Over haemodynamische verschijnselen bij stenoses
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In this thesis in vitro experiments are reported which deal with hemodynamic aspects of stenoses.

From a review of the available literature it appears that only a few workers have tried a more or less exact approach to this problem. The majority of authors restrict themselves to speculations.

Chapter 1 gives a review of the partly theoretical, partly empirical hydrodynamics. In a fluid flowing through pipe lines we can distinguish two states, laminar and turbulent flow. The first state changes into the second one when the number of Reynolds is exceeded. In a turbulent flow eddies may be formed, especially at the edges, while the central flow may be predominantly laminar. When the diameter of a pipe line changes, the occurrence of turbulence is determined by other factors. Within the physiological limits of the velocity of flow turbulence is avoided when the boundary deflection is smaller than 60°.

Friction of a flowing liquid in a certain section of a pipe line with constant diameter causes losses of energy, which are proportional to the loss of lateral pressure over this section.

The Bernoulli-law is valid in the ideal case when losses due to friction may be neglected. This principle applies to the variations of the static (lateral) pressure, when the diameter of the tube changes. From this law we have derived a formula which allows us to estimate the changes of the lateral pressures in the neighbourhood of the stenosis. Apart from the Bernoulli effect these changes of the lateral pressure are influenced by frictional losses and the contraction of the stream lines just behind the stenosis.

When a cardiac catheter is used for the determination of the pressure in a flowing liquid one may find a number of possibilities. If the catheter is moved in the direction of the flow, suction may occur near the tip of the catheter.

This suction, which we called the Siphon-effect is therefore superimposed upon the Bernoulli-effect. Because of this Bernoulli-effect a systolic gradient must be found between for example the right ventricle and the pulmonary artery, for in the ventricle the pressure is determined usually when there is little or no flow, while in the large vessels the measurements necessarily have to be performed in the stream. Especially in cases with a rapid stream in the pulmonary artery this "normal gradient" may become rather high.

With intermittent flow in elastic systems the "Windkessel" phenomena play an important role.

In the experiments we substituted glycerin solutions for blood. In a theoretical discussion of the viscosity of fluids
it is shown that there are no objections against this procedure.

The second chapter begins with a description of the apparatus which we used in our experiments.

We used metal and rubber stenosis varying in shape and diameter. With these stenoses we investigated how the resistance and the Bernoulli effect of a stenosis depend on the volume-velocity, the viscosity of the fluid, the peripheral resistance and the mean pressure in the system under circumstances of constant or intermittent flow.

First we perfused rigid systems, with different stenoses with a constant or intermittent flow. Subsequently elastic tubes were used. Because rubber tubes are not suitable for reliable measurements of the lateral pressure we used freshly excised thoracic aortas of young cattle. The intercostal arteries were canulated and connected to a manometer in order to measure the pressures on the wall in such a vessel. The thoracic aorta - in situ - of a large dog was connected to an experimental stenosis. Again the lateral pressures were registered in the intercostal arteries. In this way we obtained a natural elastic system. The pressure curves in this dog were similar to those obtained in our in vitro experimental elastic systems.

We registered the pressures in the lumen by means of a (Lehmann nr. 8) cardiac catheter with the opening at the tip. The pressures in the lumen were compared with the pressures on the wall at about the same distance from the stenosis. All pressures recorded under circumstances of intermittent flow were compared with the pressures found with constant flow, and the same volume velocity.

The influence of viscosity on the resistance of the stenosis and on the Bernoulli effect was investigated with water, a 25 percent and a 42 percent solution of glycerin in water. Some interesting phenomena were observed, caused by the consistency of the fluid on the one side and the suppression of the formation of eddies on the other side.

We recorded simultaneously the pressure on the wall and the pressure in the lumen at two cm. behind the stenoses as a function of the flow in a rigid system. Three of the curves we obtained in this way are reported here. An analysis of these curves showed some interesting points with respect to the effects of inertia of the fluid and the movements of the fluid during a contraction of the heart.

In the discussion we made a qualitative and a quantitative analysis of the results of the investigations. The results were compared with clinical observations.

On the basis of the formula proposed by Gorlin and Gorlin and the discussion by Wiggers of the resistance of stenosis we have extensively discussed the determination of the cardiac output and the velocity of flow derived from this.

We have made up a diagram from which the relationship
between the gradient due to a stenosis and its diameter can be derived for varying volume velocities.

In Chapter III the different hypothesis about the origin of post-stenotic dilatations are reviewed. These opinions can be classified into two groups. According to the first point of view the dilatation is a mechanical effect of the stenosis. The second theory considers changes in the vascular wall to be of primary importance.

The significance of mechanical factors was subjected to a critical study on the basis of our experimental data and of the histological study of the wall of arteries with or without poststenotic dilatations.

Because of the occurrence of aneurysmata in jet lesions of which a characteristic example is described, it was necessary to determine the order of the magnitude of the pressure on the wall at the site of impact of the jet.

We also had to investigate by model experiments in a quantitative manner the cavitation effect, which has been suggested as the cause of post-stenotic dilation.

Because congenital malformations of the pulmonary artery wall were considered to be the cause of post-stenotic dilatation of this vessel, the embryology of this area was reviewed.

Chapter IV deals with the augmented pulsations of the dilated pulmonary artery in cases of post-stenotic dilatation. These augmented pulsations seem to be paradoxical. The pulsations of this vessel are also augmented when there is only a dilatation with or without an increased cardiac output. On the basis of our experimental data we were able to give an explanation of this phenomenon.

This chapter also discussed some data obtained by cardiac catheterisation which were important in view of our investigations.

Conclusions:

The most important conclusions are:
1. The phenomena found by us in vitro also exist in vivo in a qualitative as well as in a quantitative manner.
2. The peripheral resistance and the mean pressure do not affect the pressure gradient of a stenosis and the magnitude of the Bernoulli effect.
3. The resistance of a stenosis increases with a decrease of the diameter and an increase of the volume velocity. The Bernoulli effect becomes more marked under these circumstances. Increase of the diameter of the stenosis or decrease of the flow cause a decrease of the gradient and a decrease of the Bernoulli-effect.
4. The resistance which a flowing fluid encounters in a diaphragm-like stenosis is higher than the resistance due to a
stenosis with a length about equal to the diameter: the pressure decreases not only at the site of the stenosis but also over a short distance beyond the stenosis.

5. With an increasing volume velocity the resistance of a stenosis with a diameter less than 10 mm increases so much that this must be a critical size. This is in striking agreement with the clinical experience of Glover.

6. Under normal circumstances the Bernoulli effect causes a pressure gradient between the ventricles and the great vessels. An increased output of the heart or one of the ventricles may result in an elevation of the gradient so that this suggests the presence of a stenosis.

We may accept the existence of a stenosis only when we have a reliable determination of the cardiac output and when there is a marked fall in pressure (at least 20 mm Hg).

7. One may find paradoxical pulsations of a blood vessel past a stenosis due to the Bernoulli-effect.

8. The formula of Gorlin and Gorlin provides a fairly close approximation of the diameter of a stenosis if the cardiac output is not excessively high or low.

9. In cardiac anomalies in which a stenosis is associated with polycythemia the correction of the polycythemia does not necessarily improve the hemodynamics.

10. It has not been demonstrated, on the contrary, that vibrations of the wall of a vessel result in damage of the elastic supporting tissue. In the region of the trunk, the wall of a normal pulmonary artery has an irregular and fragmentated elastic tissue skeleton, therefore one cannot attach significance to such an appearance as a cause of a dilatation of this artery. In the wall of an aorta which must have been vibrating during many years, we found a very regular structure of the elastic fibres.

11. The increased lateral pressure immediately beyond a stenosis, which according to Holman, is the main cause of the post-stenotic dilatation, is apparently of very short duration and not significant.

12. We believe that post-stenotic dilatation is caused by atrophy due to inactivity of a predisposed vascular wall. In addition to this factor, a significant role may be played by trophic disturbances due to an impaired blood supply of the vessel wall caused by the abnormal pulsations.