Then was of course the Woundhealing Symposium in Tübingen.

Later we got acquainted with your machines and I have never stopped to marvel at the real miracles that your Q-switched YAG laser works on each patient who has secondary cataract or pupillary block angle closure glaucoma. We can just make a hole in the iris or posterior capsule without opening the eye. We are now used to it but I still consider it as something very special.

Observations

It is the most important but not an easy task. We are dealing here with a pioneer who has made a real breakthrough in ophthalmology by a unique combination of ophthalmology, physics and mathematics. You have managed to make other people enthusiastic to cooperate with you. To mention a few names they are: P. Cibis, J. Enoch, Th. Schmidt, P. Niesel, B. Gloor, H. Bebié, J. Marshall, F. Koerner, E. van den Zypen, J. Flammer.

And to get all those people into action needs some convincing type of genius. I will not get you involved with all the details of his curriculum vitae but I should say that apart from the support of your colleagues you have had an astounding capacity for work, as Balder Gloor said at another occasion, working not only 24 hours a day but also every night. I think this is also due to the support you had from your first late wife Verena and of course from Sylvia who is supporting your efforts all the time. You were very closely associated with the University of Bern, Hans Goldmann has been your early inspiration. You have also tried distant countries – India, where you were exposed to the ophthalmology in the very difficult circumstances. Then you switched to the center of the USA to Paul Cibis and Bernard Becker in St. Louis and since 1962 you have been professor at the University of Bern. You have turned down offers to be chairman of the department being wise enough not to be loaded with additional obligations of fighting with administration. To summarise your basic research: you have started with visual physiology very early, in 1958 and I mentioned my very first contact with explanation of visual defects in choroidal nevi. You have tried to develop and succeded with developing devices like giving a calculator a voice and expression which are very difficult to achieve.

Then you have done extensive work on the light and laser coagulation and their interactions with the tissue.

And then, of course, your development of Octopus 201 and automated perimetry in 1974. Many have tried to get the perimetry into the fashion that one can standardize the findings, which one can compare to get a reasonable follow-up.

Then away from all the mechanical devices with knives and needles and so on you worked with Q-switched YAG laser Microruptor and its disruptive effect, which sounds like very destructive, but it is not. Young people now do not realise that something that used to be a major procedure in an operating room, today is a method in a range of nanoseconds. One cannot stop praising it.

You have used laser scalpels in laser sclerostomy which is a sign that you keep working and keep developing.

I will not go into the details of your publications – you have more than 300 publications, but what is nice to know is that you did get recognition. I think it is worthwhile to go through the awards.

Even before the automated perimetry you have received the Albrecht – von Graefe – Award for your basic work in physiology. Then you got a Honorary Doctorat of the University of Basel, Alfred – Vogt – Preis, Marcel Benoist Award, Alcon Research Institute Award, von Helmholz Medaille, George Weinstein Award, Wessely – Medaille, Theodor – Axenfeld – Preis and finally Albrecht von Graefe Medaille, which is presented every 10 years by the German Ophthalmological Society (Deutsche Ophthalmologische Gesellschaft).

Conclusion

I have tried to summarize my observations and now I will come to a short practical conclusion. I believe that the Tadeusz Krwawicz Foundation made a very good choice of the recepient of second Tadeusz Krwawicz Gold Medal. I have attempted to sketch a towering personality in ophthalmology not only of our time, which will have lasting effects on our field.

Countless patients will benefit and do benefit from his developments. He will remain a shining example not only of Swiss but European ophthalmology.

Wykład okolicznościowy

Klinika Oczna 1996, 98 (6): 465-470
ISSN 0023-2157 Indeks 362 646

Mechanisms related to photic hemostasis. Modelling the Thrombogenic Action of Nd:YAG Laser Light on the Vessels of the Human Eye

Mechanizmy związane z hemostazą świetlną.
Modelowanie trombogennego działania światła laseru Nd:YAG na naczynie oka

Franz Frankhauser

Key words: Laser, Nd:YAG Laser, vascular structures, irradiation, absorption, thrombogenesis, hemostasis

Słowa kluczowe: laser, laser Nd:YAG, układy naczyniowe, promieniowanie, absorpcja, trombogeneza, hemostaza

Introduction

The mechanism of hemostasis induced by laser irradiation remains an enigma. This paper is intended as a contribution towards its understanding.

With few exceptions, the hemostatic efficiency of a specific wavelength of light has been interpreted in terms of absorption of light energy by hemoglobin (Hb) and Oxyhemoglobin (HbO). While the effects of absorption may be considered an important starting point, a number of other effects must be considered. Otherwise, wrong conclusions are inevitable and one cannot attempt to explain, for instance, why krypton red or Nd:YAG laser radiation have good hemostatic properties.

We shall consider the following points in relation to photic thermothrombosis:

1. Effects related to the absorption of radiated energy by hemoglobin.
2. The basic quantities responsible for the increase of temperature within vessels irradiated by the laser beam.
3. The influence of hemodynamic factors upon intravasal temperature increase.
4. The effects of temperature increase upon the optical properties of blood.
5. Microscopic *in vitro* and *in vivo* studies of thrombogenesis induced by laser irradiation.

The hemostatic and thrombogenic effects of Nd:YAG laser light for the treatment of vascular problems in ophthalmology, contrary to what some people believe, are considerable. This has been shown with the effects of Nd:YAG laser light upon mesenteric, conjunctival, ciliary body or the retinal and choroidal vessels (8, 9, 21-27). This is an apparent contradiction to what might be expected from the absorption spectra of Hb and HbO. Other effects therefore need to be considered.

The effects of absorption of radiated energy by hemoglobin and melanin

The melanin contained in the pigmented epithelium (PE) and the choroidal melanophores absorbs laser radiation much more strongly than either Hb or HbO, when comparing equal thicknesses of absorbing layers (5). (Obviously, in comparing absorption, equal thickness of different light absorbing materials must be compared.)

It has been shown that, at wavelenghts shorter than about 625 nm and for a subretinal vascular

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membrane in contact with the PE having a thickness of about 10 microns, Hb absorption is much lower than PE absorption (5). For wavelenghts greater than about 625 nm, effects upon thin layers of blood may be almost entirely neglected. In this case, the heating of a vessel due to light energy absorption by blood and without damage to the surrounding structures may only be possible if chromophores, such as organic dyes, are added to the blood (1, 14-18). In all other cases the presence of melanin is indispensable.

The heat generated upon absorption of light energy must be transported from the absorber (the PE or the choroidal chromatophores) to the target blood vessel. Along the way, the temperature drops with increasing distance. This may be calculated both for equilibrium temperatures and for finite exposure durations (2). It may be shown e.g. that the efficiency factor η, responsible for energy transmission from PE to a vascular structure, for various exposure durations, decreases inversely as a function of the vessel-absorber distance (Fig. 1a, b). $\eta\,(x, t)=T\,(x, t)/T\,(x, \infty)$, where T is the intravasal temperature, $T\,\infty$ is the equilibrium temperature, x is the distance from absorber to the vessel and t is time.

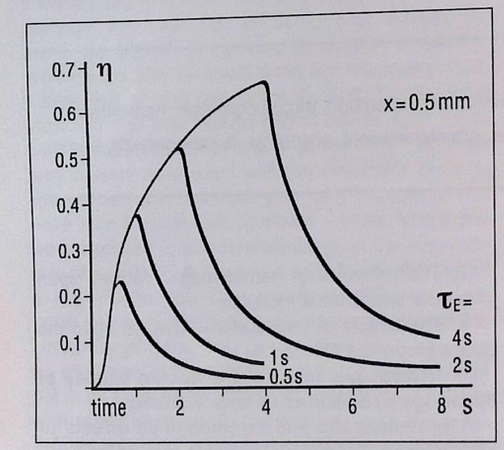

Fig. 1a. The efficiency factor as a function of time t, exposure time and distance from the pigment epithelium x. Distance x is 0,5 mm (adapted from 2)

Ryc. 1a. Efektywność jako funkcja czasu t, czasu naświetlania i odległości od nabłonka barwnikowego x wynoszącej 0,5 mm (opracowano na podstawie 2)

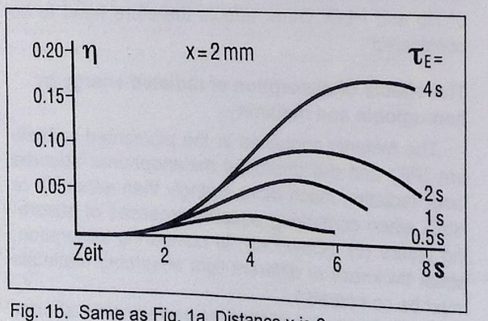

Fig. 1b. Same as Fig. 1a. Distance x is 2 mm

Ryc. 1b. Jak na ryc. 1a. Odległość x wynosi 2 mm

At large vessel-absorber distances, the heat effect upon the vessel becomes very small or – conversely – large temperatures must be created as has been shown previously (2). This is illustrated by the difficulties encountered e.g. when irradiating thin subretinal neovascular membranes. This is particularly the case at wavelengths above about 625 nm and when the PE is either absent or is only present at some distance from the sanguinous structure to be irradiated.

Thus far, we have analyzed the situation for a stationary blood column such as is more or less the case in a subretinal membrane. This may not always be a typical situation, however, and in other cases we have to deal with perfused vessels where flow velocity cannot be neglected. We therefore have to consider the consequence of a blood column in motion.

Basic factors related to increase of temperature within vessels irradiated by the laser beam

The temperature within an irradiated vessel will always be the result of an energy balance of heat generated by direct absorption by blood and by conduction from the irradiated chromophores (PE or choroidal chromatophores respectively) on the one hand and heat loss due to heat diffusion and convection on the other.

An analytical treatment relating these quantities has been given before (2) and will not be repeated here. Diffusion loss due to thermal energy transfer from extravascular absorbers has been estimated above. As a further factor, the effects of convection (movement of blood column) should be considered.

Simple calculation shows that convection losses are a critical function of blood flow velocity. This is

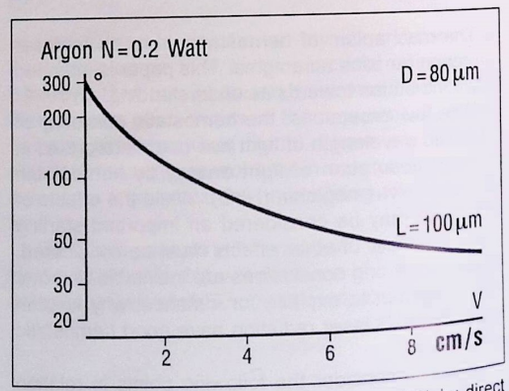

Fig. 2. Intravascular temperature increase (°C) by direct absorption of radiation within a vessel having a diameter D of 80 micron, irradiated by an argon laser bundle having a diameter L of 100 micron and a power of 200 mW as a function of velocity of blood flow (adapted from 2)

Ryc. 2. Wzrost temperatury śródnaczyniowej w wyniku bezpośredniej absorpcji promieniowania w obrębie naczynia o średnicy D=80 mikronów poddanego promieniowania wiązką lasera argonowego o średnicy L=100 mikronów i mocy 200 mW jako funkcja prędkości przepływu krwi (opracowano na podstawie 2)

shown with a computed example, i.e. the temperature within a vessel of 80 microns irradiated by an argon laser beam having a diameter of 100 microns and a power of 200 mW (Fig. 2). While a temperature of 300°C is reached for a motionless blood column, temperature goes down to about 40°C for a blood flow velocity of 9 cm/s. This computation should not be taken too literally because a number of factors have been neglected for the sake of simplification. They should therefore be considered as an estimate rather than the result of exact analysis. Even so, the critical influence of heat loss due to heat conduction by the blood flow is predicted by the model and is a current clinical observation (7). As a consequence, blood vessels with a high flow rate will require destructive high power radiation for occlusion.

The increase in temperature will eventually lead to coagulation of blood plasma, to thrombogenic phenomena and finally to hemostatis. Drop in blood flow rate is however critically related to the configuration geometry of the irradiated vessels. Hence, a brief analysis of related phenomena is indicated.

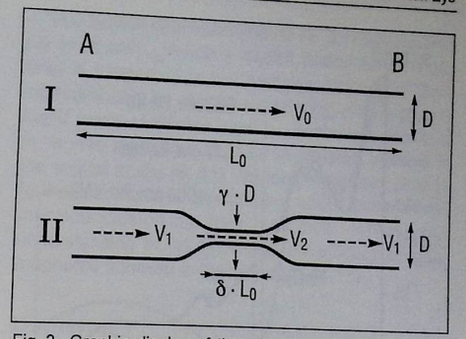

Fig. 3. Graphic display of the relevant physical quantities responsible for photic vessel occlusion. See text for details (adapted from 10)

Ryc. 3. Graficzne przedstawienie właściwych wartości fizycznych odpowiedzialnych za okluzję naczyń świetlnych. Szczegóły w tekście (opracowano na podstawie 2)

Intravasal temperature increase in relation to hemodynamic factors

The effects of hemodynamic factors are quite complex and may best be tested using simplified model assumptions (10). Such assumptions may be valid at least in the initial phase of thrombogenesis. They also appear indispensable to a first understanding of the basic mechanisms.

Blood may be considered as a first approximation to behave as a Newtonian fluid and any effects due to change in blood viscosity may be disregarded.

Because of the incompressibility of the fluid and as long as the pressure gradient across an irradiated segment of a vessel is constant, narrowing of the lumen of the vessel e.g. due to compression by a perivascular edema or by shrinking of the vessel wall, will accelerate blood flow initially at the site of compression of the lumen. However, according to Hagen-Poiseuille's law, blood velocity will eventually decrease when the extent of narrowing of the vessel has reached a critical value. This may be expressed by:

$$v_2/v_o = 1/\{\gamma^2(1-\delta)+\gamma^{-2}\delta\}$$

Here (Fig. 3) g is the ratio of compressed to non-compressed vessel diameter (D) and δ defines the fraction of L_0 (the length of the vessel considered) being compressed or constricted; v_0 is the blood velocity at the nonconstricted and v_2 at the constricted segment of the vessel.

This is illustrated in an example (Fig. 4), for the specific case when a vessel has been compressed to 20% of its original diameter. According to the assumptions made, a very local compression amounting to 1% of the length of the vessel considered will accelerate blood velocity to 16 times of its initial value and only when the length of the compression has reached about 3% of the length of the vessel length considered, will the blood velocity have returned to its preirradiation value and will then fall rapidly to lower values the longer the compressed vessel length.

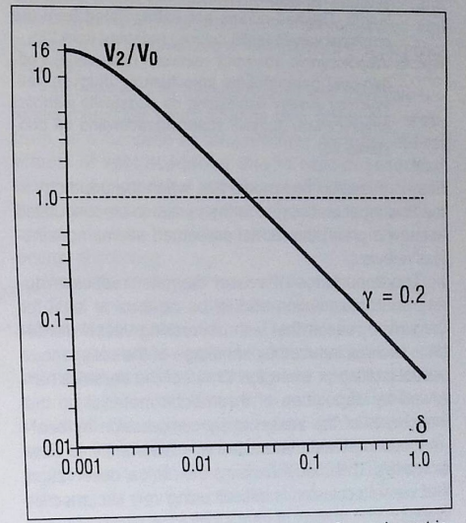

Fig 4. Ratio of blood flow velocity at site of vessel constriction v2 to flow velocity at nonconstricted site, v0 as a function of fraction of the vessel length being compressed. The ratio of compressed to noncompressed vesseel diameter is 0,2 (adapted from 10)

Ryc. 4. Stosunek prędkości przepływu krwi w miejscu zwężenia naczyń v2 do prędkości w miejscu nie zwężonym jako funkcja cząstki długości zwężonego naczynia. Stosunek średnicy naczynia zwężonego do nie zwężonego wynosi 0,2 (opracowano na podstawie 10)

According to Virchow's law, increased flow velocity disturbs clotting and adhesive processes leading to thrombogenesis. Consequently, we should attempt to decrease flow velocity as quickly as possible. Therefore, although the application of flow mechanics to photic vessel occlusion is in fact more complicated

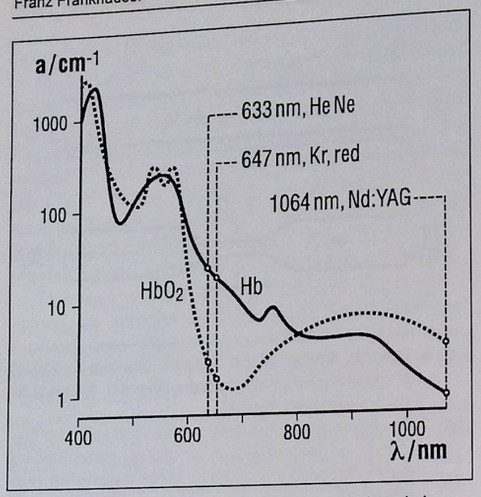

Fig. 5. Absorption coefficient of oxygenated and deoxygenated hemoglobin solution as a function of wavelength. Dashed curves are extrapolated from the empirical values (solid curves) (adapted from 28)

Ryc. 5. Współczynnik absorpcji roztworu natlenionej i odtlenionej hemoglobiny jako funkcja długości fali. Wykresy zostały naniesione na podstawie wartości empirycznych (krzywe stałe) (opracowano na podstawie 28)

than in this simplified model (i.e. when the geometry of the flow input and output to the vessel to be coagulated is know a priori) the model presented seems nonetheless relevant.

The importance of vessel diameter reduction during photocoagulation should be obvious at least for the simple reason that with decreasing vessel diameter – such as induced by shrinkage of the collagenous vessel coating or when the lumen of the vessel is narrowed by deposition of thrombotic material on the inner walls of the vessel or by compression by heat-induced perivascular oedema – resistance to flow increases. This model explains the clinical observation that vessel occlusion is difficult using very circumscript irradiation by means of very small focal spots and that a more efficient strategy should be oriented towards irradiating the vessel to be occluded over a large distance (7). In the light of this, it is also understandable that vessels, such as intravitreal vessels, which do not absorb much energy and which can be neither constricted nor compressed, are extremely difficult to occlude. Such vessels seem to be doubly handicapped, since their occlusion is neither beneficially aided by supplementary backhearing from the PE, nor is their occlusion by mechanical forces (7).

Because neither Hb nor the other constituents of blood are thermostable, we have to consider as the next step the effects of temperature upon the optical properties of blood.

The effects of temperature increase upon the optical properties of blood

It is an accepted fact that the argon laser has a strong hemostatic effect upon blood vessels of the eye. Furthermore, Hb absorbs argon laser light 17-20 times more strongly than light from a Nd:YAG laser. Nevertheless, we still manage to coagulate sanguinous structures by means of Nd:YAG laser light and we must look for further relevant mechanisms leading to thrombogenesis. For this reason we shall compare the absorption of radiation emitted by the red portion of the spectrum, such as by "krypton red" (647 nm) with the absorption by the near infrared, such as by Nd:YAG laser light (1064 nm).

As shown in Fig. 5, the absorption by HbO between wavelengths 580 nm and 805 nm is significantly lower than by Hb (28). Above 805 nm the point of intersection of the curves (isobestic wavelength), the effect is reversed, the absorption coefficient of HbO being higher than that of Hb. Unfortunately little is known about the absorption coefficient above 1000 nm, but by extrapolating the curves of Fig. 5 we can estimate the absorption coefficient at 1064 nm to be about 0,8 cm^{-1} for Hb and 6 cm^{-1} for HbO (similar values have been given by Mainster, i.e. 0,5 cm^{-1} for Hb and 5 cm^{-1} for HbO) (19).

The strong dependence of the absorption at red and infrared wavelengths raises the question of the behaviour of the oxygen saturation in blood above biological temperatures in the range of 40°C and 100°C typically induced in tissue by laser radiation. First, it may be noted that the binding of oxygen to Hb is an exothermic reaction and that HbO is thus deoxygenated at an exponential rate with increasing temperature according to Arrhenius's rule.

The oxygenation and deoxygenation of Hb is a rapid process with a reaction time shorter than 10 milliseconds and a time constant of diffusion of oxygen through the erythrocytes of the order of 100 milliseconds. Thus, when working with exposure durations of 100 milliseconds or longer (as is customary in ophthalmic applications) blood is deoxygenated when heated.

Extrapolating standard curves of the oxygen saturation dependence of hemoglobin on temperature in the range between 10°C and 43°C it may be estimated that initially 100% saturated blood will be totally deoxygenated at temperature above 80°C. As a consequence, absorption below 800 nm increases and above 800 nm decreases. Above 70°C a denaturation of the blood cells and breaking of the heme and globin bond is expected as well. In addition (or as a consequence) a dark discoloration of the irradiated blood is observed (20).

Based on these facts, a qualitative description of the processes involved is as follows (12): HbO has a somewhat higher absorption coefficient for Nd:YAG than for krypton red laser light, while absorption is nearly equal for Nd:YAG and HeNe laser radiation. As soon as blood is heated, absorption increases for HeNe laser radiation and decreases for Nd:YAG laser light. This may be one important reason why krypton red laser light is clinically effective for inducing thrombogenesis. When the temperature is elevated above the denaturation – and even more so above the coagulation point of plasma, absorption goes up for both wavelengths, though by a different amount (Table I).

Table I: Absorbance of blood samples in a capillary tube for HeNe and Nd:YAG laser radiation (adapted from 12). For details see text

Tabela I: Absorpcja próbek krwi w rurce włosowatej przy promieniowaniu laserem HeNe i Nd:YAG (opracowano na podstawie 12), szczegóły w tekście

Laser *Laser*	Capillary Diameter (mm) *Średnica włośniczki (mm)*	Blood Status *Stan krwi*	Percentage Absorbance *Procent absorpcji*
HeNe	2	oxy deoxy denatur	43,5 87,3 89,7
Nd:YAG	2	oxy deoxy denatur	47,4 12,5 50,0

This may be interpreted as follows: due to thermal denaturation and coagulation there is not only a total deoxygenation of the blood but at the same time there is a decomposition of the molecular and cellular material together with a coagulation of the plasma. This gives rise to much stronger light scattering in the samples than is observed in native blood. As a result, the mean length a photon travels through the samples becomes longer and the change of it being absorbed is high, resulting in a higher total absorbance of the sample.

A second obvious effect is that during the heating of the samples there is a loss of the liquid blood plasma due to evaporation. One may consider this process as the prephase of thermothrombosis, which can be observed in the *in vitro* model (3). In a sample of heat-degenerated blood, the ratio of the solid to the liquid blood material is thus higher than in another blood sample. This also results in higher total absorption. To obtain an estimate of this second effect, the volume of a sample in a specific experimental situation was determined before and after thermal denaturation and was found to be 87% after the heating (12). This effect may depend critically upon the absolute volume and the geometry of the irradiated vascular structure and so far we do not know how well it can be extrapolated to vessels having variable diameters and geometry nor do we know the interaction of the liberated steam with the vessel in which it is enclosed.

Microscopic *in vivo* and *in vitro* studies of thrombogenesis induced by the laser

Computation based on simplified model assumptions may be complemented by *in vitro* or *in vivo* irradiation studies. Such studies may provide good insight into what happens in a moving blood column being forced into thermothrombosis. Therefore irradiation experiments on glass capillaries, perfused by heparinized or citrated human blood have been performed (3, 16, 17). Here (3) temperature increase as a function of blood velocity and irradiation power was measured. Temperature was increased until thrombosis eventually occurred, while the phenomena due to drop in blood flow were observed with the microscope.

We believe that experiments on a relatively simple level may provide a useful notion and insight what is happening in the complex process of thermothrombosis by models consisting either of perfused glass capillaries such as described above or by *in vivo* irradiation studies on isolated vessels in the animal model (6, 11). Not only may such studies further our basic understanding about basic photic thermoocclusion but they may also increase our understanding of the thrombogenic efficacy of chromophores admixed to the blood (1, 14-18).

Discussion

An attempt is made to explain the primary effects leading to thermothrombosis induced by radiated laser energy based on simple model assumptions. They do not claim to account for effects such as enzymatic or any other secondary effects.

The observed phenomena can be explained by the current laws of absorption, energy deposition and hemodynamic effects which are interconnected and influence the mechanism of hemostasis in a complex manner.

Hemoglobin and oxyhemoglobin are thermally unstable molecules which change their absorption spectrum as a function of temperature. Also the optical effects of light scattering due to heat-degenerated blood and coagulated plasma, as well as an increase in the ratio of solid to liquid blood phases, due to evaporation, must be considered. This ratio also influences energy absorption.

Very little energy or none at all may be deposited by direct absorption in thin blood layers, such as subretinal vascular membranes. Thermothrombosis may be brought about by extravascular chromophores such as melanin contained in the pigment epithelium or the choroidal chromatophores or else by dyes added to the blood. Our model, in agreement with other models such as (4), predicts the necessity of using excessive, highly destructive energy doses, if, for pathological reasons, the pigment epithelium is absent or is no longer in immediate contact with the vascular structure to be occluded. Visual loss, following successful photocoagulation of choroidal neovascularisation may be explained by the high energy doses necessary for the thermoobliteration of such structures (13).

Because melanin, whose absorption is only a weak function of wavelength, is normally the relevant absorber, wavelength selectively may be considered as a poor predictor for clinical efficiency in irradiation tasks involving thin vascular structures. Furthermore, the thermal energy deposited in the blood column may be rapidly carried away by blood flow. Because heat dissipation in this case is very critically dependent on blood velocity, again high destructive energy doses will be necessary when blood columns with high flow velocities are to be coagulated. Hence, Doppler velocimetry may be a useful tool for predicting success or failure of a vascular occlusive irradiation operation. Hagen-Poiseuille's law predicts flow velocity at the site of irradiation, permitting formulation of an optimal irradiation strategy.

The irradiation of perfused glass capillaries or of isolated vessels in the animal experiments may be extremely useful in studying primary thrombotic effects, such as those described here.

Acknowledgement
We would like to acknowledge Dr. Sylwia Kwaśniewska for all her assistance in the preparation of this paper.

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Sprawozdanie

Sprawozdanie z V Forum Okulistyki Dziecięcej – Katowice, 24-25 maja 1996 r.

W V Forum Okulistyki Dziecięcej, które zostało zorganizowane w ośrodku kongresowym AWF w Katowicach przy ul. Mikołowskiej 72, udział wzięło 324 uczestników, w tym 25 profesorów. W czasie sześciu sesji naukowych przedstawiono 46 prac (33 referaty, 13 plakatów). Po każdym wystąpieniu miała miejsce trzyminutowa dyskusja.

Przybyłych na obrady V Forum Okulistyki Dziecięcej powitała Przewodnicząca Zarządu Sekcji Okulistyki Dziecięcej PTO, prof. dr hab. n. med. Bronisława Koraszewska--Matuszewska. Przemówienia wygłosili goście honorowi: wojewoda katowicki mgr inż. Eugeniusz Ciszak oraz prorektor Śląskiej AM prof. dr hab. n. med. Michał Tendera.

Otwarcia obrad V Forum dokonała przewodnicząca Zarządu Głównego PTO prof. dr hab. n. med. Krystyna Pecold.

I Sesja Naukowa
Temat: Zapalenie błony naczyniowej u dzieci
Skład Prezydium:
Przewodnicząca: prof. dr hab. n. med. Krystyna Pecold
Członkowie:
prof. dr hab. n. med. Teresa Baranowska-George
prof. dr hab. n. med. Krystyna Krzystkowa
prof. dr hab. n. med. Zbigniew Zagórski

Wygłoszono 7 referatów, zajmując się przedstawieniem przebiegu zapalenia błony naczyniowej u dzieci i jego powikłaniami w postaci zaćmy, otworów i odwarstwienia siatkówki oraz proliferacji szklistkowo-siatkówkowych.

W dyskusji głos zabrali: prof. B. Koraszewska-Matuszewska, prof. J. Kałużny, prof. M. Starzycka, prof. K. Pecold, prof. S. Pojda, prof. J. Nawrocki, dr Szulistkiewicz, dr Turno--Kręcicka, dr Kulesza, prof. Z. Zagórski. Pytania dotyczyły charakteru otworów i proliferacji siatkówkowych towarzyszących zapaleniu błony naczyniowej, roli kortykosteroidoterapii ogólnej i miejscowej, krioterapii i witrektomii. Dyskutowano nad celowością antybiotykoterapii w *uveitis*. Zwrócono uwagę, że zaćma występująca w przebiegu zapalenia błony naczyniowej może być również skutkiem stosowania kortykosteroidów. Omawiano wpływ materiału, z jakiego wykonane są soczewki wewnątrzgałkowe, na reakcję oka oraz dyskutowano nad problemem zmętnienia tylnej torebki soczewki, szczególnie w oku pseudofakijnym. Dyskusję podsumowała prof. K. Pecold.

II Sesja Naukowa
Temat: Etiologia i leczenie zapalenia błony naczyniowej
Skład Prezydium:
Przewodnicząca:
prof. dr hab. n. med. Helena Żygulska-Mach
Członkowie:
prof. dr hab. n. med. Wanda Andrzejewska
prof. dr hab. n. med. Maria Starzycka

Przedstawiono 5 prac oraz 1 film wideo na temat przebiegu zapaleń błony naczyniowej wywołanych *Toxoplasma gondii*, *Toxocara canis* lub wirusami. W dyskusji głos zabrali: prof. S. Pojda, prof. H. Żygulska-Mach i dr Turno-Kręcicka. Przedstawiali swoje doświadczenia w leczeniu toksokarozy ocznej metodą fotokoagulacji, laserokoagulacji i witrektomii. Podkreślali, że w przypadku wirusowego zapalenia błony naczyniowej należy rozpocząć leczenie na podstawie obrazu klinicznego, nie czekając na wyniki badań laboratoryjnych. Dyskusję podsumowała prof. H. Żygulska-Mach.

III Sesja Naukowa – Sesja Plakatowa
Skład Prezydium:
Przewodniczący:
prof. dr hab. n. med. Janusz Czajkowski
Członkowie:
doc. dr hab. n. med. Ewa Tokarz-Sawińska
doc. dr hab. n. med. Anna Kubatko-Zielińska

Sesję poświęcono plakatom, z których 5 dotyczyło zapalenia błony naczyniowej, 4 – wszczepów wewnątrzgałkowych, a pozostałe – tematów wolnych.

W dyskusji (prof. J. Czajkowski, prof. S. Pojda, prof. M. Prost, prof. B. Koraszewska-Matuszewska, prof. K. Krzystkowa, prof. T. Baranowska-George) zauważono, że zapalenia błony naczyniowej u dzieci dotyczą najczęściej tylnego odcinka gałki ocznej. Podkreślano zalety jednoczesnego wszczepiania soczewek wewnątrzgałkowych oraz usuwania zaćmy u dzieci oraz konieczność unikania soczewek przedniokomorowych. Rozważając wszczepienie soczewki wewnątrzgałkowej po usunięciu zaćmy powstałej w przebiegu zapalenia błony naczyniowej, należy uwzględnić przyczynę schorzenia, np. w młodzieńczym zapaleniu stawów powinno się unikać tego rodzaju korekcji bezsoczewkowości. Dyskutowano również nad wskazaniami do operacyjnego leczenia porażenia nerwu bloczkowego. Zwrócono uwagę na częstsze występowanie wad refrakcji u dzieci żyjących w środowisku zanieczyszczonym ekologicznie. Dyskusję podsumował prof. J. Czajkowski.

Drugi dzień obrad Forum dotyczył technik operacyjnych usuwania zaćmy u dzieci oraz wszczepiania sztucznych soczewek wewnątrzgałkowych.

IV Sesja Naukowa
Temat: Wszczepianie sztucznych soczewek wewnątrzgałkowych i techniki operacyjne usuwania zaćmy u dzieci i młodzieży
Skład Prezydium:
Przewodnicząca:
prof. dr hab. n. med. Ariadna Gierek-Łapińska
Członkowie:
prof. dr hab. n. med. Stefania Szymankiewicz
doc. dr hab. n. med. Danuta Karczewicz
prof. dr hab. n. med. Bazyli Bogorodzki

W czasie sesji przedstawiono 6 referatów, które dotyczyły leczenia chirurgicznego zaćm wrodzonych, urazowych oraz przemieszczonych soczewek. Dyskutowano problem zmętnienia tylnej torebki soczewki po pozatorebkowym usunięciu zaćmy oraz korekcji afakii soczewkami kontaktowymi. Głos zabrali: prof. S. Szymankiewicz, prof. M. Prost, prof. B. Koraszewska-Matuszewska, prof. T. Baranowska-George, prof. H. Żygulska-Mach, prof. J. Czajkowski, doc. Tokarz--Sawińska, prof. S. Pojda, prof. A. Gierek-Łapińska, prof. J. Nawrocki. Omawiano trudności techniczne w wykonywaniu