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# 120 YEARS EMBOLISM OF THE CENTRAL RETINAL ARTERY

# I. BASIC PROBLEMS AND RESEARCH FINDINGS ON RETINAL CIRCULATION

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It was the great ALBRECHT VON GRAEFE (1859) who 120 years ago, first diagnosed embolism of the central retinal artery. We have, therefore, attempted to review the history of the experimental and therapeutic concepts of this disease. The present paper will discuss some basic concepts of the retinal circulation. A second paper (SAUTTER and ROSSMANN, 1981 will then deal with clinical and therapeutic problems.

Generally speaking, there are three different methods to investigate the circulation of the retina:

Physical procedures,
studies fo the regulation of retinal circulation,
observations of the behaviour of micro-circulation.

## Physical procedures

Until to-day it is impossible to measure the exact flow rate of the retina or of parts of it, since for the time being we lack incontestable methods of *quantitative* determination. There are, however, some promising aspects that may improve

experimental flow measurements to such an extent that solely from these findings the determination of retinal blood supply may become possible. But it is fallacious to derive the exact rate of retinal blood supply purely from the velocity of flow, since we are unable to calculate the sectional area of the lumen of the vessels, which is another very important factor of POISSEUILLE's law.

In order to evaluate the rate of retinal blood supply, we therefore depend on indirect deductions using *qualitative* methods. Among these we should first mention the numerous investigations utilizing *ophthalmo-dynamometry*. In the course of some decades, these yielded remarkable results (BAILLIART, 1923; WEIGELIN and MÜLLER, 1951; WEIGELIN and LOBSTEIN, 1962; WEIGELIN et al., 1964; HAGER, 1963).

More recently, fluorescence angiography and other deye distribution studies have played a role. With such methods, however, alterations of the perfusion rate can only be obtained in experimental animals.

Finally, *calorimetric* methods must be mentioned, although these procedures give only clues to uveal perfusion. Uveal circulation itself is not uniform and varies in its components choroid, ciliary body, and iris. It is impossible to draw direct conclusions from this parameter to the behaviour of the vessels and to retinal perfusion. This is mainly due to the fact that the blood-tissue-barrier of the retina is much tighter than that of the different components of the uvea. This should be considered in all dye investigations and can be visualized under certain conditions, for example in albinotic animals.

If it were possible to enhance one of these qualitative methods to obtain quantitative data, the assessment of the physiological and pathological behaviour of retinal perfusion would be much more precise. Up to now, reliable quantitative data are only available for the uveal circulation of dogs. These were obtained by PILKERTON et al. (1964) with the $N_20$ method, whereas COHAN and COHAN (1963) used a direct procedure. The results of both methods correspondend closely and yielded an extraordinaryly high value. This even surpassed the perfusion values of the cerebral cortex, found by many authors, and approached the values of the kidney. Similar to the kidney, the $0_2$ extraction of the uvea turned out to be rather small (PILKERTON et al., 1964; COHAN and COHAN, 1963). Such a high perfusion rate is not necessary to meet a high energy demand, but to ensure a high rate of fluid secretion.

It has often been attempted to estimate retinal perfusion from the retinal $0_2$ consumption. Till now, both approaches either lead to an experimental increase of the arterial $0_2$ pressure or to *in vitro* experiments according to WARBURG. Both procedures are biased by sources of error and therefore yield only general clues. We have to assume that the retina represents a tissue with very high energy consumption. This is firstly confirmed by comparative studies of the $0_2$ saturation in the reti-

nal arteries and veins, and secondly by the anticipated fact that all *in vitro* and *in vivo* experiments revealed a remarkable aerobic glycolysis of the retina. This of course is directly related to its highly differentiated performance.

## Regulation of retinal perfusion

Much more productive were investigations into the *regulation* of retinal perfusion. According to SCHNEIDER (1953) and HIRSCH and SCHNEIDER (1967 a,b) the latter is very much in accordance with the regulation of cerebral perfusion and, therefore, one can start from here.

In the experimental animal (dog), the increase of cerebral perfusion is steeper than the increase of blood pressure and flattens out more pronouncedly than that of the extracerebral circulation. Above a level of 70 to 80 mm Hg, the cerebral perfusion rate remains constant. According to the qualitative results of PORSAA (1941) and based on clinical observations, we may assume that the retinal perfusion is similarly regulated as opposed to the uveal perfusion. In a healthy vascular system, a constant cerebral perfusion rate is thus maintained over a large pressure range and the vascular resistance increases concomitantly with pressure. This so-called BAYLISS effect is hemodynamically a very important assertion. In uncomplicated hypertension, all retinal vessels should be found to be smaller, without a mandatory conclusion from a descreased retinal perfusion. Such perfusion can develop secondaryly on the base of arteriosclerosis and arteriolosclerosis. When the blood pressure is lowered, on the other hand, the cerebral perfusion rate is maintained by autonomous vasodilatation, as long as a certain critical level is not reached. By analogy, the retinal vessels should be dilated in this case, and that is why one should not assume a decreased retinal perfusion.

HIRSCH and SCHNEIDER (1967 a,b) and others have correctly termed this behaviour autoregulation. It has been observed in other vascular areas: more pronounced in the renal cortex (but not in the renal medulla), less pronounced in the area of the external carotid artery. Besides metabolic influences, mainly an autonomous response of the smooth muscle fibres of the vessel walls seems to be responsible: The increment of the internal pressure extends and depolarizes the musculature of the vessel wall, resulting in a propagated excitability and vasoconstriction (FOLKOW, 1964). This response is so well developed in kidney, brain, and retina that within a wide range of pressure rates, a constant rate of perfusion is maintained on a critical level.

In *sudden alterations* of the blood pressure, the final rate of perfusion is certainly obtained after minutes. If, in the experimental animal for example, the arterial pressure is raised considerably and then lowered abruptly, the perfusion first ceases and after a few minutes returns to normal. A further decrease of blood pressure, however, causes a breakdown of the antiregulation mechanism and a disappearance of flow.

The most important clinical consequence of this behaviour is, as both ourselves (SAUTTER, 1953) and HAGER (1966 – 1974) have repeatedly pointed out, that a drastic treatment of hypertension — and evenmore so a vasodilatation in cases of normotension or hypotension — lead ot a sudden decline of pressure. This causes a temporary disturbance of the oxygen supply to brain und retina, and a *‚blackout'*, i. e. a loss of function or lack of consciousness, respectively, will ensue. If the pressure is decreased slowly, perfusion and oxygen supply (at least in a normal vascular system) remain constant over a wide range of time. That is why *vasodilating agents* affect cerebral or retinal circulation only to a minor degree and only a short period of time. Ensuing vasodilatation will provoke *autonomous antiregulation*, thus yielding normal perfusion values in spite of continous drug administration.

In the case of a drastic decrease of cerebral perfusion and lack of oxygen, such agents can never be effective. Although antiregulation is cancelled, an excess of metabolites in the absence of oxygen has already enforced a maximal vasodilatation.

Actually, these metabolic factors, and among these the gas concentration of the blood, play an important role in the rate of retinal perfusion. For example, an increased carbon dioxide concentration (partial pressure in the arterial blood) increases perfusion. On the other hand, a stepwise decrease of the arterial oxygen pressure induces a relative increase of the carbon dioxide concentration and, therefore, an increase of the perfusion rate. This, however, occurs only after passing through a *zone of indifference*, depending on a corresponding drop of the venous oxygen pressure. (The venous oxygen pressure is the indicator of the tissue supply.) A further decrease of the arterial oxygen pressure promotes further increase of perfusion. This increment is limited by a critical level. If the oxygen pressure is lowered to such an extend that it causes disturbances of vision and/or consciousness, the provoked increase of perfusion can only compensate for 30% of the normal values.

In this range, autoregulation becomes ineffective and is finally cancelled. This underlines that the level of blood pressure and the blood gas concentration are the most important factors for the blood supply of brain and retina.

At this point some words on the terms of *‚survival time'* and *‚reanimation time'* should be inserted. The *survival time* represents the period of complete lack of oxygen, during which the function in question can be maintained. In the eye, this is the period before the disappearance of visual function, i. e. the *black-out time*. It amounts to only a few (5 – 6) seconds, whereas loss of consciousness, according to OPITZ and SCHNEIDER (1950) and ANDERSON and SALTZMAN (1964), occurs one second later. If the interruption of blood perfusion is less final, ANDERSON and SALTZMAN found a black-out time of 10 seconds. The ERG disappears after 3 minutes, visual function being very sensitive to a lack of energy. Furthermore, ANDERSON and SALTZMAN (1964) could show that this period depends upon the oxygen supply to blood and tissues, and that it increases linearly with the

arterial oxygen pressure. Similar to other organs, excepting the muscle, anaerobic energy supply, regardless of how powerful it may be, can under no circumstances cover th energy demands of brain and retina.

*Reanimation time* represents the maximal period of complete ischaemia or anoxia, which allows recovery without singns of function or histological damage. According to HIRSCH et al. (1957), the reanimation time of cerebral tissue amounts to 8 – 10 minutes at a temperature of 37°C.

Surprisingly enough, the retinal reanimation time is considerably longer (POPP, 1955; PAPST and HECK, 1957). As an indicator for the determination of the retinal reanimation time, the re-occurence of the ERG has been used. The results of retinal reanimation time are considered to be excessive. This is based on two main factors: (i) the maintenance of a minimal basal metabolism, which guarantees a *minimum vitae* but is unable to be functionally efficient; (ii) the possibility that minute functional deficits are not detected with our methods of investigation. It should be kept in mind that a determination of the retinal reanimation time cannot be based on clinical observation. Only in rare instances is there a sudden and complete ischaemia of the whole retina.

In cases of complete occlusion of the central retinal artery a complete recovery can never be anticipated, because the retinal reanimation time is always exceeded. Therefore, this periode cannot be estimated from clinical data. In most other cases, in which functional improvement could be assessed, the ischaemia was incomplete. At this point it should be stressed that a residual circulation can expand the reanimation time considerably. As HIRSCH puts it, each drop of blood flowing through the retinal vessels, increases the reanimation time. The function is paralyzed, and it can recover even after a long period, if in the meantime a minute maintenance metabolism (metabolism of viability, *minimum vitae*) could be established (of HIRSCH and SCHNEIDER, 1967 b). The terminology and the problems of reanimation time are to be separated strictly from the term survival time, and we have to assume, that values of the retinal reanimation time are in the same range as those of the cerebral reanimation time, namely 8 – 10 minutes at a temperature of 37°C.

More and more frequently it is assumed today that perfusion disturbances of the brain and the retina accur in pre-existing *circulatory disorders* (e. g. arrhythmia or cardiac insufficiency) and are consequences of an abrupt decline of the blood pressure. In these cases the rise of blood pressure represents the most crucial part of the treatment, as has been pointed out by NEUBAUER (1961, 1965), BERNSMEIER et al. (1962), HAGER (1966 – 1974), and ourselves (SAUTTER, 1953).

The *orthostatic* element is also known to be very important. Rising up from a supine position can lead to a drop of the arterio-venous pressure gradient. Thus, the pressure of the opthalmic artery decreases by the hydrostatic difference. Venous pressure also decreases, but in most cases not to the same extent. On can, therefore, conclude that the arterio-venous pressure gradient is diminished in the cerebral circulation, even if the pressure in the brachial artery remains unchanged.

A decrease of the *arteria-venous pressure gradient* in the eye can also be induced by an *increase of the intraocular pressure*. In essence, this alteration of perfusion corresponds closely to that induced by a decreased mean arterial pressure range: If the intraocular pressure rise does not exceed a critical level, some decrease of retinal perfusion does occur, but disturbed blood supply does not supervene. This functional aspect is referred to as the *indifference zone* of arterial pressure behaviour. Analogous observations on the alterations of the cerebro-spinal fluid pressure have been published by HIRSCH and SCHNEIDER (1967 a).

As we have pointed out, the tonus of the retinal vessels is mainly maintained autonomously. The question, whether and in which way a *neural influence* on the retinal vessels can be verified, has frequently been subjected to experimental investigations. The results have been inconsistent, most likely because of methodical insufficiencies. In general, a sympathetic effect is anticipated basically. HIRSCH and SCHNEIDER (1967 b) stress the fact that this effect occurs — both in the eye and in the brain — at high stimulus thresholds and intermediate tonus, and even then it is minute.

Most interesting results have been published by THURANSZKY (1957), who conducted biomicroscopic experiments with cats. It should be noted that in these animals the opthalmic artery and all other intracerebral arteries arise from the external carotid artery. When stimulating the preganglionic peripheral cervical sympathetic nerves, THURANSZKY found an unchanged blood pressure rate, thus confirming earlier results of STREIFF and MONNIER (1946). However, a considerable acceleration of the flow velocity in the retinal vessels was detected, and an intravasal sludge (aggregation of erythrocytes), induced by experimental vasodilatation, disappeared suddenly without noticable change of the vascular diameter.

According to HIRSCH and SCHNEIDER (1967 b) neural regulation of the vessel diameter plays only a minor role, when compared with other sources of interference. Several investigations, including those of our own are in accordance with clinical experience and favour the assertion that the retinal vessels are, under certain conditions, influenced by the cervical sympathetic nerves. In the intermediate blood pressure range they react to a lesser degree in a constrictive manner, more pronouncedly by an increment of the tonus. A block of the sympathetic nerves will never be followed by a sustained improvement of the retinal perfusion.

### Behaviour of microcirculation

The existence of a *neurally-induced real angiospasm* is decidedly denied by HIRSCH and SCHNEIDER (1967 b) and this leads us to this third chapter, dealing with microcirculation phenomena. It should, however, be noted that these authors, as far as the definition is concerned, speak of vasoconstriction and not of angiospasm as long as permanent damage does not occur. Yet, we believe that this question cannot always be answered on a clinical basis.

The difference in their terminology is based on the following facts: When an elevation of the blood pressure causes vasoconstriction, this vasoconstriction cannot develop further because of accumulating metabolites. If the vasoconstriction becomes so pronounced and permanent that irreparable damage ensues, an angiospasm is present. Such an angiospasm can be released in the retina only mechanically by means of injury, stretching, caloricly, or in other ways, but not by neural stimulation.

Experimentally, we succeeded in inducing angiospasms or arterial occlusions, respectively, in experimental animals by injecting suprarenin intravitreally, directly in front of the retina. Nobody can refute that, in doing so, the mechanical irritation was the most decisive procedure. Therefore, according to HIRSCH and SCHNEIDER (1967 b), the vasoconstriction in uncomplicated hypertension cannot be regarded as an angiospasm. When, in the course of hypertension, a degeneration of the retina does occur, anatomically discernible alterations of the vessel walls are always present. Therefore, the assumption that an angiospasm is the cause of tissue damage is superfluous.

When HIRSCH suggested to use the term hypertensive retinopathy instead of angiospastic retinopathy, this terminology proved to be more appropriate. This is especially true for the definition of arteriosclerotic alterations of the fundus. For the clinician, on the other hand, the situation is different and, according to our judgement, it is not always possible to separate the terms angiospasm and vasoconstriction, since tissue damage, which in the case of angiospasm is mandatory, may escape opthalmoscopical or sensoric examination.

If vasoconstriction and angiospasm do, in fact, differ characteristically, one has to look upon vasoconstriction as a consequence of autoregulation, and upon angiospasm as a response to extravasal, possibly neural, influences. This differentiation can, however, only be made in experimental physiology, where the cause of an experimentally induced alteration of the vessel wall is known. As has been mentioned, this cause usually remains concealed to the clinician.

We are, of course, aware of the fact that several clinical phenomena can sometimes simulate an angiospasm. These, however, can be interpretated in different terms, like the arteria-venous crossings, the so-called arteriosclerotic plaques, and the paravasal alterations such as exudation of plasma or circumscribed edema of the tissue, etc. Besides, there are changes of the vascular calibre for which we have no morphological explanation at all. Amongst these we should like to mention the characteristic changes of eclampsy and pheochromocytoma, which extend, sometimes in the shape of a concertina, along the course of an arteriole. As has been pointed out by MYLIUS (1928) these conspicuous changes are completely or almost completely reversible, which supports the assumption that these excessive alterations of the vessel wall are not based on a morphological substratum.

Another microcirculatory peculiarity is erythrocyte aggregation, the so-called *sludge phenomenon*. This is a visible criterion of deceleration of flow. Secondary to the aggregation of erythrocytes, flow resistance may increase and perfusion speed may further decrease. Usually, these red cell aggregates are disrupted, when the blood pressure rises again.

A sludge phenomenon in the conjunctival vessels has frequently been used to diagnose systemic disorders and has usually been overestimated. It is not possible to extrapolate from this observation to the vasodynamic importance of the sludge in the terminal vessels of the retina. The sludge should be regarded as a consequence, rather than the cause of a critical perfusion rate.

Finally certain other microcirculatory features can influence intraretinal vasodynamics. We have still to answer the question, whether *arterio-venous capillary shunts* exist in man, similar to those that have undoubtedly been proven in animals.

Such capillary nets were demonstrated in the cat retina by *in vivo* Indian ink injection into the carotid and fixation by freezing (THURANSZKI, 1957). With modern methods such as synthetic resin injection (ASHTON, 1950; PODESTÀ and ULLERICH, 1956) and radiographic investigation (FRANÇOIS and NEETENS, 1962) it was also shown in man that the terminal vessels of the retina form a large net of capillaries and most likely microanastomoses, which are above all essential for the supply of the second and third neuron synapses. The importance of such a capillary net is evident, since it maintains a functional reservoir, which warrants a surplus of perfusion far above physiological demand, and which presents a vasodynamic reserve. Findings of LEMMINGSON (1972) point into the same direction.

Another regulation system within the retinal vasculature appears to exist in the bifurcations of the arterioles and larger capillaries.

EVANS (1933) was the first to show some confinements of the arterial branches in human eyes, and suspected a *sphincter-like organization* in these areas. Later THURANSZKY (1957) was successful in proving these indentations of arterioles at the bifurcations. He used *in vivo* Indian ink injections in cats. Together with SEITZ (1962), we pursued the question of the anatomical substratum of these findings some years ago, and came to the conclusion that these would be based on alterations of the appearance of some cells or cell groups, which can be detected at the bifurcations (cf. SAUTTER et al., 1954)

Finally, the *vascular system of the papilla* may also be of functional importance. Since we are dealing with arterio-arterial anastomoses between the central and ciliar arteries, the interconnections of the septal vessels of the lamina cribrosa are certainly not only of importance for the nutrition of this area. This has been discussed by KREIBIG (1955), GAFNER and GOLDMAN (1955), and recently by HAYREH (1975, 1978) with regard to the papilla in glaucomatous eyes. In cases of high demand, there may be a specific microcirculatory organization which, in addition to local regulation, can cover the needs of retinal nutrition.

**Summary**

Physical and physiological problems of the retinal circulation are reviewed first. Ophthalmodynamometry has been shown to be of considerable value in the determination of the retinal flow. Fluorescein angiography, which was introduced into medical research and routine more recently, can help to objectively determine disturbances of the perfusion rate, especially in animals. Calorimetric methods have also contributed in the elucidation of the blood-tissue barriers. Retinal perfusion has been estimated in comparison with the cerebral perfusion, especially in experimental animals. Special stress was placed on the autoregulation of retinal vessels and its response to drug treatment, both in animal experiments and in clinical trials. Of considerable importance is the retinal survival time, which appears to be much longer in clinical conditions than would be expected from experimental results. Neural regulation of the retinal vasculature seems to be less effective. A detailed discussion of the retinal and uveal microciruclation includes a more exact definition of the terms ‚vasoconstriction' and ‚angiospasm'. Recent results of vital staining techniques are of special significance in this context.

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## SAUTTER, H. und ROSSMANN, H. — 120 Jahre Embolie der Zentralarterie der Netzhaut.
## I. Grundlagenprobleme und Forschungsergebnisse zur Netzhautzirkulation


### Zusammenfassung

Zunächst werden physikalische und physiologische Probleme der Netzhautzirkulation untersucht. Als wesentliche Hilfe bei Bestimmung der Netzhautdurchblutungsrate hat sich die Ophthalmo-Dynamometrie erwiesen. Auch die in letzter Zeit in Forschung und Praxis eingeführte Fluoreszenzangiographie kann hilfreich sein, Störungen der Perfusionsrate, besonders bei Tieren, zu objektivieren. Schließlich haben kalorimetrische Methoden dazu geholfen, die Probleme der Blutgewebsschranke besser zu erkennen. Die retinale Perfusion konnte durch Vergleich mit der cerebralen, besonders im Tierversuch, objektiviert werden, wobei besonders der Autoregulation der Netzhautgefäße im Experiment und in der Folge auch bei klinischen Untersuchungen, zuletzt unter dem Einfluß von Medikamenten, besonderes Augenmerk gewidmet wurde. Eine besondere Bedeutung hat die Wiederbelebungszeit, die im klinischen Bereich wesentlich länger zu sein scheint, als dies experimentelle Untersuchungen erwarten ließen. Neurale Regulationen der Netzhautgefäße sind dagegen offenbar von geringerer Bedeutung. Bei der Untersuchung der Mikrozirkulation der Netz- und Aderhautgefäße wird versucht, die Begriffe Vasokonstriktion und Angiospasmus exakter zu definieren, wobei die Ergebnisse von Untersuchungen mit Vitalfarbstoffen, wie sie in jüngerer Vergangenheit durchgeführt wurden, eine nicht unerhebliche Rolle spielen.


## SAUTTER, H. et ROSSMANN, H. — Histoire de l'embolie de l'artère centrale de la rétine au cours des 120 dernières années.
## I. Problèmes fondamentaux et recherche sur la circulation rétinienne.


### Resumé

Des problèmes physiques et physiologiques concernant la circulation rétinienne sont discutés. L'ophtalmo-dynamométrie a été d'un grand secours dans l'étude de la circulation rétinienne. Il en est de même de la fluoro-angiographie, qui a permis d'objectiver les troubles de la perfusion sanguine, entre autres chez les animaux. Des méthodes calorimétriques ont, en outre, contribué a mieux connaître la barrière hémato-tissulaire. La perfusion rétinienne a pu être objectivée en la comparant à la perfusion cérébrale, surtout dans l'expérimentation animale, ce qui a permis de mieux connaître l'autorégulation, sous l'influence des médicaments, des vaisseaux rétiniens tant au point de vue expérimental que clinique. Une signification particulière doit être attribuée au temps de récupération, qui semble être plus long en clinique que dans les expériences animales. La régulation neurale des vaisseaux rétiniens

est de moindre importance. Les concepts de vasoconstriction et d'angiospasme doivent être mieux définis pour les études sur la microcirculation de la rétine et de la choroïde. Les colorants vitaux, qui ont été utilisés dans le passé récent, jouent dans ce domaine un rôle qui n'est pas insignifiant.

**SAUTTER, H. y ROSSMANN, H. — Historia de la embolia de la arteria central de la retina en el curso de los últimos 120 anos. I. Problemas fundamentales e investigaciones sobre la circulación retinal.**

**Resumen**

Se discuten los problemas físicos y fisiológicos concernientes a la circulación retinal. La oftalmodinamometría ha sido de una gran ayuda en el estudio de la circulación retinal. Lo mismo puede decirse de la angiofluoresceinografía, que ha permitido objetivar los trastornos de la circulación sanguinea entre otros en los animales. Los métodos colorimétricos han contribuido a conocer mejor la barrera hematotisular. La perfusión retinal pudo ser objetivada comparándola con la circulación cerebral, sobretodo en la experimentación animal, lo que ha permitido conocer mejor la autoregulación de los vasos retinales bajo la influencia de medicamentos, tanto desde el punto de vista experimental como clínico. La regulación neural de los vasos retinales es de menor importancia. Los conceptos de vasoconstricción y angioespasmo deben ser mejor definidos por los estudios de la microcirculación de la retina y de la coroides. Los colorantes vitales que han sido utilizados en el pasado reciente, juegan en este dominio un rol que no debe desdenarse.

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