CHAPTER I

INTRODUCTION

As the average length of human life increases, ophthalmological problems associated with advancing years become increasingly important.

The fundus lesion known for more than a century, since the invention of the ophthalmoscope in 1850, as retinal venous thrombosis, still forms a serious threat to the visual acuity of aging persons.

In the early 1940's it was thought that an effective medicament had been found for retinal venous thrombosis: heparin. The first report of improvement with this medicament supported the theory that the condition was caused by a thrombus which was based on pathological findings in eyes enucleated on account of haemorrhagic glaucoma, nowadays known as neovascularization glaucoma. It very soon became apparent, however, that the results obtained with anticoagulants were not always favourable and ultimately turned out to be disappointing. It also appeared that the 'thrombotic' fundus picture could be caused by other conditions than arteriosclerosis but the poorest results were obtained in patients suffering from arteriosclerosis.

More recent histological studies (Seitz, 1968; Rabinowicz et al., 1968) and more particularly fluorescence angiography (Oosterhuis, 1968) have shown that a thrombus is not primarily involved in the development of the pathological fundus picture. From experiments (Hayreh, 1965; Dollery et al., 1969; Kohner et al., 1970) it appears that the condition is predominantly the result of chronic hypoxigenation of the retina.

Sclerosis leads to increasing narrowing of the lumen of the retinal artery, retinal vein or both, resulting in chronic impediment of the blood flow in the retina. The resulting hypoxigenation causes the blood-retinal barrier to break down. Oedema and haemorrhages are not the result of back-pressure in the vein which was thought to be blocked by a thrombus, but are caused by the breakdown of the blood-retinal barrier by hypoxigenation.

It is therefore better to speak of 'occlusion of the retinal vessels', as suggested by Ennema & Zeeman in 1953, than of 'retinal venous thrombosis', since the name 'venous thrombosis' has originated from and has led to misunderstandings about the aetiology of the circulatory disorder.

As the symptoms are predominantly localized on the venous and capillary side of the retinal vasculature we prefer the name 'retinal vein occlusion'.

This thesis has been written as a contribution to the evaluation of the recently developed photocoagulation as a therapy in retinal vein occlusion. Further, 'carbogen' (95% O2 and 5% CO2) inhalation has been added as a new element to the treatment of retinal vein occlusion.
CHAPTER II

HISTORICAL SURVEY

The ophthalmoscopic picture of 'thrombosis' of the central retinal vein was first described by Liebreich in 1855, five years after the invention of the ophthalmoscope by Helmholtz. Subsequently, many publications have come out in which this picture is explained as being the direct result of an obstruction on the venous side of the retinal circulation.

Von Michel (1878) presumed that a thrombus in the vein was the cause of the fundus picture. In his publications of 1899 he affirms this and states that the condition is secondary to a proliferative inflammatory condition of the veins (phlebitis proliferans). Angelucci (1878) thought that thrombophlebitic foci were the cause of the occlusive process. Reimar (1899) and Hertel (1901) postulated an obstruction caused by endothelial proliferation. Harms (1905) assumed that a primary inflammation of the venous wall in the form of a meso-endophlebitis proliferans was the causative factor, which would lead either to closure of the vessel by progressive proliferation or to thrombus formation causing vessel closure either in situ or further downstream. Verhoeff (1907) thought that the vein occlusion was caused by endothelial or sub-endothelial proliferation.

Coats (1905 and 1913) defended the theory that in nearly all cases the picture of central retinal vein occlusion is the result of a thrombus; he considered endothelial proliferation and organization as secondary phenomena. However, he introduced a new element into the discussion by being the first to draw attention to the importance of sclerotic changes and circulatory disturbances on the arterial side of the retinal vascular system in the pathogenesis of retinal vein occlusion. Since then attention has remained focussed also on the arterial component. Bauer, as early as 1909, speaks of a primarily arterial condition, but one in which with the ophthalmoscope the changes are more readily visible in the veins.

Later on Leber, Coats & Harms (1915) together developed a different theory: arterial thrombotic processes would lead to multiple micro-emboli in the retina, causing slowing down of the blood stream and ischaemia, which in turn would result in thrombus formation in the retinal veins. They considered thrombophlebitis to be a cause of vein occlusion in young people only. Scheerer (1923) stated that in most, if not all, cases of central retinal vein occlusion lesions of the central retinal artery are also significant for the pathogenesis, even if they are not visible on ophthalmoscopy. The theory that sclerosis of the central retinal artery is an aetiological factor in the clinical picture of vein occlusion is also supported by later publications (Braend-