane is composed of collagen fibers on which thrombocytes (platelets) can adhere. The process of platelet adherence is a matter of surface receptors. Each adhered thrombocytes is activated, and many factors are released from this thrombocytes, one of them is ADP. ADP evokes **the adherence of other thrombocytes**. This procedure is continuously repeated. The platelet adherence will lead to and inflammatory process in the basal membrane. There will be the formation of **non bacterial thrombotic endocarditis**. It can be a completely benign process. Thrombocytic micro thrombi can be stabilized by fibrin. Due to unknown causes, in patients suffering from cachexia and malignancies there might be the formation of non bacterial thrombotic endocarditis, which is marked as marantic endocarditis.

Endocardial injury is the first condition for the occurrence of infective endocarditis. Consequently we may say that infective endocarditis is connected to the non bacterial thrombotic endocarditis. **The microorganisms get into the circulation** during many diagnostic and therapeutic procedures. These might be genitourinary diagnostic procedures such as intravenous pyelography (IVP), and even some dental procedures. There is a great opportunity for the entrance of the microorganisms during haemodyalisis. In long lasting intravenous infusions and in surgical procedures. It might be skin infection or just simply post prandial infection.

Adhesive microorganisms are actually provided by the current non bacterial thrombotic endocarditis. Although there are some known cases of infective endocarditis where no evidence of non bacterial thrombotic endocarditis could be proven to exist. Colonization of the endocardium is not an easy event. Most probably could be *performed* only by pathogenic strains of the microorganisms, that have the ability to survive in the presence of circulating complement system, antibodies, and thrombocytic aggregation on the endocardial surface. It is very likely that bacteria form polysaccharides like dextran and fibronectin, that disturb the non fluidized endocardial surface. After the process of colonization the microorganisms start to multiply. After 3–6 hours aggregations of the microorganisms, thrombocytes, and fibrin are formed. After 24 hours the infecting vegetation are markedly enlarged. Microorganisms, thrombocytes, and fibrin are sandwiched and joined together. Bacteria may accelerate **the processor fibrin formation via activating the blood coagulation cascade**. the growing bacterial colonies participate by this way in the formation of fibrin network, that contains only few cells having the ability to phagocytose and lose their defensive ability. The whole endocardial surface is continuously washed by blood that contains antibodies and protective monocytes and polymorphonuclear leucolyses. Bacterial colonies are protected by the fibrin network. This process can take place anywhere in the endocardium. The most common sites are the valvular edges.

Infective endocarditis can cause the most serious **changes in the valvular apparatus of the heart**. It is commonly a problem that concerns the whole organism. the classical picture of infective endocarditis has the following parameters: fever, cardiac murmur which changes very fast, petechiae on the skin, conjuctiva, and oral mucus membrane. The cooperation of the whole organism is caused by the continuous **spread of the infected micro emboli** and immunocomplexes to the whole organism. Apart from fever there is anorexia, loss of weight, lumbar pain, night sweating, the appearance of new changing murmurs, petechiae, positive haemoculture, increasing erythrocyte sedimentation, abnormal urine exam and usually a quick onset of the disease.

Infective endocarditis can affect the conductive

system of the heart. We can then notice different types of atrioventricular heart blocks. Atrioventricular blocks, bundle branch blocks or fascicular blocks. The displaced vegetation can reach the coronary arteries, and then evoke a picture of acute myocardial infarction.

The process of treatment can be obtained by antibiotics to destroy the bacteria. Macrophages then remove the destroyed and useless **material**. Fibroblast form new collagen and in few weeks or few months time the endocardial surface is covered with epithelium. Even through we may find later during transplantation of values the presence of microorganisms on those injured valves. The affected valve is usually thickened and calcified. During endocarditis the affected valve can be perforated.

The most frightening complication is heart failure. It occurs nearly in 75 % of the affected individuals. The second serious complication is **meningoencephalitis and cerebral embolism**. Renal disorders are mainly caused by the embedded immunocomplexes in the basal membrane of the glomerular capillaries. In the acute form of infective endocarditis the patient is threatened by a huge distraction of the affected valves, including perforation. In long-lasting intensive use of drugs there might be a progression to a sub acute form of infective endocarditis. After the replacement of a valve (implantation) infective endocarditis occur within 60 days. During pregnancy infective endocarditis may follow infections in patients having already some kind of congenital heart disease. The untreated infective endocarditis is always a fatal disease.

3.13 Pathological changes of the blood pressure

3.13.1 Functional anatomy of the circulation

The circulation can perform its basic function in an optimal way only when the amount of the blood flowing through the capillaries of each tissue, or organ per a time unit is fair enough to keep the homeostasis of that organ, so that it can perform its function adequately. the blood flow per minute via the capillaries of the given tissue or organ is the most important parameter of the blood flow (haemodynamics)

The vessels from the functional point of view can be devided into:

1. **Compliance vessels**, that form the large and intermediate arteries. Their function is to provide a continuous flow of blood. Ensure a fast transport of blood to the peripheries.

2. **Resistant vessels** are the major determinants of the general peripheral vascular resistance and by this even the regional blood flow.

   The whole peripheral vascular resistance is an important factor upon which the intermediate arterial blood pressure depends. It includes: The elastic resistance in the arterial system, the peripheral resistance of the resistant vessels, and the resistance which is imposed by the pre capillary sphincter. We recognize two types of the resistant vessels:

   (a) **pre capillary resistant vessels** – small arteries and arterioles – which form about one half of the value of the peripheral vascular resistance.

   (b) **post capillary resistant vessels** – venules and small veins That form a small part of the resistance. The participate in the changes of the potential volume of the capacity field.

3. **precapillary sphincter** is that part of the vessel that regulates blood flow into the capillaries and selectively distributes blood into those capillaries. By opening and closing these segments we can determine the number of transition capillaries in a given organ or tissue. The pre capillary sphincter under goes systemic and local effects. That determine the metabolic of the tissue or organ.

4. **capacitance vessels** (volume) are mainly the large systemic veins. They represent the reservoir for heart filling.